

## The Need for a Valid Theory of Dyslexia

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### Description

Developmental dyslexia is a biologically 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 implementing 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. Familial studies indicate an etiological origin in a complex and largely unknown interplay of genetic, epigenetic, and environmental factors [2]. As a result, parents, clinicians and educators have no clear theoretical understanding of the disorder. The emotional trauma on individuals and the costs to society are considerable.

On a positive note, the development of non-invasive neuroimaging techniques to study the human brain in vivo holds great promise to eventually provide the theoretical data base that we need to formulate more successful interventions. Functional and structural imaging research has now mapped the brain circuitry implicated in reading [3]. These studies with average readers support a distributed, attentionally-controlled, multi-dimensional, cortical-subcortical, interhemispheric reading network. Studies with individuals with dyslexia have pinpointed the neuronal areas within this circuitry that are underdeveloped and presumably associated with the disability.

This research focus has led to a resurgence of interest in variations of the traditional notion [4] that dyslexia may be related to atypical interhemispheric processing between the left and right cerebral hemispheres [5,6]. Specifically, in findings that have been replicated, two underdeveloped brain networking regions appear to be centrally involved in the disorder: (1) left arcuate fasciculus, which is the direct white matter route connecting Broca's and Wernicke's territories in the left hemisphere [7-10]; and (2) the posterior region of the corpus callosum, which drives the lateralization process and interconnects the bilateral parietal cortices with frontostriatal projection tracts, supporting top-down cognitive control of behavior [3,11].

Such research findings will be accretive to forging a comprehensive theoretical account of the underlying neurobiology of dyslexia. These specific findings are all the more intriguing in view of research showing that the structures of both the left arcuate fasciculus and the posterior

corpus callosum are enhanced and strengthened by learning to read, suggesting an environmentally sensitive avenue for future instructional interventions [12,13]. In summary, our knowledge of dyslexia is accumulating at a faster pace than ever before. We sorely need a valid theoretical account before we can begin to endorse newer and, hopefully, more effective remedial and/or preventive efforts. That time may not be too far in the future.

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Received July 06, 2015; Accepted August 14, 2015; Published August 26, 2015

Citation: Kershner JR (2015) The Need for a Valid Theory of Dyslexia. *Brain Disord Ther* 4:179. doi:10.4172/2168-975X.1000179

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