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Differential Genetic Etiology of Reading Difficulties as a Function of IQ: An Update

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Abstract

In order to test the hypothesis that the genetic etiology of reading disability differs as a function of IQ, composite reading performance data from 308 pairs of identical (monozygotic, MZ) twins and 440 pairs of fraternal (dizygotic, DZ) twins (254 same-sex and 186 opposite-sex) in which at least one member of each pair was classified as reading-disabled were subjected to multiple regression analysis (DeFries and Fulker, 1985, 1988). In the total sample, heritability of the group deficit in reading performance (h^2_g) was .61 ($\pm .06$). However, results of fitting an extended regression model to reading performance and IQ data suggested that the genetic etiology of reading disability differs as a linear function of IQ ($p \leq .04$). When the basic regression model was fitted separately to data from twin pairs with Wechsler (1974, 1981) Full Scale IQ scores in the upper and lower 25% of the sample, resulting estimates of h^2_g were .75 ($\pm .12$) and .50 ($\pm .10$), respectively ($p \leq .045$). These results suggest that reading difficulties in children with a higher IQ are due substantially to genetic influences and may require intensive remediation efforts.

Keywords

Genetics; Reading disability; Dyslexia; IQ; General cognitive ability; Twin studies

Introduction

As shown in Table I, definitions of dyslexia (reading disability or reading difficulties, RD) have expanded and evolved in recent years, in part due to refined understanding obtained through research and in part due to controversy that has arisen over the role of cognitive abilities in the definition and classification of RD. Although some reference to cognitive/intellectual ability has historically been included in most definitions of dyslexia, either in the form of discrepancy scores (e.g., Critchley, 1970; Rutter & Yule, 1975) or IQ cut-off (e.g., Siegel, 1989), the past two decades have brought increasing criticism of these models (e.g., Gustafson & Samuelsson, 1999; Lyon, 1989, 1995; Siegel, 1998, 2006; Stanovich & Siegel, 1994; Van den Broeck, 2002) and a tendency toward exclusion of IQ from the definition of RD (e.g., Aaron et al., 2008; Siegel, 2006). However, even the most recent working definition of dyslexia (Lyon et al., 2003) adopted by the International Dyslexia Association and the National Institute on Child Health and Human Development includes the phrase "...

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Dedication: This paper is dedicated to Gerald E. McClearn, who reinforced the third author's nascent interest in behavioral genetics at the University of California, Berkeley, in 1963-64, invited him to join the newly founded Institute for Behavioral Genetics, University of Colorado, in 1967, and fostered the second author's interest in dyslexia in the 1970's.

is often unexpected in relation to other cognitive abilities...” Although the use of discrepancy scores or an IQ cutoff is intended to ensure that learning difficulties are not due to general cognitive deficits (Lyon, 1996; Stanovich, 1986), this “assumption of specificity” (Stanovich, 1986) implies that specific reading disability is etiologically distinct from reading deficits associated with more general learning difficulties (Olson et al., 1991; Lyon et al., 2003). Thus, measures of general cognitive ability may provide additional information or context from which to understand better the reading deficits of children with RD.

One possible test of the specificity assumption is to assess the extent to which genetic influences on reading difficulties vary with general cognitive ability (e.g., Olson et al., 1991; Wadsworth et al., 2000). Results from previous behavioral-genetic studies using data from identical and fraternal twins have revealed that approximately half of the group deficit in reading is due to genetic influence (for reviews, see DeFries and Alarcón, 1996; Fisher & DeFries, 2002; Plomin & Kovas, 2005). However, the degree and/or type of genetic influence may also vary across individuals within the reading-disabled group.

Several studies have addressed this issue and will be described briefly (for a more detailed review of the early studies, see Wadsworth et al., 2000). In the first study designed specifically to test the hypothesis of differential genetic etiology of reading deficits as a function of IQ, Olson et al. (1991) analyzed composite reading data and measures of IQ adjusted for their relationship to word reading (i.e., IQ residuals calculated from the regression of IQ scores onto word reading scores) from 247 same-sex twin pairs participating in the Colorado Reading Project. Data from twin pairs in which at least one member scored at least one standard deviation below the mean of the control group on the word recognition composite were subjected to DeFries-Fulker multiple regression analysis for use with selected samples (DF analysis; DeFries & Fulker, 1985, 1988). Although the heritability of the reading deficit was substantially higher among those with higher full-scale IQ ($h^2_g=.67$) than among those with lower IQ ($h^2_g=.40$), this group difference was not significant ($p \leq .08$). However, Olson et al. (1999) subsequently analyzed composite word recognition data from a much larger sample (279 MZ and 334 DZ twin pairs) and found that the heritability of deficits in word recognition differed significantly as a function of a residual IQ variable ($p \leq .05$).

DeFries and Light (1996) obtained similar results using a broader composite of reading performance data (Reading Recognition, Comprehension and Spelling subtests of the Peabody Individual Achievement test) to test the hypothesis that the etiology of reading disability differs between children with IQ scores within the normal-range (90 and above) and those with lower (80-90) IQ. Although the sample with lower IQ was too small for regression analysis to yield reliable parameter estimates (28 pairs, 17 MZ and 11 DZ), the estimate of heritability of the group deficit in reading ability ($h^2_g=.29 \pm .25$) was substantially lower than that obtained from 260 pairs (149 MZ, 111 same-sex DZ) having an IQ of 90 or above ($h^2_g=.51 \pm .10$).

In the analyses by DeFries and Light (1996) and Olson et al. (1999), a minimum IQ cut-off of 80 or 85 was employed, restricting the range of IQ scores used in the regression analyses. Moreover, Olson et al. (1999) included opposite-sex twin pairs. Any quantitative or qualitative sex differences in etiology could potentially have affected the heritability estimates obtained for the IQ groups. Thus, in an effort to test the hypothesis of differential genetic etiology of reading deficits more rigorously, Wadsworth et al. (2000) subjected composite reading performance data from a larger sample of same-sex twin pairs than had previously been available (i.e., 223 pairs of MZ twins and 169 pairs of same-sex DZ twins in which at least one member of each pair was classified as reading-disabled) to DF analysis. In contrast to previous analyses, no minimum IQ restriction was imposed. Further, IQ scores

for members of a pair were averaged, so that when concordant pairs were double-entered (once with Twin 1 as the proband and once with Twin 2 as the proband), the same pair could not be represented in different IQ groups depending on which twin was considered the proband. When the basic model (DeFries & Fulker, 1988) was fitted separately to data from subjects with higher IQ scores (100 and above) and lower IQ (below 100), resulting estimates of h^2_g were .72 and .43, respectively, a significant difference ($p \leq .03$, one-tailed). When an extended regression model (DeFries & Fulker, 1985), including IQ as a covariate along with its appropriate interactions, was fitted to reading performance and continuous IQ data, results indicated that the genetic etiology of reading disability differed as a linear function of IQ ($p \leq .007$, one-tailed).

In a novel extension of these previous analyses of Colorado Twin Study data, Knopik et al. (2002) employed both the basic and extended DF model to test for differential genetic etiology of word recognition, phonological decoding, orthographic coding and phoneme awareness as a function of IQ in 465 twin pairs with a history of reading problems (201 MZ, 160 same-sex DZ and 104 opposite-sex DZ pairs). In addition, data from 168 sibling pairs (including DZ twin pairs and their siblings as well as non-twin siblings of MZ twins) were subjected to a sib-pair linkage adaptation of the DF model (Fulker et al., 1991) to test the hypothesis that the quantitative trait locus (QTL) for reading deficits on the short arm of chromosome 6 (Fisher & DeFries, 2002) also influences other reading component processes. This model was further extended to test for differential effects of the QTL as a function of IQ. As in the analyses of Wadsworth et al. (2000), twin pairs were placed into higher (IQ = 100 or above) and lower (IQ below 100) groups based on the average score of the twin pair. Results of the DF analyses of twin data provided evidence for a differential genetic etiology as a linear function of IQ for all of the reading and language measures. Specifically, group heritabilities for word recognition were .82 for the higher IQ group and .45 for the lower IQ group, a highly significant difference ($p \leq .0014$, one-tailed). Moreover, results of DF linkage analyses suggested that a QTL on chromosome 6p may differentially influence the reading and language measures as a function of IQ, though the small sample sizes for some of the measures resulted in limited power to detect significance.

Despite using different measures, different IQ cut-offs, different criteria for placement of twin pairs into IQ groups, residualization of IQ scores, and inclusion or exclusion of opposite-sex DZ pairs, results of the previous studies collectively suggest that genetic factors are more important as a cause of reading disability among children with higher IQ than among those with lower IQ. However, these studies examined the etiology of reading difficulties as a function of high and low IQ groups that were either overlapping or divided based on the average or median IQ score, so that there was little distinction between “higher” and “lower” IQ. In every case, the groups would have been too small to conduct meaningful analyses of the upper and lower ends of the IQ distribution. Further, in all but two of the previous studies (Olson et al., 1999; Knopik et al., 2002), analyses were limited to same-sex pairs. Because analyses of differential heritability of reading deficits as a function of gender have failed to find either quantitative or qualitative sex differences in these samples (Hawke et al., 2007; Wadsworth & DeFries, 2005), there remains no compelling reason to exclude opposite-sex DZ twin pairs from these analyses. Finally, the two most recent studies with the largest samples (Wadsworth et al., 2000 and Knopik et al., 2002) used the average IQ of members of a twin pair to avoid placement of concordant pairs in different IQ groups with double entry. Subsequent analyses of twin data from the CLDRC and content-free simulations (Cross et al., 2002) have indicated that use of the average of proband and cotwin scores for a subtype variable (in this case, IQ) which is positively correlated with the reading measure introduces a possible “cotwin-regression artifact” that almost always results in higher heritability for the high subtype group. Thus, the purpose of the current study is to test the hypothesis of a differential heritability of reading deficits as a

function of IQ more rigorously by analyzing data from a much larger sample of twins participating in the Colorado Learning Disabilities Research Center, including opposite-sex twin pairs, and using proband's residualized IQ score (Olson et al., 1991, 1999), rather than the proband-cotwin average.

Methods

Participants and Measures

The subjects were participants in either the Colorado Reading Project (CRP; DeFries, 1985; DeFries et al., 1991) or the ongoing Colorado Learning Disabilities Research Center (CLDRC; DeFries et al., 1997). Twin pairs are systematically ascertained through 27 cooperating school districts in the state of Colorado. To minimize the possibility of ascertainment bias, twin pairs are initially identified without regard for reading performance. Parents' permission is then sought to review the children's records for evidence of reading problems (e.g., low reading achievement test scores, referral to resource rooms or reading therapists, reports by classroom teachers or school psychologists, etc.). Twin pairs in which at least one member has a positive history of reading problems are invited to participate in the study at the Institute for Behavioral Genetics, Boulder, Colorado. Participants are administered an extensive battery of psychometric tests, including the Wechsler Intelligence Scale for Children--Revised (WISC--R; Wechsler, 1974) or the Wechsler Adult Intelligence Scale--Revised (WAIS--R; Wechsler, 1981) and the Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970).

Data from the Reading Recognition, Reading Comprehension, and Spelling subtests of the PIAT are used to compute a discriminant function score (READ) for each child, based on coefficients estimated from an independent sample of 140 reading-disabled and 140 control singletons tested in the Colorado Reading Project (DeFries, 1985). For the purposes of the present study, twin pairs were included in the proband sample if at least one member had a positive school history of reading problems, scored at least 1.5 standard deviations below the mean of the comparison sample, had no evidence of serious emotional or behavioral problems, and no uncorrected visual or auditory acuity deficit. Because the current study examines the etiology of reading disability as a function of IQ, only those subjects with both verbal and performance IQ below 70 (9 individuals, representing 7 pairs) were excluded from the analyses.

Because the CLDRC is an ongoing study which includes data from twin pairs tested as early as 1983, subjects tested in earlier years of the project may have scored lower than those tested in later years (Flynn, 1984), thereby affecting group placement. However, in the current study, those probands tested before 1990 had a mean full scale IQ of 96.29 (± 10.09), those tested between 1990 and 2000 had a mean of 96.36 (± 10.35), and those tested from 2000 onward had a mean of 95.11 (± 12.55). None of these differences were significant. Further, year of testing is not a significant predictor of IQ when analyzed as a continuous variable ($p > .1$).

Zygosity is determined using selected items from the Nichols and Bilbro (1966) questionnaire, which has a reported accuracy of 95%. In ambiguous cases twin pairs are genotyped using polymorphic DNA markers. The twins were reared in primarily English-speaking, middle-class homes, and were between 8.0 and 20.2 years of age at the time of testing. The current sample consists of 308 pairs of identical (monozygotic, MZ) twins and 440 pairs of fraternal (dizygotic, DZ) twins (254 same-sex and 186 opposite-sex) tested through November 30, 2008. Because this is an augmented sample, there is partial overlap with samples from previous reports.

Analysis

When testing for genetic etiology of a qualitative, dichotomous trait, a comparison of MZ and DZ concordance rates is appropriate. However, DeFries and Fulker (1985) proposed that when probands are selected because of deviant scores on a *continuous* variable, such as reading performance, the differential regression of MZ and DZ cotwin means toward the mean of the unselected population provides a more appropriate test of genetic etiology. If the deficit of the probands is due to genetic influences, DZ cotwins (who share half their segregating genes, on average) are expected to regress more toward the mean of the unselected population than are MZ cotwins (who are genetically identical to the MZ probands). Thus, if the mean scores of MZ and DZ probands are approximately equal, a t-test of the difference between MZ and DZ cotwin means would provide a sufficient test of genetic etiology. However, fitting the following multiple regression model to selected twin data provides a more statistically powerful and versatile test.

$$C = B_1P + B_2R + K \quad (1)$$

where C is the cotwin's score, P is the proband's score, R is the coefficient of relationship (1.0 for MZ pairs and 0.5 for DZ pairs), B_1 and B_2 are the regression coefficients, and K is the regression constant. When this model is fitted to data from selected twin pairs, B_2 estimates twice the difference between means of the MZ and DZ cotwins after covariance adjustment for any the difference between MZ and DZ proband means; thus, B_2 provides a direct test of genetic etiology. Further, when each individual's score is appropriately transformed (i.e., expressed as a deviation from the mean of the unselected population and divided by the difference between the mean of the probands and that of the unselected population), $B_2 = h^2_g$, an estimate of the extent to which the deficit of the probands is due to heritable influences (DeFries & Fulker, 1988).

Because truncate ascertainment of subjects was used, concordant pairs were double-entered for the regression analyses, in a manner analogous that used in the calculation of probandwise concordance rates in which each member of the concordant pair is considered a proband (DeFries & Gillis, 1991). Standard error estimates and significance levels were adjusted accordingly.

In the current study, equation 1 was fitted first to transformed discriminant function data (READ) from probands and their cotwins, irrespective of IQ, to assess the genetic etiology of reading disability in the total sample. To test for differential genetic etiology of RD as a linear function of IQ, the following extended model was then fitted to the READ and proband IQ scores:

$$C = B_1P + B_2R + B_3IQ + B_4P * IQ + B_5R * IQ \quad (2)$$

where IQ is the proband's residualized IQ score (calculated from the regression of IQ scores onto word reading scores), $P * IQ$ is the product of the proband's READ and IQ score, and $R * IQ$ is the product of IQ and the coefficient of relationship. When proband's IQ is entered as a continuous covariate, the B_5 coefficient tests for differential etiology of RD as a linear function of IQ across the full range of scores. Equation 2 can also be fitted to dichotomously coded data as described below.

To illustrate the differential etiology of reading difficulties as a function of IQ, probands were then selected from the upper and lower 25% of the IQ distribution. Equation 1 was

then fitted to transformed READ data from each IQ group separately, in order to assess the genetic etiology of RD in each of the two IQ groups. To test the significance of the difference between the estimates obtained for the higher and lower IQ groups, equation 2 was then fitted to dichotomously coded data from the two groups simultaneously. When the groups are coded -.5 for twin pairs whose proband IQ is at or below the 25th percentile and .5 for twin pairs whose proband IQ is at or above the 75th percentile, the B_5 coefficient provides a direct estimate of the difference between the estimates for the 2 groups as well as a test of the significance of the difference between the two estimates.

Finally, in order to test for a possible differential h^2_g as a quadratic function of IQ, the following regression model was fitted to reading performance and continuous proband IQ data from the entire sample:

$$C = B_1P + B_2R + B_3IQ + B_4P * IQ + B_5R * IQ + B_6IQ^2 + B_7P * IQ^2 + B_8R * IQ^2 \quad (3)$$

where IQ^2 is the square of each proband's residualized IQ. Consequently, when equation 3 is fitted to selected twin data, B_8 provides a test of the significance of differential genetic etiology of reading disability as a quadratic function of IQ.

Although previous analyses have found no evidence for differential sex effects on the etiology of reading deficits, sex was included as a covariate to control for any possible mean differences. Because there was an *a priori* hypothesis about the direction of linear effects, one-tailed tests of significance were used for the extended models.

Results

Mean discriminant function (READ) scores, expressed in standard deviation units from the mean of 1423 control twins, and Full Scale IQ scores are presented in Table II for probands and cotwins in the total MZ and DZ samples as well as for the lower and higher IQ groups. Means of both MZ and DZ probands are more than 2.5 standard deviation units below the control mean. The mean of the MZ cotwins has regressed only slightly toward the control mean (.22 standard deviation units on average), whereas that of the DZ cotwins has regressed approximately 1 standard deviation, supporting previous evidence of a genetic etiology for reading deficits. Of particular interest to the present study, the differential regression of the MZ and DZ cotwin means is greater in the higher IQ group than in the lower IQ group, suggesting a greater genetic influence among individuals with reading problems and higher IQ.

Results of fitting the basic and extended regression models to the data are provided in Table III. When the basic model (equation 1) was fitted to the full proband sample, the B_2 estimate was $.61 \pm .06$ ($p = .00$). Differential genetic etiology of reading disability as a linear function of proband's IQ was tested by fitting equation 2 to data from the full sample of selected twins, with the IQ residual included as a continuous measure. The resulting B_5 estimate ($.10 \pm .06$) indicated significant differential genetic etiology of reading disability as a linear function of IQ ($p \leq .04$).

When equation 1 was fitted to data from the higher and lower IQ groups separately, the B_2 estimate obtained for the higher IQ group was substantially higher than that obtained for the lower IQ group (.75 and .50, respectively), a significant difference ($B_5 = .25$; $p \leq .045$). These results suggest that genetic factors may be more important as a cause of reading difficulties in individuals with higher IQ. In contrast, when equation 3 was fitted to the data, there was

no evidence for differential genetic etiology of reading disability as a quadratic function of IQ ($p \geq .45$).

Discussion

Historically, the reading deficits of children with low IQ have been assumed to be etiologically distinct from those of children with normal-range to high IQ. However, as previously noted, recent decades have seen considerable controversy over the use of IQ and/or discrepancy scores in the diagnosis and classification of RD. Although working definitions of dyslexia have evolved over the past half-century to minimize the importance of general cognitive ability, none has eliminated the construct from the definition. If reading deficits in the presence of normal-range to high cognitive ability are, in fact, etiologically distinct from those associated with lower IQ, the heritability of reading deficits may differ as a function of IQ level. Previous studies have provided support for this hypothesis, but differences in measures and methods make it difficult to compare results across studies, and methodological limitations diminish the validity of the findings. For example, three of the previous studies either analyzed data from overlapping high and low IQ groups (Olson et al., 1991), or divided their samples at the average or median IQ [i.e., subjects with IQ of 100 and above were placed in the higher IQ group, and those with IQ scores below 100 were placed in the lower IQ group (Wadsworth et al., 2000; Knopik et al., 2002)]. Thus, there was little distinction between “higher” and “lower” IQ. Further, in all but two of the previous studies (Olson et al., 1999; Knopik et al., 2002), analyses were limited to same-sex pairs. Recent analyses of gender differences in these samples provide no support for either quantitative or qualitative differences in etiology as a function of gender (Hawke et al., 2007; Wadsworth & DeFries, 2005). Thus, exclusion of opposite-sex pairs unnecessarily reduces sample size, an important consideration for analyses of high and low ends of the IQ distribution. Finally, two studies (Wadsworth et al., 2000 and Knopik et al., 2002) used the average IQ of members of twin pairs to determine IQ group placement of each pair. More recent analyses of twin data from the CLDRC and content-free simulations (Cross et al., 2002) have indicated that averaging proband and cotwin scores of a variable which is positively correlated with the reading measure (in this case, IQ) introduces a possible “cotwin-regression artifact” that generally results in higher heritability for the high subtype group. Thus, the corroborating results of these two studies could have been due to a bias introduced by averaging the IQ scores of the members of each twin pair.

It is noteworthy and encouraging that results of the previous studies have been consistent, despite the differences in measures and methods and in spite of the limitations of each of these studies. However, it is important to test the hypothesis of a differential heritability of reading deficits as a function of IQ in the absence of methodological limitations. Consequently, in the current study we attempted to incorporate the “best” practices of each study. Thus, the present study included both same-sex and opposite-sex twin pairs, used proband's residualized IQ rather than the proband-cotwin average for group designation, included sex as a covariate in the extended models to control for any possible sex differences, and analyzed the largest sample to date of twins selected for reading difficulties.

Although, the purpose of this study was to test the hypothesis of differential genetic etiology of reading deficits as a linear function of IQ across the full range of IQ scores, the current sample is large enough to examine the heritability of reading deficits at both ends of the IQ distribution. Thus, for illustrative purposes, we also assessed the genetic etiologies of reading deficits among twin pairs scoring in the upper and lower 25% of the sample.

The overall heritability of the group deficit in reading performance in this sample, irrespective of IQ, was .61, suggesting that over half of the group deficit was due to heritable

influences. However, when IQ was treated as a continuous covariate, and the extended model (equation 2) was fitted to data from all twin pairs simultaneously, the genetic etiology of reading deficits differed as a linear function of IQ. Further, when the twin pairs were divided into two groups--those with proband IQ scores in the upper 25% of the distribution and those in the lower 25%--the h^2_g estimates were .75 and .50, respectively, a significant difference. However, the test for differential h^2_g as a quadratic function of IQ was not significant. These findings are consistent with those of previous studies of differential heritability of reading deficits as a function of IQ. Therefore, in spite of the differences in methods and definitions used among the various studies, the finding of differential heritability of reading deficits as a function of IQ appears to be robust.

Results of the current study suggest that genetic influences may be more important as a cause of reading disability among subjects with higher IQ. However, the methods used in this study do not address the issue of whether there are *different* genetic influences on reading disability as a function of IQ. The same genetic factors could be operating irrespective of IQ level, but the proportion of variance accounted for might differ because the nature and degree of influence of environmental factors may vary as a function of IQ. For example, the environment for reading development could be both more favorable and more homogeneous, on average, for subjects with higher IQ, with the result that the environment would have less of an impact in producing individual differences in these children (Wadsworth et al., 2000). Consistent with these findings, Friend, DeFries & Olson (2008) found that parental education, which is correlated not only with IQ, but with other environmental variables as well, moderated genetic influences on reading deficits such that genetic influences were greater and environmental influences lesser among those whose parents had higher levels of education. Of course, those genetic factors that influence parents' education level may be correlated with both their children's IQ and reading environment. However, it is important to note that the differential etiology of reading difficulties as a function of IQ is not mediated by parental education level. When maternal and paternal education levels were included as covariates in Equation 2, the test for differential h^2_g as a function of IQ (i.e., B_5) remained significant ($p = .05$).

The results of the current study suggest that IQ scores may tell us something about the causes of children's reading deficits. They do not, however, imply that IQ scores should be used for diagnosis via discrepancy scores or strict cut-offs. It has been frequently noted that for both practical and psychometric reasons, the use of IQ and/or discrepancy scores can be problematic (e.g., Aaron et al., 2008; Fletcher et al., 2005; Gustafson & Samuelsson, 1999; Lyon et al., 2003; Siegel, 2006). Therefore, in recent years, this practice has been strongly challenged. A currently popular alternative to defining dyslexia and diagnosing children as dyslexic is Response to Instruction (RTI; Fletcher et al., 2004). RTI involves early identification of reading difficulties and subsequent classroom-based intervention with additional instruction for those with continuing difficulties. Our results suggest that the reading difficulties of children with a higher IQ are due substantially to genetic influences. Thus, IQ may help identify those children whose reading deficits may require intensive remediation efforts.

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Table I

Evolving definitions of dyslexia¹

1968	<p>“A disorder manifested by difficulty in learning to read, <i>despite conventional instruction, adequate intelligence, and sociocultural opportunity</i>. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin.”</p> <p>(World Federation of Neurology; Critchley, 1970)</p>
1994	<p>“...one of several distinct learning disabilities. It is a specific language-based disorder of constitutional origin characterized by difficulties in single word decoding, <i>unexpected in relation to age and other cognitive and academic abilities</i>; they are not the result of generalized developmental disability or sensory impairment....”</p> <p>(The Orton Dyslexia Society Research Committee, 1994, <i>op.cit.</i> Lyon, 1995)</p>
2003	<p>“...a specific learning disability that is neurobiological in origin. It is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often <i>unexpected in relation to other cognitive abilities</i> and the provision of effective classroom instruction....”</p> <p>(Lyon, Shaywitz & Shaywitz, 2003).</p> <p>(Adopted by the International Dyslexia Association and the Nat'l. Inst. on Child Health and Human Dev)</p>

¹ Italics added.

Table II
Mean READ and FSIQ scores ($\pm$ SD) of probands and cotwins by zygosity and IQ group[/]

	READ		FSIQ		N Pairs
	Probands	Cotwins	Proband	Cotwins	
Identical (total)	-2.65 $\pm$.81	-2.43 $\pm$ 1.00	95.37 $\pm$ 14.62	95.92 $\pm$ 13.97	308
Lower IQ	-2.66 $\pm$.82	-2.51 $\pm$ 1.03	82.85 $\pm$ 6.40	85.85 $\pm$ 9.54	80
Higher IQ	-2.68 $\pm$.83	-2.37 $\pm$ 1.00	108.97 $\pm$ 6.71	106.80 $\pm$ 8.93	74
Fraternal (total)	-2.64 $\pm$.84	-1.62 $\pm$ 1.32	96.93 $\pm$ 14.43	101.97 $\pm$ 13.06	440
Lower IQ	-2.64 $\pm$.86	-1.84 $\pm$ 1.25	83.39 $\pm$ 5.87	95.87 $\pm$ 11.58	104
Higher IQ	-2.65 $\pm$.88	-1.36 $\pm$ 1.27	109.51 $\pm$ 6.32	107.63 $\pm$ 11.77	118

[/] Ns for higher and lower IQ groups do not equal the totals for MZ and DZ pairs because only the upper and lower 25% are included in the IQ groups, whereas all MZ and DZ pairs are included in the totals.

Table IIIResults of fitting DF models to data from full, higher IQ and lower IQ samples¹

	$B_2 / B_5 / B_8$	p
Basic model – full sample	$B_2 = .61 \pm .06$	0.00
Extended basic model, continuous, linear– full sample	$B_5 = .10 \pm .06$	$\leq .04$
Higher IQ	$B_2 = .75 \pm .12$	$< .001$
Lower IQ	$B_2 = .50 \pm .10$	$< .001$
Extended basic model, dichotomous, IQ groups	$B_5 = .25 \pm .15$	$\leq .045$
Extended basic model, continuous, quadratic – full sample	$B_8 = .03 \pm .04$	$\geq .45$

¹ Upper and lower 25% of full scale IQ distribution