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Dyslexia: competing theories explained

DYSLEXIA: COMPETING THEORIES EXPLAINED

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If you are able to see this string of symbols as a line of meaningful units, you are performing an extraordinary task. Many people take for granted the ability to read, however, not everyone can do it so effortlessly. Dyslexia in its broadest sense is consistent difficulty reading despite having the necessary education, intelligence, and opportunity. Definitions of the disorder that are more specific and in-depth have been heavily debated and revised countless times since the term 'dyslexia' was coined in the 1800s. One theory on the cause of dyslexia would lead to it being overly diagnosed while the next would question its very existence. In the past few decades, advances in neuroimaging technologies have allowed researchers to study the neural mechanisms of Dyslexia, enabling them to test predictions made on the basis of long-standing psychological theories formed over the years. The two prevailing theories are the Phonological Deficit Hypothesis and the Magnocellular Theory. Both are supported by extensive experimentation and concrete evidence but neither has been agreed upon as the universal explanation of the disorder. The latest Dyslexia research aims at determining which, if either, of these two theories is an appropriate model of Dyslexia.

DYSLEXIA DESCRIBED

As is the case for many complex disorders, there is no single definition of Dyslexia. Decades of study have led to the development and modification of many descriptions of the disorder, each based on the distinctive findings of different research groups with different goals.[1] Despite their subtle differences, most current descriptions have certain aspects in common. First, many institutions use the phrase 'Developmental Dyslexia' rather than simply 'Dyslexia.' This variation is used because the condition that is traditionally associated with the word 'dyslexia' is persistent and presents itself at a young age, as opposed to conditions with similar symptoms that are caused by a discrete postnatal brain injury.[2] Many descriptions specify this by indicating that Dyslexia is a result of a genetic risk factor.[3] For our purposes, 'Dyslexia' and 'Developmental Dyslexia' will be used interchangeably. Secondly, most groups identify Dyslexia as a learning disability, which the National Center for Learning Disabilities specifies as a neurological disorder rather than "a 'difference' or 'difficulty' with learning."[4] This distinction is essential because Dyslexia has historically been mistaken as the result of low general intelligence or lack of proper instruction. Although a child with dyslexia and a child without dyslexia could have the same reading skills, the cause of their impairments is not the same. Accordingly, the core definition of Dyslexia is that it entails a difficulty with reading despite otherwise normal intellectual functioning and educational opportunities.[5] Finally, descriptions of Dyslexia include symptoms like trouble with spelling, difficulty identifying words, and problems learning to read in general. The International Dyslexia Association integrates all of the commonalities with the following definition:

> "Dyslexia is a specific learning disability that is neurological in origin. It is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede the growth of vocabulary and background knowledge."[6]

This description and many others like it do not mention other symptoms associated with Dyslexia. This is due to the diversity of symptoms and to the inconsistency in which secondary symptoms appear. Perceptual problems in the visual, auditory and tactile domains, and problems with balance and motor control are among the symptoms that have been observed in many, but not all Dyslexics.

LEVELS AT WHICH DYSLEXIA IS CHARACTERIZED

Beneath its basic description and diverse set of symptoms, Dyslexia is a highly multifaceted disorder. Researchers attempt to characterize Dyslexia at four different levels: genetic, neurobiological, cognitive, and behavioral. They seek to relate findings at each level and map pathways of those relationships, ultimately revealing any causal connections between the levels.[7] Other problems associated with Dyslexia include problems with phonology, which is the mental representation and processing of speech sounds. It has been estimated that one-third of Dyslexics exhibit phonological processing deficits that consist of problems that limit their ability to separate out speech sounds and recombine them in a new order.[8] This inconsistency could be explained by the fact that Dyslexia is frequently comorbid with other neurodevelopmental disorders like Specific Language Impairment (SLI) and Attention Deficit Hyperactivity Disorder (ADHD).[9] But this explanation begs the question: why are we concerned with secondary symptoms like auditory deficits if they appear infrequently and can be accounted for by comorbidity? The answer lies in the fact that researchers are still trying to confidently identify the cause of Dyslexia. It has been found that 1/3 of people with Dyslexia who exhibit an auditory impairment also have a more severe phonological deficit. This discovery indicates that there may be a connection between reading impairment, which is the core characteristic of Dyslexia, and deficiencies in phonological processing.[10] To make sense of the varying symptoms, determine their relationships, and identify a cause of Dyslexia, researchers typically follow one of two theoretical approaches. These two approaches mark a significant difference between the Phonological Processing Deficit Hypothesis and the Magnocellular Theory of Dyslexia.[11]

PHONOLOGICAL DEFICIT HYPOTHESIS

The Phonological Deficit Hypothesis, which was first proposed in the 1970s, approaches the varied symptoms associated with Dyslexia by concentrating on the one cognitive system that is believed to have the most causal role in the disorder. As its name indicates, this theory focuses on phonological processing and treats all other potential symptoms of Dyslexia as comorbid markers with no causal relationship to reading disability. At the biological level, the Phonological Deficit Hypothesis proposes that Dyslexia is caused by a genetic risk factor that, combined with events occurring during gestation, causes physical anomalies in the language-processing area of the left hemisphere.[12] In terms of cognition, the Phonological Deficit Hypothesis is based on the process by which we learn to read. In order to learn an alphabetic writing system, the brain must associate individual phonemes (i.e., the smallest units of sound in a language) with the corresponding graphemes (i.e., alphabetic letters) that are written but represent spoken sound. The brain needs to be able to distinguish individual phonemes in order for reading to be carried out properly. Consequently, this process relies heavily on the brain's ability to reliably hear, represent, and recall phonemes. The Phonological Deficit Hypothesis maintains that the core cognitive deficit of Dyslexia lies in the formation and manipulation of these crucial phonological representations. This essentially leads to poor mapping of letters to sounds and thus leads to difficulty reading and performing phonological tasks.[13] In terms of behavior, the Phonological Deficit Hypothesis cites poor grapheme-phoneme mapping as the cause of the most common characteristic of Dyslexia: poor reading. It is not concerned with the sensory and motor symptoms that can accompany reading disability in Dyslexics. Instead, the Phonological Deficit Hypothesis considers all non-phonological symptoms to be comorbid and unrelated to the core cognitive deficit.[14] This distinction sets the Phonological Deficit Hypothesis apart from its rival hypothesis, the Magnocellular Theory of Dyslexia.

MAGNOCELLULAR THEORY

The Magnocellular Theory, which was proposed years after the Phonological Deficit Hypothesis, takes a different theoretical approach to the complex and varied symptoms of Dyslexia. It identifies a high-level sensory processing pathway as the primary cause of the disorder. Unlike the Phonological Deficit Hypothesis, the Magnocellular Theory maintains that some of the symptoms not consistently seen in Dyslexics are evidence of the underlying cause of Dyslexia and are not merely symptoms of a comorbid disorder.[15] The Magnocellular Theory is based on the concept that our visual system is divided into two neuronal pathways.[16] The first is the parvocellular pathway, which responds most strongly to stimuli that are different in color, high in contrast, and reverse contrast slowly. The other is the magnocellular pathway, which responds best to stimuli that are color neutral, low in contrast, and reverse contrast quickly.[17] The theory points to a widespread dysfunction of cells in the visual magnocellular pathway and in the associated auditory magnocellular pathway.[18] In terms of reading disability, this dysfunction is thought to have two important consequences. First, the deficient auditory pathways cause difficulty in processing sounds, which leads to a phonological deficit. This deficit described by the Magnocellular Theory does not have the same origin as the deficit described by the Phonological Deficit Hypothesis, yet it would cause the same reading disability. Second, the deficient magnocellular pathways cause difficulties with visual perception and binocular control, both of which could cause or exacerbate poor reading.[19] By giving higher order sensory processing pathways a causal role in Dyslexia, the Magnocellular Theory is also able to account for the presence of the many secondary visual and auditory manifestations. The Magnocellular Theory ultimately explains the presence of reading disability through both an auditory-phonological deficit and a visual deficit.

THEORIES COMPARED

The differences between the Phonological Deficit Hypothesis and Magnocellular Theory are apparent even in the short descriptions above. They take opposing theoretical approaches to Dyslexia, identify causes in completely different neurological systems, and acknowledge unique sets

Artwork by
Alexandra Maxim +
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of symptoms. Despite these differences, there are different bodies of literature and study that support (or refute) both theories. Much of the current study of Dyslexia is therefore focused on determining which theory is a more accurate model. At first glance, the Magnocellular Theory appears to be a better model of Dyslexia because it extends the Phonological Deficit Hypothesis. The Magnocellular Theory provides a neurological cause of the disorder and accounts for the high incidence of sensory symptoms in Dyslexics instead of segregating them as comorbid markers. Additionally, it is the culmination of many theories that cite auditory or visual dysfunction as the cause of reading disability in Dyslexia.[20] Following this line of thought, it appears that the Magnocellular Theory is the more accurate hypothesis, but I don't believe this to be the case.

The Magnocellular Theory may not actually be the most appropriate model of Dyslexia, particularly because it doesn't explain the inconsistent appearance of sensory symptoms both within and across different populations of individuals with Dyslexia. While studying the hypothesis, I had to wonder: If dysfunction of the visual magnocellular pathway were the cause of a phonological deficit and thus reading disability, wouldn't all Dyslexics exhibit some degree of visual impairment, especially those with severe reading disability? And if that dysfunction also existed in the auditory magnocellular pathway, wouldn't some extent of auditory impairment be consistently found in people with Dyslexia? Many studies have been performed over the past few years in an attempt to shed light on this question by determining the extent of sensory and motor deficits and, ultimately, evaluating the validity of the Magnocellular Theory. One study for example, administered a battery of psychometric, phonological, auditory, visual, and cerebellar tests to a group of 16 Dyslexic college students. It was found that all 16 Dyslexic students suffered from a phonological deficit while 10 had an auditory deficit, four had a motor deficit, and two had a visual (magnocellular) deficit. Researchers concluded that these results supported the Phonological Deficit Hypothesis more strongly than the Magnocellular Theory.[21] To me, this study and many similar to it only help to confirm my belief that the Phonological Deficit Hypothesis is a more appropriate model of Dyslexia. It may not be comprehensive enough on its own, but it has the potential to serve as an excellent foundation for future theories. While I have more confidence in a hypothesis that accurately accounts for only some of the complexities of Dyslexia, other people believe that a hypothesis with less support and more breadth is a better fit. As research continues, the hypotheses are further refined and we come closer and closer to understanding the cause of the phonological deficit and ultimately to understanding the mysteries of Dyslexia.

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