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The magnocellular theory of developmental dyslexia.

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Abstract

Low literacy is termed 'developmental dyslexia' when reading is significantly behind that expected from the intelligence quotient (IQ) in the presence of other symptoms--incoordination, left-right confusions, poor sequencing--that characterize it as a neurological syndrome. 5-10% of children, particularly boys, are found to be dyslexic. Reading requires the acquisition of good orthographic skills for recognising the visual form of words which allows one to access their meaning directly. It also requires the development of good phonological skills for sounding out unfamiliar words using knowledge of letter sound conversion rules. In the dyslexic brain, temporoparietal language areas on the two sides are symmetrical without the normal left-sided advantage. Also brain 'warts' (ectopias) are found, particularly clustered round the left temporoparietal language areas. The visual magnocellular system is responsible for timing visual events when reading. It therefore signals any visual motion that occurs if unintended movements lead to images moving off the fovea ('retinal slip'). These signals are then used to bring the eyes back on target. Thus, sensitivity to visual motion seems to help determine how well orthographic skill can develop in both good and bad readers. In dyslexics, the development of the visual magnocellular system is impaired: development of the magnocellular layers of the dyslexic lateral geniculate nucleus (LGN) is abnormal; their motion sensitivity is reduced; many dyslexics show unsteady binocular fixation; hence poor visual localization, particularly on the left side (left neglect). Dyslexics' binocular instability and visual perceptual instability, therefore, can cause the letters they are trying to read to appear to move around and cross over each other. Hence, blanking one eye (monocular occlusion) can improve reading. Thus, good magnocellular function is essential for high motion sensitivity and stable binocular fixation, hence proper development of orthographic skills. Many dyslexics also have auditory/phonological problems. Distinguishing letter sounds depends on picking up the changes in sound frequency and amplitude that characterize them. Thus, high frequency (FM) and amplitude modulation (AM) sensitivity helps the development of good phonological skill, and low sensitivity impedes the acquisition of these skills. Thus dyslexics' sensitivity to FM and AM is significantly lower than that of good readers and this explains their problems with phonology. The cerebellum is the head ganglion of magnocellular systems; it contributes to binocular fixation and to inner speech for sounding out words, and it is clearly defective in dyslexics. Thus, there is evidence that most reading problems have a fundamental sensorimotor cause. But why do magnocellular systems fail to develop properly? There is a clear genetic basis for impaired development of magnocells throughout the brain. The best understood linkage is to the region of the Major Histocompatibility Complex (MHC) Class 1 on the short arm of chromosome 6 which helps to control the production of antibodies. 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. But the genes that underlie magnocellular weakness would not be so common unless there were compensating advantages to dyslexia. In developmental dyslexics there may be heightened development of parvocellular systems that underlie their holistic, artistic, 'seeing the whole picture' and entrepreneurial talents.

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