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Developmental dyslexia: Convergence of all theories towards a multifactional model, and the role of ICTs

Stasinou Dimitra *

Department of Greek Philology, Democritus University of Thrace, Greece.

Abstract

Dyslexia is a neurodevelopmental disorder that is characterised by slow and inaccurate word recognition. This article intends to summarize the current understanding of developmental dyslexia's etiology, brain bases, neuropsychology, and social context. In this article are discussed the progress has been made the last years in domains involving definition and classification, neuropsychological correlates, neurobiological factors, and intervention of dyslexia. It is also referred to the neuropsychological approaches to dyslexia have evolved and the importance of an interdisciplinary perspective for understanding dyslexia.

Dyslexia is caused by multiple genetic and environmental risk factors as well as their interplay. Several candidate genes have been identified in the past decade. At the brain level, dyslexia is associated with aberrant structure and function, particularly in left hemisphere reading/language networks. The neurocognitive influences on dyslexia are also multifactorial and involve phonological processing deficits as well as weaknesses in other oral language skills and processing speed.

Contextual issues such as the correlation of dyslexia with an individual's IQ or age, the manifestation of dyslexia across languages and social classes as well as what treatments are best supported are being examined. This article is aimed at highlighting the exciting new research that cuts across levels of analysis. Such work promises eventually to provide a comprehensive explanation of the disorder as well as its prevention and remediation.

In this recent contribution to the field of Dyslexia, we will be mentioned to some historical information, as well as we will be faced with significant issues in the Phonological Theory of Dyslexia. The single versus will multiple deficit accounts. In the field of the etiology of Dyslexia, we will be mentioned to the Behavioural Genetics, to the Molecular Genetics and to Environmental Influences. Individuals with dyslexia show functional abnormalities in both posterior and anterior language networks.

This approach is intended to promote scientific knowledge and draws attention to the complexity of the developmental dyslexia (DD).

Keywords: Developmental dyslexia; Learning disorders; Reading impairment; Multifactor-interactive model

1. Introduction

Dyslexia is a reading disorder in children and adults identified in part by difficulties with single-word reading and spelling (Lyon et al., 2003; Pennington, 2009). Prevalence estimates range from 6 to 17% of the school age population depending largely on criteria for the severity of reading difficulties (Fletcher et al., 2007). There is male preponderance,

* Corresponding author: Stasinou Dimitra

with a ratio of about 1.5:1 but lower than historical estimates of about 3–4:1 (Pennington, 2009; Rutter et al., 2004). The origins of dyslexia are neurobiological with strong evidence for heritability, but environmental factors also shape and ameliorate risk for dyslexia; it can be prevented in many children with early intervention (Fletcher et al., 2007; Pennington, 2009).

Dyslexia is extremely common. Estimates of its prevalence range from five to as high as 20%. The precise figure depends on exactly how it is defined and diagnosed. But such high prevalence in a strongly hereditary condition suggests the operation of a 'balanced polymorphism'.

According to the ICD-10 criteria, developmental dyslexia (DD) is diagnosed in children who fail to develop normal reading skills in spite of normal intelligence, adequate motivation and schooling. Reading difficulties constitute the most visible dysfunction of a more composite picture of a defective neurocognitive profile, which is likely to extend across different functions. Although phonological deficits have been described as the most reliable and specific distinctive features of dyslexic readers, several other functions were shown to be specifically impaired in dyslexia, including long-term and short-term verbal memory or working memory, visual and auditory perception and visual attention.

Since the German ophthalmologist Oswald Berkhan first described the symptoms of dyslexia in 1881 and Rudolf Berlin introduced the term "dyslexia" , numerous theories have been proposed about its causes and treatments. The magnocellular theory of dyslexia, the theory of unusual foveal and parafoveal processing of letters including an unusual crowding effect, and the temporal summation theory regard developmental dyslexia (DD) as a visual perceptual disorder. Other theories assume that DD results from an impaired ability to process auditory stimuli or is caused by impaired control of reading eye movements.

Vidyasagar and Pammer tried to explain dyslexia as a deficit in visuo-spatial attention due to poor control of the dorsal visual pathway. This shift in attention is believed to be controlled by the mainly magnocellular pathway of the dorsal stream including the posterior parietal cortex. Dyslexia is believed to be caused by a dysfunction of magnocellular neurons in the dorsal pathway.

But why do magnocellular systems fail to develop properly? That underlie magnocellular weakness likely involve interaction of several genes, together with the impact of adverse environmental factors, such as immunological attack and poor nutrition. Six predisposing candidate genes have been identified, and evidence shows gene by environment interaction. Together these influences may compromise magnocellular neuronal development, both in utero and later in infancy. There is a clear genetic basis for impaired development of magnocells throughout the brain. The development of magnocells may be impaired by autoantibodies affecting the developing brain. Magnocells also need high amounts of polyunsaturated fatty acids to preserve the membrane flexibility that permits the rapid conformational changes of channel proteins which underlie their transient sensitivity.

It is clear that genetic vulnerability lies at the heart of dyslexia. Numerous studies have shown that reading ability has a high heritability/. A percentage of 60% of the variability in reading attainment between subjects can be explained by the genes they inherit and the rest 40% of the variability to be caused by environmental influences (Olson, 2006).

Dyslexia has neurobiological and neurocognitive bases. It has been developed behavioural, neuropsychological, neurobiological, and causal levels of analysis in the past few years. From a neuropsychological perspective, the phonological theory remains the most compelling, although phonological problems also interact with other cognitive risk factors.

Of all the neurodevelopmental disorders, dyslexia has been the most studied and is the best understood. There has been considerable progress over the last years in understanding dyslexia's cross-cultural manifestation, causes, neuropsychology, and neurobiology. Much of the most significant work includes an interdisciplinary focus across these different specialties.

2. Historical background

For many years, research on dyslexia proceeded on the basis that it was a specific learning difficulty. Initially conceived of as a measure of General Cognitive Ability an individual's IQ. The most widely used measures of IQ were the Wechsler Scales (Wechsler, 1974). Dyslexia can occur across the IQ range and that poor decoding skills require the same kinds of intervention irrespective of IQ (Snowling, 2020).

Neuropsychologists assessed symptoms of brain dysfunction to understand the etiology of dyslexia as a prerequisite to treatment. Thus, Benton (1975) identified eight neuropsychological correlates of dyslexia, including deficits in visuoperceptual and audioperceptual functions, directional sense, right–left discrimination, finger recognition, and generalized language deficiencies. He identified putative brain mechanisms for reading disorders involving focal maldevelopment of the parietal lobes or overall organization of the cerebral hemispheres.

It is well established that in cognitive terms, dyslexia is caused by problems at the level of phonological representation (e.g. Shankweiler et al., 1979; Snowling & Hulme, 1994). This hypothesis has its roots in long-standing clinical observations, arguably starting with Orton (1937) and continuing with Bannatyne (1974), who noted difficulties in 'auditory sequencing, auditory discrimination, and associating auditory symbols with sequences of visual symbols'.

A considerable body of research has since detailed the nature of 'auditory' problems in dyslexia, narrowing the deficit to one affecting the sound (phonological) structure of speech (Griffiths & Snowling, 2001; Vellutino et al., 2004): problems with phonology lead to difficulty in learning mappings between orthography and phonology and other difficulties which include problems learning new spoken words, poor verbal short-term memory and problems with word retrieval and picture naming (see Snowling, 2019).

Nonetheless, despite the strength of the phonological deficit hypothesis, it seems that phonological difficulties are neither necessary nor sufficient to account for dyslexia (Pennington, 2006). According to the influential theory by Pennington (2006), dyslexia is the outcome of multiple risks which accumulate towards a threshold for what is usually termed 'diagnosis'.

There were efforts for scientific understanding of how children develop word recognition skills. The major breakthrough was the discovery that the link between oral language and written language resided in the phonological structure of speech (Shavelson & Towne, 2002).

Historically, several alternatives to the phonological theory have been pro-posed. There are theories involving the cerebellum, low-level vision and speech processing, and other domains (Fletcher et al., 2007; Pennington, 2009; Vellutino et al., 2004). Harkening back to Doehring (1978), univariate theories abound and much depends on identification criteria.

Today there is the conviction that a single phonological deficit does not seem to be sufficient to cause dyslexia. Many children with other disorders of language development (e.g., speech sound disorder, language impairment) have normal-range reading abilities despite phonological deficits. Phonological difficulties are very important in the development of dyslexia, and they probably interact with other neurocognitive risk factors and protective factors.

To sum up, some of the complexity associated with dyslexia arises because the predominant proximal cause – a phonological deficit – is often not the only deficit that is observed. Moreover, phonological skills are themselves dimensional and can affect one or more aspects of reading.

3. Theories

Many alternative theories have garnered substantially less support than the phonological theory, such as low-level sensory theories (auditory and visual), the cerebellar theory, and the magnocellular theory. Convergent evidence shows that sensorimotor problems are correlated with dyslexia but are not causal.

A core deficit model is inherently at odds with a multifactorial model; Reconciling the many disparate theories of reading disability remains a formidable challenge. Furthermore there are several variants of the cascading deficit theory—one is the magnocellular deficit theory of dyslexia, in which a low-level impairment in the motion-sensitive magnocellular pathway of the visual system is said to disrupt reading skill development (Stein, 2001, 2019; Stein & Walsh, 1997). Proponents of this theory have argued that sensitivity to transient sensory information may not be restricted to vision, but could also affect auditory processing (Stein & Talcott, 1999; Van Ingelghem et al., 2001; Witton et al., 1998).

One important cause of dyslexic reading problems is probably difficulty with auditory and visual sequencing - auditory sequencing of the sounds in a word and visual sequencing of its letters (Stein and Walsh, 1997, Vidyasagar and Pammer, 2010). Successful sequencing depends on accurate timing of auditory and visual sensory inputs; this is often known as temporal processing.

Magnocells create a stream that called the magnocellular dorsal (M-D) attention stream, which is specialised for the deployment of visual attention and for visuomotor control. The visual motion area V5/MT lies at, not beyond the front of the occipital lobe and is supplied mainly by the M system. M-D system locates and times the order of visual events such as inspecting the letters in a word when reading. Thus, a rapid and accurate M system is crucial for sequencing letters correctly. Reduced M cell sensitivity could be irrelevant to reading and it might be a result of failing to learn to read (Huettig et al., 2017). The great majority of studies demonstrate a causal relationship between Magnocellular Dorsal stream deficits and Developmental Dyslexia(Gori et al., 2015).

The sensitivity of a person's auditory magnocellular system seems to be under the same control as its visual equivalent, and, like the latter, it is also a highly important determinant of overall reading ability. Reading also requires the ability to rapidly sequence the sounds in words, in syllables and in the phonemes within syllables. This requires hearing accurately the changes in the amplitude and/or frequency of the sounds (amplitude or frequency modulations – AM or FM) and remembering their order led to sequence the sounds correctly. Detecting such changes in sound frequency and amplitude is mainly mediated by large neurons in the auditory brainstem; they could be called 'auditory magnocellular' neurons (MGN), like visual magnocells. Thus reduced AM and FM sensitivity in dyslexics may be due to impaired development of the auditory 'magnocellular system'.

However, people with developmental dyslexia have been shown to be less sensitive to these changes in sound frequency and intensity (Colling et al., 2017, Kraus et al., 1996, McAnally and Stein, 1996a, McAnally and Stein, 1996, Menell et al., 1999). MGN neurons have been shown to be reduced in size in dyslexics, particularly on the left side (Galaburda et al., 1994) and MGN activity during reading predicts phonological skill (Díaz et al., 2012).

The right cerebellum, which is preferentially connected to the left hemisphere, is now known to be very important for speech & reading comprehension, not just in speech output (Stoodley, 2012). The magnocellular systems all project strongly to the cerebellum because it needs accurate timing information for its control functions; hence dyslexics show abnormal cerebellar anatomy and chemistry (Rae et al., 1998) and impaired activation of the cerebellum during reading (Stoodley and Stein, 2011). The cerebellum receives a large input from the brain's magnocellular systems for timing and sequencing, and it is a part of the brain that is one of the most underactive in dyslexics. This explains their coordination problems and their poor embodied cognition. In fact, training children to be aware of how their body moves (mindful movement) has been shown to greatly help their cognitive development, including their reading and their social interactions (McClelland et al., 2014).

Magnocellular neurons form networks specialised for temporal processing, hence for sequencing. They track changes in light, sound, position, etc., for the direction of attention and the control of movement. They are found throughout the whole brain; in the visual system, the auditory system, the touch and proprioceptive systems, in the cerebral cortex, hippocampus, cerebellum and brainstem.

Impaired magnocellular development has been found in developmental dyslexia, ADHD, ASD, Williams syndrome, and even in schizophrenia and bipolar depression (Whitford et al., 2017).

Assuming that impaired magnocellular development is seen as a fundamental contributory cause of developmental dyslexia, then clearly an important question is what causes this magnocellular deficit to arise. There are three important contributions, namely genetic, immunological and nutritional.

It is possible that some of these theories could explain other aspects of a broader phenotype (e.g., fluency problems) or even why some children with dyslexia have problems in the motor system unrelated to reading (Denckla et al., 1985). However, phonological awareness, along with rapid naming and verbal working memory, is most consistently linked to the word reading disorder regardless of comorbidities (Willcutt et al., 2005), thus helping to explain the word reading problem that is the cardinal feature of dyslexia.

In parallel, there is a broad literature characterizing dyslexia as the consequence of a fundamental deficit that supersedes phonological processing. There are many reports indicating that people with dyslexia perform poorly in experiments targeting various aspects of visual (Stuart et al., 2006; Talcott et al., 2002) and auditory processing (Hämäläinen et al., 2013; Noordenbos & Serniclaes, 2015), as well domain general mechanisms such as processing speed and statistical learning (Gabay et al., 2015; Vandermosten et al., 2018). These findings have spurred competing theories that explain dyslexia as the consequence of cascading effects from a fundamental deficit in the neural systems that process sensory information (either visual, auditory, or both; Goswami, 2015), or the ability to make optimal use of sensory information (e.g., Ahissar, 2007; Ramus & Ahissar, 2012). Sensory systems are organized in a hierarchy and the information that is encoded by the eyes and ears is processed in a sequence of stages in the brain. Generally, these

"cascading deficit" theories contend that deficits in one of the early stages of sensory processing disrupt the development of phonological processing and, by this mechanism, disrupt reading development.

Notably, these two branches of research remain largely distinct. Presently, hypotheses positing a core deficit with cascading effects are the focus of many neuroscientific and psychophysical studies of reading disability (Casini et al., 2018; Colling et al., 2017; Frey, François, Chobert, Besson, et al., 2019; Frey, François, Chobert, Velay, et al., 2019; Gori et al., 2016; Krause, 2015; Lieder et al., 2019; Nicolson & Fawcett, 2018; Vidyasagar, 2019).

Distinct from these sensory processing theories, proponents of the statistical-learning hypothesis argue that a domain-general deficit in sensory learning and perceptual decision-making more broadly could explain why people with dyslexia perform poorly on myriad psychophysical tasks (Ahissar, 2007; Nicolson & Fawcett, 2018; Ziegler, 2008).

Today, the literature remains inconclusive for several reasons. First, various cascading deficit models contradict one another as each posits distinct mechanisms for disrupting phonological processing and the few studies focusing on individual patterns across a battery of diverse tasks do not encourage much hope for a uniform profile (Amitay et al., 2002; Ho et al., 2002; Menghini et al., 2010; Ramus et al., 2003; White et al., 2006). The cascading deficit models remain at the forefront of the dyslexia debate, particularly for theories that hold a central role for sensory processing deficits (reviewed in Goswami, 2015).

As O'Brien G. & Yeatman J. (2020) test core-deficit and multifactorial models of dyslexia, they examine the role of sensory processing and perceptual decision-making deficits in dyslexia. Specifically, by capitalizing on the drift diffusion model to analyse performance on a visual motion discrimination experiment, it is showed that deficits in visual motion processing, perceptual decision-making, and phonological processing manifest largely independently. Based on statistical models of how variance in reading skill is parcelled across measures of visual processing, phonological processing, and decision-making, our results challenge the notion that a unifying deficit characterizes dyslexia. Instead, these findings indicate a model where reading skill is explained by several distinct, additive predictors, or risk factors, of reading (dis)ability. The Yeatman´s research highlights are that a single-mechanism model of dyslexia cannot account for the range of linguistic and sensory processing outcomes in children. The authors propose an additive risk factor model where different aspects of sensory, cognitive, and language function each contribute independently to reading development.

According to a study of Pennington, B. F., Santerre-Lemmon, L., Rosenberg, J., et al (2012), the hybrid model provided the best overall fit to the data and contribute severe to the early detection-diagnosis of dyslexia. The overall goals of their study were to test single versus multiple cognitive deficit models of dyslexia (reading disability) at the level of individual cases and to determine the clinical utility of these models for prediction and diagnosis of dyslexia. Five cognitive models of dyslexia were being tested (two single-deficit models, two multiple-deficit models, and one hybrid model), in two large population-based samples. The cognitive deficits included in these cognitive models were in phonological awareness, language skill, and processing speed and/or naming speed. The results of this study shown that roughly equal proportions of cases met both tests of model fit for the multiple deficit models (30–36%) and single deficit models (24–28%); hence, the hybrid model provided the best overall fit to the data. This article nicely contributes to the field of the hybrid model and complements other work by Jason D. Yeatman.

4. Epidemiology

4.1. Socioeconomic Status

Epidemiology prevalence estimates depend on the definition of dyslexia. A common definition sets the cutoff for reading achievement 1.5 standard deviations below the mean for age and identifies 7% of the population as dyslexic; a similar IQ–achievement discrepancy definition identifies a similar proportion. A small but significant male predominance exists (3:1).

4.2. Cross-Cultural Findings

Historically, research on dyslexia has focused mainly on the English language. Among alphabetic languages, English is especially difficult to learn because the mapping between letters and sounds is less consistent than in most other languages. Dyslexia exists in every language studied, but clinically significant difficulties are less common in languages with consistent orthographies than in languages with inconsistent orthographies. Reading problems in consistent orthographies are characterised more by fluency problems than by accuracy problems.

Cognitive predictors of early reading were similar for many European orthographies. Particularly, phonological awareness (PA) was the main predictor of reading in each language, although it had more of an effect in consistent than in less consistent orthographies. Other predictors, such as rapid serial naming (RSN), vocabulary knowledge, and verbal short-term memory (VSTM) made smaller contributions than did phono logical awareness. Thus, phonological awareness (PA) is the main predictor of reading skill in inconsistent orthographies, whereas rapid serial naming is the main predictor in consistent orthographies. Today it is believed that phonological awareness is a key predictor of reading skill in both more and less consistent orthographies and rapid serial naming predicts reading speed across languages. Across countries and languages, many cognitive–linguistic constructs—including semantics, syntax, phono logical awareness, verbal short-term memory, and rapid serial naming—consistently predict dyslexia later in life.

Cross-cultural similarities extend to Chinese, a logo-graphic language. By contrast with alphabetic languages, in which letters represent phonemes (individual sounds), the smallest written units in Chinese are characters representing monosyllabic morphemes (units of language that convey meaning). However, phonology is not irrelevant to reading in Chinese and dyslexia could exist because of deficits in amplitude envelope tasks - beat perception task, in which auditory rise time is an important cue.

The neural correlates of poor reading seem to be very consistent across cultures. A neuroimaging study showed that weak readers in English, French, and Italian had similar patterns of aberrant neural activation (underactivation in left temporal and occipital regions) during a reading task. Another study compared Chinese and English dyslexic and typical readers in a functional MRI framework. . However, the effect of dyslexia was very similar in both languages with reduced activation in posterior and anterior left hemisphere regions (LH).

To summarize, cross-cultural work suggests universality in the neurobiological and neurocognitive causes of dyslexia. The neural basis of dyslexia is similar across alphabetic languages and includes disruption of posterior and anterior language regions in the left hemisphere. The neural basis of dyslexia in Chinese is somewhat different and includes disruption of the left middle frontal gyrus.

4.3. Comorbidities

Dyslexia is comorbid with other special learning difficulties.

Comorbidity refers to the co-occurrence between two (or more) disorders in the same individual. Rates of comorbidity between reading disorder and other neurodevelopmental disorders vary widely but, on average, about 40% of the children with a reading disorder will have another disorder as well (Moll et al., 2020).

Some children with dyslexia meet criteria for Developmental Language Disorder (DLD), a disorder characterised by persistent difficulties in expressive and/or receptive language (Bishop et al., 2017). McArthur et al. (2000) showed that among children with specific reading difficulties, some 40% had significant language impairments. On the other hand, children who enter school with DLD are at high risk of literacy difficulties (Bishop & Adams, 1990). These language problems are under-diagnosed but are likely to affect response to intervention (Adlof & Hogan, 2019).

Dyslexia is also often comorbid with attentional and motor coordination problems (Gooch et al., 2014; Rochelle & Talcott, 2006). There is also an overlap between dyslexia and speech sound disorder (Pennington & Bishop, 2009), socio-emotional and behavioural disorders (Carroll et al., 2005) and internalising problems such as anxiety and depression (Francis et al., 2019).

Another disorder that is frequently comorbid with dyslexia is mathematics disorder (or dyscalculia). Like reading disorder, mathematics disorder is classified as a specific learning disorder in DSM5 (American Psychiatric Association, 2013). it is comorbid with dyslexia in between 30% and 70% of the cases (Landerl & Moll, 2010).

None of these comorbidities should be viewed as 'core' features of dyslexia, but they can complicate both its presentation and response to intervention (Rose, 2009). The clinical and educational reality is that for many children, poor reading sits within a constellation of difficulties each of which represents a dimension.

To summarize, in addition to comorbidity with attention-deficit hyperactivity disorder (DHD), it is also comorbid with two other disorders of language development—language impairment (LI) and speech sound disorder (SSD). These comorbidities are clinically significant because by paying attention to deficit hyperactivity disorder, speech sound disorder, and language impairment, which are all likely to be apparent earlier, we can predict a child's risk for later reading problems.

5. Brain bases of dyslexia - neuropsychology of dyslexia

Early theories of dyslexia postulated a basic deficit in visual processing and focused on the reversal errors commonly made by individuals with the disorder.

Since then, much research has made clear that dyslexia is a language-based disorder whose primary underlying deficit involves problems in phonological processing. For many years, a single-deficit phonological theory of dyslexia was most prominent. Dyslexia is caused by an underlying deficit in phonological representations, which causes poor performance on various oral language tasks and is the source of the phonological coding deficit. The phonological deficit impairs language processing at the level of the phoneme, and thus interferes directly with the establishment of phoneme–grapheme mappings.

Importantly, many theories posit that dyslexia reflects a cascade of impairments emanating from a single "core deficit". Competing theories of dyslexia posit that reading difficulties arise from impaired visual, auditory, phonological, or statistical learning mechanisms (O´Brien, B. & Yeatman J.2020).

Interest remains in alternative accounts, and visual attention has been a particular focus. Much of the relevant research has important limitations, such as use of linguistic stimuli to measure visual attention. However, visual attention weaknesses could be an additional risk factor that interacts with a phonological deficit. Also, problems in speech perception and phonological development are not limited to phonemes.

6. Neural substrates - Functional & Structural findings

As reading is a linguistic skill, we would expect it to involve activation of brain structures used in oral-language processing and some additional structures associated with visual-object processing and establishment of visual–linguistic mappings.

Children at risk for dyslexia showed activation abnormalities across a widely distributed set of bilateral cortical and bilateral subcortical regions. Many functional imaging studies have shown aberrant activation patterns in the regions that are related with brain structures used in oral-language processing and some additional structures associated with visual-object processing. The most common findings encompass abnormalities of a distributed left hemisphere (LH) language network.

Consistent under-activations have been reported in two posterior left hemisphere regions; a temporoparietal region believed to be crucial for phonological processing and phoneme– grapheme conversion, and an occipitotemporal region, including the so-called visual word form area (VFWA), which is thought to participate in whole word recognition. Abnormal activation of the left inferior frontal gyrus (IFG) is also commonly reported.

Results of several qualitative reviews showed abnormal activation in people with dyslexia, with underactivation in left temporoparietal regions relative to both chronological-age and reading-age controls. The main finding was that poor reading was associated with a reduction in activation in the bilateral temporoparietal cortex; no effects were found in occipitotemporal regions.

Recent neuroimaging studies have found that dyslexic readers showed weaker speech-brain coupling than normal readers. Specifically dyslexic readers present weaker coupling between low-frequency auditory oscillations and speech according to Goswami's theory.

Speech comprehension has been proposed to critically rely on oscillatory cortical tracking, that is, phase alignment of neural oscillations to the slow temporal modulations (envelope) of speech. Speech-brain entrainment is readjusted over time as transient events (edges) in speech lead to speech-brain phase realignment. Auditory behavioral research suggests that phonological deficits in dyslexia are linked to difficulty in discriminating speech edges.

The speech signal includes slow temporal fluctuations in the 1–10 Hz band that are closely related to phrase and syllable features in the acoustic signal. Tracking such temporal structures, both at phrasal and syllabic rates, is crucial for speech segmentation (Greenberg et al., 2003; Poeppel, 2003; Poeppel et al., 2008). The phase of low-frequency delta (1–3 Hz) and theta (4–7 Hz) oscillations in auditory cortex synchronizes to the phrasal and syllabic patterns of speech, respectively (Molinaro & Lizarazu, 2018; Lizarazu et al., 2019; Bourguignon et al., 2013).

Recent neuroimaging studies (Jiménez-Bravo et al., 2017) have found that dyslexic readers showed weaker speech-brain coupling than normal readers, mainly in the delta band and right auditory cortex (Cutini et al., 2016; Molinaro et al., 2016; Power et al., 2013, 2016). Van Hirtum, Ghesquière, & Wouters, (2019) also analysed neural synchronization in normal and dyslexic readers. They found reduced neural synchronization in the alpha, beta, and low-gamma frequency ranges in dyslexia.

Lizarazu M. et al, evaluated whether neural mechanisms involved in phase realignment to speech edges are affected in developmental dyslexia. It was found that speech-entrained brain oscillations at different frequency bands are hierarchically coupled to mediate the encoding of the phonological structure of continuous speech (Leong & Goswami, 2015). Specifically, the phase of low-frequency (delta and theta) oscillations modulates the amplitude of high-frequency (gamma) oscillations in auditory regions (Poeppel, 2003; Gross, Hoogenboom, et al., 2013; Lizarazu et al., 2019).

Much research has explored white matter correlates of dyslexia. The most consistent findings have included local white matter changes in left temporoparietal regions and in the left interior frontal gyrus (IGF). Studies have consistently reported correlations between white matter integrity and reading skill.

Kotz and Schwartze (2010) suggest that 'temporal processing mechanisms (i.e., mechanisms underlying the explicit encoding, decoding and evaluation of temporal information) need to be involved in the interpretation of the temporal structure of speech'. Timing originates in evolutionary primitive brain structures such as the cerebellum and the basal ganglia (Buhusi & Meck, 2005). Interestingly, the cerebellar theory of dyslexia (Nicholson, Fawcett, & Dean, 2001) has already highlighted a dysfunction of the cerebellum as a possible biological cause of dyslexia.

However, while this theory has focused on the cerebellum's role in skill automatization, research by Casini L, Pech-Georgel C, and Ziegler, suggests that it might be deficient temporal processing in the cerebellum that causes abnormal speech processing and phonological development. The survey of Casini, Pech-Georgel & Ziegler examines the temporal processing deficits in dyslexia in French children. In this survey temporal processing with dyslexia was evaluated in three tasks: a word identification task requiring implicit temporal processing, and two explicit temporal bisection tasks, one in the auditory and one in the visual modality. . Children with dyslexia exhibited robust deficits in temporal tasks whether they were explicit or implicit and whether they involved the auditory or the visual modality. The results showed that children with dyslexia presented larger perceptual variability when performing temporal tasks, whereas they showed no such difficulties when performing the same task on a non-temporal dimension (intensity). This dissociation suggests that their difficulties were specific to temporal processing and could not be attributed to lapses of attention, reduced alertness, faulty anchoring, or overall noisy processing. In the framework of cognitive models of time perception, these data point to a dysfunction of the 'internal clock' of dyslexic children. These results are broadly compatible with the recent temporal sampling theory of dyslexia. The study link temporal processing deficits in dyslexia with psychophysical methods and models of time perception.

Temporal processing deficits have a long history in dyslexia research (Farmer & Klein, 1995; Tallal, 1984; Tallal & Benasich, 2002; Wright, Brown, & Zecker, 2000), and various studies converge to suggest that children with dyslexia have deficits in temporal processing, temporal alignment, temporal sequencing and temporal sampling (Goswami et al., 2002, 2010; Thomson & Goswami, 2008; Thomson, Fryer, Maltby, & Goswami, 2006; Vandermosten et al., 2010, 2011).

A recent theory of speech processing has proposed that accurate perception of the speech signal at multiple temporal scales is important for the efficient extraction of meaningful phonological elements, and that oscillatory entrainment mechanisms may contribute to this process (Poeppel, 2003; Ghitza & Greenberg, 2009). Based on such theorizing and on the behavioral relationships observed between rise time discrimination, rhythmic performance and phonological difficulties, the temporal sampling framework proposed that the phonological deficits found in dyslexia may arise in part because of atypical 'temporal sampling' of the speech signal by neuroelectric oscillations (Goswami, 2011).

In regard to the neural entrainment and sensorimotor synchronization, relevant article/research (Colling et al, 2017) investigates the neural mechanisms that may underpin the developmental relationships between the precision of beat synchronization, children's phonological awareness, and their progress in reading. Tapping in time to a metronome beat (hereafter beat synchronization) shows considerable variability in child populations, and individual differences in beat synchronization are reliably related to reading development. Children with developmental dyslexia show impairments in beat synchronization. These impairments may reflect deficiencies in auditory perception of the beat which in turn affect auditory-motor mapping or may reflect an independent motor deficit.

In conclusion, the data reported here are supportive of atypical neural beat entrainment by the dyslexic brain in the delta band (~1–3 Hz), with the children with dyslexia showing a significantly different preferred phase both during

passive listening and during beat synchronization. Overall, the data are supportive of an interpretation of developmental difficulties in beat synchronization driven by impaired auditory perception of the beat. So, there is evidence that the accuracy of neural synchronization to an external beat is related to progress in reading and phonological development.

Precise specification of neural mechanisms should enable the optimization of remedial programmes based on improving temporal synchronization in children, for example programmes based on drumming and other forms of rhythm production, which have been shown to enhance both children's phonological awareness and their reading development (Overy et al., 2003; Degé and Schwarzer, 2011; Bhide et al., 2013; Slater et al., 2014; Flaugnacco et al., 2015; Serrallach et al., 2016).

7. Definition of dyslexia

Researchers became increasingly aware of the heterogeneity of LDs (Rourke, 1975) and this led them to definition and classification issues in order to understand the etiology of dyslexia. Because of these efforts, research has evolved to a point where there is a good understanding of the neuropsychological and behavioural correlates, and an emerging understanding of the neurobiological and environmental factors that cause this complex disorder.

Individuals with developmental dyslexia have difficulties with accurate or fluent word recognition and spelling despite adequate instruction and intelligence and intact sensory abilities. The ultimate purpose of reading is comprehension. Dyslexia is defined by difficulties with decoding whereas comprehension is more intact. Poor comprehenders show the opposite profile of adequate decoding but poor understanding. Dyslexia is independent and irrelevant to intelligence quotient (IQ).

Much discussion was whether the diagnostic threshold for dyslexia should be relative to age or intelligence quotient (IQ). However, published work does not support the external validity of the distinction between age-referenced and IQ-referenced definitions in terms of underlying neuropsychology or appropriate treatments. Although the two definitions overlap (some people with clinically significant reading problems meet only IQ-discrepancy criteria, whereas others meet only age-discrepancy criteria), children who meet either definition should be identified and treated.

8. Etiology

The etiology of dyslexia is being identified among Behavioural Genetics, Molecular Genetics and Environmental Influences.

Like all behaviourally defined disorders, the cause of dyslexia is multifactorial and is associated with multiple genes and environmental risk factors. Dyslexia is familial and moderately heritable and has been linked to nine risk loci (DYX1–DYX9) through significant studies.

The main advance in the genetics of dyslexia has been the identification of six candidate genes (DYX1C1 in the DYX1 locus on chromosome 15q21; DCDC2 and KIAA0319 in the DYX2 locus on chromosome 6p21; C2Orf3 and MRPL19 in the DYX3 locus on chromosome 2p16–p15; and ROBO1 in the DYX5 locus on chromosome 3p12–q12) and studies of their role in brain development. Four of these candidate genes, all except the two DYX3 candidate genes, which are DYX1C1, DCDC2, KIAA0319, and ROBO1 have been identified to affect neuronal migration and axon guidance and coregulate each other. Very little is known about the functions of the two DYX3 candidate genes. Two other studies have identified three new candidate genes for dyslexia (MC5R, DYM, and NEDD4L) on chromosome 18 and one shared with language impairment (CMIP), but these results need to be replicated.

Despite this important progress, much remains to be done to fully understand the causes of dyslexia. Genetic contribution to dyslexia in families increases with a high level of parent education (a bioecological gene by environment interaction).

Recently, there has been growing adoption of the view that dyslexia, a reading disability, is probabilistic in nature; children with a family history of dyslexia are considered "at-risk", and compensatory skills such as strong oral language or executive functions may be "protective factors" (Haft et al., 2016; Hulme et al., 2015; Muter & Snowling, 2009; Pennington, 2006).

On this well documented basis, Fletcher, J. (2009) emphasises that factors related to poverty and the family's orientation to literacy represent risk factors for dyslexia. However, another critically important factor is instruction. At a classroom level, the quality of reading instruction varies considerably. Genetically controlled studies investigating environmental contributions to the development of reading problems and many reviews have shown that children at risk for reading problems require instructional approaches that are more explicit. That means translation of the alphabetic principle into instruction through methods like phonics needs to be intentionally laid out in an organized fashion in order for at-risk children to make explicit what is inherently an implicit understanding of the relation of print and sound (Rayner et al., 2002).

As Stein J. (2019) points out, however, it seems to be necessary to look at individuals' nutrition. M- cells' high dynamic sensitivity requires high membrane flexibility so that their ionic channels can open and close very fast. This flexibility is provided by the local lipid environment, particularly by the incorporation into the membrane of one very important omega 3 long chain fatty acid, Docosahexaenoic acid (DHA) (Haag, 2003, Muskiet et al., 2004). This is normally provided in our diet by consuming oily fish. Green vegetables, flax or rape seed and seaweed all contain the shorter chain omega 3, alpha linolenic acid, but humans do not convert this into DHA very efficiently.

Dyslexia negatively affects school performance, mental health, and socioeconomic outcomes, burdening families and society (Hulme and Snowling, 2016; Peterson and Pennington, 2015). The causes of dyslexia are uncertain and multifactorial (Peterson and Pennington, 2015). Environmental factors are critical aspects of its etiology. Recently, a growing body of literature has identified the adverse effects of environmental pollutants on dyslexia, providing important clues (Liu et al., 2022; Xie et al., 2022; Xue et al., 2020). As competitive inhibitors of the sodium iodide symporter (NIS), perchlorate, nitrate, and thiocyanate can disrupt iodide uptake into the thyroid and further interfere with its function (De Groef et al., 2006; Tonacchera et al., 2004; Willemin and Lumen, 2017).

The approach taken by the Chinese scientists (Liu et al., 2022; Xie et al., 2022; Xue et al., 2020) provided quantitative research framework that nicely complements environmental factors. They determined the urinary concentrations of perchlorate, thiocyanate, and nitrate in children in China and assessed the association between exposure to these three analytes and Chinese dyslexia. The results demonstrated that children were ubiquitously exposed to the selected analytes. It was observed that elevated urinary thiocyanate levels were significantly associated with an increased risk of dyslexia in Chinese children, especially in boys.

9. Prediction – diagnosis

In this multifactorial framework, most cases of dyslexia cannot be explained by a single cognitive deficit. Despite this heterogeneity, it is broadly accepted that phonological awareness (PA) and rapid automatized naming (RAN) are two of the strongest—if imperfect—predictors of reading development (Pennington et al., 2012; Wolf & Bowers, 2000).

Some researchers have advocated the use of 'response to intervention' as an approach to diagnosis (Fletcher & Vaughn, 2009). To date, the evidence suggests that the most effective interventions for children with dyslexia are phonologically based, involving training in phoneme awareness and letter knowledge combined with structured reading practice (McArthur et al., 2012). However, there is a dearth of evidence for the efficacy of interventions to improve spelling and writing fluency – future research must address these important questions as a matter of urgency.

Comprehensive approaches that also include reading practice to build fluency and explicit teaching of comprehension strategies and vocabulary usually result in higher levels of overall reading proficiency (Stuebing et al., 2008). They should include evaluations of classroom programs, prevention programs, and remedial programs.

Response to intervention models link prevention and remedial interventions through multitiered approaches to service delivery in schools, including universal screening for reading (and math and behavior problems), monitoring progress of at-risk children through frequent assessment probes using reading fluency tasks, and providing increasingly intense intervention based on the child's progress (Fletcher & Vaughn, 2009).

9.1. Treatment and the role of ICTs

The development of evidence-based treatments for dyslexia has benefited from our understanding of the neuropsychology of the disorder, and the best interventions provide intensive, explicit instruction in phonological awareness, the alphabetic principle and phonics, word analysis, reading fluency, and reading comprehension.

Much more is known about effective remediation of reading problems in younger than in older children. There are some evidence shows that reading problems can be prevented with appropriate intervention in kindergarten and first grade (age range 5–7 years), at least in the short term. Professionals should not wait until children are formally diagnosed with dyslexia or experience repeated failures before implementation of reading treatment because remediation is less effective than early intervention.

Work on treatments for reading failure supports the following conclusions: intervention is most effective when provided in a one-to-one or small group setting; successful interventions heavily emphasise phonics instruction; and other important treatment elements include training in phonological awareness, supported reading of increasingly difficult connected text, writing exercises, and comprehension strategies. Many effective treatments are low cost, which further draws attention to the importance of early identification, prevention, and treatment of dyslexia for public health.

The number of interventions–imaging studies investigating how remediation of dyslexia alters brain activity is increasing. Briefly, effective intervention seems to pro mote normalisation of activity in the left hemisphere reading and language network. Additionally, increased right hemisphere activation has been reported after dyslexia treatment, which is sometimes interpreted as showing compensatory processes.

According to Stein J. (2019), use of filters can be proved beneficial to the dyslexia. Those who primarily suffer blur due to defocus, together with reduced convergence and double vision (diplopia) may be helped by using yellow filters. On the other hand, those who see words and letters appear to move around, jitter or 'fizz' due to their apparent rapid random motion, and who may also tend to lose concentration and suffer from headaches or eyestrain when trying to read, may be helped most by using blue filters. (Stein J.,2019). On the other hand, auditory magnocellular sensitivity can be improved by relatively simple techniques such as training children to drum actively and rhythmically.

Last but not least, we emphasize the significance of digital technologies in the educational and dyslexia domain, which are very productive and successful, and how they facilitate and improve assessment, intervention, and educational procedures via mobile devices that bring educational activities everywhere [18-19], various ICTs applications that are the main supporters of education [20-32], and AI, STEM, and ROBOTICS that raise educational procedures to new performance levers [33-35] and games [36-37]. Additionally, ICTs are being improved and combined with theories and models for cultivating emotional intelligence, mindfulness, and metacognition [38-50], accelerates and improves more than educational practices and results, especially for students with dyslexia, treating domain and its practices like assessment and intervention.

10. Conclusions

Dyslexia is multifactorial and is associated with multiple genes and environmental risk factors. Several preceding studies have attempted to investigate multiple candidate mechanisms of dyslexia, including auditory, visual, and motor processes.

Unfortunately, the results cannot validate any particular causal mechanism. It is possible that each factor represents clusters of symptoms that indicate underlying abnormalities in a processing system but are not a direct cause of reading difficulties themselves). Broadly speaking, skilled reading requires rapid communication among a distributed network of visual, auditory, and language processing systems and an impairment in any one of these systems, or the connections between them, could cause difficulties learning a complex skill like reading (Wandell & Yeatman, 2013).

The results of the survey of O'Brien G., Yeatman J., (2020) demonstrate that a core phonological deficit model is insufficient to account for many cases of developmental dyslexia. Instead, dyslexia should be conceptualized as a disorder that may arise from several distinct loci. Pennington and colleagues research (which has capitalized on large samples) demonstrate that individuals with dyslexia have a heterogeneous profile of cognitive and linguistic impairments (Pennington, 2006; Pennington et al., 2012; Peterson & Pennington, 2015). Their main conclusion is a lack of concordance with either a single deficit or cascading deficit model. As such, the results claims that a single mechanism, either phonological, visual, or non-sensory, can be considered the "fundamental" or "core" deficit of dyslexia.

Moving forward, it is proposed an additive risk factor mode of dyslexia in which multiple dimensions of sensory, cognitive, and linguistic processes contribute distinct risk for reading difficulties. Probably, they will be dimensions that we have not explored here, as there is growing evidence for a unique role of oral language and vocabulary skills in reading development (Catts et al., 2017; Snowling, 2008; Snowling & Melby-Lervåg, 2016).

The clinical implications of this multifactorial model are an important target for future research. Whether or not different risk profiles predict outcomes for children enrolled in competing intervention programs is an empirical question that cannot be readily inferred from correlational data.

In sum, an additive model outperforms cascading deficit models or models that only consider measures of phonological processing without considering the role of sensory processing. Rather than continuing to seek a single underlying cause of dyslexia, the field should systematically build toward a more complete model of the factors that add risk (or protection) for reading difficulties. Our data and model necessitate a shift toward theories that explain skilled and disabled reading as emerging from a high-dimensional space determined by several distinct processing systems.

According to Peterson R. & Pennington B. (2012), future research should be in the field of:

- The nature of the phonological deficit and its interaction with other linguistic and non-linguistic risk factors.
- The developmental course of neural abnormalities and how these predict treatment response.
- Which environmental risk factors contribute to the development of poor reading and whether these are the same across demographic groups.
- Testing of the hypothesis that visual attention problems underlie difficulties with rapid serial naming and reading fluency.
- Identification of additional risk loci to account for missing heritability.
- Clarification of which risk loci are unique to dyslexia and which overlap with comorbid disorders.
- Investigation of newly discovered genetic mechanisms, such as copy number variations, parent-of-origin effects, and epigenetic effects.

Therefore, the suggestion made by Fletcher, J. (2009) is that research in the future should focus on children whose single-word deficits are resistant to intervention, with comparisons to typical and at-risk children. Such studies may shed new light on the neuropsychological and neurobiological factors. We need to prioritize intervention and link our scientific and professional practices to the goal of enhancing adaptive functions in children with or at risk for LDs.

Compliance with ethical standards

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