Developmental Contributions to Reading Disabilities

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The term "reading disability" [RD] is characterized by the failure to develop reading skills at an age-appropriate level, in spite of normal intelligence, adequate instruction in reading, appropriate motivation, and a lack of any overt neurological handicap (Pugh, Mencl, Jenner, Katz, Frost, Lee, Shaywitz, & Shaywitz, 2000; Francks, MacPhie, & Monaco, 2002). In educational terms, reading is most frequently viewed as a cognitive process, and is often treated as such when difficulties arise in students. Cognitively speaking, reading is a multicomponent process (Francks et al., 2002) in which a subject receives visual input and processes and decodes it for orthography, phonology, meaning and context. As valid an explanation as this is (however cursory), the current methods of assessing RDs are insufficient to diagnose root causes (Francks et al., 2002). In other words, a student's performance on a test of single-word-reading ability may confirm that he does have an RD, but it does not explain why or how the disability came to be. An alternate approach to studying the process of reading and its associated disabilities is to view it from the biological standpoint.

The neurobiologist examines what physiological functions or structures fuel these cognitive processes. Reading-related cognition has been linked to high activation of cortical regions in the left hemisphere, some of which have been identified as integral to language processing (Francks et al., 2002). Sensorimotor coordination comes into play when the eyes progress along a line of text and chunk the visual stimuli into words. The stimuli travels through the retina, to the primary visual cortex, and then on to the aforementioned regions of the brain (Francks et al., 2002). If researchers have been able to identify physical regions of the brain that aid in reading and language development, could that research not be furthered in search of remediation or prevention?

Studying reading disability from a neurobiological perspective is important because it provides a tangible realm of study for diagnosis, if not remediation, of these disabilities. Researchers attempt to identify root causes of these disabilities and can then develop measures to prevent them from occurring in other children, rather than merely confirming the existence of a disability within the paradigm of "hypothetical cognitive constructs" (Francks et al., 2002, p. 485). While RDs can be caused by a number of external factors ranging from comorbid visual processing problems (or simply poor eyesight) to trauma to the brain, there is a sizeable body of evidence pointing to genetic, neurobiological, and prenatal factors involved in the development of reading disabilities.

Hereditary and Genetic Factors

A genetic basis for learning and reading disabilities has been positively established. In twin studies, monozygotic twins are more likely to display common heritable traits than dizygotic twins (Francks et al., 2002), since monozygotic twins share twice as many genes as dizygotic twins. Twin studies focusing on the heritability of reading disorders have demonstrated that if one monozygotic twin has an RD, the likelihood of the other twin having a similar disability is 68 % (Fiedorowicz, 1999); dizygotic twins retain a 40 % rate of common RDs (Fiedorowicz, 1999). These RDs are believed to be transmitted on the genetic level, most significantly on chromosome 6 (Fiedorowicz, 1999) and 18 (Francks et al., 2002). By itself, chromosome 6 has only shown weak (low significance) linkage results to sample groups, but it retains some importance because of the role it played with chromosome 18 in demonstrating the two strongest linkage signals in a study of 195 sibling pairs affected by dyslexia (Francks et al., 2002). Francks et al. (2002) also cite chromosomes 1, 2, 3, 13, and 15 as having weak, but to date underexplored, influence on reading ability.

Fiedorowicz (1999) also references broader studies on the familial transmission of reading disabilities, which have provided evidence that RDs tend to run in families. Francks et al. (2002) supply a caveat to Fiedorowicz's claim, stating that the passing on of dyslexia, probably the most common RD, does not segregate and follow a typical Mendelian pattern of dominant/recessive transmission to offspring. Francks et al. (2002) posit that as genetic relatedness to a dyslexic increases, the ability to perform reading-related cognitive tasks decreases. In other words, the closer one is related to someone with dyslexia, the more likely one is to also have an RD. According to the researchers, this suggests that there may be several underlying genetic factors at work in influencing the development of an RD in a developing human.

Brain Development

Provided that in a given zygote, no genetic predisposition to RD was passed on, RD can still manifest itself in the physical development of the brain. The brains of people with RDs and people without RDs have long been compared to one another in an ongoing effort to determine what, if any, causal or correlative links exist between brain physiology and RD. While this is by no means a new technique (Fiedorowicz, 1999), it has evolved from its original state.

One method of comparing brain structures is through postmortem examinations. Although this is a fairly superficial method of comparison, it provided the first tangible clue that there may be a connection between brain structure and RD, and certainly inspired researchers to delve deeper, in methods to be discussed later. Fiedorowicz (1999) notes that according to postmortem findings, brains of people without RD were asymmetrical, a condition which is considered to be quite normal. In this asymmetry, the temporal lobe in the left hemisphere was found to be larger than the temporal lobe in the right hemisphere. Brains of people with RD, typically dyslexics, have temporal lobes that are of equal size or possibly reverse asymmetry, in which the left temporal lobe is smaller, not larger, than the right (Wiertelak, Whitcomb, Winer, Tobin, Nguyen, Cooper, & Beise, 2003). Wiertelak et al. (2003) also call attention to the corpus callosum in people with RD. The corpus callosum is much shallower and narrower in brains of people with RD, and there is little separation between the hemispheres, which allows for greater interhemispherical activity (Wiertelak et al., 2003). These, and other structural abnormalities in brains of people with RD, all relate to perceptual deficits, specifically in phonology with regard to impaired coding (Wiertelak et al., 2003).

Medical and technological advances have allowed researchers to move beyond examining the exterior structure of the brain and turn to the interior; specifically, neuronal activity. Disruptions in the range of neural systems used in reading have been found to have a number of specific effects, ranging from simple sensory impairments to deficits in complex cognitive functioning (Francks et al., 2002). Fiedorowicz (1999) describes the many ways researchers have examined these systems, using electroencephalograms [EEGs] and event related potentials [ERPs] to gauge electrical activity in the brain and several different functional neuroimaging techniques to measure brain activity by observing blood flow to different parts of the brain while a subject is engaged in an activity, like reading. Fiedorowicz (1999) indicates that single photon emission computed tomography [SPECT] scans, for example, demonstrate that subjects with RD show underfunctioning in the occipital lobe while reading.

Through functional neuroimaging, Pugh, Mencl, Jenner, Katz, Frost, Lee, Shaywitz, & Shaywitz (2000) were able to draw some specific parallels between neuronal activity (or lack thereof) and RD. In one trial, Pugh et al. (2000) used a functional magnetic resonance imaging [fMRI] scan to observe brain activity in both RD and non-RD subjects as they performed various tasks that placed demands on the aforementioned cognitive constructs of visual-spatial processing, orthographic processing, phonological assembly, lexical-semantic processing, and simple phonologic analysis. Their findings mirror the suggestion of Fiedorowicz (1999): non-RD readers showed a "systematic increase in activation as orthographic-to-phonologic processing demands increased, while RD readers failed to show such systematic modulation in their activation patterns in response to the same task demands" (Pugh et al., 2000, p. 210). As phonological demand increased on RD readers, more activity was noted in the inferior frontal gyrus and other frontal lobe areas. Pugh et al. (2000) propose that this re-assigning of neural duty, so to speak, occurs on several levels in RD readers, and becomes a sort of vicious cycle, in which an already weak occipito-temporal system is made weaker by the continued disruption in temporo-parietal functioning. In more observable terms, although the RD brain attempts to compensate for existing weaknesses by redirecting efforts to the inferior frontal gyrus, this shift does not support fluent processing, and the subject's RD becomes more evident. This is but one example of the depths to which functional neuroimaging can and has demonstrated neurobiological and physiological abnormalities that have at least correlative, if not causal, links to reading disabilities.

Teratogens

If abnormalities in the physiological development of the brain can lead to RD, then what causes those abnormalities to occur? For decades, common wisdom has held that any potentially harmful substance ingested by an expectant mother will have a detrimental effect on the embryo or fetus. These substances are classified as teratogens, agents that harm the development of a fetus. Unfortunately for the mother and her future child, teratogens abound in everyday life: environmental pollution and second-hand cigarette smoke are but two examples. Other environmental factors such as physical trauma to the mother and the frequent combination of low socio-economic standing [SES] and low level of education can also impact the child's development insofar as the mother's willingness or ability to seek prenatal care. While environmental factors are not always within the mother's control, what she puts into her body most certainly is.

Effects of Alcohol on Developing Reading Areas

Thankfully, expectant mothers have been actively discouraged from drinking alcohol for several decades now. In addition to the myriad of physical and developmental problems that can occur as a result of fetal alcohol syndrome [FAS], Dr. Claire Coles (1991) observed significant reductions in many areas of intellectual functioning, including reading, among children whose mothers drank until the third trimester of pregnancy. In her study, children were classified by three groups: “never drank,” “stopped drinking” (drank throughout first and second trimester, but stopped just before third), and “continued to drink” (children were exposed to alcohol throughout entire pregnancy).

It seems logical to guess that students in the “never drank” group would score much higher on intellectual tasks than the other two groups. While that was the case for mathematical tasks (as assessed by the Kaufman Assessment Battery for Children) and tests of sequential processing skills and short term memory processing, reading and decoding scores were only significantly lower for the “continued to drink” group (average score of 91.6, as compared to 101.8 in the “stopped drinking” group) (Coles, 1991). Dr. Coles (1991) concluded from these findings that it is the exposure to alcohol in the third trimester that has the greatest effect on the hippocampus, thus leading to deficits in the ability to encode visual information, a key element of reading. These results also suggest that it is in the third trimester when the reading areas of the brain experience their most significant periods of neurological growth, and to introduce a teratogen such as alcohol at that point is tantamount to asking for an RD.

Effects of Prenatal Cigarette and Marijuana Exposure on Developing Reading Areas

The 1994 National Institute on Drug Abuse [NIDA] Monitoring the Future survey result data suggested a disturbing trend: after a downward trend in the extent of regular usage of cigarettes and marijuana in the late 1980s and early 1990s among women of reproductive age (including adolescents), that usage was increasing (Fried, Watkinson, & Siegel, 1997). Fried et al. (1997) conducted a ten-year longitudinal study among the children of maternal cigarette and marijuana users to determine the extent of damage to reading and language capabilities. The children’s reading ability was assessed by performance on several standardized tests and subtests such as the WISC-III, WRAT Reading subtest, Woodcock Passage Comprehension tasks, and many others (Fried et al. 1997). After adjusting for several variables such as SES, parental education, and mother’s personality, a significant negative dose-response association was noted between in utero cigarette exposure and reading, with specific regard to passage comprehension (Fried et al., 1997). This skill was impacted most significantly, but phonological processing, syntactic awareness, and the ability to read pseudowords were also moderately affected by cigarette exposure. This is significant because it suggests that cigarette exposure also affects higher level cognitive skills (the meaning and context processors) rather than just the mechanical act of interpreting single words (Fried et al., 1997). To a teacher, the most immediate observable effects of maternal smoking are the difficulties children experience with phonological processing and syntactical awareness. Therefore, Fried et al. (1997) suggest that many children who are labeled dyslexic (and only thought to have these phonological problems), also have cognitive disorders as a result of prenatal exposure to cigarette smoke. Unfortunately, these tend to go unexamined and untreated.

Ironically enough, the effects of the illegal drug in Fried et al.'s (1997) study (marijuana) had far less of an effect on reading ability than the legal one. Researchers failed to find statistically significant deficits among children who had been exposed to marijuana in utero when compared to the control group (Fried et al. 1997). While no mother should be encouraged to smoke marijuana while pregnant for numerous other health reasons, in terms of reading ability, children of moderate marijuana users were no more impaired in reading or language functions than children of heavy users or non-users (Fried et al., 1997). Fried et al. (1997) note that, surprising as it was to the researchers, this finding among the 9- to 12-year old age group was consistent with the earlier study done with the children several years earlier, when they were in the five- to six-year age range.

As stated in the present study, teratogens surround expectant mothers on a daily basis. While not all of them can be avoided as consistently as the mothers may like, the demonstrated effects of alcohol and cigarettes on reading and cognitive ability, to say nothing of the other, more immediately recognizable and profoundly harmful effects on general health and well-being, should be enough to dissuade any mother from drinking or smoking during her pregnancy.

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