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Genetic and environmental etiologies of reading difficulties: DeFries-Fulker analysis of reading performance data from twin pairs and their nontwin siblings

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Abstract

Reading performance data from 254 pairs of identical (MZ) and 420 pairs of fraternal (DZ) twins, 8.0 to 20.0 years of age, were subjected to multiple regression analyses. An extension of the DeFries-Fulker (DF) analysis (DeFries & Fulker, 1985, 1988) that facilitated inclusion of data from 303 of their nontwin siblings was employed. In addition to providing estimates of heritability, this analysis yields a test of the difference between shared environmental influences for twins versus siblings (Astrom et al., 2011). Results suggest that proband reading deficits are due substantially to genetic factors ($.67 \pm .07$, $p < .001$), and that shared environmental influences are significantly higher for members of twin pairs than for those of twins and their nontwin siblings (viz., .25 versus .17, $p = .02$).

Keywords

DF analysis; twins; siblings; reading; heritability

1. Introduction

The heritable nature of reading difficulties has long been established (e.g., DeFries et al., 1991; DeFries and Alarcón, 1996; Harlaar et al., 2005; Stevenson et al., 1987; Wadsworth et al., 2007). Early twin studies compared concordance rates in pairs of identical and fraternal twins as a test for genetic etiology. A pair is considered concordant if both members are affected with the same disorder or discordant if only one member of the pair is affected. Because members of MZ twin pairs are genetically identical, while DZ pairs share, on average, only half of their segregating genes, MZ concordance is expected to be greater than DZ concordance if a condition is heritable. Such differences in MZ and DZ concordance rates were obtained in several early studies of reading disability (Bakwin, 1973; Stevenson et al., 1987; Zerbin-Rudin, 1967; Decker & Vandenberg, 1985).

Although a comparison of concordance rates in MZ and DZ twin pairs provides evidence for a genetic etiology, reading disability is diagnosed in part on the basis of quantitative measures with somewhat arbitrary cut-off points (Stevenson, et al., 1987). Thus, when a continuous measure, such as reading performance, is transformed into a categorical variable (e.g., reading disabled versus non-reading disabled) information pertaining to the range of

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variation in reading performance is inevitably lost. Consequently, DeFries and Fulker (1985, 1988) proposed fitting a multiple regression model in which a co-twin's score is predicted from the proband score and the coefficient of relationship (1.0 for MZ, 0.5 for DZ twins) to data from selected twin pairs to assess genetic influences on deviant scores. Based on the differential regression of MZ and DZ cotwin means to the mean of the unselected population, this method accounts for variation in continuous variables (e.g., reading performance). Using this method, an early study from the Colorado Learning Disabilities Research Center (CLDRC; DeFries et al., 1997) assessed the genetic etiology of reading disability in a sample of 191 MZ, 143 same-sex DZ, and 99 opposite-sex DZ twin pairs in which least one member of the pair was classified as reading disabled. Although the MZ and same-sex DZ proband means were highly similar, the MZ co-twin mean regressed only 0.20 standard deviation units on average toward the control mean, whereas that of the DZ co-twins regressed 0.94. When the basic regression model (DeFries & Fulker, 1985, 1988) was fitted to the transformed data, h^2_g (an index of the extent to which reading deficits are due to genetic influences) was 0.56 (p < .001), suggesting that more than half of the average reading performance deficit of probands was due to heritable influences. Results obtained from subsequent analyses of data from twin pairs ascertained for reading difficulties have been highly similar (e.g., Harlaar et al., 2005; Harlaar et al., 2007; Wadsworth et al., 2010).

DeFries-Fulker (DF) analysis is powerful and flexible. It has been adapted to test for differential heritability as a function of covariates such as IQ (Knopik et al., 2002; Wadsworth et al., 2000, 2010), or gender (Hawke et al., 2007; Wadsworth et al., 2005), as well as for bivariate analyses (e.g., Light et al., 1995), including longitudinal applications (Astrom et al., 2007, 2011), and for analysis of quantitative trait loci (Fulker et al., 1991). The DF method has become a cornerstone of behavior genetic analysis.

Most recently, DF analysis has been adapted to accommodate data from both twins and their non-twin siblings. In a recent application of the bivariate longitudinal extension of the DF model, Astrom et al. (2011) investigated the etiology of the stability of reading deficits using a novel extension of the DF method which incorporates sibling data and facilitates a 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). The sample included 33 MZ and 64 DZ twin pairs, and 44 of their nontwin siblings, who participated in the Longitudinal Twin Study of Reading Disability (LTSRD) approximately five years after their initial participation in the CLDRC. In order to incorporate sibling data, a simple extension of the basic DF model was employed in which the co-twin or co-sib score was predicted from the proband score and the coefficient of relationship (1.0 for MZ, 0.5 for DZ twins and siblings) and a dummy-coded variable to differentiate data from twin pairs and twin-sibling pairs. The model was simultaneously fitted to transformed data from selected twins, their co-twins and co-sibs. Results of fitting the basic DF model to twin data from the initial assessment yielded an h^2_g estimate of .67 (p = .004), indicating that the proband deficit in this sample was due principally to genetic influences. When the extended DF model was fitted to both twin and sibling data, a measure of the extent to which shared environmental influences for members of twin pairs differ from those for twin-sibling pairs (i.e., $c^2_{g(t)} - c^2_{g(s)}$ = .14) was non-significant (p = .167), but not trivial. Results of the bivariate DF analysis indicated that 70% of the observed stability was due to genetic influences.

In the CLDRC, data have been collected from siblings of approximately half of the MZ and DZ probands. Using the novel application of DF multiple regression analysis described by Astrom et al. (2011), we have included sibling data in the present analysis. Thus, the primary objectives of the present study were twofold: (1) to examine the etiology of reading disability using the full sample of CLDRC twin pairs; and (2) to estimate the heritability of

reading deficits in the full CLDRC sample using a novel extension of the DF method (DeFries-Fulker, 1985, 1988) which incorporates sibling data and facilitates a test for "special twin environments" (e.g., Koeppen-Schomerus et al., 2003; Medland et al., 2003; VanGrootheest et al., 2007; Young et al., 2006). We expect that results will support previous findings of substantial and significant genetic influences on reading deficits and that the test for special twin environments will be significant in this much larger sample.

2. Methods

2.1. Participants and Measures

Subjects in the present study were tested in the ongoing CLDRC (DeFries et al. 1997). Twin pairs were systematically identified through 27 different school districts within the state of Colorado. Pairs in which at least one member demonstrated a history of reading problems were invited to participate in the study at the University of Colorado, Boulder, and at the University of Denver (For a complete description of subject ascertainment, please see Astrom et al., 2011). The subjects were administered an extensive battery of psychometric tests which included 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 were used to compute a discriminant function score (DISCR) for each subject (DeFries, 1985). In order for an individual to be included in the current proband sample, he or she must have a positive history of reading problems and be classified as reading-disabled by the discriminant function score. Additional selection criteria include a minimum IQ score of 85 on either the Verbal or Performance Scale of the WISC-R or WAIS-R, no evidence of neurological, serious emotional or behavioral problems, and no uncorrected visual or auditory acuity deficit. Control twin pairs are matched to probands on the basis of age, gender, and school district, and both members of the pair must have a negative history of reading problems. Zygosity of same-sex twin pairs is established using selected items from the Nichols and Bilbro (1966) questionnaire which has a reported accuracy of 95%. In ambiguous cases, zygosity is determined by analysis of blood or buccal samples. All variables were age-adjusted prior to inclusion in the analyses.

The current sample included 254 MZ pairs (128 male and 126 female), and 420 DZ pairs (131 same-sex male, 138 same-sex female, and 151 opposite-sex) in which at least one twin met proband criteria (average age 11.23 years), and 303 of their non-twin siblings (175 male and 128 female, average age 13.37 years). For standardization and transformation of the variables, the control sample comprised 728 twin pairs. Informed consent and assent was obtained and the study protocol was approved by the Institutional Review Board of the University of Colorado, Boulder.

2.2. Analyses

When probands have been ascertained because of extreme scores on a continuous measure, the scores of their co-twins are expected to regress toward the mean of the unselected population. To the extent that the trait is heritable, this regression to the mean should differ for the MZ and DZ co-twins (see Figure 1). Thus, when 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, multiple regression analysis facilitates a more flexible and statistically powerful test for genetic etiology (DeFries & Fulker, 1985, 1988). The basic DF model is as follows:

$$C = B_1 P + B_2 R + A \qquad [1]$$

where C is the co-twin's score, P is the proband's score, R is the coefficient of relationship (1.0 for MZ and 0.5 for DZ twin pairs), and A is the regression constant. When the basic model is fitted to selected twin data, B_1 is a measure of the average MZ and DZ twin resemblance. B_2 estimates twice the difference between the means of MZ and DZ co-twins after covariance adjustment for any difference between the means of the MZ and DZ probands. Thus, B_2 provides a test for genetic etiology which is more general and statistically powerful than a comparison of concordance rates. 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 extent to which the deficit of the probands is due to genetic influences (DeFries & Fulker, 1985; 1988).

To incorporate sibling data, the following extended basic model can be simultaneously fitted to transformed data from probands, their co-twins and co-sibs (Astrom et al., 2011):

$$C=B_1P+B_2R+B_3S+A \tag{2}$$

where C is now the co-twin's or co-sib's score, R is the coefficient of relationship (1.0 for MZ pairs and now, 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. When this model is fitted to the data, B_3 estimates the difference between the DZ co-twin (CDZ) and co-sib (CS) means and, therefore, provides a direct test of significance for the difference between environmental influences shared by members of DZ twin pairs ($c^2_{g(t)}$) and those of twin-sib pairs ($c^2_{g(s)}$) As in the basic model, B_2 estimates h^2_g, derived only from the twin data.

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 non-significant, S may be dropped from the extended model, and Equation 1 may be fitted to the combined data set of twins and siblings. In such cases, B_2 will estimate h^2_g from both the twin *and* co-sib data, and not only the twin data. Conversely, if B_3 is significant or relatively large, h^2_g should be estimated from fitting Equation 2 to the combined data set.

Because truncate selection was employed (DeFries & Gillis, 1991), pairs in which both members met criteria for reading disability were double-entered. 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 significance were adjusted accordingly.

3. Results

Mean reading performance scores for MZ and DZ probands, their co-twins and co-sibs, expressed as standard deviation units from the mean of the control twins, are presented in Table 1. The MZ and DZ proband means are highly similar (approximately 2.5 standard deviations below the control mean). Furthermore, there is a differential regression of the MZ co-twin, DZ co-twin and co-sib means toward the mean of the control twins. The MZ co-twin mean regressed 0.21 standard deviation units toward the control mean, whereas those of the DZ co-twins and co-sibs regressed 1.02 and 1.25 standard deviation units, respectively.

Corresponding transformed proband, co-twin and co-sib means are presented in Figure 2.

Results of fitting Equations 1 and 2 to the twin-only data and twin-sibling data are presented in Table 2. When the basic model (Equation 1) was fitted to the transformed proband and co-twin 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 also .67. However, the B_3 coefficient, while relatively small, was significant (.08 ± .04, p = . 02), suggesting that shared environmental influences for members of twin pairs are greater than those of the less contemporaneous twin/nontwin sibling pairs. Although B_3 could not be dropped from the model, results of fitting the more parsimonious model (Equation 1) to the combined twin and sibling data are presented in Table 2 for illustrative purposes. As expected, since $c^2_{g(t)}$ is significantly larger than $c^2_{g(s)}$, the estimate of h^2_g (.74 ± .06) is substantially larger than when Equation 2 was fitted to those data (.67 ± .07).

As illustrated in the Appendix, estimates of h^2_g and differential c^2_g may also be readily calculated from the transformed co-twin and co-sib means. Given obtained estimates of h^2_g, it may be seen that $c^2_{g(t)}$ = .25 for members of MZ and DZ twin pairs, $c^2_{g(s)}$ = .17 and B_3 = . 25–.17 = .08, the difference between shared environmental influences for members of twin pairs versus twin/sib pairs.

4. Discussion

The primary goals of the present study were to assess genetic influences on reading difficulties using data from the full CLDRC sample, and to fit a novel extension (Astrom et al., 2011) of the basic DF multiple regression model (DeFries & Fulker, 1985, 1988) to reading performance data from both twins and their nontwin siblings, thereby assessing "special twin environments." The current sample of reading-disabled twin pairs and siblings tested in the ongoing CLDRC is much larger than that previously analyzed by Astrom et al. (2011), providing more rigorous tests of both the etiology of reading deficits and of special twin environments.

When the basic model (Equation 1) was fitted to transformed reading performance data from MZ and DZ twin pairs with reading difficulties, h^2_g was estimated at .67. This result, similar to those of previous studies, suggests that about two-thirds of the reading deficit of the probands is due to heritable influences. When the extended model (Equation 2) was fitted to data from twins and siblings, B_3 was small but significant (B_3 = .08 ± .04, p = .02), suggesting that members of twin pairs share more environmental influences related to reading deficits than do members of twin-sibling pairs.

Although the B_2 estimate from Equation 2 (.67) in the present study was the same as that obtained when Equation 1 was fitted to twin data only, the standard error of the B_2 term estimated from Equation 2 was slightly larger than that for Equation 1. To rule out the possibility that this increased standard error was due to multicollinearity of the independent variables in our models, tolerance levels were examined and found to be within acceptable range (i.e., .82 for the addition of the B_3 term to the model).

This result differs from that of Astrom et al. (2011), which had suggested that the addition of sibling data improves power. Our present results indicate that this is not always the case. In fact, inclusion of data from siblings in twin studies may result in a reduction in power because the multiple R-squared may be decreased if data from pairs of relatives with less familial resemblance are included in the analysis (Zieleniewski et al, 1987). However, the basic DF analysis of extreme scores and the extension described here are quite powerful. For example, in our current sample, power to detect significance of the B_2 term (h^2_g) was 1.00 both with and without sibling data, even with an alpha level of .001. Given these same parameters and a sample half this size, power is still greater than .90 in both cases. Further,

power may increase when B_3 is small and Equation 1 is fitted to combined twin and sibling data, and this effect may be magnified in small samples. For example, in a sample of 100 twin pairs, given an h^2_g estimate of .67, such as that obtained in the current study, a B_3 estimate near zero at .01, a change in R^2 of .10, and $\alpha = .001$, power to detect significance of h^2_g is .65. Given these same parameters, when the sample size is increased with the addition of data from 50 twin-sibling pairs, power increases to .84. This is consistent with the power estimates previously reported by Astrom et al. (2011).

In conclusion, results of the current study provide further confirmation of substantial genetic influences on reading deficits. In addition, results of fitting the extended DF model to data from twin pairs and their siblings suggest that members of twin pairs share more environmental influences related to reading deficits than do members of twin-sibling pairs. Additional studies including detailed measures of the home and school environments are needed to establish the nature of these shared environmental influences. Further, DF analyses, both with and without the inclusion of sibling data are quite powerful. However, the effect of sibling data on the power of twin analyses of proband deficits has not been established. Therefore, more comprehensive power analyses of including sibling data in twin analyses of proband deficits are clearly warranted.

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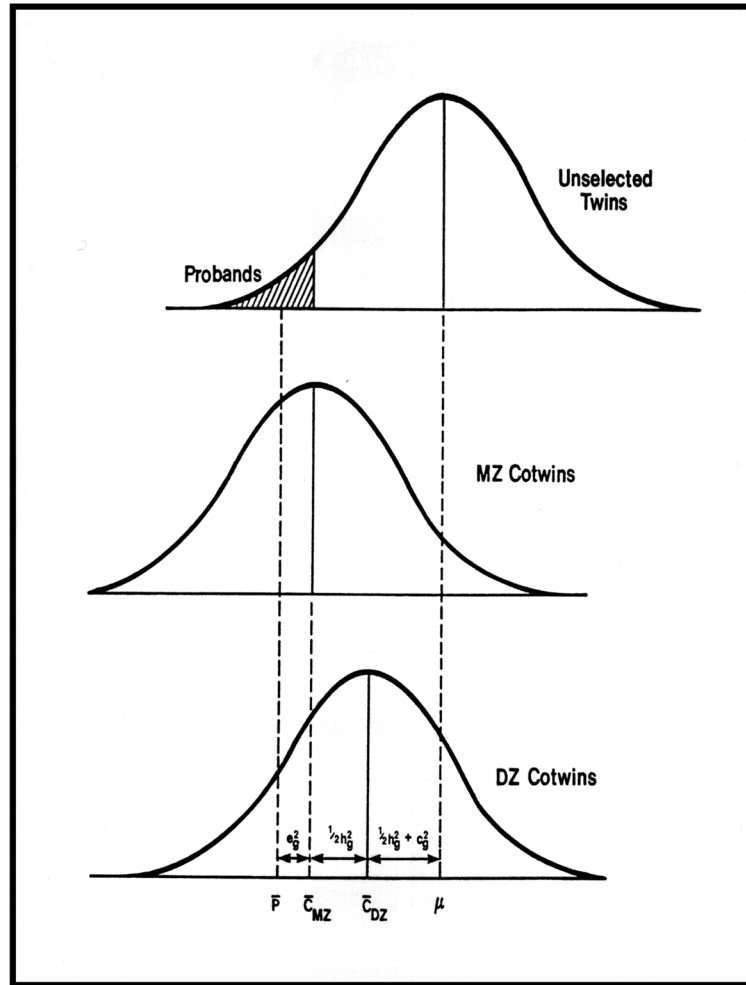
Figure 1.
Hypothetical distributions of reading performance of an unselected sample of twins (with mean μ) and of the identical (MZ) and fraternal (DZ) co-twins of probands with a reading disability. Proband and co-twin means are symbolized $\bar{P}$ and $\bar{C}$, respectively. The deficit of probands ($\bar{P} - \mu$) is due to heritable influences (h^2_g) and to environmental influences that are either shared (c^2_g) or not shared (e^2_g) by members of twin pairs. The differential regression of the MZ and DZ co-twin means toward the mean of the unselected population (μ) provides a test of genetic etiology (After DeFries and Fulker, 1988).
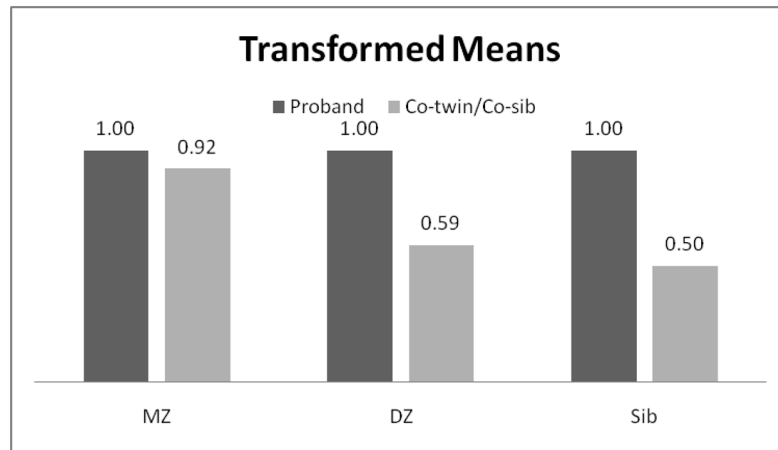
Figure 2.
Transformed proband, and co-twin and co-sib means.

Table 1

Mean standardized reading performance scores (± SD) of probands, co-twins and co-sibs

	MZ		DZ		Twin/sib	
	M	SD	M	SD	M	SD
Proband	−2.529	.766	−2.463	.854	−2.525	.811
Co-twin/co-sib	−2.324	.973	−1.442	1.336	−1.272	1.391
N pairs	254		420		303	

Table 2

Comparison of twin and twin-sibling results of DF analysis

Subjects	Model	$B_2 \pm$ S.E.	p	$B_3 \pm$ S.E.	p
Twins only	$C = B_1P + B_2R + A$	$.667 \pm .067$	.001	----	----
Twins & siblings	$C = B_1P + B_2R + B_3S + A$	$.667 \pm .070$	.001	$.082 \pm .035$	.020
Twins & siblings[1]	$C = B_1P + B_2R + A$	$.736 \pm .063$	.001	----	----

[1]Ignoring DZ co-twin versus co-sibling status

Appendix

Expected transformed[1] co-twin and co-sib means and parameter estimates

Expectations and parameter estimates
Transformed means
MZ Co-twin mean (CMZ) $h^2_g + c^2_{g(t)} = .9190$
DZ Co-twin mean (CDZ) $\frac{1}{2}h^2_g + c^2_{g(t)} = .5856$
Co-sib mean (CS) $\frac{1}{2}\, h^2_g + c^2_{g(s)} = .5039$
$h^2_g = 2(\text{CMZ}-\text{CDZ}) = 2(.9190 - .5856) = .6668$
Differential c^2_g
$c^2_{g(MZ)}$ $(\text{CMZ} - h^2_g) = (.9190 - .6668) = .2522$
$c^2_{g(DZ)}$ $(\text{CDZ} - \frac{1}{2}\, h^2_g) = (.5856 - .3334) = .2522$
$c^2_{g(s)}$ $(\text{CS} - \frac{1}{2}\, h^2_g) = (.5039 - .3334) = .1705$
B_3 $c^2_{g(t)} - c^2_{g(s)} = (.2522 - .1705) = .0817$