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Etiology of covariation between reading and mathematics performance: a twin study

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The etiology of the observed relationship between reading and mathematics performance was examined by analyzing data from samples of same-sex twin pairs tested in the Colorado Learning Disabilities Research Center. Bivariate phenotypic and genetic structural equation models were fitted to data from 526 twin pairs selected for reading deficits (290 identical and 236 same-sex fraternal) and 355 control pairs (220 identical and 135 same-sex fraternal). Subtests of the Peabody Individual Achievement Test (PIAT; Reading Recognition, Reading Comprehension, and Spelling) were used as measures of reading performance, and scores from the Wechsler Intelligence Scale for Children-Revised (WISC-R) or Wechsler Adult Intelligence Scale-Revised (WAIS-R) Arithmetic subtest, the Wide Range Achievement Test Arithmetic subtest, and the PIAT Math subtest were used as indices for mathematics performance. The results of these confirmatory factor analyses indicate that genetic and environmental covariances between reading and math latent factors do not differ significantly for twin pairs in the proband and control groups. Estimates of heritability for reading performance in the proband and control samples were 0.81 and 0.69, respectively, and those for math performance were 0.88 and 0.67, respectively. Moreover, genetic influences accounted for 83% of the covariation between the reading and math factors in the proband group and for 58% of the covariation between these two latent variables in the control group; in contrast, shared environmental influences did not contribute significantly to the relationship between the reading and math latent factors nor to their independent variation.

Keywords: genetics, math performance, reading performance, structural equation modelling, twin studies

Reading and mathematics deficits frequently co-occur. For example, Badian[1] reported that 56% of elementary and junior high school children with reading disability (RD) also manifested poor math achievement, and that 43% of the children with math disability (MD) also had reading difficulties. Subsequently, Rourke[2] noted that children with reading and/or spelling disability have difficulty remembering mathematical tables or remembering a particular step in the correct procedure for solving a problem and tend to avoid problems that require the reading of printed words. Thus, deficits in reading and math could both be due in part to language-based deficits or problems with short-term memory.[3,4]

The genetic and environmental etiologies of the comorbidity between reading and mathematics deficits were first examined by Light and DeFries.[5] Data from a sample of twin pairs selected for reading deficits were fit to a bivariate extension of the basic multiple regression model for the analysis of selected twin data (DeFries and Fulker[6,7]). The resulting estimate of bivariate heritability (an index of the extent to which the proband reading deficit is due to genetic factors that also influence math disability) was 0.26 ± 0.11, suggesting that proband reading deficits are due at least in part to genetic factors that also influence math performance. Moreover, the ratio of bivariate heritability to the standardized covariance between reading and mathematics performance indicated that over half (0.55) of their observed covariance was due to genetic influences.

Knopik et al[8] subsequently employed this methodology to assess the etiology of the comorbidity of reading and mathematics deficits in two twin samples: one selected for reading deficits and one selected for math deficits. Results of these analyses provided additional evidence for the heritable nature of reading and math deficits, and also suggested that the observed comorbidity between reading and math difficulties is due substantially to genetic influences.

In order to assess the etiology of the covariation between individual differences in reading and mathematics performance, Gillis et al[9] conducted a confirmatory factor analysis on data from twin pairs tested in the Colorado Twin Study of Reading Disability. Reading performance was measured by

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the Peabody Individual Achievement Reading Recognition, Reading Comprehension, and Spelling subtests, whilst mathematics performance was indexed by the Peabody Individual Achievement Math subtest, the Wechsler Arithmetic subtest, and the Primary Mental Abilities Spatial Test. Individual differences in reading and mathematics performance were found to be highly heritable in both a sample of twins with reading disability (heritability = a^2 = 0.78 and 0.51 for reading and math, respectively) and in twin pairs with no learning difficulties (a^2 = 0.74 and 0.60, respectively). Moreover, results of this confirmatory factor analysis also suggested that genetic influences account for almost all (98%) of the observed correlation between reading and math performance within the sample of twins with reading disability, and for 55% of the observed correlation in the control sample. Therefore, it was concluded that individual differences in both reading and mathematics performance are highly heritable and appear to be caused by many of the same genetic influences (Gillis et al[9]). These results are highly similar to those of a twin study of scholastic achievement by Thompson et al[10] in which the genetic correlation between reading and math performance was 0.98.

The primary objective of the present study is to assess the etiology of the covariation between individual differences in reading and mathematics performance by analyzing data from two samples of twins tested in the Colorado Learning Disabilities Research Center (DeFries et al[11]): an updated sample of twins ascertained for reading deficits and previously analyzed by Gillis et al[9]; and a control sample of twins without learning difficulties. By fitting a bivariate (two-factor) structural equation model (Neale and Cardon[12]) to the twin data, the proportions of phenotypic variance due to additive genetic, shared environmental, and non-shared environmental influences were estimated for reading and mathematics performance in both samples. In addition, the phenotypic correlation between the two factors was partitioned into components due to genetic, shared environmental, and non-shared environmental influences.

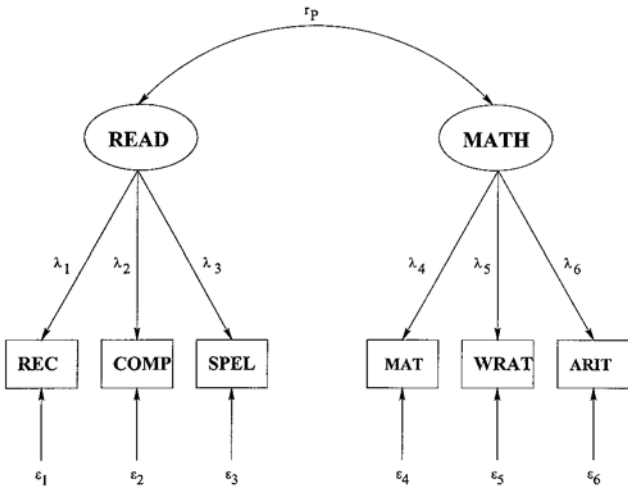

Figure 1 Path diagram for bivariate phenotypic model

Methods

Participants and measures

The data for this analysis were collected from twin pairs tested by 31 August 1998 in the Colorado Learning Disabilities Research Center, which ascertains twin pairs from 27 school districts in the state of Colorado. In order to minimize the possibility of ascertainment bias, all twin pairs in a school were identified, without regard to reading status. Parental permission was then sought to review the twins' school records for evidence of reading problems (eg referral to a reading therapist, low reading achievement test scores). Twin pairs in which at least one member of the pair exhibited a positive school history of reading problems and a comparison sample of twins with no learning difficulties were invited to laboratories at the University of Colorado to complete an extensive battery of psychometric tests measuring various cognitive abilities. This battery included the Wechsler Intelligence Scale for Children – Revised (WISC-R, Wechsler[13]) or the Wechsler Adult Intelligence Scale – Revised (WISC-R, Wechsler[14]), the Peabody Individual Achievement Test (PIAT, Dunn and Markwardt[15]), and the Wide Range Achievement Test (WRAT, Jastak and Wilkinson[16]). Complete data were available on all measures with the exception of WRAT Arithmetic, which was added to the test battery at a later date. Scores for each of these variables were age-adjusted using regression deviation scores and then standardized by expressing each subject's score as a deviation from the corresponding control mean and dividing by the control standard deviation. The PIAT Reading Recognition (REC), Reading Comprehension (COMP), and Spelling (SPEL) subtests were used as measures of reading performance, whilst PIAT Mathematics (MAT), WISC-R or WAIS-R Arithmetic (ARIT), and WRAT Arithmetic (WRAT) subtests were used as indices of mathematics performance.

Pairs were included in the reading disabled (RD) sample if at least one member exhibited a positive school history of reading problems. The control sample was matched, when possible, to the RD sample on the basis of age, gender and zygosity. However, both members of control pairs have a negative school history for reading problems.

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Because of the possibility of sex-limitation for these measures (Knopik et al[17]), the present study incorporates only those data obtained from same-sex twin pairs.

All twin pairs included in this analysis were raised in English-speaking, predominantly middle-class homes, and ranged in age from 8 to 20 years, with the RD and control samples both having a mean age of 11.73 years. Zygosity of the same-sex twin pairs was determined by selected items from the Nichols and Bilbro[18] questionnaire, which has a reported accuracy of 95%. In cases of doubtful zygosity, blood or buccal samples were analyzed. As of 31 August 1998, the RD sample included a total of 290 pairs of MZ twins and 236 pairs of same-sex DZ twins. A total of 220 MZ twin pairs and 135 same-sex DZ pairs were included in the control sample.

Analyses

The bivariate phenotypic and genetic structural equation models, and their corresponding submodels, were fitted to the twin data using the Mx statistical modelling package (Neale[19]). Because of the variability in patterns of missing data for the WRAT Arithmetic subtest, all available raw data were analyzed by creating a variable length record and using the following fit function (Neale[19]):

$$RM = -k \log(2\pi) + \log|\Sigma| + (x_i - \mu_i)'\Sigma^{-1}(x_i - \mu_i),$$

where RM is the raw maximum likelihood. The appropriate mean vector μ and covariance matrix Σ are automatically created by Mx for each observation. In addition, this procedure calculates twice the

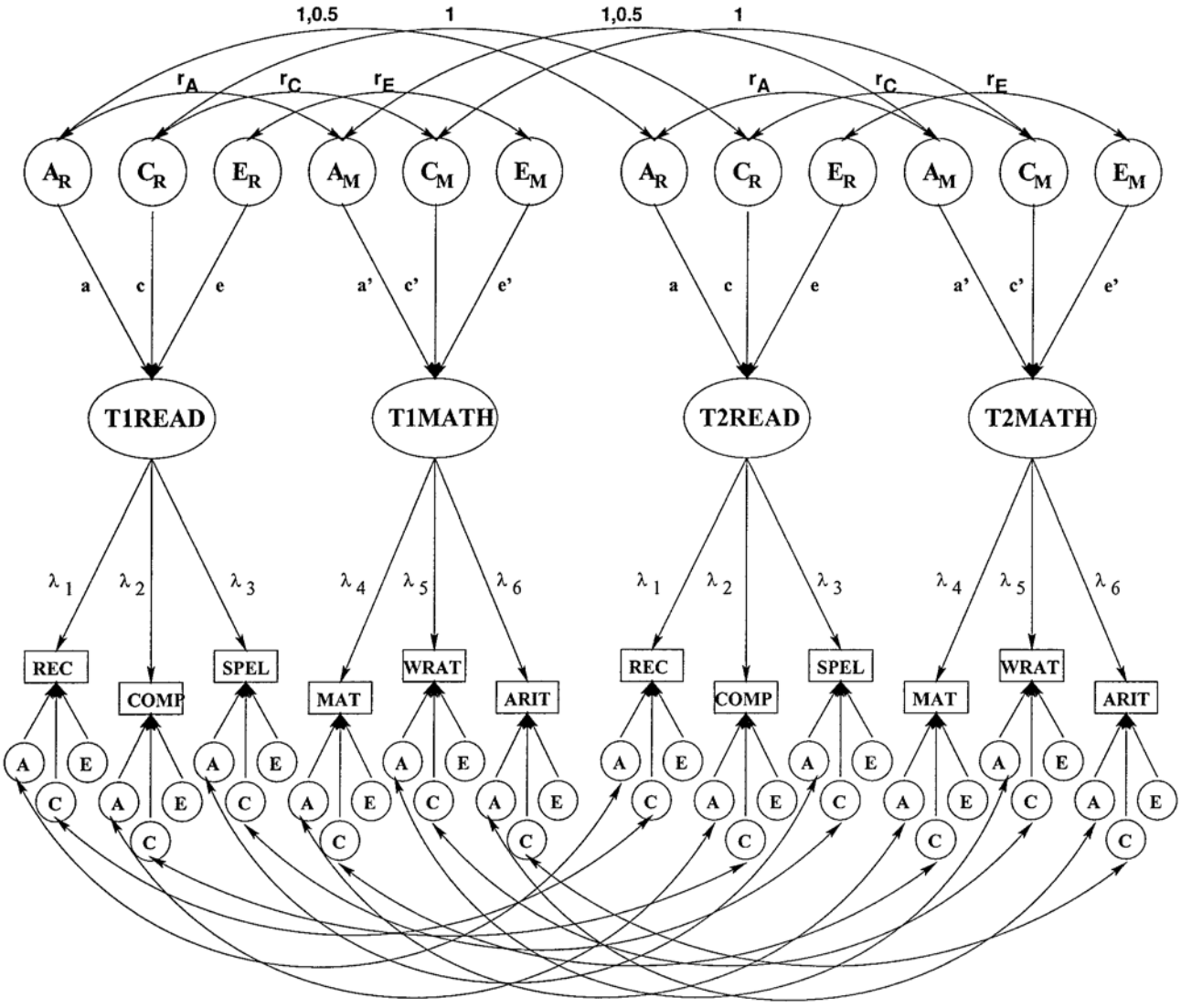

Figure 2 Path diagram for bivariate twin model

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negative log-likelihood for each data point. Because there is no observed covariance matrix to which the expected covariance matrix can be compared, evaluation of the fit of the model is based on a comparison of a constrained model that is nested within a more general model. The difference between the log-likelihood estimates for these nested models is distributed as a χ^2 and thus provides a goodness-of-fit index for model comparison. Because estimates of χ^2 are highly sensitive to sample size, even small differences in large samples may result in significant changes in χ^2 ($\Delta\chi^2$). Therefore, we also employed a ratio of χ^2 to the degrees of freedom to assess the model fit, with ratios of five or less indicating a reasonable fit to the data (Wheaton et al,[20] Hayduk[21]).

Phenotypic models

A phenotypic model with two correlated factors was fitted simultaneously to the variable length record files of the RD and control groups to provide separate parameter estimates for the two samples. The reading factor loaded on the three PIAT subtests (REC, COMP, and SPEL), and the mathematics factor loaded on the PIAT Math (MAT), WISC-R or WAIS-R Arithmetic (ARIT), and WRAT Arithmetic (WRAT) subtests. In addition, one residual for each of the measured variables was included to account for specific influences not shared with the factors. The model, depicted in Figure 1, includes the phenotypic correlation between the two latent variables as well as loadings for each of the measures on the corresponding reading and math factors. In order to test for homogeneity of variances and covariances between the two samples, parameter estimates for the RD and control groups were equated and the resulting log-likelihood estimate was compared to that for the full model.

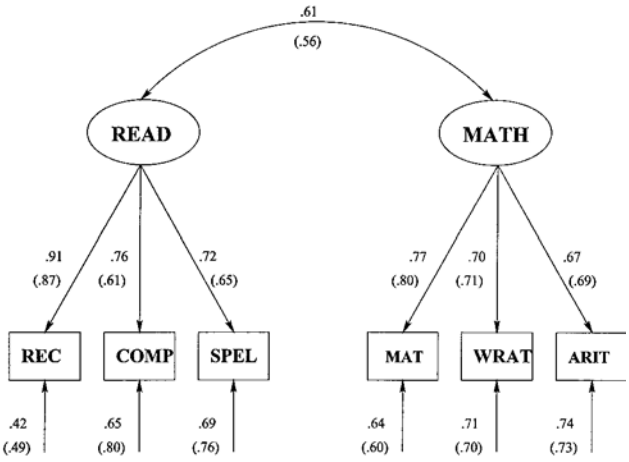

Figure 3 Parameter estimates for the full bivariate phenotypic model of data from individuals in the Colorado Learning Disabilities Research Center. Estimates for the control group are shown in parentheses

Genetic models

As depicted in Figure 2, the bivariate twin model partitions the phenotypic variances and covariances into genetic and environmental components. For a given variable (eg READ), the phenotypic correlation between members of MZ twin pairs is due to both additive genetic (a^2) and shared environmental (c^2) influences ($r_{MZ} = a^2 + c^2$). The corresponding phenotypic correlation for DZ twin pairs is $0.5\,a^2 + c^2$, because DZ twin pairs share only half of their segregating genes but all of the shared environmental influences. Thus, twice the difference between the MZ and DZ observed correlations estimates heritability (a^2). Consequently, subtracting the heritability estimate from the MZ correlation provides an estimate of the proportion of the total variance due to shared environmental influences (c^2), and subtracting the MZ correlation from one provides an estimate of the proportion of the variance due to non-shared environmental factors (e^2). The bivariate model simply extends these basic principles and provides estimates of univariate parameters (ie a^2, c^2 and e^2 for each of the traits) in addition to cross-trait correlations, which can also be partitioned into genetic and environmental components.

For the bivariate twin model (Figure 2), the additive genetic, shared environmental, and non-shared environmental influences on reading performance are represented by A_R C_R, E_R, respectively. A_M, C_M, and E_M are the corresponding additive genetic, shared environmental, and non-shared environmental influences on the mathematics performance

Table 1 Model comparisons of phenotypic models for twin data from the Colorado Learning Disabilities Research Center

Model	–2log likelihood	NPAR	vs	χ^2	df	P	χ^2/df	AIC
1 Saturated	24238.57	360						
2 Full	24584.98	158	1	346.41	202	≤0.005	1.71	–57.59
3 RD = CONT	24654.76	145	2	69.78	13	≤0.005	5.37	43.78
4 $read_{RD} = read_{CONT}$	24637.93	152	2	52.95	6	≤0.005	8.83	40.95
5 $math_{RD} = math_{CONT}$	24600.86	152	2	15.88	6	≤0.025	2.65	3.88
6 $r_{RD} = r_{CONT}$	24586.20	157	2	1.22	1	≥0.250	1.22	–0.78

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Table 2 Parameter estimates obtained from fitting the full phenotypic model to reading and mathematics performance data

Parameter	RD	95% CI	Control	95% CI
λ_1	0.91	(0.88, 0.93)	0.87	(0.82, 0.92)
λ_2	0.76	(0.73, 0.80)	0.61	(0.54, 0.66)
λ_3	0.72	(0.68, 0.76)	0.65	(0.59, 0.70)
λ_4	0.77	(0.72, 0.82)	0.80	(0.74, 0.86)
λ_5	0.70	(0.64, 0.76)	0.71	(0.63, 0.78)
λ_6	0.67	(0.62, 0.72)	0.69	(0.63, 0.74)
Residuals				
ε_1	0.42	(0.36, 0.48)	0.49	(0.38, 0.57)
ε_2	0.65	(0.61, 0.69)	0.80	(0.75, 0.84)
ε_3	0.69	(0.65, 0.73)	0.76	(0.71, 0.81)
ε_4	0.64	(0.58, 0.69)	0.60	(0.52, 0.67)
ε_5	0.71	(0.65, 0.77)	0.70	(0.62, 0.78)
ε_6	0.74	(0.69, 0.78)	0.73	(0.67, 0.78)
Phenotypic correlation				
r_P	0.61	(0.55, 0.67)	0.56	(0.46, 0.64)

factor. The path coefficients for additive genetic, shared environmental, and non-shared environmental influences on the reading variables are symbolized by a, c, and e, respectively. The corresponding coefficients for the math variables are a', c', and e', respectively. The cross-trait genetic (r_A), shared environmental (r_C), and non-shared environmental (r_E) correlations are also depicted in Figure 2, as are the residuals for each measure partitioned into specific additive (A), specific shared environmental (C), and specific non-shared environmental (E) components. The proportions of variance due to genetic and environmental influences for the RD and control groups are obtained by squaring the respective path coefficients.

Genetic and environmental parameters, as well as 95% confidence intervals, were estimated by fitting both full and reduced models using the Mx statistical modelling package (Neale[19]). Because genetic and environmental parameter estimates are not expected to be negative, boundary constraints were placed on the parameters in order to ensure positive estimates. However, such boundaries can present interpretational problems for the computation of confidence intervals (Neale and Miller[22]). Therefore, for the present analysis, confidence intervals with lower bounds equal to zero should be interpreted with caution.

The full models were compared to a saturated model, which acocunts for all of the variance, to determine goodness-of-fit. These model comparisons and those for corresponding nested submodels were evaluated using χ^2 difference tests and the χ^2/df ratio; however, Akaike's information criterion (AIC = $\chi^2 - 2df$[23]) was also employed, with lower Akaike's values representing better fitting models (Neale and Cardon[12]). The following submodels were examined: (1) submodels in which various parameter estimates for the RD and control groups are constrained to be equal; (2) a genetic model in which r_C, c, and c' are constrained to be zero; and (3)

Table 3 Twin correlations of standardized variables for MZ and DZ twin pairs in the RD sample (DZ correlations below the diagonal)

	T1REC	T1COMP	T1SPEL	T1MAT	T1WRAT	T1ARIT	T2REC	T2COMP	T2SPEL	T2MAT	T2WRAT	T2ARIT
T1REC	1.00	0.67	0.65	0.28	0.31	0.14	0.73	0.49	0.52	0.21	0.19	0.10
T1COMP	0.73	1.00	0.47	0.37	0.38	0.23	0.55	0.54	0.35	0.31	0.30	0.22
T1SPEL	0.72	0.57	1.00	0.29	0.37	0.18	0.46	0.32	0.54	0.24	0.24	0.09
T1MAT	0.48	0.51	0.49	1.00	0.44	0.48	0.24	0.44	0.26	0.62	0.40	0.49
T1WRAT	0.50	0.43	0.48	0.59	1.00	0.38	0.34	0.35	0.20	0.39	0.76	0.43
T1ARIT	0.45	0.44	0.46	0.55	0.51	1.00	0.06	0.18	0.12	0.39	0.28	0.48
T2REC	0.28	0.25	0.16	0.07	0.10	0.14	1.00	0.63	0.59	0.25	0.37	0.19
T2COMP	0.16	0.24	0.08	0.09	0.04	0.14	0.72	1.00	0.42	0.43	0.39	0.31
T2SPEL	0.22	0.20	0.22	0.12	0.19	0.11	0.68	0.49	1.00	0.24	0.23	0.13
T2MAT	0.17	0.26	0.14	0.32	0.21	0.25	0.45	0.44	0.44	1.00	0.46	0.52
T2WRAT	0.18	0.28	0.19	0.30	0.42	0.23	0.48	0.39	0.52	0.69	1.00	0.49
T2ARIT	0.20	0.21	0.11	0.18	0.20	0.32	0.48	0.42	0.39	0.55	0.57	1.00

Table 4 Twin correlations of standardized variables for MZ and DZ twin pairs in the control sample (DZ correlations below the diagonal)

	T1REC	T1COMP	T1SPEL	T1MAT	T1WRAT	T1ARIT	T2REC	T2COMP	T2SPEL	T2MAT	T2WRAT	T2ARIT
T1REC	1.00	0.57	0.61	0.32	0.46	0.25	0.59	0.42	0.46	0.28	0.39	0.30
T1COMP	0.63	1.00	0.38	0.33	0.32	0.33	0.41	0.48	0.31	0.30	0.28	0.31
T1SPEL	0.48	0.21	1.00	0.32	0.37	0.31	0.57	0.38	0.62	0.30	0.29	0.33
T1MAT	0.39	0.35	0.36	1.00	0.58	0.65	0.31	0.43	0.22	0.67	0.50	0.57
T1WRAT	0.31	0.20	0.24	0.54	1.00	0.50	0.33	0.27	0.20	0.52	0.68	0.32
T1ARIT	0.30	0.29	0.26	0.40	0.50	1.00	0.31	0.32	0.21	0.55	0.37	0.56
T2REC	0.31	0.24	0.19	0.19	0.29	0.24	1.00	0.44	0.61	0.39	0.38	0.33
T2COMP	0.34	0.34	0.12	0.16	0.11	0.11	0.52	1.00	0.41	0.41	0.31	0.39
T2SPEL	0.20	0.05	0.27	0.15	0.17	0.13	0.59	0.25	1.00	0.30	0.19	0.24
T2MAT	0.13	0.18	0.06	0.29	0.26	0.27	0.33	0.38	0.18	1.00	0.55	0.55
T2WRAT	0.23	0.07	0.33	0.24	0.47	0.29	0.35	0.25	0.21	0.45	1.00	0.42
T2ARIT	0.25	0.23	0.11	0.21	0.14	0.17	0.32	0.43	0.22	0.58	0.45	1.00

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submodels that test whether specific genetic and environmental effects, independent of those that contribute to the covariance of the factors, significantly influence the reading and math variables.

Results

Phenotypic analysis

The parameter estimates resulting from the fit of the full phenotypic model to the data are presented in Figure 3, and the goodness-of-fit indices for the full and corresponding submodels are shown in Table 1. Parameter estimates and 95% confidence intervals for the full model, which yielded separate parameter estimates for the RD and control groups, are also presented in Table 2. Due to the large sample size (9773 observed data points for 1762 individuals), the $\Delta\chi^2$ value for the full model compared to the saturated model is large ($\Delta\chi^2$ = 346.41, df = 202, $P \leq 0.005$); however, both the χ^2/df ratio (1.71) and the Akaike's information criterion (–57.59) are low, suggesting an acceptable fit to the data.

As shown in Figure 3, the phenotypic correlation between the reading and mathematics performance factors was 0.61 in the RD group and 0.56 in the control group, suggesting a substantial relationship between the latent variables in both samples.

As shown in Table 1, the factor structure for both reading and math performance is significantly different for the RD and control samples (model 3). When the parameter estimates were equated across groups, the change in χ^2 was significant ($\Delta\chi^2$ = 69.78, df = 13, $P \leq 0.005$). In addition, the factor structure for reading performance (model 4), and the factor structure for math performance (model 5) significantly differed for the RD and control groups ($P \leq 0.005$ and $P \leq 0.025$, respectively). These results are due, at least in part, to the greater variation in the reading-disabled sample (Alarcón and DeFries[24]).

In contrast, when equating the correlation between reading and math (model 6) for the two groups, a nonsignificant change in χ^2 was achieved ($\Delta\chi^2$ = 1.22, df = 1, $P \geq 0.250$), suggesting that the standardized covariance of reading and math performance is similar in individuals from the RD and control samples.

Table 5 Genetic and environmental contributions to the variance of the latent reading and math factors estimated from the full model

	RD[a]			Control[b]		
Measure	a^2	c^2	e^2	a^2	c^2	e^2
READ	0.81	0.00	0.19	0.69	0.13	0.18
MATH	0.88	0.00	0.12	0.67	0.21	0.11

[a] r_A = 0.61, r_C = 0.97, r_E = 0.71
[b] r_A = 0.47, r_C = 1.00, r_E = 0.50

Genetic analyses

Tables 3 and 4 present the twin correlations for the observed reading and math variables. As shown, the univariate MZ twin correlations (eg T1REC with T2REC) are substantially higher than DZ twin correlations for both groups, indicating that individual differences in these measures are due at least in part to heritable influences. Although less striking, the pattern of cross-twin bivariate correlations (eg T1REC with T2MAT) also suggest genetic covariation between measures.

For the genetic analyses, the full phenotypic model was partitioned to include additive genetic, shared environmental, and non-shared environmental contributions to the variances of the latent factors, as well as to the covariance among the measures. The proportions of variance due to additive genetic, shared environmental, and non-shared environmental sources obtained from the full bivariate twin model are presented in Table 5. In order to ensure identification of the model, the variances of the latent variables were constrained such that $a^2 + c^2 + e^2 = 1$ and $a'^2 + c'^2 + e'^2 = 1$.

Results from the full genetic model suggest that although reading performance is highly heritable in both the RD sample (0.81) and in the control sample (0.69), reading performance is slightly more heritable in the RD sample. These estimates are highly similar to those found by Gillis et al.[9] In contrast, shared environmental influences on reading performance account for no variance in the reading-disabled group (0.00) and only a small proportion of the variance in the control group (0.13).

Mathematics performance is also substantially heritable in both groups (0.88 for the RD sample and 0.67 for controls). Although these estimates of heritability for math performance are somewhat higher than those reported by Gillis et al,[9] different indices of math performance were used for the present analysis. As is the case for the reading latent factors, shared environmental influences account for relatively little variance of math performance in both samples (0.00 for the RD group, and 0.21 for controls).

The parameter estimates for the full bivariate twin model are shown in Figure 4 and Table 6. The genetic correlation (r_A) between reading and math is substantial in both the reading-disabled and control samples (r_A = 0.61 and 0.47, respectively). Moreover, 83% of the phenotypic correlation (r_P = 0.62 in the full genetic model) between reading and math performance in the RD sample is due to genetic influences, ie $ar_a a'/r_P = (0.90)(0.61)(0.94)/(0.62)$,

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where a and a' are the standardized genetic parameter estimates. In the control sample, 58% of the phenotypic relationship (r_P = 0.55 in the full genetic model) between reading and math is due to genetic influences. Although the estimates of r_C are close or equal to one in both groups, both estimates are not significantly different from zero (95% CI = 0.00–1.00 in both groups, Table 6).

The results of goodness-of-fit tests for the various twin model comparisons are presented in Table 7.

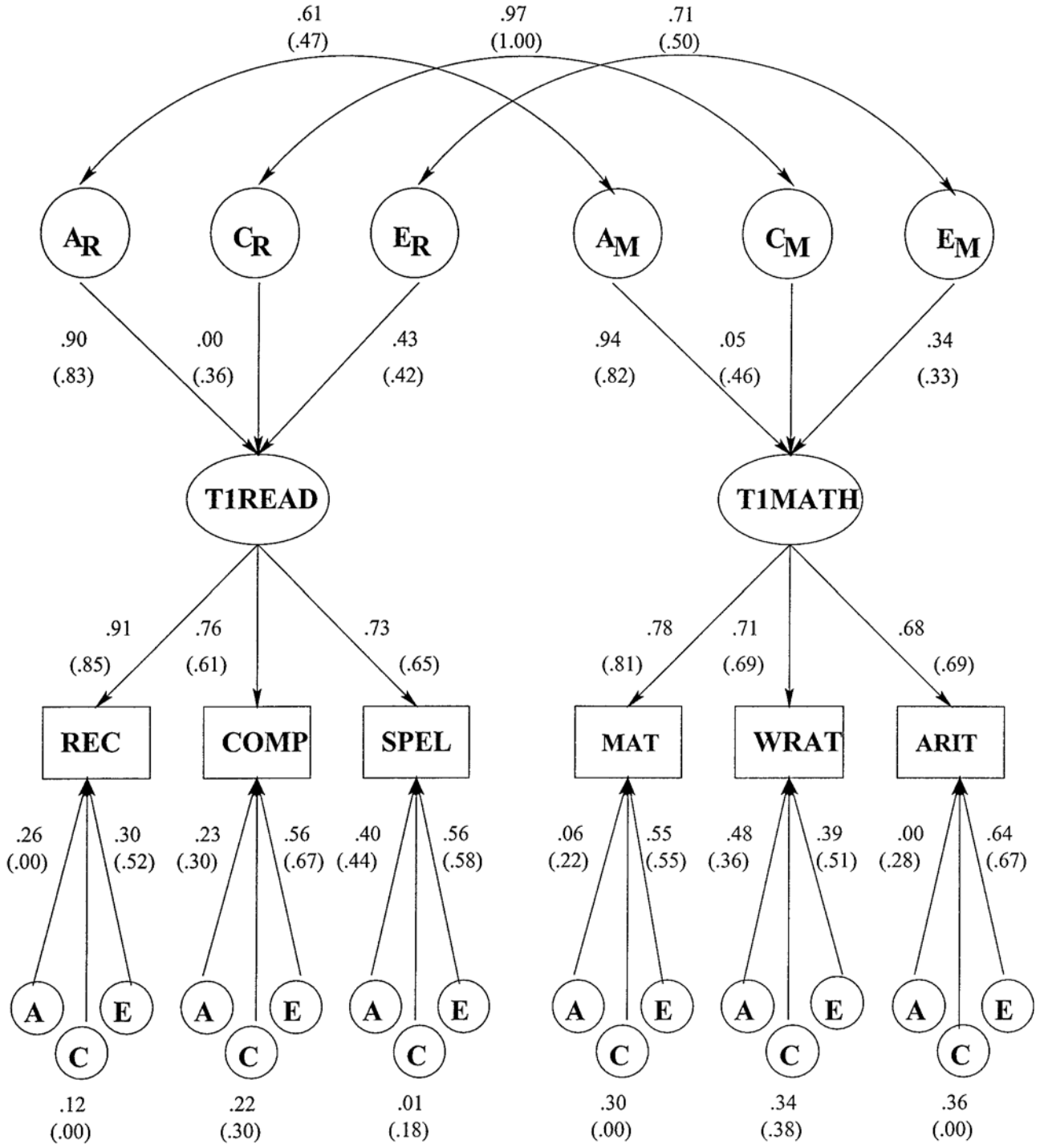

Figure 4 Parameter estimates for the full bivariate twin model (one twin only). Estimates for the control group are shown in parentheses

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Table 6 Parameter estimates obtained from fitting the full bivariate twin model to reading and mathematics performance data

Parameter	RD	95% CI	Control	95% CI
a	0.90	(0.84, 0.93)	0.83	(0.62, 0.94)
c	0.00	(0.00, 0.29)	0.36	(0.00, 0.65)
e	0.43	(0.37, 0.50)	0.42	(0.33, 0.52)
a′	0.94	(0.81, 0.97)	0.82	(0.58, 0.97)
c′	0.05	(0.00, 0.47)	0.46	(0.00, 0.73)
e′	0.34	(0.25, 0.42)	0.33	(0.21, 0.43)
λ_1	0.91	(0.88, 0.94)	0.85	(0.81, 0.89)
λ_2	0.76	(0.73, 0.80)	0.61	(0.55, 0.67)
λ_3	0.73	(0.69, 0.76)	0.65	(0.59, 0.71)
λ_4	0.78	(0.73, 0.82)	0.81	(0.75, 0.86)
λ_5	0.71	(0.65, 0.76)	0.69	(0.60, 0.76)
λ_6	0.68	(0.63, 0.72)	0.69	(0.63, 0.75)
r_A	0.61	(0.52, 0.68)	0.47	(0.20, 0.74)
r_C	0.97	(0.00, 1.00)	1.00	(0.00, 1.00)
r_E	0.71	(0.49, 0.94)	0.50	(0.17, 0.87)

The full model provides a reasonable fit to the data when compared with a fully saturated model with a $\Delta\chi^2$ of 420.40 for 246 degrees of freedom (χ^2/df = 1.71, AIC = –71.60). Because we could equate the correlation between the reading and math latent factors in the phenotypic model, and based on inspection of the 95% confidence intervals of the parameter estimates for the full genetic model, we equated the common genetic and environmental parameters for the two samples (ie $a_{RD} = a_{CONT}$, $r_{A(RD)} = r_{A(CONT)}$, $a'_{RD} = a'_{CONT}$, etc.). As expected, model 3 did not significantly reduce the model fit ($\Delta\chi^2$ = 4.20, df = 9, P ≥ 0.750, AIC = –13.80) nor did model 4, which tested whether shared environmental factors significantly influenced the relationship between the reading and math latent factors ($\Delta\chi^2$ = 0.03, df = 3, P = 0.995, AIC = –5.97).

After examination of the 95% confidence intervals, specific shared environmental variances were dropped from the model (model 5). The change in χ^2 was not significant ($\Delta\chi^2$ = 8.30, df = 12, P ≥ 0.750, AIC = –15.70); thus, shared environmental effects neither contribute significantly to the covariation between the traits, nor account for significant independent variation in the reading and math variables. Finally, in order to determine whether genetic influences independent of those shared by the two factors significantly influence the measured variables, the specific additive genetic effects were dropped from the model. Model 6 fits the data significantly worse than model 5 ($\Delta\chi^2$ = 312.28, df = 12, P ≤ 0.005, AIC = 288.28), indicating that genetic influences independent of those shared by the factors significantly contribute to the measures of reading and mathematics.

Discussion

The etiology of the relationship between reading and mathematics performance was examined by fitting structural equation models to twin data from the Colorado Learning Disabilities Research Center. In the RD sample, at least one member of the twin pair (290 MZ and 236 same-sex DZ) had a positive school history of reading problems. In contrast, neither member of each pair in the control sample (220 MZ and 135 same-sex DZ) had a school history of reading deficits. The proportions of observed variance due to genetic and environmental influences were estimated for the various measures, and the phenotypic correlation between reading and math performance was partitioned into components due to genetic, shared environmental, and non-shared environmental influences in both groups. Specific genetic and environmental influences, independent of the covariation between the two factors, were also examined.

Reading performance is highly heritable both in the proband (a^2 = 0.81) and control (a^2 = 0.69) samples, suggesting that 80% of the phenotypic variance in this latent variable is due to genetic influences in the RD group, and almost 70% of reading performance in the control group is accounted for by genetic factors. Similar results were found for math performance, with heritability estimates of 0.88 and 0.67 for the RD and control groups, respectively. In addition, reading and math performance appear to be due substantially to the same genetic influences in both groups (r_A = 0.61 and 0.47 for the RD and control samples, respectively), supporting results previously reported by Gillis et al,[9] Light and DeFries,[5] and Knopik et al.[8]

Despite similar estimates in the proband and control groups, phenotypic parameter estimates for the two samples could not be equated. It is important, however, to note that the phenotypic loadings are similar for the two groups, as are those in the

Table 7 Model comparisons of twin models for data from the Colorado Learning Disabilities Research Center

Model	–2log likelihood	NPAR	vs	χ^2	df	P	χ^2/df	AIC
1 Saturated	24238.57	360						
2 Full	24658.97	114	1	420.40	246	≤0.005	1.71	–71.60
3 Equate common ACE	24663.17	105	2	4.20	9	≥0.750	0.47	–13.80
4 $c = c' = r_c = 0$	24663.20	102	3	0.03	3	≥0.995	0.01	–5.97
5 specific C = 0	24671.50	90	4	8.30	12	≥0.750	0.69	–15.70
6 specific A = 0	24983.78	78	5	312.28	12	≤0.005	26.02	288.28

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genetic model. Horn et al[25] argue that exact metric invariance, that is, identical loadings across groups, is scientifically unrealistic. Cunningham[26] also suggests that models requiring strict metric invariance are rarely supported by empirical data, and those that do show reasonable fit usually have a contrived character. Configural invariance, which requires only that the configuration of zero loadings and salient loadings remain the same between two groups, is therefore a more realistic goal. Employing this criterion, it appears that configural invariance is achieved for both the genetic and environmental parameter estimates in the RD and control samples. Therefore, although the phenotypic parameter estimates cannot be equated between groups, the underlying genetic and environmental factor structures for measures of reading and mathematics performance appear to be highly similar.

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