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DeFries-Fulker analysis of longitudinal reading performance data from twin pairs ascertained for reading difficulties and from their nontwin siblings

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Abstract

Reading difficulties are both heritable and stable; however, little is known about the etiology of this stability. Results from a preliminary analysis of data from 56 twin pairs who participated in the Colorado Longitudinal Twin Study of Reading Disability (Astrom et al., 2007) suggested that about two-thirds of the proband deficit at follow-up was due to genetic factors that also influenced deficits at their initial assessment. Although our proband sample is now nearly twice as large, it is still relatively small; thus, to increase power, we subjected data from probands, co-twins and their nontwin siblings to a novel extension of DeFries-Fulker analysis (DeFries & Fulker, 1985; 1988). In addition to providing estimates of univariate and bivariate heritability, this analysis facilitates a test of the difference between shared environmental influences for twins versus siblings. Longitudinal composite reading performance scores at 10.6 and 15.5 years of age, on average, were analyzed from 33 MZ and 64 DZ twin pairs in which at least one member of each pair had reading difficulties, and from 44 siblings of the probands. Scores were highly stable ($.86 \pm .03$, across probands, cotwins and siblings) and heritability of the group deficit at initial assessment was $.67 \pm .22$. Longitudinal bivariate heritability was $.59 \pm .21$, suggesting that nearly 60% of the proband reading deficit at follow-up is due to genetic factors that influenced reading difficulties at the initial assessment. However, tests for special twin environmental influences were nonsignificant.

Keywords

DF analysis; twins; siblings; reading; longitudinal

Introduction

During the past few decades, much has been learned about the heritable nature of reading ability and disability. For example, twin and adoption studies have shown that individual differences in reading performance are highly heritable, although estimates of heritability have ranged from .18 to .81 for subjects 7 to 16 years of age (Alarcón et al., 1995; Harlaar et al., 2007; Stevenson et al., 1987; Wadsworth et al., 2006; Wadsworth et al., 2007). At comparable ages, heritability estimates for reading deficits range from .37 to .72 (e.g., DeFries & Alarcón, 1996; DeFries & Gillis, 1991; Harlaar et al., 2005). Moreover, results obtained from longitudinal studies have shown that stability correlations for reading

performance are considerable, ranging from .23 to .96 over intervals of 1 to 8 years (e.g., Bast & Reitsma, 1998; DeFries & Baker, 1983; Shaywitz et al., 1992; Wadsworth et al., 2006; Wadsworth et al., 2007; Wagner et al., 1997). When reading performance was estimated by Hulslander et al. (2010) as a latent trait from multiple measures of word recognition, the stability correlation between age 10 and 15 years was .98.

Although studies have consistently yielded evidence that both individual differences in reading performance and reading deficits are heritable and stable, little is known about the genetic and environmental etiologies of this stability. Only a small number of studies have evaluated the etiology of stability utilizing genetically informative designs, and they have primarily assessed the etiology of individual differences in reading performance within the normal range. For example, in the Colorado Adoption Project, word recognition was examined at ages 7, 12, and 16 in a sample of adoptive and nonadoptive sibling pairs (Wadsworth et al., 2006). Results indicated substantial stability for reading performance with stability correlations of .62 between ages 7 and 12, .71 between ages 12 and 16, and .58 between ages 7 and 16. Moreover, the proportion of observed stability attributable to shared genetic influences was 53%, 62%, and 86%, respectively.

Analyzing data from a younger sample participating in the International Longitudinal Twin Study (ITLS), Byrne et al. (2007) found that genetic influences accounted for approximately 90% of the observed stability between word reading in kindergarten and first grade. However, Grade 1 reading was also influenced by a second genetic factor, suggesting both genetic stability and genetic change at this developmental milestone. Environmental contributions to stability were limited to nonshared environmental influences, which were also a significant source of change. In contrast, shared environment was important only for kindergarten word reading, and did not contribute to stability.

Petrill et al. (2007) assessed the longitudinal stability of reading-related skills and reading outcomes of children who were tested on two occasions in the Western Reserve Reading Project (WRRP): in kindergarten or first grade, and a year later. Measures of reading related skills included phonological awareness (PA), expressive vocabulary (VOCAB) and rapid automatized naming (RAN); outcome was measured by performance on tests of letter knowledge (LET), word knowledge (WORD), phonological decoding (PD) and passage comprehension (COMP). Results of a series of Cholesky decomposition analyses suggested that, with the exception of RAN, genetic influences accounted for a significant proportion of the observed stability (as much as 42%, depending on the measure) for all measures of reading outcome at the two assessments. Shared environmental influences contributed to stability of PA, VOCAB, and LET. Nonshared environment had small, but significant, effects on the stability of RAN, WORD, PD and COMP.

These results are highly similar to those of Harlaar et al. (2007) who examined the genetic and environmental influences on reading performance and reading stability from the Twins Early Development Study (TEDS). For this study, reading achievement was measured by teacher assessments of subjects at ages 7, 9, and 10 years, using a rating scale of general reading achievement that referenced U.K. National Curriculum (NC) achievement goals for literacy. Results of the study showed substantial heritabilities for reading performance ranging from .57 to .67. In addition, NC scores were found to be significantly correlated across all three age groups; .62 for ages 7 to 9, .59 for ages 7 to 10, and .63 for ages 9 to 10, with genetic influences contributing to 77%, 68%, and 77% respectively, of the observed stability.

To our knowledge, only one study has assessed the etiology of stability of reading deficits (Astrom et al., 2007). Subjects from this preliminary longitudinal analysis of reading

disabilities of school-aged children (aged 8 – 16 years) were tested in the Colorado Learning Disabilities Research Center (CLDRC, DeFries, et al., 1997) and again approximately 5–6 years later in the Longitudinal Twin Study of Reading Disability (LTSRD, Wadsworth et al., 2007). Probands scored approximately two standard deviations below the mean of the control sample at both measurement occasions (Astrom et al., 2007), and a stability correlation of .84 was obtained between the two assessments. A bivariate extension of the DeFries-Fulker basic multiple regression model for analysis of selected twin data (DF; DeFries & Fulker, 1985; Light & DeFries, 1995) yielded an estimate of bivariate heritability of .65 ($\pm .32$), suggesting that nearly two-thirds of the proband reading deficit at follow-up was due to genetic factors that also influenced reading at initial assessment (Astrom et al., 2007).

Although our current longitudinal sample of MZ and DZ twin pairs who met criteria for inclusion in the proband sample at their initial assessment is now nearly twice as large, it is still somewhat underpowered for more complex analyses. However, since the inception of the CLDRC, we have also collected data from siblings of approximately half of the MZ and DZ probands. Consequently, using a novel application of the DF multiple regression method, we have included data from siblings of probands in the present analysis. Thus, the primary objectives of this study were twofold: (1) to assess more rigorously the etiology of the stability of reading deficits in a larger sample of twin pairs than was previously analyzed by Astrom et al. (2007); and (2) to apply an extension of the DF method (DeFries and Fulker, 1985; 1988) to analyze both twin and sibling data and test for “special twin environments” (i.e., a measure of the extent to which shared environmental influences for members of twin pairs differ from those for nontwin-sibling pairs). Based on previous findings of genetic influences on the stability of reading performance within the normal range, as well as our preliminary findings regarding the stability of reading deficits, we hypothesize that largely the same genetic influences on reading deficits are manifested at initial and follow-up assessments. Moreover, given the results obtained from a small twin and sibling study of individual differences in reading-related measures (Zieleniewski, et al., 1987), we hypothesize that shared environmental influences for reading deficits are similar for members of twin pairs and between twins and their nontwin siblings.

Methods

Participants and Measures—Subjects in the present study were first tested in the ongoing CLDRC between September 1996 and March 2003, and also participated in follow-up testing approximately five years later in the LTSRD. In order to minimize the possibility of ascertainment bias, school personnel in 27 different school districts within the state of Colorado identify twin pairs without knowledge of reading status. Parental permission is then sought to review the school records of the twins for any evidence of reading problems (i.e., low reading achievement test scores, referral to resource rooms or reading therapists, reports by classroom teachers, school psychologists, and/or parents). If either member of the twin pair displays a history of reading problems, both twins and their siblings are invited to participate in the study at the University of Colorado, Boulder, and at the University of Denver. The subjects complete a comprehensive battery of psychometric tests which includes the Wechsler Intelligence Scale for Children–Revised (WISC-R; Wechsler, 1974) or the Wechsler Adult Intelligence Scale–Revised (WAIS-R; Wechsler, 1981), the Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970), and measures of reading and language processes and executive functions. A discriminant function score (DISCR) is computed for each subject employing discriminant weights estimated from an analysis of data from the Reading Recognition, Reading Comprehension, and Spelling subtests of the PIAT obtained from an independent sample of 140 non-twin children with reading

disabilities and 140 non-twin children without reading problems (DeFries, 1985). In order for an individual to be diagnosed as reading-disabled in the current study, he or she must have a positive history of reading problems and score at least 1 standard deviation below the mean of the control sample on the discriminant score. Additional criteria include a minimum IQ score of 85 on either the Verbal or Performance Scale of the WISC-R (Wechsler, 1974) or the WAIS-R (Wechsler, 1981); no evidence of neurological problems or severe behavioral or emotional problems; and no uncorrected visual or auditory acuity deficits. Control twin pairs are matched to probands on the basis of age, gender, and school district. For a twin pair to be included in the control sample, both members of the pair must have a negative history for reading problems. Selected items from the Nichols and Bilbro (1966) questionnaire are used to determine zygosity of same-sex twin pairs. For indeterminate cases, zygosity is confirmed by analysis of blood or buccal samples.

The current sample included 33 MZ and 64 DZ twin pairs in which at least one twin met proband criteria at initial assessment in the CLDRC, and 44 of their nontwin siblings. Selected twins and both their co-twins and siblings underwent follow-up testing approximately 5 years after their initial participation (average age of 10.6 years at initial assessment and 15.5 years at follow-up). Because concordant twin pairs were double entered, the siblings were paired with both twins in those cases. As a result, there were 58 twin/sib pairings; in 21 of those pairings the twin was selected from an MZ pair and in 37 pairings the twin was selected from a DZ pair. For standardization and transformation of the variables at initial assessment, the control sample comprised 284 subjects tested in the CLDRC during the same time period as those subjects who participated in follow-up testing. At follow-up the control sample included 171 control twins who participated in follow-up testing.

Analyses

Multiple Regression Analysis—Early twin studies of reading difficulties compared concordance rates for deviant scores in identical and fraternal twin pairs to test for genetic etiology (DeFries & Alarcón, 1996). However, because reading performance is a continuous trait, a comparison of concordance rates of “affected” and “unaffected” pairs does not make optimal use of the data. Thus, DeFries and Fulker (1985, 1988) proposed a multiple regression analysis of twin data to assess the etiology of deviant scores, as well as individual differences within selected groups. This method has become a standard of behavioral genetic analysis and the method of choice for analyzing data from selected samples. This multiple regression method is particularly appropriate when analyzing data from probands who are selected because of deviant scores on a continuous variable such as reading performance; the differential regression of MZ and DZ co-twin scores toward the mean of the unselected population provides a test of genetic etiology (DeFries & Fulker, 1985). As MZ twins are genetically identical and DZ twins share only half of their segregating genes on average, the scores of DZ co-twins should regress more toward the mean of the unselected population if the condition is heritable. Consequently, when the MZ and DZ proband means are approximately equal, a simple *t*-test of the difference between the MZ and DZ co-twin means provides a test of genetic etiology. However, DeFries and Fulker (1985, 1988) proposed that a multiple regression analysis of such data, in which a co-twin’s score is regressed on both the proband’s score and the coefficient of relationship, facilitates a more flexible and statistically powerful test.

The basic DF model is as follows:

$$C = B_1P + B_2R + A \quad [1]$$

where C is the co-twin's score, P is the proband's score, R is the coefficient of relationship ($R = 1.0$ for MZ and 0.5 for DZ twin pairs), and A is the regression constant. The B_1 coefficient is the partial regression of the co-twin's score on the proband's score, a measure of the average MZ and DZ twin resemblance (DeFries & Fulker, 1985; 1988). The B_2 partial regression coefficient estimates the differential regression of co-twin scores by zygosity and equals twice the difference between the MZ and DZ co-twin means after covariance adjustment for any difference between the MZ and DZ proband means. Therefore, B_2 provides a direct test for genetic etiology. Moreover, when the data are appropriately transformed prior to multiple-regression analysis (i.e., each score is expressed as a deviation from the mean of the unselected population and then divided by the difference between the proband and population means), B_2 provides a direct estimate of heritability of the group deficit, h^2_g , an index of the degree to which the observed proband deficit is heritable.

In order to incorporate sibling data, a simple extension of the basic DF model can be simultaneously fitted to transformed data from selected twins, their co-twins and co-sibs. The expected transformed MZ and DZ co-twin means (see DeFries & Fulker, 1988) and the corresponding transformed co-sib means are presented in Table 1. From these expected values, it may be seen that the difference between the DZ co-twin mean and the co-sib mean is a simple function of the difference between shared environmental influences in twins versus sibling pairs.

Thus, to test for differential c^2_g between twins and siblings, the following extended basic model can be simultaneously fitted to transformed data from selected twins, their co-twins and co-sibs:

$$C = B_1P + B_2R + B_3S + A \quad [2]$$

where C is now the co-twin's or co-sib's score, P is the proband's score, R is the coefficient of relationship (1.0 for MZ pairs and 0.5 for both DZ pairs and twin/sib pairs), and S is a dummy code for pair type, i.e., twin pair versus twin-sibling pair ($+0.5$ for MZ twins, $+0.5$ for DZ twins and -0.5 for twin-sib pairs). When this model is fitted to the data and all three partial regression coefficients are estimated simultaneously, B_3 estimates the difference between $c^2_{g(t)}$ and $c^2_{g(s)}$ which equals the difference between the DZ co-twin (CDZ) and co-sib (CS) means, as can be observed in the Appendix. As a result, B_3 provides a direct test of significance for the difference between environmental influences shared by DZ twin pairs versus those shared by twin-sib pairs, i.e., a test for "special twin environment". As in the basic model, B_2 estimates h^2_g from twice the difference between the MZ and DZ co-twin means.

Because B_3 estimates the difference between $c^2_{g(t)}$ and $c^2_{g(s)}$, its significance is relevant for obtaining an estimate of h^2_g based upon an analysis of the combined twin and co-sibling data. If B_3 is small and not significant, S may be dropped from the extended model, and Equation 1 fitted to the combined data set of twins and siblings. In that case, B_2 will estimate h^2_g from both the twin and co-sib data, rather than from only the twin data. However, if B_3 is significant or relatively large, h^2_g should be estimated from fitting Equation 2 to the combined data set.

Because subjects were not reselected at follow-up, univariate DF models were fitted only to data from the initial assessment. Then, to assess the heritable nature of the *stability* of reading deficits a bivariate extension of the basic DF model was fitted to data from the initial and follow-up assessments as follows:

$$C_y = B_1 P_x + B_2 R + A \quad [3]$$

where C_y is the co-twin's or co-sib's composite reading score at follow-up, P_x is the proband's initial composite reading score, R is the coefficient of relationship, and A is the regression constant. B_1 is the partial regression of the co-twin's or co-sib's follow-up reading score on the proband's initial reading score and is a measure of average MZ and DZ cross-variable twin resemblance. Thus, B_1 estimates the extent to which co-twin and co-sib scores on the follow-up measure are related to proband scores on the initial measure across zygosity. B_2 is the partial regression of the co-twin's or co-sib's follow-up reading score on the coefficient of relationship. When the data are transformed prior to multiple-regression analysis, the bivariate B_2 coefficient is a function of the square roots of the group heritabilities for reading performance at the two time points and the genetic correlation (r_G) between them (i.e., $h_{\text{initial}} \times h_{\text{follow-up}} \times r_G$; Light & DeFries, 1995). Therefore, B_2 provides an estimate of "bivariate heritability" ($h^2_{g(Biv)}$), an index of the extent to which the proband reading deficit at follow-up is due to heritable factors which also influenced the reading deficit at the initial assessment. Further, the proportion of the phenotypic stability correlation (r_p) attributable to genetic influences can be obtained by dividing the B_2 estimate by r_p . Finally, when the following extended bivariate model is fitted to both twin and sibling data (Equation 4), B_3 provides a test of significance for the difference between bivariate shared environmental influences in twins and siblings:

$$C_y = B_1 P_x + B_2 R + B_3 S + A \quad [4]$$

Because truncate selection was employed (DeFries & Gillis, 1991), pairs in which both members met criteria for RD were double-entered for all regression analyses. This is analogous to the computation of probandwise concordance rates, in which both affected members of concordant pairs are included as probands. Standard error estimates and tests of significance were adjusted accordingly. Models were fit using linear regression in SPSS for UNIX server.

Results

Table 2 presents the standardized mean reading performance scores for MZ and DZ probands, as well as those of their co-twins and co-sibs at each assessment. The MZ and DZ proband means are highly similar at both initial and follow-up assessments (averaging about 2 standard deviations below the respective control means) suggesting that the deficit of the probands is highly stable. In addition, at each assessment there is a differential regression of the MZ co-twin, DZ co-twin and co-sib means toward the mean of the control twins. At the initial assessment, the MZ co-twin mean regressed 0.23 standard deviation units toward the control mean on average, whereas that of the DZ co-twin regressed 0.92 standard deviation units. In addition, the co-sib mean regressed 1.30 standard deviation units. Similarly, at follow-up the MZ co-twin mean regressed 0.26 standard deviation units toward the control mean on average, whereas the DZ co-twin and co-sib means regressed 0.75 and 1.00 standard deviation units suggesting that reading deficits are both stable and substantially due to heritable influences.

Corresponding transformed proband, co-twin and co-sib means are presented in Figure 1.

Table 3 presents results of the basic univariate regression analysis of twin-only data and twin-sibling data. When the basic model incorporating data from twins only (Equation 1) was fitted to the transformed proband and co-twin initial scores, the B_2 estimate was .67,

confirming that the proband reading deficit in this sample is due substantially to genetic influences. Similarly, as expected, when the extended model was fitted to data from both twins and siblings (Equation 2), the B_2 estimate was again .67, but slightly more significant. In contrast, B_3 was nonsignificant, but not trivial (.14). Therefore, although the more parsimonious model (Equation 1) was also fitted to the combined twin and sibling data for illustrative purposes, as shown in Table 3 the resulting estimate of h^2_g was biased upward (.79). However, if B_3 were nonsignificant and relatively small, fitting the more parsimonious model to the combined data set would be appropriate.

When the bivariate model (Equation 3) was fitted to the transformed proband initial scores and co-twin follow-up scores, the estimate of bivariate h^2_g was .59, suggesting that approximately 60% of the proband deficit in reading at follow-up is due to genetic factors that also influenced reading difficulties at the initial assessment. As expected, when the bivariate model was extended to include siblings (Equation 4), the resulting B_2 estimate was also .59, but more highly significant. Moreover, the ratio of B_2 to the observed correlation between initial and follow-up scores (.86) suggests that common genetic influences account for nearly 70% (.59/.86 = .69) of the stability of reading difficulties in this sample. However, the estimate of bivariate B_3 was relatively small (.08) and non-significant ($p = .415$). Table 3 also presents results of fitting the more parsimonious model to the longitudinal twin and sibling data, combining the data from DZ co-twins and co-siblings.

As shown in the appendix, estimates of both univariate and bivariate h^2_g and differential c^2_g may also be readily calculated from the transformed co-twin and co-sib means.

Discussion

Results obtained from previous studies have shown that reading deficits are stable and heritable; however, the genetic and environmental etiologies of the stability of reading deficits have not been well characterized. Therefore, the primary objectives of the present study were (1) to assess more rigorously genetic and environmental influences on the stability of reading deficits using data from a larger sample of twin pairs and their siblings tested in the CLDRC and re-tested approximately 5 years later in the LTSRD and (2) to fit a novel extension of the DeFries-Fulker multiple regression model (DeFries & Fulker, 1985) to reading performance data from both twins and siblings, potentially increasing power and facilitating a test for “special twin environments.”

In the current study, the average reading performance of the probands at their initial assessment was approximately two standard deviations below those of the controls, and this deficit persisted at follow-up. The stability correlation of probands was $.72 \pm .04$ and that for controls was $.75 \pm .03$. This result is consistent with those of previous studies that have found both reading deficits and reading performance within the normal range to be highly stable (e.g., Bast & Reitsma, 1998; DeFries, 1988; DeFries & Baker, 1983; Hulslander, et al., 2010; Shaywitz et al., 1992; Wadsworth et al., 2006; Wadsworth et al., 2007; Wagner et al., 1997).

Results of univariate DF analysis of data from twins and siblings at their initial assessment suggested that about two-thirds of the proband deficit in this sample was due to genetic influences. Because subjects were not reselected at follow-up, univariate DF analyses were not conducted for follow-up data. When the bivariate extension of the multiple regression model was fitted to proband scores at initial assessment and co-twin/co-sib scores at follow-up, combining DZ co-twin and co-sibling status, the resulting estimate of bivariate heritability was $0.66 (\pm .20, p = .001)$, suggesting that about two-thirds of the proband deficit at follow-up was due to genetic influences which also influenced reading deficits at

the initial assessment. Further, common genetic influences accounted for approximately 70% of the observed stability between reading scores at the initial and follow-up assessments.

These results are highly consistent with our previous longitudinal analysis of the stability of reading deficits which obtained an estimate of bivariate heritability of $.65 (\pm .32)$ and found that about 75% of the stability of reading difficulties between initial and follow-up assessments was due to common genetic influences (Astrom et al., 2007). However, our previous study included only twins. Results of the current study suggest that including sibling data increases power at least to some extent. For example, given the effect size estimated from the basic univariate model and sample of 97 twin pairs, power to detect significance of the B_2 term, i.e., h^2_g , is about .85. However, when the 44 siblings are also included and the basic model is fitted to the combined data sets, power improves to .97. Similarly, power for the bivariate model using only twin data is .79; including the sibs, assuming the same parameters, power is increased to about .95. Because the basic DF twin model is quite powerful, even with relatively small samples, the addition of sibling data may be especially beneficial when fitting more complex models, such as those testing hypotheses of differential genetic etiology.

The similarity of results from Astrom et al. (2007) and the current study may be expected as there is considerable overlap between the two samples. However, previous studies of individual differences in reading performance also support these findings. For example, Byrne et al. (2007) found that 90% of the observed stability of word reading in twin pairs between kindergarten and grade one was due to common genetic influences. Results from the TEDS study (Harlaar et al., 2007) were also highly similar with more than two-thirds of the phenotypic stability being genetically mediated. Although Petrill et al. (2007) reported somewhat lower estimates (at most, 42% of the phenotypic stability was due to genetic influences), their study examined the genetic variance of individual subtests of several different measures utilizing Cholesky decomposition models. Our findings are also highly similar to results from the Colorado Adoption Project (Wadsworth et al., 2006) in which data from related and unrelated sibling pairs were analyzed, and 86% of the phenotypic correlation between reading performance at ages 7 and 16 was accounted for by genetic influences. These remarkably similar results across different studies with different types of subjects, ages, measures and analytical methods suggest that this is a highly robust finding.

The extended DF model for analyzing data from both twins and siblings also provides a test for “special twin environments” (e.g., Koeppen-Schomerus et al., 2003; Medland et al., 2003; Van Grootheest et al., 2007; Young et al., 2006). In fact, B_3 provides a direct estimate of the difference between shared environmental influences for members of twin pairs versus those for less contemporaneous twin/co-sibling pairs. Moreover, its magnitude and significance indicate whether the estimate of h^2_g should be based upon an analysis of the combined twin and co-sibling data. If B_3 is small and not significant, S may be dropped from the extended model, and Equation 1 fitted to the combined data from twins and siblings. However, if B_3 is significant or relatively large, h^2_g should be estimated from fitting Equation 2 to the data set. In the current study, no significant differences were found between twin and sibling shared environmental influences. However, the parameter estimate of .14 for differential c^2_g in the univariate model is not trivial. Consequently, when the B_3 term was dropped from the extended twin/sib model, the resulting estimate of h^2_g was inflated. Thus, results of fitting the more parsimonious model (Equation 1) to the combined twin and sibling data are presented for illustrative purposes only.

Finally, although our findings suggest that reading deficits are highly stable and that this stability is due principally to genetic influences, this should not deter our best efforts with

regard to environmental intervention and remediation. There are multiple pathways to poor reading (Gilger & Kaplan, 2001), and the relative contributions of genetic and environmental influences may differ depending on the measure (Gayán & Olson, 2001). Reading disabilities do not generally segregate in families in a simple Mendelian fashion; thus, multiple genetic and environmental factors almost certainly influence reading abilities and may also interact (e.g., Friend, DeFries, & Olson, 2008). Nevertheless, our results suggest that intensive remediation efforts may be needed to compensate for genetic and other biological constraints on learning rates (Olson, et al., in press, a). Therefore, in order to identify and treat at risk children more effectively and account for their individual abilities and disabilities, additional longitudinal studies of the genetic and environmental etiologies of reading disability are clearly warranted (Olson, et al., in press, b).

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Figure 1.
Mean proband, co-twin and co-sib transformed reading scores at initial and follow-up assessments.

Table 1Expected transformed^{*l*} co-twin and co-sib means

Subjects	Model
MZ Co-twins	$h_g^2 + c_{g(t)}^2$
DZ Co-twins	$\frac{1}{2}h_g^2 + c_{g(t)}^2$
Co-sibs	$\frac{1}{2}h_g^2 + c_{g(s)}^2$

^{*l*} Scores are expressed as a deviation from the unselected population mean and then divided by the difference between the proband and population means (see DeFries & Fulker, 1988).

Table 2
Mean standardized reading performance scores ($\pm$ SD) of probands, co-twins and co-sibs at initial¹ and follow-up test sessions²

	Initial					
	MZ pairs		DZ pairs		Twin/sib pairs	
	M	SD	M	SD	M	SD
<i>Proband</i>	-2.15	$\pm .95$	-2.11	$\pm .87$	-2.24	$\pm .95$
<i>Co-twin/sib</i>	-1.92	± 1.17	-1.19	± 1.39	-0.94	± 1.22
	Follow-up					
	M	SD	M	SD	M	SD
<i>Proband</i>	-2.00	$\pm .67$	-1.93	$\pm .64$	-2.14	$\pm .70$
<i>Co-twin/sib</i>	-1.74	$\pm .99$	-1.18	± 1.16	-1.14	± 1.08

¹ Standardized against the mean of 284 Control twins participating in the initial assessment in the CLDRC.
² Standardized against the mean of 171 Control twins participating in the LTSRD at follow-up.

Table 3
Comparison of twin and twin-sibling results of univariate and bivariate DF analysis

Model	Subjects	Model	$B_2 \pm \text{S.E.}$	p	$B_3 \pm \text{S.E.}$	p
Univariate	Twins only	$C = B_1P + B_2R + A$	$.67 \pm .23$	$= .004$	----	----
	Twins & siblings	$C = B_1P + B_2R + B_3S + A$	$.67 \pm .22$	$= .003$	$.14 \pm .10$	.167
	Twins & siblings ^I	$C = B_1P + B_2R + A$	$.79 \pm .20$	$\leq .001$	----	----
Bivariate	Twins only	$C_y = B_1P_x + B_2R + A$	$.59 \pm .22$	$= .008$	----	----
	Twins & siblings	$C_y = B_1P_x + B_2R + B_3S + A$	$.59 \pm .21$	$= .006$	$.08 \pm .10$	.415
	Twins & siblings ^I	$C_y = B_1P_x + B_2R + A$	$.66 \pm .20$	$= .001$	----	----

^I Ignoring DZ co-twin versus co-sibling status

Appendix

Parameter estimates calculated from the expectations in Table 1 and the transformed co-twin and co-sib means

Univariate	
Transformed means	
MZ Co-twin mean (CMZ)	$h_g^2 + c_{g(t)}^2 = .8947$
DZ Co-twin mean (CDZ)	$\frac{1}{2}h_g^2 + c_{g(t)}^2 = .5620$
Co-sib mean (CS)	$\frac{1}{2}h_g^2 + c_{g(s)}^2 = .4199$
	$h_g^2 = 2(\text{CMZ} - \text{CDZ}) = 2(.8947 - .5620) = .6654$
Differential c_g^2	
$c_{g(MZ)}^2$	$(\text{CMZ} - h_g^2) = (.8947 - .6654) = .2293$
$c_{g(DZ)}^2$	$(\text{CDZ} - \frac{1}{2}h_g^2) = (.5620 - .3327) = .2293$
$c_{g(s)}^2$	$(\text{CS} - \frac{1}{2}h_g^2) = (.4199 - .3327) = .0872$
B_3	$c_{g(t)}^2 - c_{g(s)}^2 = (.2293 - .0872) = .1421$
Bivariate ¹	
Transformed means	
MZ Co-twin mean (CMZ)	$h_{g(biv)}^2 + c_{g(biv)(t)}^2 = .7934$
DZ Co-twin mean (CDZ)	$\frac{1}{2}h_{g(biv)}^2 + c_{g(biv)(t)}^2 = .4989$
Co-sib mean (CS)	$\frac{1}{2}h_{g(biv)}^2 + c_{g(biv)(s)}^2 = .4203$
	$h_{g(biv)}^2 = 2(\text{CMZ} - \text{CDZ}) = 2(.7934 - .4989) = .5890$
Differential c_g^2	
$c_{g(biv)(t)}^2$	$(\text{CMZ} - h_{g(biv)}^2) = (.7934 - .5890) = .2044$
$c_{g(biv)(t)}^2$	$(\text{CDZ} - \frac{1}{2}h_{g(biv)}^2) = (.4989 - .2945) = .2044$
$c_{g(biv)(s)}^2$	$(\text{CS} - \frac{1}{2}h_{g(biv)}^2) = (.4203 - .2945) = .1258$
B_3	$c_{g(biv)(t)}^2 - c_{g(biv)(s)}^2 = (.2044 - .1258) = .0786$

¹ Co-twin and co-sib mean reading scores at follow-up are transformed based on the proband mean at initial assessment.