



DYSLEXIA: IS IT ALL IN YOUR MIND?

Recent neurological research provides new insight into the mechanisms and etiology of developmental dyslexia, although there is still much to learn and discover. In this article, we will consider the latest neuroanatomical findings that may, in part, be responsible for the functional difficulties that challenge individuals with dyslexia. The conjecture explored here is that there is a disruption of the cerebral architecture during gestation that sets in motion a cascade of events resulting in reorganization of neuronal circuits and networks. This reorganized anatomical substrate is not optimally organized for language acquisition and does not flourish in the typical environment/education system. Learning difficulties may result depending on the severity and location of brain alterations, the neural plasticity of the system, available compensatory cognitive strategies, and environmental conditions.

Anatomical Differences

In 1979, Albert Galaburda and Thomas Kemper examined a brain removed during an autopsy from a 20-year-old man with dyslexia and reported that there were nerve cells in unusual parts of the cerebral cortex. Subsequent studies at the Dyslexia Research Laboratory at Beth Israel Hospital in Boston, MA, of four dyslexic males and three dyslexic females showed that in the males (less so in females) clusters of “ectopic” neurons are consistently seen in the outside layer of the cerebral neocortex. This layer usually is devoid of nerve cell bodies. Most ectopias were in the frontal and perisylvian language regions. Ectopias are produced before six months of gestation

when there is a breach in the pial-glia border, which normally prevents neurons from migrating too far. Although female dyslexics had only a few ectopias, large numbers of gliotic regions representing areas of neuronal loss were present in the cortex.

Etiology of the Anatomical Changes

Ectopias result from the disruption of the developing cerebral cortex before neuronal migration ends at mid-gestation. The focal gliotic regions in female dyslexics may be the outcome of a similar pathological process acting during the third trimester of early postnatal period after neuronal migration is completed. An insult spanning the two periods could produce both ectopias and areas of neuronal loss. Because autoimmune disorders (work begun by the late Dr. Norman Geschwind in 1982) may be increased in individuals with dyslexia, it was suggested that maternal auto-antibodies might injure the developing brain during gestation, leading to the type of neuropathology seen in dyslexia. This view is not supported by work in experimental animal models. Further, new findings in the human and in experimental models point to the importance of genetic factors. An exciting finding recently by Dr. Bruce Pennington and colleagues is that a region on chromosome 6 may be related to dyslexia. It is intriguing that this area contains many genes related to immune function.

How Might Anatomical Changes Affect Function?

Ectopias are densely and aberrantly connected with other brain areas. Thus, one result of ectopia formation is the alteration of brain organization. One such alteration in dyslexia is the lack of asymmetry in a language-related cortical region called the planum temporale, an auditory area that lies on the superior surface of the temporal lobe. In control subjects, the planum temporale is usually larger in the left hemisphere. However, the dyslexics discussed above showed symmetry of the region. Another change involves one subsystem (magnocellular system) of the visual pathway that may be *functionally deficient* in individuals with dyslexia (Livingstone, Galaburda, and colleagues). The visual processing disturbance could interfere with normal reading ability. Likewise, similar deficits in other sensory pathways, such as the auditory system (refer to the work of Paula Tallal), could interfere with the

normal acquisitions of phonological skills. The visual and auditory systems both show related anatomical changes in organization and neuronal size. However, the functional meaning of these changes is not always clear. For example, Margaret B. Rawson and Thomas West both have emphasized that differences in brain organization sometimes may impart a processing advantage. Albert Einstein and Thomas Edison would undoubtedly concur.

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