

Is Dyslexia Scientifically Confirmed?

Or is it caused by the ineffective teaching of reading?

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For centuries, teachers noticed that some students experience unusual difficulty in learning to read. A term for this disability, congenital word blindness, appeared over 100 years ago. It was deduced then that the cause of students' problems in acquiring reading skills was inherited neurological disease, malfunction, or damage.

Today, extraordinary difficulty in learning to read is called dyslexia. However, a potential danger in classifying students as dyslexic is readily apparent. If children's failure to learn to read capably is due to physical disabilities, rather than malpractice in reading instruction, teachers obviously escape having to assume responsibility for pupils' backwardness in this ability.

That ineffective teaching is the cause of dyslexia is suggested by findings that certain students' reading test scores are significantly lower than their intelligence (IQ) scores. This "reading-IQ discrepancy" formula for detecting dyslexia no longer is applied, however. For one thing, routine measurements of students' IQs are prohibited in many public schools because IQ tests supposedly are culturally biased against non-Asian minority students.

After a comprehensive review of relevant experimental studies, educational psychologist Keith Stanovich concludes that the neurological and cognitive functioning of retarded readers, who have high IQs, is not significantly different from that of below-average readers with low IQs. Stanovich notes that "whatever distinct causes actually exist" for dyslexia, they are not closely associated with IQ.

Further confusing the what-causes-dyslexia question are the students identified as victims of the disorder. They usually are from middle and upper socioeconomic class families and attend modern, attractive schools staffed with mature reading teachers. On the other hand, students from families with low incomes are not as often diagnosed as dyslexic. More of them are subject to emotional and physiological impairment, socioeconomic disadvantage, racial and cultural biases, poorly funded schools, and relatively inexperienced teachers. Any item on this list could cause dyslexia.

In this regard, I recently examined what 47 textbooks, designed for university courses on basic or remedial reading instruction, had to say on the matter. More than half of the texts subtly reject the term dyslexia by making no reference at all to it. About a quarter of the volumes openly disapprove of its application to children, complaining it has neither diagnostic nor prescriptive powers. Presence of the personal handicaps that low-income students suffer makes legitimate diagnosis of them as dyslexics impossible, it is held.

Tests designed to detect dyslexia in students do not distinguish them from reading underachievers in general, it also is pointed out. It further is noted that reading handicaps displayed by youngsters called dyslexic are those that almost all students learning to read undergo, at one time or another.

In that respect, so-called dyslexic children have little conscious awareness of letters and speech sounds, and few written word recognition skills. They thus exhibit erratic oral reading, slow silent reading, and many spelling errors. They fail to make correct inferences when reading, rely on sentence context cues to recognize written words, and are unaware of the organization of written material and its author's intent in writing it.

This list describes specific reading weaknesses that readily can be prevented by experimentally confirmed direct, intensive, systematic, early, and comprehensive (DISEC) instruction of a prearranged hierarchy of discrete reading skills (especially phonics). In this regard, it is found that as many as two-thirds of children identified as dyslexic can shed this label through the application of DISEC reading tuition; one study found only 28 percent of first-graders designated as dyslexic still had that designation in grade three.

On the other hand, I found about a third of 36 recent university textbooks designed for learning disabilities courses stubbornly maintain there is a significant causal relationship between brain damage or dysfunction and dyslexia. These volumes thus contend that dyslexia has a biological, i.e., organic origin. Neurological impediments can be distinguished from other causes of students' incompetent reading, it is held, so students with dyslexia can be differentiated from other disabled readers.

This view dismisses experimental findings that the incidence of specific neurological abnormalities among learning disabled children, and among youngsters at large, is about the same. Nevertheless, support for the contention there is a biological basis of dyslexia is encouraged by studies such as one in the prestigious journal, *Science*, for October 1994.

The authors of the 1994 report contend they found a marker (a "quantitative trait locus") on chromosome 6 of 8th-grade dyslexic students that was not there for students with average reading ability or above. This defective chromosome was cited as the cause of students' backwardness in reading.

However, the validity of this finding hinges on whether the study identified truly dyslexic subjects. The study held that dyslexics are (1) the "5 to 10% of school children" whose reading performance is at least two years below that expected for those of their age group, and (2) children who have received "adequate educational opportunities" to learn to read.

To the discredit of the study, it does not describe what is "adequate" reading instruction. That is a critical omission since ineffective reading teaching easily can account for two years retardation in 8th-grade students' standardized reading test performance. For example, the federally funded National Assessment of Educational Progress in 1998 reported that 26 percent of America's 8th-graders read below the basic level of competency. They are as retarded in reading as were the dyslexic subjects in the 1994 Science study.

The study thus accepted mere attendance in reading classes in schools as legitimate evidence that students receive “adequate” reading instruction. The investigators in the study doubtless were unaware that adequate instruction often was absent in many public schools prior to 1994. The experimentally discredited Whole Language approach to development of students’ reading ability was highly popular in the 1980s and 1990s.

Another report of the supposed biological origin of dyslexia appeared in *Science* for March 2001. There have been several favorable references to the accuracy of its findings in the media. For example, the March 26, 2001 issue of *Time* magazine claims that the study helps “establish a universal neurological basis for dyslexia.” The study acknowledges that “differential response to the written word is the most widely agreed defining behavioral feature of dyslexia.” There is no argument with that broad definition.

However, the study goes on to claim that “dyslexia is increasingly acknowledged to be a disorder of genetic origin with a basis in the brain.” There is “considerable agreement that a causal link [exists] between brain abnormality and reading difficulties.” These sweeping statements about neurological irregularities as the cause of dyslexia cry out for critical analysis.

The “dyslexic” subjects in the 2001 *Science* study were from Italy, France, and the U.K. They “all had achieved tertiary levels of education,” i.e., they were “university-level adults.” They thus “could perform the simple word-reading task” demanded for inclusion in the study “to a satisfactory level.”

It consequently was held that this reading performance “ruled out certain causes of reading impairment,” including “poor general ability,” or “poor education” in reading that dyslexic subjects of the study had been provided. However, the study’s assumption that dyslexic subjects from the U.K. had received effective reading instruction is highly debatable. During the 1980s and 1990s, the experimentally discredited Whole Language approach to reading instruction was highly fashionable in the U.K.

Despite their advanced educational status, the French and British dyslexic subjects in the study previously “had been diagnosed as dyslexic and had documented histories of reading and spelling difficulties.” That is not an impressive statement, however, considering the fact diagnoses of dyslexia often are misleading in that reading difficulties they observe easily could have been caused by ineffective instruction.

The subjects in Italy selected for inclusion in the 2001 dyslexia study were ones who scored “in the bottom 10%” on three of six tests: spelling accuracy; ability to mark the stressed syllable in words; reading speed; digit naming; “short-term memory”; and “spoonerisms” (ability to transpose speech sounds in words, e.g. blushing crow for crushing blow). The assumption here, that low scores on any of these tests are equally valid signs of dyslexia, is not convincing, rationally nor experimentally.

The 2001 study gathered data from PET brain scans of 36 “dyslexic” subjects and from a like number of “normal control” subjects. It was found that normal subjects have greater “regional cerebral blood flow activation” in the language area of their brains during oral and silent reading than is the case for dyslexic subjects.

On the basis of this finding, the study proclaims that “our results are clear-cut.” That is to say, “they show that dyslexia has a universal basis in the brain and can be characterized by the same neurological deficit” that “causes literacy problems.”

The deficit was deduced to have less impact on subjects from Italy than from France or the U.S., because Italian is spelled more regularly. Dyslexia “is less severe in a shallow orthography,” such as Italian, a spelling system that spells words in more predictable ways, the study concludes.

That is a deduction about which there is no dispute. Nonetheless, the 2001 *Science* study displays further shortcomings in its design that raise suspicions about the authenticity of its conclusions. For example:

Its subjects were the wrong age. If PET scans are used to accurately determine whether dyslexia is caused by organic abnormalities, they must be administered to determine whether dyslexia is caused by organic abnormalities, they must be administered before formal reading instruction is begun.

The PET scans in the study did not indicate that there was disease of a physical or chemical nature in the brains of its dyslexic subjects, but absent in those of its normal ones. The scans thus do not confirm that reading disability is caused by physical abnormalities caused by disease.

People who have had effective reading instruction concentrate more blood flow into the language area of their brains when reading than do those who receive ineffective instruction says eminent neurologist Fred Baughman. Ineffectively taught persons, in a desperate struggle to recognize written words, dissipate their neural energy, i.e., their blood flow, to other areas of the brain when they read. Thus inept teaching is likely the cause of the diffused blood flow observed in dyslexic subjects of the 2001 study, rather than genetic brain disorders.

In conclusion, scientific studies to date do not allow reading teachers to avoid responsibility for the 2000 NAEP report that 37 percent of our 4th-graders lack even basic reading skills, and that two out of three of them are not proficient readers. Nor can teachers be so excused for the 2001 findings from the Organization for Economic Cooperation and Development that U.S. young adults are the least capable readers in the industrialized world. Instead, a controversy remains as to whether dyslexia is caused by students’ brain abnormalities, or by instructional incompetence. Lacking so far is proof that reading teachers are innocent in this regard.

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