Types of hypothesis about dyslexia psychology essay

Dyslexia is the most common disability in children characterised by difficulties in acquiring language skills like reading, writing and spelling(S. Shaywitz and B. Shaywitz, 1999). Other symptoms of dyslexia include difficulties in skill automatisation[Nicholson R. I, 1990]. According to the U. S national institute of health 'Dyslexia is a brain-based type of learning disability that specifically impairs a person's ability to read. These individuals typically read at levels significantly lower than expected despite having normal intelligence'[US national institute of health(May 6 2010)]. Studies show that dyslexia is hereditary[B. F. Pennington, 1996]. Genes responsible for dyslexia has been shown to be located on chromosomes 6, 15 [E. L. Grigorenko, 1997]and 2[T. Fagerheim, 1999]. The theories that explain the cause of dyslexia are given below.

THE PHONOLOGICAL DEFICIT HYPOTHESIS(PDH)

According to this hypothesis dyslexia is caused due to the impairment of the phonological module which disrupts the ability to sequence the written word into its phonetic constituents[D. Shankweiler et al, 1995]. Reading is a complex process that involves two steps: decoding and comprehension[P. B. Gough, 1986]. The process of comprehension requires higher order cognitive abilities like intelligence, vocabulary[D. L. Share, 1995] which are left unaffected in dyslexia[Shaywitz SE, 2001]. Decoding involves the conversion of graphemes to phonemes. As a result the written word is not decoded properly which makes the individual difficult to comprehend it. Support for this hypothesis comes from an fmri study conducted by Georgiewa[P. Georgiewa, 2002] in which both the dyslexic and control subjects were asked to read silently the given linguistic stimuli. There was a significant activation

of Inferior frontal gyrus(IFG) in the control subjects[P. Georgiewa, 2002]. In the dyslexic subjects there was activation in 3 areas in addition to significant activation in the Broca's area. The hyper activation of Broca's area in dyslexics when compared to the controls could possibly be due to increased effort in phonological decoding. Recent anatomical evidence shows that there is a significant underactivation in Wernicke's area, angular gyrus and striate cortex and overactivation in the Inferior frontal Gyrus in dyslexics when performing a phonological task[Shaywitz SE, 1998]. Critics of this hypothesis argue that it doesn't account for symptoms not related to phonological decoding. Another criticism of this theory is that these phonological skills can be recovered on extensive training.

THE MAGNOCELLULAR DEFICIT HYPOTHESIS(MDH)

This hypothesis states that the symptoms in dyslexia are causes due to the impairment of the magnocellular pathway of the Lateral Geniculate nucleus(LGN)[Livingstone M, 1991]. In their post mortem study of 5 dyslexic brains, Galaburda and their colleagues found that the M cells of the LGN were 20% smaller than that in normal people. The LGN is a six layered structure with the layers 1 and 2 known as magnocellular layer and the layers 3, 4, 5, 6 known as the paarvocellular layer[Carlson, 2007]. The Magnocellular layer is known as the faster processing pathway as they respond faster and responsible for contrast sensitivity at low spatial frequencies and low luminance levels[XuX, IchidaJM 2001, WIESELT,. N. & D. H. HUBEL1. 1966]. As this functional segregation of the LGN is maintained upto the cortical association areas,(posterior parietal cortex)this M-layer defect could cause impairments in reading[M. Kinsbournen 1962, Morris, R. K

1991, Olson 1991]. The theory is supported by the fact that the flicker fusion rate in dyslexics is low at conditions operated by the magnocellular layer[W. J. Lovegrove, 1980]. Since the major target of M cells is the Cerebellum there are possibilities where this defect in specific line of M-layer could account for the motor symptom defects in dyslexics[John Stein and Vincent Walsh, 1997]. The Magnocellular hypothesis has some criticisms. There is evidence against the theory that dyslexics perform worse not only in low spatial frequencies and luminance but also on high frequencies and luminance levels[Skottun, B. C, 2000].

THE DOUBLE DEFICIT HYPOTHESIS(DDH)

According to this hypothesis the reading impairment caused in dyslexia is due to two deficits which are phonological and rapid naming and both are different[Wolf, M, 1999]. Rapid naming deficit is a deficit in which the subjects have problem in naming different classes of stimuli when presented visually. A typical test which assesses rapid naming is Rapid Automatized naming(RAN)[Denckla MB, 1974]. According to Wolf[Wolf, M, 1999], Dyslexics perform poorly on RAN. Support for this theory also comes from other researchers like Tallal, Temple[Temple 2000] who argue that dyslexic children process sounds very slowly than normal people. But Critics say that the task of rapid naming is also phonological since naming is done by spelling and producing sounds and so it involves the Brain's phonological system.

THE AUTOMATISATION DEFICIT HYPOTHESIS(ADH)

Automatization is a process by which the process of acquisition of skill becomes finer and refined so that the skill can be performed easily. This hypothesis states that the dyslexics perform badly in any task that requires skill automatization[Nicolson, 1990]. For example dyslexic children perform poorly in balancing task and the performance is poor only when they are not allowed to compensate for it consciously[Nicolson R. I and Angela J. Fawcett(1995)]. A normal child would automatize the process of hearing phonemes in a word after some period, which is difficult for dyslexic children.

THE CEREBELLAR DEFICIT HYPOTHESIS(CDH)

The poor performance of dyslexics on tasks that require motor skill automatization(balance task) raises a possibility that Cerebellum might the involved in Dyslexia since cerebellum is primarily involved in motor learning[J. F. Stein and M. Glickstein, 1992] and error based learning. This theory states that Dyslexia is characterised by cerebellar impairment[Nicolson, 2001]. Almost 80% of the cases of Dyslexia is characterised by Cerebellar impairment. If the cerebellum is impaired in Dyslexia then the dyslexic patients should show classic cerebellar syndromes like dystonia and ataxia[R. S. Dow and G. Moruzzi, 1958]. It was found that the performance of the dyslexics were worse than the control subjects on all the cerebellar tests[A. J. Fawcett et al., 1996]. Direct evidence for the involvement of cerebellum in dyslexia is based on the experimental paradigm of a PET study conducted by Jenkins[I. H. Jenkins et al, 1994]. In their study the subjects were made to learn a sequence of key presses by trial and error and using auditory feedback. It was found that the Cerebellum was active when the subjects learned new sequence and when the subjects

were performing a prelearned sequence. The dyslexics showed less cerebellar activation (ipsilateral) both during learning of new sequence and performance of a prelearned sequence[Nicolson, 2001]. One important question to answer is how cerebellar impairment could cause specific cognitive deficits in Dyslexia. The answer would be Cerebellar impairment causes problem in the child's articulatory speed which leads to reduced 'working memory' which in turn causes language acquisition problems[S. A. Gathercole et al, 1992]. A recent study on the morphology of Cerebellum correlates cerebellar symmetry with the degree of phonological deficit in dyslexics[Rae C, 2002] indicating that there is a connection between cerebellum and phonological deficit. A lot of neuroimaging studies say that the Cerebellum is involved in language processing tasks[J. E. Desmond and J. A. Fiez 1998, S. G. Kim, K. Ugurbil and P. L. Strick 1994]. Also abnormalities in the fronto cerebellar network is related to double deficit in dyslexics[Mark A. Eckert and Christiana M. Leonard 2003]. Given all these evidences it becomes clear that cerebellum is involved in Dyslexia. But the Cerebellar deficit hypothesis also has some unanswered questions. The Cerebellum is a large structure receiving inputs from a lot of regions of the brain and so the primary impairment might be located somewhere else in the brain causing a disruption of Cerebellar processing[T. Zeffiro and G. Eden, 2001]. Recent research also suggests that the actual impairment might be located in the perisylvian neocortical regions[Eden, G. F. and Zeffiro, T. A. 1998, Klingberg, T. et al. 2000], which sends and receives projections from the Cerebellum.

CONCLUSION

Dyslexia is characterised by a wide range of symptoms and so it highly possible that the underlying defects are in multiple brain systems. Given the role of Cerebellum in Language and motor tasks, it is quite clear that cerebellum is involved in dyslexia. That said one cannot deny the PDH and MDH. Many problems in dyslexia are phonological which is in accord with the PDH. The MDH accounts for rapid processing deficits. Also Anatomical evidences described above shows that there is sufficient underactivation in the angular gyrus, Wernicke's area and the magnocellular layer and the striate and extra striate cortices in dyslexics which account for the phonological and rapid processing deficits. One way of unifying these three theories is by using the fact that the Cerebellum is connected with many parts of the brain [J. D. Schmahmann and D. N. Pandya 1997, H. C. Leiner, A. L. Leiner and R. Dow 1993]. Thus I conclude by stating that Dyslexia is an impairment of multiple Brain systems.