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THE PREVALENCE OF SPECIFIC READING DISABILITY

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Knowing the prevalence of specific reading would facilitate planning, improve the allocation of resources, and enhance targeted treatment approaches. However, the prevalence of the disorder has not been determined because of a shifting conceptual base, lack of a universally acceptable operational definition, and the lack of biological markers. Systems for case identification are biased and confounded by age, socioeconomic factors, intelligence, co-morbid conditions, and treatment. Analyses of the failure to determine the prevalence of Specific Reading Disability raise questions about the fundamental assumptions that subsume this diagnosis.

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BACKGROUND

Reading is fundamental to our society. Reading and writing skills are keys to a lifetime learning process in our society, in which job requirements change continually [Miller, 1988]. As technological requirements increase, demands for literacy increase.

Even though the social costs of the inability to read have long been recognized, the number of adults who are not functionally literate is estimated to be between 54 and 64 million [Hunter and Harman, 1979]. Illiteracy is not randomly distributed. About one-fifth of all young adults and about one-half to one-third of minority young adults in the United States read below the eighth grade level [Kirsch and Jungeblut, 1986].

Students' deficient acquisition of reading skills is a major challenge to schools. There are 2.3 million children with learning disabilities [Roush, 1995]. Of the approximately 120,000 students per year who are diagnosed as having a "learning disability," 80% are "dyslexic" [Roush, 1995]. The impact of specific reading disability is not limited to these numbers. Adverse outcomes associated with specific reading disability include juvenile delinquency, dropping out of school, emotional disorders, and socioeconomic disadvantage as adults [Schonhaut and Satz, 1983].

PREVALENCE

Early data on the prevalence of extreme or pathological cases of reading disability stand in contrast to the "epidemic" currently being described in the press. Early descriptions of congenital word blindness in no way suggested a large problem needing special accommodations by schools [Doris, 1993]. Thomas [1905] estimated that one in 2,000 children were congenitally word blind. He later increased his estimate to one in 1,000 [Thomas 1908], but Hinshelwood [1917] challenged this figure as being too high. On the other hand, subsequent authors reported higher rates of dyslexia. Wallin [1921] found dyslexia in 0.7% of a series of school children. Schmitt [1917/1918] found 13 cases within a Chicago population of 42,900 school children.

In the 1920s, the focus shifted from word blindness to a range of reading disabilities [Doris, 1993]. Consequently, the prevalence rates increased. Hallgren [1950] estimated that specific dyslexia affected 10% of the Swedish population. Critchley [1970] also considered this estimate to be reasonable and cited eight additional studies from 1948–1962 that reported dyslexia in 3–25% of school children.

With passage of the Education for All Handicapped Children Act 1975 (Public Law 94–142), the number of children receiving special education services increased from 1.8% in 1976–1977 to nearly 5% in 1988–1989. A disproportional increase in the number of children receiving services for learning disability, from 22% to almost 50% of those receiving special education services, occurred during the same time span [Zigmond, 1993]. By contrast, recent studies have not found a similar increase in dyslexia. The Connecticut Longitudinal Study [Shaywitz et al., 1990] found that 8.2% of second-grade students and 7% of third-grade students were identified by their schools as having specific reading disability. Miles and Haslum [1986], in a study of 12,905 10-year-olds from the British Births Cohort Study, found a 2.3% prevalence of reading underachievement. Yule [1976] found prevalences of reading retardation that were similar to those of the previously cited studies depending on where the cohort was derived: The cohort from the Isle of Wight had a prevalence of 3.9%, while that from an inner-London borough had a prevalence of 9.9%.

CASE DEFINITIONS

Diagnosis

The fundamental properties of dyslexia that define a group of effected children support the still-unproved assertion that

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children who are bright and experience an isolated reading difficulty are fundamentally different from the larger population of children who experience reading difficulty. The features of dyslexia have changed little since Hinshelwood [1917] enumerated them: onset during childhood of severe difficulty in reading, despite normal cognition and adequate educational experience. Differing interpretations and applications of these characteristics have yielded varying estimates of the prevalence of dyslexia.

The early descriptions focused on adolescents and young adults who were capable of learning but failed to read. Intellectual capacity was not questioned in these reports. In the 1920s, Orton drew on his experiences in Greene County and noted that although only two of his cases fit strict criteria for "word blindness," all 15 children with special reading problems showed similarities in reading behavior [Doris, 1993]. Expansion of the definition of reading disabilities to include children who would learn to read complicated the identification process. Later authors sought to improve diagnostic ability by refining operational definitions of "imperfect ability," normal cognition, and adequate educational experience. The time and effort clinicians spent to establish a diagnosis of specific reading disability is staggering [Shepard and Smith, 1983].

The most common form of specific reading disability presents early in childhood as poor word recognition (decoding). Deficits in phonological tasks are associated with this form of specific reading disability. However, phonological deficits also occur with other forms of specific reading disability (such as dyslexia), which are distinguished from the more common form by the severity and persistence of children's difficulty with reading. These latter types of specific reading disability are rare, as noted in the early studies, and their diagnosis is not considered until children reach early adolescence, when time demarcates them from the more common form.

Children can also have specific reading disability in the absence of poor decoding. These children usually present at older ages than the children with decoding difficulties and manifest deficient comprehension relative to IQ. This form of specific reading disability is more likely to be detected by timed tests that assess the ability to read longer passages.

The Adequacy of Discrepancy

Formulas that quantify the difference between reading achievement and intellectual potential were devised to identify children with specific learning disabilities who would qualify for special education. The general approach applied to specific reading disability was the same as that applied to other specific learning disabilities. Although the discrepancy formulas incorporated the characteristics that distinguished dyslexia, they were restrictive in order to exclude children whose deficiency was likely to be transient or not severe. The formulas also sought to prevent the provision of special education to children who would be served under other categories. During the past decade, the concept of specific reading disability has been reexamined. Much of the knowledge gained since the passage of the Education for All Handicapped Children Act of 1975 (Public Law 94-142) has challenged the original precepts that underlie specific reading disability. The most frequent target for challenge, diagnosis via discrepancy formulas, has been faulted for statistical as well as conceptual reasons.

Nevertheless, considering that only 17% of children classified as dyslexic in the first grade were so classified in the sixth grade [Shaywitz et al., 1992], one cannot endorse the use of discrepancy formulas to identify children whose reading disability will persist.

Perhaps the most common reason for faulting the discrepancy formulas on statistical grounds is that they have poor classification abilities. Discrepancy formulas do not adequately identify children with specific reading disability. In an unselected cohort of 230 7.5-year-old children, application of five discrepancy formulas correctly identified between 0 and 48% who received special education [Shapiro et al., 1990]. Discrepancy formulas using standard scores under-identified not only young students, but African-American students as well [McLeskey et al., 1990]. Another study that used eight discrepancy formulas to identify learning disabled children between the ages of 7.5 and 13.1 years found sensitivities ranging from 10.9 to 37%. Almost half of the 92 children were not identified by any formula. Two or more formulas agreed with regard to only about a third of the children when the diagnosis of learning disability was made [Forness et al., 1983].

In addition to the issues related to classification, discrepancy formulas do not have sufficient predictive validity to forecast children's later reading disability. Poor predictive validity may not result from poor measurement, but may, instead, reflect instability in the behavior being measured. Nevertheless, considering that only 17% of children classified as dyslexic in the first grade were so classified in the sixth grade [Shaywitz et al., 1992], one cannot endorse the use of discrepancy formulas to identify children whose reading disability will persist.

Conceptual Challenges

The existence of dyslexia as an independent condition continues to be questioned. Early challenges during the 1920s and 1930s held that dyslexia is an extreme point on the continuum of reading function and the differences were in degree, not kind [Doris, 1993]. Later authors described dyslexia as a hypothesis requiring scientific validation, but expressed the view that specific reading retardation involved no theoretical assumptions [Rutter, 1978].

The early reports of word blindness dealt with categorical entities: children could not read and were not intellectually limited. As dyslexia evolved into a dimensional diagnosis, IQ was established as a benchmark to which reading skills were compared. Siegel [1989] reviewed four assumptions that are fundamental to the use of IQ test scores in the definition of reading disability: IQ scores measure intelligence; intelligence and achievement are independent; IQ scores predict reading; and children with reading disability who have different IQ scores also have different cognitive processes and information skills. Siegel concluded that IQ is irrelevant to the definition of reading disability. Empirical data from an epidemiological study of child development showed that IQ did not limit rates of progress or level of achievement. Lower IQ was associated with lower reading achievement, but the relationship was too weak to permit predictions for individual children [Share et al., 1989]. Siegel's conclusion was supported by a study that found that abandoning IQ in classification had little impact on the number of children identified as having a reading disorder, even though the methods used

identified different children [Rispens et al., 1991].

Current reports continue to question the existence of fundamental differences in etiology, neurology, or reading-related cognition in poor readers with and without IQ-reading discrepancy. In their epidemiological study in Connecticut, Shaywitz et al. [1992] failed to find evidence of two populations. They concluded that dyslexia is part of a continuum that includes normal reading ability and is not an all-or-none phenomenon. In a study that compared dyslexic students to poor readers, no differences were found in reading, spelling, phonological processing, or memory or language tasks [Siegel, 1992]. Stanovich [1994] has reviewed the field and concluded that:

> 1) reading-IQ discrepancy measurement fails to identify a distinct phenotypic pattern of word recognition subskills; 2) reading-IQ discrepancy measurement does not identify a group of children with significantly different heritability values for core information processing deficits; 3) there are as yet no indications the neuroanatomical anomalies that are associated with reading disability are more characteristic of high-IQ than of low-IQ poor readers (p 588).

CASE ASCERTAINMENT

Referral Bias

The process of identification must be addressed in studies of dyslexia. Case ascertainment is independent of the definition of a disorder and is dependent on the setting from which subjects are recruited. Studies of populations derived from clinical settings, schools, and epidemiologic surveys yield different information. Even within a single setting, there may be differing methods of case ascertainment.

Early studies of dyslexia were derived from clinical populations and, consequently, are limited by bias of ascertainment. The hypothesis that children who demonstrated a discrepancy between their IQ and reading achievement were different from others was based on a truncated population of poor readers. For example, Hinshelwood's [1917] estimate of the prevalence of word blindness was based on patients who were referred to his practice. Many other individuals with reading difficulty just went to work and were not referred. Contemporary reports of the prevalence of dyslexia that were based on school populations were higher [Schmitt, 1917/1918].

Reports from school-based populations are also limited by case identification, although the bias is less systematic. In the school setting, referral is required for case ascertainment. The decision to refer is a function of capacities that are intrinsic to the behaviors of children and teachers. Students with similar capabilities may succeed in one environment and fail in another. Low achievement alone may not result in referral. Referral is a transactional process: a breakdown in the interaction between student and teacher, between learner and environment [Zigmond, 1993]. Referral bias was found to decrease the prevalence of reading disability in girls [Shaywitz et al., 1990].

Bias can also increase the apparent prevalence of disorders. In a review of the identification of students with learning disability in Colorado, slightly less than half of the children that had been identified actually met criteria for learning disability. The others met criteria for other handicaps, were non-English dominant, or were better described as being slow learners or behaviorally disturbed [Shepard and Smith, 1983]. Inclusive case identification is necessary to understand the natural history of these disorders. Much of the current research on mechanisms of specific reading disability has focused on adults with persisting difficulties. How the findings of these studies relate to children remains to be demonstrated. Similarly, the study of the relationship of phonology to reading requires determination of whether or not there are good readers who demonstrate poor phonologic skills.

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Within-Subject Variation

Specific reading disability is not a constant diagnosis. In a longitudinal study of Norwegian children, the prevalence of severe reading disability dropped from 5% in the second grade to 2% at the end of ninth grade [Gjessing and Karlsen 1989]. Children have differing rates of development and, therefore, may grow into or out of the diagnosis [Korhonen, 1991; Shaywitz et al., 1992].

Treatment influences the manifestations of specific reading disability. Children who receive reading therapy progress and may come to read appropriately [Kurzweil, 1992], although therapeutic efficacy is yet to be proved. Rosenberger [1992] summarized this issue: "Dyslexic children who receive proper tutorial support acquire essential literacy skills despite a shortage of the aptitude that makes learning to read easier than their normal classmates" (p 193).

Not all children improve enough to grow out of the diagnosis of dyslexia. Some children fail to maintain their rate of cognitive growth, so that the discrepancy between IQ and reading achievement is no longer significant [Siegel, 1992]. Stanovich [1988] has posited a mechanism, the Matthew effects, to describe this phenomenon. The Matthew effects, which are dependent on cognitive academic interactions, hold that the early acquisition of reading skills results in reading and academic experiences that facilitate the development of other cognitive structures that lay the foundation for successful reading achievement at more advanced levels [Stanovich, 1988].

Confounding Variables and Co-morbidity

It was not by chance that the early studies of dyslexia chose older children. Sufficient academic exposure must be experienced to confirm the delay in the acquisition of reading skills, although the neuropsychological dysfunction associated with specific reading disability may be identified earlier. The difficulty arises in trying to distinguish specific reading disability from intellectual limitation, developmental language disorders, attention deficit hyperactivity disorder, and idiosyncratic patterns of development that result in transient delays in reading acquisition. Each of these disorders may present as delayed reading skills and obscure the diagnosis of specific reading disability. These neurodevelopmental disorders are dimensional, lack unique diagnostic criteria, and evolve as a child ages. The early diagnosis of specific reading disability also may be confounded by social, emotional, temperamental, and behavioral factors.

Specific reading disability is rarely seen in isolation. It is common for

children to manifest several neurodevelopmental disorders simultaneously. Comorbidity—for example, the coexistence of attention deficit hyperactivity disorder and specific reading disability—changes the nature and complicates the diagnosis of each disorder. Cognitive-achievement discrepancies may not be apparent. The interaction of co-morbid conditions with social, emotional, temperamental, and behavioral factors challenges the existence of a "garden variety" poor reader. How multiple disorders affect treatment is unknown.

Threshold Effects

Reading is not a single entity. Young readers do not read like older readers. Children with poor decoding do not always have poor comprehension. In young children, reading requires the attainment of a series of thresholds. One model of reading acquisition posits three phases: a logographic phase wherein words are learned as visual forms; an alphabetic phase in which children acquire and learn to use the grapheme-phoneme correspondence; and an orthographic phase wherein children can directly analyze words into orthographic units (e.g., the suffixes -ed or -able) [Frith, 1985]. This model is not well quantified using interval scales. Small changes in the reading performance of young children is accompanied by relatively large changes in standard scores on reading tests. Thresholds may delay the identification of children with specific reading disability because a child may decode, but not comprehend well. The attainment of literacy by an adult who, as a child, was diagnosed as having a specific reading disability challenges the foundations of the diagnosis. On a conceptual level, it is difficult to be considered individuals to be "disabled" if they have achieved literacy. Literacy is viewed in a categorical fashion: age is no longer of interest once literacy is attained. On a practical level, the diagnosis of specific reading disability is difficult to establish in literate adults because the level of reading attainment cannot be meaningfully quantified. Consequently, comparison of reading attainment and cognitive potential is precluded.

CODA

The concept of specific reading disability is still evolving, but its fundamentals are unchanged. Specific reading disability is still defined as occurring in children who experience significant difficulty in the acquisition of reading abilities despite having adequate intellect and educational experience. Current studies are limited by the unproved conceptual assumptions that define specific reading disability, the inadequacy of discrepancy formulas, comorbidity, threshold effects exerted by age and intelligence, the effects of treatment, and incomplete case ascertainment. It is not possible, at present, to determine the prevalence of specific reading disability. ■

REFERENCES

Critchley M (1970): The Dyslexic Child. Springfield IL: Charles C. Thomas, pp 94–96.

Doris JL (1993): Defining learning disabilities. A history of the search for consensus. In Lyon GR, Gray DB, Kavanagh JF et al. (eds): Better Understanding Learning Disabilities. Baltimore: Paul H. Brookes, pp 97–115.

Forness SR, Sinclair E, Guthrie D (1983): Learning disability formulas: Their use in actual practice. Learn Disabil Q 6:107–114.

Frith U (1985): Beneath the surface of developmental dyslexia. In Patterson KE, Marshall JC, Coltheart M (eds), Surface Dyslexia. Hillsdale NJ: Erlbaum, pp 301–331.

Gjessing HJ, Karlsen B (1989): A Longitudinal Study of Dyslexia. New York: Springer-Verlag, p 272.

Hallgren B (1950): Specific dyslexia ("congenital word blindness") a clinical and genetic study. Acta Psychiatrica at Neurologica Scandinavica S 65:1–287.

Hinshelwood J (1917): Congenital Word Blindness. Chicago: Medical Book Co.

Hunter C St. J, Harman D (1979): Adult Illiteracy in the United States. New York: McGraw-Hill.

Kirsch IS, Jungeblut A (1986): Literacy: Profiles of America's Young Adults. Princeton: Educational Testing Service.

Korhonen TT (1991): Neuropsychological stability and prognosis of subgroups of children with learning disabilities. J Learn Disabil 24:48–57.

Kurzweil SR (1992): Developmental reading disorder: Predictors of outcome in adolescents who received early diagnosis and treatment. J Dev Behav Pediatr 13:399–404.

McLeskey J, Waldron NL, Wornhoff SA (1990): Factors influencing the identification of black and white students with learning disabilities. J Learn Disabil 23:362–366.

Miles TR, Haslum NB (1986): Dyslexia: Anomaly or normal variation? Ann Dyslexia 36:103–117.

Miller GA (1988): The challenge of universal literacy. Science 241:1293–1299.

Rispens J, van Yperen, van Duijn GA (1991): The irrelevance of IQ to the definition of learning disabilities: some empirical evidence. J Learn Disabil 24: 434–437.

Rosenberger PB (1992): Dyslexia: Is it a Disease? N Engl J Med 326:192–193.

Roush W (1995): Arguing over why Johnny can't read. Science 267:1896–1898.

Rutter M (1978): Prevalence and types of dyslexia. In Benton A, Pearl D (Eds) Dyslexia. An Appraisal of Current Knowledge. New York: Oxford University Press, pp 5–28.

Schmitt C (1917/1918): Developmental alexia: Congenital word blindness, or inability to learn to read. Elementary School J 18:680–700, 757–769.

Schonhaut S, Satz P (1983): Prognosis for children with learning disabilities: A review of followup studies. In Rutter M (ed): Dev Neuropsychiatry. New York: Guilford Press, pp 542–563.

Shapiro BI, Palmer FB, Antell SE, et al. (1990): Detection of young children in need of reading help. Clin Pediatr 29:206–213.

Share DL, McGee R, Silva P (1989): IQ and reading progress: A test of the capacity notion of IQ. J Am Acad Child Adolesc Psychiatry 28:97–100.

Shaywitz SE, Escobar MD, Shaywitz BA, et al. (1992): Evidence that dyslexia may represent the lower tail of a normal distribution of reading ability. N Engl J Med 326:146–150.

Shaywitz SE, Shaywitz BA, Fletcher JM, et al. (1990): Prevalence of reading disability in boys and girls. Results of the Connecticut Longitudinal Study. JAMA 264:998–1002.

Shepard LA, Smith ML (1983): An evaluation of the identification of learning disabled students in Colorado. Learn Disabil Q 6:115–127.

Siegel LS (1989): IQ is irrelevant to the definition of learning disabilities. J Learn Disabil 22:469–478.

Siegel LS (1992): An evaluation of the discrepancy definition of dyslexia. J Learn Disabil 25:618–629.

Stanovich KE (1988): Explaining the differences between the dyslexic and the garden-variety poor reader: The phonological-core variable difference model. J Learn Disabil 21:590–612.

Stanovich KE (1994): Annotation: Does dyslexia exist? J Child Psychol Psychiatry 35:579–595.

Thomas CJ (1905): Congenital word-blindness and its treatment. Ophthalmoscope 3:380–385.

Thomas CJ (1908): The aphasias of childhood and education hygiene. Public Health 21:90–100.

Wallin JEH (1921): Congenital word blindness. Lancet 1:890–892.

Yule W (1976): Issues and problems in remedial education. Dev Med Child Neurol 18:675–682.

Zigmond N (1993): Learning disabilities from an educational perspective. In Lyon GR, Gray DB, Kavanagh JF, et al. (eds): Better Understanding Learning Disabilities. Baltimore: Paul H. Brookes, pp 251–272.