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JOURNAL OF NEUROLOGY AND NEUROSCIENCE
ISSN 2171-6625

2015
Special Issue

A Mini-Review: Toward a Comprehensive Theory of Dyslexia

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Abstract

Current research suggests that the neurobiological substrate of dyslexia involves the dysfunctional orchestration of a multi-dimensional and hierarchical circuitry of at least three neuronal networks. This circuitry principally involves the posterior corpus callosum, left arcuate fasciculous, and the right frontostriatal attentional control network. The key to understanding the disability and in forging a comprehensive theory of dyslexia may be found in investigations aimed at interactions among all three networking territories.

Keywords: Dyslexia; Corpus callosum; Arcuate fasciculus; Frontostriatal attention control

Developmental dyslexia is a hereditary, neurocognitive-based learning difficulty, usually identified early in children's primary education when young children struggle to acquire proficiency in beginning reading skills. Prevalence estimates vary, ranging from 5% to as high as 20% [1]. After more than a century of research and the implementation of a broad range of remedial strategies, this disability, which affects individuals irrespective of their level of intelligence, motivation to learn and adequate educational and social circumstances, remains relatively intransigent to educational approaches. The reading, spelling, and array of related cognitive difficulties found in children with dyslexia persist into adulthood [2]. Familial studies indicate an etiological origin in an intricate interplay of neurobiological, genetic, epigenetic, and environmental factors [3].

To further complicate current clinical and educational overtures to the problem, the term "dyslexia" itself has become the subject of current controversy, with several scholars preferring "reading disability" [4]. This suggested change in terminology is motivated partly to broaden the category to include greater numbers of subtypes representing a wider variety of theoretical accounts of the disability [5]. Notwithstanding the longer-term value of expanding the benefits of counselling and remedial services to more affected individuals, there remains a strong experimental and scientific basis supporting the more restrictive "phonological deficit hypothesis" [6]. Over the last 20 years, this assumption has guided clinical practice and research [7,8]. According to this view, dyslexia is an accepted term, referring to poor readers who struggle with (1) initially forming, or accessing and retrieving, phonemic and phonological representations, (2) poor verbal short-term memory, and (3) rapid lexical access.

This review will work backwards from this premise, but will identify three key brain regions that may be central to the metacognitive modulation of phonological processing, suggesting a more comprehensive distributed neurobiological framework. Numerous subtyping models have been suggested as alternatives, but the predominant view is that individuals with left hemisphere localized sub-lexical phonological deficits make up the majority of poor readers [2,9,10]. On the other hand, studies have produced strong support in some individuals with dyslexia for deficits in spatial and auditory attention [11,12], visual magnocellular-dorsal pathway deficits [13], deficits in cerebellar functions [14], and deficits in interhemispheric processing [15]. However, while each of these behavioral and neurological factors may be clinically important when considering intervention and counselling, each has also been linked with phonological deficits, suggesting a deficiency in phonological processing may constitute a common final pathway of the disability. Therefore, this review will focus on phonological processing as the most common academic difficulty in dyslexia and will show preference selectively for research that has been replicated. This review will not address the important issue of gender effects in dyslexia. The literature on gender is controversial and highly inconsistent, warranting exclusive analysis in a separate report. The review will present the broad outlines of a theory of dyslexia that attempts to integrate the variety of subtypes and putative etiological factors into a unified circuitry of interconnected neuronal networks: a tripartite widely distributed, attentionally-controlled, cortical-subcortical,

interhemispheric networking of intercommunicating neuronal areas. Such an ambitious undertaking is necessarily preliminary, with conjectural inferences, but will be accretive to clinical knowledge and suggest clear directions for future research.

Research in dyslexia has identified three specific brain regional networks within this circuitry that, together, may provide the foundations for a comprehensive theoretical account. Phonological processing requirements and computational routines are implicit in the functions of each of these neuronal networks.

Arcuate fasciculus

The most common finding revealed by functional magnetic resonance imaging (fMRI) studies is that poor readers underactivate attentionally-controlled left hemisphere posterior areas, principally including dorsal temporoparietal and ventral occipitotemporal cortex [1]. Left inferior prefrontal cortex has shown both underactivation and overactivation. Structural imaging studies are largely in agreement in showing lower diffusion tensor imaging (DTI), fractional anisotrophy (FA) values in these left hemisphere posterior and frontal regions and their interconnections [7]. FA values are thought to measure the integrity of axonal microstructural properties, mainly myelination and packing density [16]. In addition, several derived FA measurements can also reflect axon properties such as the magnitude of diffusion and the alignment of axons. In particular, studies investigating these left hemisphere regions have pinpointed what appears to be a hallmark processing bottle-neck for dyslexia: underdevelopment of the left arcuate fasciculus, which is the direct white matter route connecting Broca's territory in prefrontal cortex with Wernicke's territory in posterior temporoparietal cortex.

The left arcuate functionally engages a sensory-motor phonological processing loop [17]. The left inferior frontal cortex, interacting with left posterior cortex, may be recruited in language processing to form articulatory programs for speech (phonemic segmentation); whereas the left posterior-temporal and angular gyrus, interacting with left frontal cortex, maps phonological representations to semantics [18], and supports phoneme-grapheme integration [19]. Thus, the left arcuate fasciculus acts reciprocally as an anterior-posterior intercommunicating unit in high-level phoneme and phonological computations.

In typical adults, the left arcuate is asymmetrically better developed than the right, containing relatively greater fiber volume and enhanced microstructural integrity [20,21]. Moreover, its functional and structural strength is associated in normal adults with the ability to learn new words [22] and is related to superior phonological skill in normal children [23,24].

In individuals with dyslexia, DTI studies are highly consistent in implicating underdevelopment of the left arcuate and its topologically connected terminations in left temporoparietal and frontal regions [7]. However, a longitudinal DTI study indicates a complex and little understood maturational trajectory. The results suggest that poor readers may undergo earlier development of the left arcuate which shortens the typical extended period of plasticity important for learning and maximizing development [25]. Even in pre-kindergarten children, the earlier establishment of left language specialization is associated with inferior language abilities [26]. Nontheless, in adults with dyslexia, the resulting inefficiency in signal strength and computational power of the left arcuate is correlated with poor phonemic awareness and significant underperformance on tests of word reading, verbal short-term memory, speech perception and lexical access [8,27].

An important question is whether anomalous development of the arcuate is a cause or consequence of underachievement in reading? Studies have shown that reading instruction with illiterate adults, and intensive remedial training with children who are poor readers can produce structural improvements of the left arcuate concomitant with gains in reading [28,29]. On the other hand, prior to reading instruction, nine of 40 kindergarten children who were at risk for dyslexia because of poor phonemic awareness, were found to have a smaller and less mature arcuate [30]. In addition, studies have found that the initial coherence of the direct segment of the left arcuate was (1) already anomalous in prereaders at familial risk for dyslexia [31] and (2) predictive in normal children of their reading ability over a three year period [32]. Thus, the structural integrity of the left arcuate fasciculus appears to be receptive to reading instruction, but also a significant cause of reading failure in children and adults.

Corpus callosum

The second structural neuronal mechanism that appears to play a critical role in dyslexia is the corpus callosum, the main commissure coordinating facilitative and inhibitory processing between hemispheres. Corpus callosum neurons are largely excitatory. They may, however, target inhibitory interneurons at their points of origin/termination [33]. A study by Frye et al. [34], found that individuals with dyslexia demonstrated higher FA in the splenium area of the posterior corpus callosum. The splenium, which is the most caudal segment of the corpus callosum, interconnects its foremost axons to posterior temporal and parietal association cortex, while primary and secondary visual areas are interconnected by hindmost splenium fibers [35]. Apparently, the integrity of the corpus callosum partly reflects the maturational architecture of topologically connected cortico-cortical regions [15,36-38]. For instance, atypical maturation of the corpus callosum is thought to reduce the drive for left hemisphere lateralization of the reading network [7] and, because the two hemispheres compete in development by mutual competitive inhibition, we would expect disinhibition and accelerated maturation of homologous regions in the right hemisphere [39]. Thus, there may be a hierarchical relationship with callosal maldevelopment precipitating overdevelopment of connected right hemisphere regions and underdevelopment of the arcuate fasciculus. Other studies are consistent with this possibility, reporting higher corpus callosum FA and poor phonological skills in children and adults who are poor readers [40,41]. Related studies have also reported a larger and abnormally shaped corpus callosum associated with inferior phonological ability in children and adults with dyslexia [42,43].

Finally, Rumsey et al. [44] were the first to suggest that the apparent "advanced" development of axonal fibers traversing through the posterior corpus callosum may be due to a shortened

period of plasticity and reduced synaptic pruning of the right angular gyrus. In effect, a better connected callosum (i.e., larger with less diffusivity) may reflect abnormally early but truncated maturation compromising the interhemispheric functions of both hemispheres. In this regard, it is important to point out that the hypertrophied callosum in dyslexia stands in sharp contrast to other developmental disorders, i.e., schizophrenia, autism, alien hand syndrome, ADHD, bipolar disorder, and borderline personality disorder, all of which have demonstrated a smaller callosum with sparser connectivity [45].

Cognitive control

In typical development, the maturation of the dorsal left hemisphere reading network and the posterior corpus callosum, both serving phonological computations, undergoes a prolonged period of growth which co-occurs with the extended maturation of projection and association tracts of the third key processing area of dysfunction in dyslexia: the frontostriatal-parietal, cognitive control system [46]. Late maturation of this system's network dynamics, which are responsible for the supervisory metacontrol of other distinct executive and attentional networks, ensures an extended period of plasticity [47]. Plasticity in all three appears to be greatly reduced or absent in dyslexia.

Petersen and Posner [48], based on over 20 years of basic research, have presented a detailed account of how such a system may modulate the attentional control of cognitive processes. They have identified three attentional networks, i.e., alerting, orienting, and executive, which are anatomically and neurochemically independent of information processing and which subserve cognitive processes. All are highly bonded in a unified, feed-forward and feed-backward synchronous organization. A variety of related attentional models formulated on the centrality of dorsal and ventral networks have been published [49,50]. However, a common feature of each is the significant part played by areas of bilateral frontal and parietal cortex acting as the "command center" in exercising higher-order control over goal-directed behavior [51]. More specifically, a majority of researchers are in agreement that the right hemisphere system assumes dominance over the left when cognitive control is recruited to regulate attentional and information processing resources [52-56]. In evolution and in ontogenetic development, some degree of asymmetrical control may be necessary to unify behavioral management over a potentially competing bilateral system. Such an arrangement ensures maximizing adaptiveness to rapidly changing environmental circumstances. A more detailed exposition of these models is beyond the scope of this review. However, this preface leads to two important considerations. Firstly, extensive evidence suggests that the right system manages the distribution of endogenous and exogenous attention and, hence, the magnocellular dorsal visual pathways connecting posterior parietal to frontal cortex may be within its organizational jurisdiction. Moreover, a deficit in the right cognitive control system theoretically may also negatively affect interhemispheric and/or cerebellar functions [57]. Secondly, evidence suggests that the right hemisphere system may subserve the left hemisphere phonological lexicon.

For example, research in dichotic listening using consonant-vowel stimuli demonstrates the importance of efficient recruitment of cognitive control in modulating categorical speech perception and basic auditory processing [58]. Dichotic listening usually produces a right ear advantage recalling simultaneously presented verbal material, which inheres in the language specialization of the left hemisphere. This inference is based on (1) stronger, more direct contralateral ear-hemisphere pathway connections and (2) the inducement, by the verbal nature of the stimuli, of a rightward attention and left hemisphere arousal bias [59]. The area of the left planum temporale houses consonant and vowel representations and is adapted to the phonological rules of one's spoken language [60].

Kompus et al. [58] engaged cognitive control by increasing the difficulty of the dichotic task by forcing attention to one or other ear, requiring suppression of the stimuli arriving at the unattended ear. In the more difficult forced left condition, the fMRI results showed distinct activations of left inferior prefrontal and caudate; whereas forcing attention to both left and right activated the right inferior frontal sulcus, caudate, and the right inferior parietal lobule. Thus, this study in concert with the immense dichotic literature [59], suggests that right hemisphere processes can be reliably recruited for phoneme perception and retrieval in situations requiring cognitive control. Indeed, irrespective of design variations in processing requirements, dichotic recall from the left ear must engage the right hemisphere and posterior corpus callosum in interhemispheric access to the phonological lexicon.

Research in dyslexia indicates anomalous development of the right hemisphere cognitive control system. It has been suggested that the overdevelopment and abbreviated period of regressive synaptic pruning of the corpus callosum in dyslexia may have compromised the development of the left lateralized language network. It has also been suggested that such callosal overdevelopment would be expected to be associated with a similar overdevelopment of connected areas of the right hemisphere. Morphological studies with male children with dyslexia (ave. age of 11) showing increased numbers of right Heschl's gyrus and reversed hemispheric asymmetries of the surface area of the planum temporale with right larger than left are consistent with this prediction [61]. Also, children with dyslexia have shown overactivation of the right temporoparietal cortex which was related to lower reading scores [62]. Measures of cognitive control were not included in these studies, so they may or may not turn out to be relevant to the present thesis. However, other research using the dichotic paradigm has provided indirect and direct support for the hypothesis of a right hemisphere cognitive control dysfunction in dyslexia.

Dichotic research has been consistent in finding children who are poor readers to be better than good readers in simple left ear recall [63]. In contrast, however, they are just as consistent in underperforming when the task is made more difficult by forcing a reorientation to the left ear after attending to the right [64]. A plausible interpretation of this paradox is poor readers may develop the right attention networking system precociously, giving them an advantage when the task is relatively easy, but compromising performance when increasing attentional

demands call for cognitive control. Support for this inference was provided by Bowen and Hynd [65] who reported that good readers eventually catch up to poor reader's earlier superior left ear performance. My colleagues and I have provided a more direct test in a programmatic series of forced attention dichotic studies. We found evidence of dysfunctional frontostriatal cognitive control in dyslexia in four replications with different samples, consisting of children and adults and including males and females [64,66,67]. Two of these studies included reading-level controls, which discounts the possibility that the deficit in cognitive control may be a secondary consequence rather than a cause of their reading disability.

In summary, the high connectivity found in posterior corpus callosum in individuals struggling with dyslexia suggests its typical inhibitory function in suppressing right hemisphere processes to favor left may be compromised. Such a possibility would be expected to cultivate premature but pretermitted maturation of the right hemisphere cognitive control system and, simultaniously, underdevelopment of the left lateralized language network. Thus, the neurobiological substrate of dyslexia may involve the dysfunctional orchestration of a multi-dimensional and cross-model neuronal circuit. This circuitry principally involves the posterior corpus callosum, left arcuate fasciculus, and right frontostriatal control system. Research studies in dyslexia have focused almost exclusively on one or two of these regions; whereas the key to more accurately understanding the disability may be found in investigations aimed at interactions among all three networking territories. Developmentally informed, longitudinal research will be required to unravel the complexities involved in answering how and why these three interconnected networks fail to mature in individuals with dyslexia. Without question, a comprehensive theory of dyslexia will need to acknowledge that dyslexia implicates a developmental breakdown in a widely distributed circuitry of interrelated processing territories.

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An Original publication of ImedPub in a Special Issue-**Current Trends in Neurodegenerative Diseases and Medical Procedures**, Edited by **Dr. Jianqi Cui**, Department of Biochemistry and Molecular Biology, School of Basic Medical Sciences, Ningxia Medical University (NXMU), PR China.