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Annotation: Does Dyslexia Exist?

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Introduction

Obviously, in order to answer the question posed in the title, we must specify what we mean by the term dyslexia. And, in doing so, we immediately encounter the crux of the problem. This problem is a recurring one in the field of developmental disabilities, and it arises because the field has repeatedly displayed a preference for terminology that connotes unverified theories about causation. For example, in this journal Bishop (1992) has recently noted how the terms developmental dysphasia and developmental aphasia have “fallen into disfavour in the U.K. and U.S.A., largely because they misleadingly imply that we are dealing with a single condition with a known neurological basis” (p. 3). Likewise, the term dyslexia is out of favor in many educational and research communities within North America—and for similar reasons. As we shall see, “dyslexia” carries with it so many empirically unverified connotations and assumptions that many researchers and practitioners prefer to avoid the term.

Indeed, it does seem that reading research could benefit from adopting more neutral terms for the phenomena that it studies. Terminology that is less likely to carry with it a speculative theory is to be preferred in the early stages of scientific investigation. The reading field seems unnaturally prone to popularizing terminology that carries with it unproven theory. For example, publications in early literacy research in North America are currently littered with the terms “emergent literacy” and “invented spelling”. But, just as with “dyslexia”, these are not neutral terms. They are not descriptions of certain operationally-defined performance patterns in early literacy. These terms convey a theory of early literacy acquisition (e.g. that it is natural and will normally progress without much formal tuition) that is without empirical support (Adams, 1990; Liberman

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& Liberman, 1990; Perfetti, 1991). The theory carried with the term "dyslexia" seems similarly to have outrun the evidence.

With these strictures in mind, we will begin our discussion at the beginning. Whether or not there is such a thing as "dyslexia", there most certainly *are* children who read markedly below their peers on appropriately comprehensive and standardized tests. In this most prosaic sense, poor readers obviously exist. Controversy begins only when we address the question of whether, within this group of poor readers, there are groups of children who are "different". Terms like congenital word-blindness and dyslexia were coined to describe groups of children who were thought to be different from other poor readers in their etiology, neurological makeup, and cognitive characteristics. From the very beginning of research on reading disability, it was assumed that poor readers who were of high intelligence formed a cognitively and neurologically different group. Investigators who pioneered the study of the condition then known as congenital word-blindness were at pains to differentiate children with this condition from other poor readers. Hinshelwood (1917) stated clearly that he intended the term congenital word-blindness not for all poor readers but instead for those who were high functioning in other cognitive domains:

When I see it stated that congenital word-blindness may be combined with any amount of other mental defects from mere dullness to low-grade mental defects, imbecility or idiocy, I can understand how confusion has arisen from the loose application of the term congenital word-blindness to all conditions in which there is defective development of the visual memory center, quite independently of any consideration as to whether it is a strictly local defect or only a symptom of a general cerebral degeneration. It is a great injustice to the children affected with the pure type of congenital word-blindness, a strictly local affection, to be placed in the same category as others suffering from generalized cerebral defects, as the former can be successfully dealt with, while the latter are practically irremediable. (pp. 93-94).

Similarly, the term dyslexia has often been reserved for children displaying discrepancies between intelligence and reading ability. In the 1970s and 1980s, proponents of the generic term learning disabilities—coined largely as a school service-delivery category (Kirk, 1963; Lerner, 1985)—continued the tradition of assuming that there were important etiological, neurological and cognitive differences between high-IQ and low-IQ poor readers, despite the fact that there existed no more evidence for this assumption in 1970 than there was in Hinshelwood's day.

One might have thought that researchers would have begun with the broadest and most theoretically neutral definition of reading disability—reading performance below some specified level on some well-known and psychometrically sound test—and then proceeded to investigate whether there were poor readers with differing cognitive profiles *within* this broader group. Unfortunately, the history of reading disabilities research does not resemble this logical sequence. Instead, early definitions of reading disability *assumed* knowledge of differential cognitive profile (and causation) within the larger

sample of poor readers and defined the condition of reading disability in a way that actually served to preclude empirical investigation of the unproven theoretical assumptions that guided the formulation of these definitions!

This remarkable sleight-of-hand was achieved by tying the definition of reading disability to the notion of aptitude/achievement discrepancy (Reynolds, 1985; Shepard, 1980; Siegel, 1989; Stanovich, 1991). That is, it was assumed that poor readers of high aptitude—as indicated by IQ test performance—were cognitively different from poor readers of low aptitude and that they had a different etiology. The term dyslexia, or reading disability, was reserved for those children showing significant discrepancies between reading ability and intelligence test performance. Such discrepancy definitions have become embedded in the legal statutes governing special education practice in many states of the United States (Frankenberger & Fronzaglio, 1991; Frankenberger & Harper, 1987) and they also determine the subject selection procedures in most research investigations (Stanovich, 1991). The critical assumption that was reified in these definitions—in almost total absence of empirical evidence—was that degree of discrepancy from IQ was meaningful: that the reading difficulties of the reading-disabled child with reading-IQ discrepancy (termed specific reading retardation in the classic investigation of Rutter & Yule, 1975) were etiologically and neurologically distinct from those characterizing the reading-disabled child without IQ discrepancy (termed general reading backwardness in the Rutter & Yule, 1975 study).

Quite early in the history of research on dyslexia, researchers adopted a strong theoretical bias by tying an intuition about differential causation so closely to the notion aptitude/achievement discrepancy (see Pennington, Gilger, Olson & DeFries, 1992; Taylor & Schatschneider, 1992). It was simply assumed that reading difficulty unaccompanied by low IQ was a distinct entity from other reading problems. It was not until the mid-1970s that we had the data from the ground-breaking epidemiological comparison of poor readers with and without reading-IQ discrepancy conducted by Rutter and Yule (1975), and only in the past decade has their data been supplemented by that from other investigations of a similar type.

From a total sample of 2300 9-year-old children, Rutter and Yule (1975; Rutter, 1978) defined two groups of poor readers who were equal in reading achievement (each approximately 33 months below the general population mean). However, the mean IQ of the specific reading retardation group (102.5) was significantly higher than that of the reading backwardness group (80). Rutter and Yule (1975) reported some significant differences between the two groups, but also several similarities. The specific reading retardation group was less likely to have organic brain damage or to display various neurological abnormalities. The backward group was more likely to display a variety of motor abnormalities and to show left/right confusion. The groups had similar proportions of family members with histories of reading difficulties and similar histories of delays in language development. Rutter and Yule were careful to

point to the fact that many aspects of the classic "dyslexic syndrome" were *not* found disproportionately in their specific reading retardation group (e.g. left/right confusion, neurological signs, directional confusion and familial linkage, see also Taylor, Satz & Friel, 1979).

Some of the differences that were uncovered in the Isle of Wight studies have been difficult to replicate. For example, the data on differential prognosis for reading are contradictory. Rutter and Yule (1975) found differential growth curves for the specifically disabled and the general retardation groups. The latter displayed greater growth in reading but less growth in arithmetic ability than the specifically disabled children. However, this finding of differential reading growth rates has failed to be replicated in some other studies (Bruck, 1988; Labuda & DeFries, 1989; McKinney, 1987; Share, McGee, McKenzie, Williams & Silva, 1987; van der Wissel & Zegers, 1985).

Are There Distinct Etiologies for Some Cases of Reading Disability?

Nevertheless, as Pennington *et al.* (1992) argue, the issue of "whether RD is just the lower tail of the multifactorially determined, normal distribution of reading skill, or whether some cases of RD represent an etiologically distinct disorder" (p. 562) is separable from the issue of whether there are differences between poor readers with and without reading-IQ discrepancy. We must ask first whether there is evidence that some children within the entire group of poor readers display evidence for a distinct etiology. At that point, we are in a position to address the question of whether poor readers *with* reading-IQ discrepancy children happen to *be* those with a distinct etiology. As Pennington *et al.* (1992) note, "If no cases of RD represent an etiologically distinct disorder or syndrome, then it is pointless to argue about how to define a syndrome that does not exist!" (pp. 562-563).

The issue of a distinct etiology for some cases of reading difficulty is, in fact, a confusing one. Much attention has focused on the issue of whether there is a statistically discernible "hump" in the lower tail of the distribution of reading ability. Some studies have found evidence for such a hump (Rutter & Yule, 1975; Stevenson, 1988), but others have not (Rodgers, 1983; Shaywitz, Escobar, Shaywitz, Fletcher & Makugh, 1992). Resolving the "hump issue", however, will not give the definitive answer to the distinct etiology question because, even if such a hump is not found "a few etiologically distinct factors, plus noise, can give rise to a normal phenotypic distribution" (Pennington *et al.*, 1992, p. 563).

Genetic epidemiology provides a sounder basis for establishing distinct causation for some cases of reading disability. Here the evidence is more definitive. In several examples, Pennington *et al.* (1991) found evidence for sex limited, autosomal additive or dominant transmission of reading disability, although there is evidence for genetic heterogeneity (see Pennington, 1990; Smith, Kimberling & Pennington, 1991; Stevenson, 1992a). Twin studies have

also consistently indicated a moderate heritability for the group deficit in reading ability displayed by the twin probands—as well as significant genetic covariance between the group deficit and phonological coding and awareness skills (DeFries, Fulker & LaBuda, 1987; Olson, Wise, Conners, Rack & Fulker, 1989; Pennington *et al.* 1992; Stevenson, 1992a, 1992b). Pennington, Van Orden, Kirson and Haith (1991) summarize the evidence: “These behavior genetic analyses are consistent with the view that the heritable component in dyslexia at the written language level is in phonological coding and the heritable precursor to this deficit in phonological coding is a deficit in phoneme awareness” (p. 183).

Further evidence that reading disability might have distinct etiology comes from neuroanatomical studies (see Hynd, Marshall & Gonzalez, 1991, and Hynd, Marshall & Semrud-Clikeman, 1991 for reviews). For example, both post mortem and *in vivo* studies have indicated that atypical symmetry in the planum temporale is associated with reading disability (Galaburda, 1991; Galaburda, Sherman, Rosen, Aboitz & Geschwind, 1985; Larsen, Høien, Lundberg & Odegård, 1990; Steinmetz & Galaburda, 1991). Larsen *et al.* (1990) found that the atypical symmetry was directly associated with the phonological coding deficit that is the primary phenotypic indicator of reading disability (see below). Additional cortical anomalies have been identified in other studies (see Hynd, Marshall & Gonzalez, 1991; Semrud-Clikeman, Hynd, Novey & Eliopoulos, 1991).

Does Discrepancy Measurement Identify Poor Readers With a Distinct Etiology?

Thus, there is some support for a distinct etiology for at least some cases of reading disability. Reading difficulty appears to be moderately heritable. At least some cases of reading difficulty appear to be genetically transmitted in an autosomal dominant pattern. Some cases also appear to be associated with distinct neuroanatomical anomalies. All of these findings provide a foundation for a principled concept of dyslexia, but they are not—in and of themselves—enough to justify current definitional practice. There is still one critical link missing in the chain of evidence. The phenotypic performance pattern that defines the concept of dyslexia must be reliably and specifically linked with these indicators of distinct etiology. As discussed in the introduction, both research and educationally-based definitions of dyslexia have incorporated the notion of reading-IQ discrepancy (Stanovich, 1991). This practice arose because of the intuition that children with reading-IQ discrepancies would be more likely to display a distinct etiology. Thus, identifying reading-IQ discrepancies was viewed as an easy way of selecting those children characterized by this distinct etiology. The basic assumption was that there were fundamental etiological, neurological and (reading-related) cognitive differences between poor readers with and without IQ-reading discrepancy. It is this assumption that is presently without empirical support.

Reading disabled children display a characteristic profile of cognitive skills (to be discussed below); reading disability displays moderate heritability; evidence of a number of different modes of genetic transmission has been found (Pennington, 1990; Stevenson, 1992a); and some reading disabled children display atypical neuroanatomical features. However, the problem is that there is not one bit of evidence indicating that these characteristics are more true of poor readers with IQ-reading discrepancy than of poor readers without such discrepancies.

For example, genetic linkage studies have usually employed a discrepancy criterion in defining cases of reading disability. However, we have no knowledge of whether similar evidence of genetic linkage would be found if reading disability were defined without reference to discrepancy in such studies. Likewise, no extant study has systematically related the neuroanatomical correlates of reading disability to degree of reading-IQ discrepancy. There is again no evidence in the literature indicating that similar relationships between neuroanatomical features such as symmetry of the planum temporale and reading disability would not be found if reading disability were defined without reference to IQ-reading discrepancy. For instance, the Larsen *et al.* (1990) study of planum temporale symmetry defined reading-IQ discrepancy in their sample using the Raven Matrices test. The use of a nonverbal test which displays very low correlations with reading and other verbal skills (Stanovich, Cunningham & Feeman, 1984) might well have resulted in a sample containing several subjects with depressed verbal IQs and/or below average full scale IQs (Stanovich, 1991; Stanovich, Nathan & Vala-Rossi, 1986). Such subjects might well have been classified as nondiscrepant, or "garden-variety" poor readers (see Gough & Tunmer, 1986, and Stanovich, 1988, 1991) had other verbally-loaded aptitude measures been used in discrepancy assessment (see Stanovich, 1991, for an extensive discussion of the implications of using different aptitude benchmarks). Thus, this particular study might well be providing indirect evidence *against* the hypothesis that these atypical symmetries are unique to poor readers with reading-IQ discrepancy and would not be found in poor readers without such discrepancies. Further negative evidence comes from the finding that neurological disorders are no more common among poor readers with reading-IQ discrepancies. If anything, the opposite appears to be the case (Ingram, Mason & Blackburn, 1970; Ruter & Yule, 1975; Silva, McGee, Williams, 1985).

The issue of differential etiology for children with reading delays that are, or are not, discrepant with IQ has been directly addressed in twin studies of genetic influence. Olson, Rack, Conners, DeFries and Fulker (1991) did find that the heritability of the group deficit of high-IQ (full scale) reading disabled twins (.67) was higher than the heritability of the group deficit for low-IQ reading disabled twins (.40), but this difference was not statistically significant. A parallel analysis based on verbal IQ rather than full-scale IQ revealed heritability values of .59 and .49, a difference that was again not statistically significant. Pennington *et al.* (1992) defined two groups of reading disabled children: one using a

reading/IQ regression equation and the other using an age-only discrepancy. The group heritability for low scores on the IQ-discrepancy criterion was .46 and the group heritability for low scores on the age-discrepancy criterion was .49. The authors concluded that "These values indicate that approximately 50% of the deficit in scores for both diagnostic continua is due to heritable factors. The similarity in values suggests that the estimated proportion of genetic variance contributing to RD is essentially the same, regardless of the manner in which RD is identified. Thus, there is no evidence here for differential external validity of the two phenotypes" (p. 567). Pennington *et al.* (1992) also found that the genetic covariance between phonological coding ability and the IQ-discrepancy diagnosis (.60) was slightly higher than the corresponding covariance for phonological coding and the age-discrepancy criterion (.47), but this difference was not significant. The investigators concluded that "The heritability analyses are primarily consistent with the hypothesis that the same genes influence each diagnostic phenotype" (Pennington *et al.*, 1992, p. 570).

Other investigations have also failed to provide strong evidence for markedly different heritability of deficits among high- and low-IQ reading-disabled children who are reading at the same level (Stevenson, 1991, 1992b; Stevenson, Graham, Fredman & McLoughlin, 1987). Stevenson (1991) did find that the heritability of a group deficit in spelling ability tended to be greater for spelling scores residualized on IQ than for raw spelling scores, but this tendency was not present in most of his measures of reading ability. Taken collectively, the findings from all of these studies "do not refute a possible biological basis for reading disability—only the hypothesis that the biological basis is different for children who meet IQ-based discrepancies" (Fletcher, 1992, p. 547).

IQ-Discrepancy and the Reading Disability Phenotype

In summary, although genetic and neuroanatomical studies may be narrowing in on a syndrome of dyslexia, that syndrome does not seem to be strongly correlated with degree of IQ-discrepancy in the reading-disabled population. It is really not so surprising that genetic and neuroanatomical correlates have not been found to be differentially associated with the presence or absence of a reading-IQ discrepancy. This is because IQ-discrepancy appears to be at best weakly correlated with the primary phenotypic indicators of reading disability. What are those indicators?

Although there may be small groups of children who have specific comprehension difficulties (Oakhill & Garnham, 1988), there is a great deal of converging evidence indicating that most cases of reading disability arise because of difficulties in the process of word recognition (e.g. Bruck, 1988, 1990; Morrison, 1991; Perfetti, 1985; Siegel, 1985; Siegel & Faux, 1989; Siegel & Ryan,

1989; Snowling, 1991; Stanovich, 1981, 1986, 1988). These difficulties are, in turn, due to deficiencies in processes of phonological coding whereby letter patterns are transformed into phonological codes. Problems with phonological coding lead to the most diagnostic symptom of reading disability: difficulty in pronouncing pseudowords (e.g. Bruck, 1988, 1990; Felton & Wood, 1992; Manis, Custodio & Szeszulski, 1993; Olson *et al.* 1989; Siegel, 1989; Siegel & Ryan, 1988; Snowling, 1981, 1991). In contrast to phonological coding, processes of orthographic coding—where words are recognized via direct visual access—appear to be relatively less impaired in disabled readers (Frith & Snowling, 1983; Holligan & Johnston, 1988; Olson, Kliegl, Davidson & Foltz, 1985; Olson *et al.* 1989; Pennington *et al.* 1986; Rack, 1985; Siegel, 1993; Stanovich & Siegel, 1994; Snowling, 1980).

The precursor to the phonological coding difficulty appears to be a deficit in segmental language skills sometimes termed phonological awareness or phonological sensitivity (e.g. Bentin, 1992; Bowey, Cain & Ryan, 1992; Bradley & Bryant, 1978, 1985; Bruck, 1990, 1992; Bruck & Treiman, 1990; Bryant, Maclean, Bradley & Crossland, 1990; Goswami & Bryant 1990; Olson *et al.*, 1989; Stanovich, 1982, 1992; Stanovich, Cunningham & Cramer, 1984; Vellutino & Scanlon, 1987; Wagner & Torgesen, 1987). Becoming aware of the segmental structure of language appears to be a prerequisite to rapid reading acquisition in an alphabetic orthography. Lack of phonological awareness inhibits the learning of the alphabetic coding patterns that underlie fluent word recognition (Bryant *et al.*, 1990; Goswami & Bryant, 1990; Stanovich *et al.*, 1984; Tunmer & Hoover, 1992; Tunmer & Nesdale, 1985).

As previously indicated, the most distinctive indicator of the phonological coding deficits that are characteristic of reading disability is difficulty in naming pseudowords (Rack, Snowling & Olson, 1992). Reading-disabled children not only perform worse than chronological age peers on pseudoword tasks, but they also underperform reading-level controls, i.e. younger nondisabled children equated on word recognition skill. This pseudoword deficit in a reading-level match is one of the most distinctive indicators of the reading-disabled phenotype (Olson *et al.*, 1989; Rack *et al.* 1992; Stanovich & Siegel, 1994). However, several studies that have compared the performance of poor readers with and without reading-IQ discrepancy have found that they display equivalent pseudoword deficits (Felton & Wood, 1992; Fredman & Stevenson, 1988; Share *et al.* 1990; Siegel, 1988, 1989, 1992; Stanovich & Siegel, 1994). This primary indicator of reading disability does not distinguish disabled readers with IQ-discrepancy from those without such discrepancies (see also, Fletcher *et al.*, 1994).

Likewise, measures of orthographic processing, on which reading-disabled children are less impaired, show no differences between poor readers with and without reading-IQ discrepancy (Fredman & Stevenson, 1988; Siegel, 1992; Stanovich & Siegel, 1994). Finally, the spelling-sound regularity effect, often interpreted as an indicator of the relative reliance (although not necessarily of relative skill, see Rack *et al.*, 1992) on phonological and orthographic coding

processes appears to be of a similar magnitude in reading-disabled children and younger reading-level controls. This also appears to be true for both poor readers without (Beech & Harding, 1984; Stanovich, Nathan & Zolman, 1988; Treiman & Hirsh-Pasek, 1985) and for poor readers with reading-IQ discrepancy (Baddeley, Logie & Ellis, 1988; Ben-Dror, Pollatsek & Scarpati, 1991; Brown & Watson, 1991; Bruck, 1990; Holligan & Johnston, 1988; Olson, Kliegl, Davidson & Foltz, 1985; Siegel & Ryan, 1988; Watson & Brown, 1992).

Thus, there is no indication that the nature of processing within the word recognition module differs at all for poor readers with and without IQ-discrepancy. Their relative strengths in phonological and orthographic coding processes, and their relative reliance on these subskills, appears to be nearly the same. The relative tradeoff between phonological and orthographic subskills—one of the most reliable phenotypic behavior patterns associated with reading disability—does not distinguish poor readers with and without reading-IQ discrepancy. This finding is consistent with the lack of evidence for a difference between these two groups in genetic and neuroanatomical studies.

Not surprisingly, there *are* cognitive differences between poor readers with and without reading-IQ discrepancy outside of the word recognition module (Ellis & Large, 1987; Siegel, 1992; Stanovich, 1988; Stanovich & Siegel, 1994), because these children differ in intelligence. Some of these cognitive differences may be related to comprehension processes. Thus, there may well be reading comprehension differences between the two groups when they are equated on word recognition ability (Bloom, Wagner, Reskin & Bergman, 1980; Ellis & Large, 1987; Jorm, Share, Maclean & Matthews, 1986; Silva, McGee & Williams, 1985), although even this expectation has not always been borne out (Siegel, 1988, 1989; Felton & Wood, 1992). These differences might well relate to certain educational issues such as the reading level to be expected of a student subsequent to remediation of their primary word recognition problem (Stanovich, 1991). However, it is important to note that any such differences are not indicators of the core processing problem that caused the word recognition deficit that triggered the diagnosis of reading disability: Phonological coding difficulties probably resulting from deficient phonological awareness. Thus, such differences outside of the word recognition model provide no rationale for a definition of reading disability based on IQ-discrepancy. Such definitions would only give the mistaken impression that children with reading-IQ discrepancy have distinctive genetic/neurological etiology. Indirect validation of the idea of differentiating poor readers on the basis of reading-IQ discrepancies would come from data showing that high- and low-IQ poor readers are differentially sensitive to specific educational interventions. There is, however, no body of evidence indicating that poor readers with reading-IQ discrepancy respond differently to various educational treatments than do poor readers without such discrepancies.

Whither Dyslexia?

Thus, the research literature provides no support for the notion that we need a scientific concept of dyslexia separate from other, more neutral, theoretical terms such as reading disabled, poor reader, less-skilled, etc. Yes, there is such a thing as dyslexia if by dyslexia we mean poor reading. But if this is what we mean, it appears that the term dyslexia no longer does the conceptual work that we thought it did. Indeed, whatever conceptual work the term is doing appears to be misleading. The concept of dyslexia is inextricably linked with the idea of an etiologically distinct type of reading disability associated with moderate to high IQ. Certainly an extreme form of this belief can be seen in the promotional activities of many advocacy groups and in media portrayals of "dyslexia". The typical "media dyslexic" is almost always a very bright child. Indeed, this media portrayal has now entered the realm of folk belief, for there exists a popular myth that dyslexia is the "affliction of geniuses" (Adelman & Adelman, 1987; Coles, 1987), if anything, *more* likely to occur in very bright people. This folk belief has even subtly affected the thinking of researchers who, without much thought, appear to have embraced the unverified assumptions about the meaning of reading-IQ discrepancy promulgated in school and clinic-based definitions and often derived from pragmatic considerations rather than scientific ones.

In fact, it appears 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 that neuroanatomical anomalies that are associated with reading disability are more characteristic of high-IQ than of low-IQ poor readers. As Taylor and Schatschneider (1992) argue, "IQ criteria were imposed primarily as a means of ruling out confounding variables and for assisting in the search for specific cognitive antecedents" (p. 630). It appears that the intuition that IQ discrepancy measurement would provide such assistance is mistaken. IQ discrepancy does not carve out a unique information processing pattern in the word recognition module that is the critical locus of reading disability. If there is a special group of reading disabled children who are behaviorally/cognitively/genetically "different" it is becoming increasingly unlikely that they can be quickly picked out using reading-IQ discrepancy as a proxy for the genetic and neurological differences themselves.

Rather than becoming further distracted by the IQ issue, it may well be more fruitful for the field to explore the implications of conceptualizing reading disability as residing on a continuum of developmental language disorder (see Bishop & Adams, 1990; Catts, 1991; Gathercole & Baddeley, 1987; Kamhi, 1992; Kamhi & Catts, 1989; Scarborough, 1990). For example, Gathercole and Baddeley (1987) argue that "although language problems are typically detected prior to the children receiving reading instruction . . . it is possible that the

alphabetic literacy skills required in reading may be more sensitive to the adequacy of speech analytic skills than other aspects of normal linguistic development, such that a mild deficit may only be detectable in reading performance. More severe subjects may result in the more generalized symptom complex associated with developmental language disorder . . . This is also clearly consistent with the notion that the two populations may quantitatively differ rather than qualitatively" (p. 464).

In light of these attempts to conceptualize reading disability as a milder form of language disability, it is interesting to note that the question of whether a discrepancy-defined disability is different from a disability defined purely in terms of chronological age occurs in analogous form in the area of developmental language disorder (Aram, Morris & Hall, 1992; Cole, Dale & Mills, 1990). Cole *et al.* (1990) describe how prior to more recent concerns about the relation between cognition and language "Any child who demonstrated a discrepancy between chronological age and language age would generally have been considered a candidate for language intervention by speech-language pathologists" (p. 291). However, an assumed tight link between language and cognition has recently led to what is called the Cognitive Referencing model, which has the implication that "Children who have developed language skills at a level equal to their cognitive skills are not considered to be language delayed, even if their language skills are significantly below chronological age" (p. 292). However, just as in the area of reading disability, Cole *et al.* point out that "it is surprising that there is little or no empirical evidence for evaluating the Cognitive Referencing model" (p. 292).

In summary, the search for neurological and genetic correlates of reading disability is being conducted with vigor in research laboratories around the world. The moral of the tale told in this Annotation is that there appears to be no reason for such investigations to restrict their research samples in advance based on reading-IQ discrepancy. Indeed, our ability to map the multidimensional space of reading-related cognitive skills would be impaired by such a procedure. Likewise, the argument put forth here should not be read as an argument for eliminating the inclusion of IQ measures in research studies of reading disability. Researchers may well want to investigate whether relationships with genetic or environmental variables are differentially related to reading raw scores and reading scores residualised on IQ (Stevenson, 1991; Stevenson & Fredman, 1990). For example, in a previously mentioned study, Stevenson (1991) found that the heritability of a group deficit in spelling ability tended to be greater for spelling scores residualised on IQ than for raw spelling scores. Such findings are often *not* primarily viewed as establishing a separate genetic etiology for one group of poor readers (or spellers). Instead, they may be viewed as establishing that the genetic factors influencing spelling are not the same as those influencing IQ.

To conclude, no one disputes the logical possibility of distinct etiologies within the population of poor readers. Obviously, if a group of children were not

taught to read and not exposed to print, their reading disability would have a distinct causation different from that in the general disabled population. The point, instead, is that it has yet to be demonstrated that whatever distinct causes actually exist are correlated with the degree of reading-IQ discrepancy. Because the term dyslexia mistakenly implies that there is such evidence, the reading disabilities field must seriously consider whether the term is not best dispensed with.

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