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Overlap and specificity of genetic and environmental influences on mathematics and reading disability in 10-year-old twins

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Background: To what extent do genetic and environmental influences on reading disability overlap with those on mathematics disability? Multivariate genetic research on the normal range of variation in unselected samples has led to a Generalist Genes Hypothesis which posits that the same genes largely affect individual differences in these abilities in the normal range. However, little is known about the etiology of co-morbidity for the disability extremes of reading and mathematics. **Method:** From 2596 pairs of 10-year-old monozygotic and dizygotic twins assessed on a web-based battery of reading and mathematics tests, we selected the lowest 15% on reading and on mathematics. We conducted bivariate DeFries–Fulker (DF) extremes analyses to assess overlap and specificity of genetic and environmental influences on reading and mathematics disability defined by a 15% cut-off. **Results:** Both reading and mathematics disability are moderately heritable (47% and 43%, respectively) and show only modest shared environmental influence (16% and 20%). There is substantial phenotypic co-morbidity between reading and mathematics disability. Bivariate DF extremes analyses yielded a genetic correlation of .67 between reading disability and mathematics disability, suggesting that they are affected largely by the same genetic factors. The shared environmental correlation is .96 and the non-shared environmental correlation is .08. **Conclusions:** In line with the Generalist Genes Hypothesis, the same set of generalist genes largely affects mathematical and reading disabilities. The dissociation between the disabilities occurs largely due to independent non-shared environmental influences. **Keywords:** Mathematical disability, reading disability, twin method, genetic correlation, etiology, behavioral genetics, child development, comorbidity, learning difficulties. **Abbreviations:** MZ: monozygotic; DZ: dizygotic; TEDS: Twins' Early Development Study; PIAT: Peabody Individual Achievement Test.

According to the 'Generalist Genes' Hypothesis of learning abilities and disabilities (Plomin & Kovas, 2005), most genetic effects for scholastic achievement and cognitive abilities are general rather than specific. That is, the genes that affect one area of learning, such as mathematics performance, are largely the same genes that affect other abilities, although there are some genetic effects that are specific to each ability. The main purpose of the present study is to test the Generalist Genes Hypothesis in the domain of mathematics and reading disabilities. We used bivariate genetic analysis to assess the extent to which genetic effects on mathematics disability at 10 years of age overlap with genetic effects on reading disability at the same age.

We acknowledge that the choice of appropriate labels for children's low performance is controversial, with no agreement on defining deficit, challenge, delay, difficulty, disorder, and impairment in mathematics and reading. For the purposes of this study we use the word *disability* with its semantic link to the word *ability* because recent research suggests that common learning disabilities are the low end of the normal distribution of learning abilities (Plomin & Kovas, 2005).

Nearly all previous bivariate genetic research has used unselected samples and thus considered the aetiology of the entire range of normal variation (ability) in mathematics and reading performance rather than performance at the low extreme of the distribution (disability). Mathematics and reading abilities covary phenotypically (Knopik & DeFries, 1999), and heritabilities are substantial for both traits, although the estimates of heritability vary widely (Markowitz, Willemsen, Trumbetta, van Beijsterveldt, & Boomsma, 2005). Even though individual differences in mathematics and reading are influenced by genes, it is possible that completely different sets of genes affect these domains. Bivariate genetic analysis, which addresses the etiology of the covariance between two traits rather than the variance of each trait considered on its own, can estimate the extent to which the genetic factors that influence individual differences in mathematics are also involved in shaping individual differences in reading.

Previous multivariate genetic studies addressing the extent to which mathematics and reading abilities are influenced by the same genetic factors (genetic correlation) suggest substantial overlap. Four twin studies with different sample sizes and participants of different ages found genetic

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correlations between reading and mathematical abilities ranging from .40 to .98 (average correlation of .68) (Thompson, Detterman, & Plomin, 1991; Knopik & DeFries, 1999; Light, DeFries, & Olson, 1998; Kovas, Harlaar, Petrill, & Plomin, 2005; Markowitz et al., 2005). In an adoption study, the genetic correlation between reading and mathematical performance was .80 in a parent–offspring analysis (Wadsworth, DeFries, Fulker, & Plomin, 1995a) and .83 in a sibling analysis (Wadsworth, DeFries, Fulker, & Plomin, 1995b).

These high genetic correlations between reading and mathematics ability led us to predict that genetic overlap is also substantial for reading and mathematics disability. Only one multivariate genetic study of reading and mathematics disability has been reported (Knopik, Alarcón, & DeFries, 1997; Light & DeFries, 1995). The first report from this study focused on children selected for reading disability who were then selected for mathematics disability, yielding a sample of 148 MZ and 111 DZ twin pairs of a wide age range (Light & DeFries, 1995). Twin cross-concordances (i.e., reading disability in one twin and mathematics disability in the co-twin) were 68% for MZ twins and 40% for DZ twins, suggesting substantial genetic influence. In a bivariate DF extremes analysis of reading disability using the mathematics variable as a continuous score, MZ and DZ cross-trait group correlations (the extent to which the mean standardized quantitative trait score of co-twins on trait Y is similar to the mean standardized score of selected probands on trait X) were .92 and .66, respectively. Bivariate heritability (the extent to which the genetic factors account for the mean difference between the probands and the population) was .55, suggesting substantial genetic overlap between reading disability and mathematics ability.

Because the twins were all selected for reading disability, no genetic correlation could be calculated. In a follow-up analysis, twins were selected both for reading disability (102 MZ and 77 same-sex DZ twin pairs) and for mathematics disability (42 MZ and 23 DZ pairs) (Knopik et al., 1997). Bivariate DF extremes analysis for reading disability probands versus mathematics ability yielded results similar to those described above. Analysis of mathematics disability probands versus reading ability also yielded similar results. This was the first report in which a genetic correlation was calculated from bivariate DF extremes analysis. The genetic correlation between reading disability and mathematics disability was estimated as .53.

The present study provides the first analysis of the overlap and specificity of genetic and environmental influences on reading and mathematics disability in a large sample of twins of the same age. A large sample is needed to provide the necessary statistical power to estimate the genetic correlation between the two disabilities and therefore, web-based tests, which facilitate data collection in large, geographically dispersed samples, were used to assess mathematics and reading disability.

Method

Participants and procedure

Participants were part of the Twins Early Development Study (TEDS), a longitudinal study involving a representative sample of all twins born in England and Wales in 1994, 1995, and 1996 (Oliver & Plomin, in press; Trouton, Spinath, & Plomin, 2002). When the twins were 10 years old, tests of a wide variety of cognitive measures were administered on the web to 7442 children born between January 1994 and August 1996. This number refers to all children who took part in the battery after specific medical exclusion criteria were applied (see Kovas et al., 2005 for details). From this sample, data from both twins in a pair for mathematics and reading were available for 2596 pairs of same-sex and opposite-sex twins.

In order to examine the effect of attrition, we compared National Curriculum math scores for children who completed the math battery and those who did not. Using standard scores based on the entire TEDS sample (after medical exclusions), we found that children who completed our web-based math battery performed only slightly better than average on NC math (mean standard score of .08 and SD = .92). Those who did not complete the NC math battery had slightly lower than average math scores (–.18, SD = 1.03). These mean differences account for less than 1% of the variance. Analyses of reading produced similar results. Zygosity was ascertained by parental ratings of physical similarity, supplemented by DNA genotyping for difficult zygosity diagnoses (for details see Freeman et al., 2003). Informed consent was obtained in writing from all of the families who agreed to take part in the study.

Measures

Using web-based assessment, reading was assessed by an adaptation of the Peabody Individual Achievement Test (PIAT-Revised; Markwardt, 1997) Reading Comprehension scale, and Mathematics by three subtests from the nferNelson Math 5–14 Series (2001): Understanding Number, Non-Numerical Processes, and Computation and Knowledge. Based on our previous research, showing high phenotypic (.62 on average) and genetic (.84 on average) correlations among different aspects of mathematics (Kovas, Petrill, & Plomin, in press b), we created a composite score using the mean of the percentage scores of the three tests. Both reading and mathematics tests scores obtained on the web correlated highly (.83 and .93 respectively) with standard paper-and-pencil versions administered one to three months later to a subsample of TEDS (Haworth et al., 2007). Further information about the measures, test administration, and validity and reliability of the measures can be found in Kovas, Haworth, Petrill, and Plomin (in press a) and Oliver and Plomin (in press).

Web-based assessment has been shown to be well suited for testing school-age children, as well as reli-

able, valid, and highly convenient for use in large samples (Birnbaum, 2004; Gosling, Vazire, Srivastava, & John, 2004). In TEDS, 80% of the families have daily access to the internet, which is similar to the results of market surveys of UK families with adolescents. Most children without access to the internet at home have access in their schools and local libraries.

Analyses and results

Descriptive statistics and further exclusions

The data were first explored using descriptive statistics analyses in SPSS. Descriptive statistics for the three categories of mathematics (Understanding Number, Non-Numerical Processes, and Computation and Knowledge) are available from the authors. Means and standard deviations for the Mathematics composite score and PIAT reading score are shown in Table 1 separately by sex and zygosity. The means and standard deviations for MZ and DZ twins, and for male and female twins were highly similar.

Analysis of variance (ANOVA) was performed in order to assess the mean effects of sex and zygosity on mathematical and reading ability in our sample. It revealed a significant main effect of zygosity for mathematics, with DZ twins performing better; however, this effect explained less than 1% of the variance. The main effect of sex was significant for the PIAT, with boys on average performing better than girls. However, this effect was also negligible, accounting for less than 1% of the variance. No significant sex-by-zygosity interactions were found.

For subsequent analyses, standardized residuals correcting for age and sex were used because the age of twins is perfectly correlated across pairs, which means that, unless corrected, variation within each age group at the time of testing would contribute to the correlation between twins and be misrepresented as shared environmental influence (Eaves, Eysenck, & Martin, 1989). This regression procedure is standard in analyses of twin data (McGue & Bouchard, 1984).

Genetic analysis of abilities

The twin method addresses the origins of individual differences by estimating the proportion of variance that can be attributed to genetic, shared environment (contributing to twin similarity), and non-shared environment (contributing to twin differences) factors (Plomin, DeFries, McClearn, & McGuffin, 2001).

Genetic influence on a specific trait can be estimated by comparing intraclass correlations for monozygotic (MZ) twins, who are genetically identical, and dizygotic (DZ) twins, whose genetic relatedness is on average .50. The phenotypic variance of a trait is attributed to genetic variance (called heritability) to the extent that the MZ twin correlation exceeds the DZ twin correlation. The relatedness for shared (common) environmental influences is assumed to be 1.0 for both MZ and DZ twin pairs who grow up in the same family because they experience similar prenatal and postnatal environments. Shared environmental influences are indicated to the extent that DZ correlation is more than half of the MZ correlation. The rest of the variance is attributed to non-shared environmental factors, which include measurement error.

In this sample, the twin intraclass correlation for mathematics was .68 for MZ twins ($N = 727$ pairs) and .44 for DZ twins ($N = 1265$ pairs); and the correlation for reading was .64 for MZ twins ($N = 931$ pairs) and .44 for DZ twins ($N = 1610$ pairs). The results for the two measures are similar and are consistent with those previously reported for both teacher-assessed global measures of mathematics and reading and for tests of reading (Oliver et al., 2004; Kovas et al., 2005; Gayan & Olson, 2003). These correlations suggest at least moderate genetic influences for the two traits (.48 for mathematics and .40 for reading), with environmental factors being primarily non-shared.

In order to assess whether being in the same class and having the same teacher increased similarity between co-twins and affected the genetic findings, we re-ran our correlational analyses splitting the data by same vs. different teacher. The two groups were nearly equal in size. The correlations were highly similar for the two groups, suggesting that being in the same classroom and being taught by the same teacher did not increase the twins' similarity in performance in reading and mathematics, at least as measured by our tests. In another report using the

Table 1 Means (standard deviations) for % of correct items and ANOVA results by sex and zygosity for the Mathematics Composite and PIAT

	MZ, *M* (*SD*)	DZ, *M* (*SD*)	Males, *M* (*SD*)	Females, *M* (*SD*)	ANOVA		
					Sex	Zyg	Sex*Zyg
Math	−.03 (1.01)	.02 (1.00)	.08 (.99)	−.07 (1.00)	$p = .158$	$p < .001$	$p = .587$
	$n = 1941$	$n = 3407$	$n = 2413$	$n = 2935$	$\eta^2 < .001$	$\eta^2 = .005$	$\eta^2 < .001$
PIAT	−.06 (1.00)	.03 (1.00)	.02 (1.04)	−.01 (.97)	$p = .001$	$p = .307$	$p = .871$
	$n = 2110$	$n = 3698$	$n = 2646$	$n = 3162$	$\eta^2 = .002$	$\eta^2 < .001$	$\eta^2 < .001$

Note: Descriptive statistics are reported on the whole sample after the medical exclusions, N = 7442. MZ = monozygotic twins, DZ = dizygotic twins (same and opposite sex), Math = Mathematics Composite score. η^2 = the proportion of the total variance that is attributed to an effect. In the ANOVA analysis zygosity had 3 levels: MZ, DZ same-sex and DZ opposite-sex.

same sample and measures (Davis et al., 2007), we investigated sex differences in etiology of individual differences in mathematics and reading. Sex-limitation model-fitting (Neale, 1997) yielded no significant sex differences in the extent of genetic and environmental influences or in comparisons between same-sex and opposite-sex twins for either reading or the three components of mathematics measured. This finding is consistent with other previous research in TEDS and with the 'gender similarities' that has recently been proposed based on the extensive meta-analysis and review of available literature (Hyde, 2005; Spelke, 2005). For this reason and to maximize power, we performed all analyses in this study combining males and females as well as same-sex and opposite-sex twins.

Although covariation between mathematics and reading *abilities* is not the focus of this study, we estimated phenotypic and genetic correlations between the two abilities in the whole sample. The phenotypic correlation between mathematics and reading was .50 ($N = 2667$, one twin from each pair). Bivariate heritability for the two abilities was .49 and the genetic correlation was .52 ($N = 2602$ pairs). For shared environment, the bivariate estimate was .41 and the correlation was 1.00. Finally, for non-shared environment, the bivariate estimate was .10, and the correlation was .16. These results are similar to those of a paper that focuses on these abilities in the whole sample (Davis et al., submitted).

Genetic analysis of disabilities

Probands and probandwise concordances. We defined probands as the lowest-performing 15% of the whole sample. We chose this cut-off for three reasons. First, performance one standard deviation below the mean, which corresponds to a 15.9% cut-off in a perfectly normal distribution, is an accepted, although not the only, cut-off used for common disorders (e.g., Eisenmajer, Ross, & Pratt, 2005). Second, for the UK National Curriculum, a 15% cut-off corresponds to children identified as performing below their grade expectation and failing items that are solved correctly by the majority of much younger children (Kovas et al., in press a). Third, in TEDS, a 15% cut-off strikes a balance between extremity of scores and sample size needed to attain reasonable power in DF extremes analysis.

Table 2 shows the means and standard deviations (standardized scores) for probands for the two measures, indicating that the probands are on average more than 1.5 standard deviations below the population mean. Of the mathematical probands, 33.8% were also reading probands (263 out of 789), and of the reading probands, 33.3% were also mathematical probands (263 out of 789). Because bivariate DF extremes analysis is an analysis of co-morbidity, it is most informative when probands are representative of co-morbidity in the population. It would not be useful, for example, to conduct such analyses using twins with reading problems who were selected as not having problems with mathematics and vice versa.

Probandwise concordances (the ratio of the number of probands in concordant pairs to the total number of probands) were calculated for reading and mathematics separately for MZ and DZ twins. Probandwise concordances represent the risk that a co-twin of a proband is affected (Plomin et al., 2001). Table 2 shows that concordances for MZ twins are higher than for DZ twins, suggesting genetic influence.

Because they are based on categorical information, twin concordances by themselves cannot be used to estimate genetic and environmental parameters as they do not include information about the population incidence. Moreover, twin concordances do not provide confidence intervals for the estimates.

Univariate DF extremes analysis. Rather than assessing twin similarity in terms of individual differences on a quantitative trait of ability or in terms of concordance for a diagnostic cut-off, DF extremes analysis (DeFries & Fulker, 1988) assesses twin similarity as the extent to which the mean stan-

Table 2 Univariate DF extremes analysis of mathematics and reading: MZ and DZ standardized means (SDs), probandwise concordances, twin group correlations, and h^2g, c^2g and e^2g parameter estimates using a 15% cutoff

	Proband mean		Co-twin mean (SD)		Probandwise concordance		Twin group correlation		DF extremes estimates		
	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ	h^2g (SE)	c^2g (SE)	e^2g
10-year Math	−1.86 (.68)	−1.87 (.66)	−1.19 (1.10)	−.75 (1.16)	.46	.36	.64	.40	.47 (.10)	.17 (.07)	.37
10-year PIAT	−1.64 (.47)	−1.63 (.47)	1.03 (.90)	−.68 (1.04)	.47	.36	.63	.42	.43 (.11)	.20 (.08)	.37

Note: The proband and co-twin means are expressed as standard scores; for example, the MZ proband mean is 1.86 standard deviations below the population mean. As expected, proband means are highly similar for MZ and DZ probands. The twin group correlation (Plomin, 1991) is the ratio between the co-twin mean and the proband mean (e.g. for MZ twins: −1.19 ÷ −1.86 = .64), which is an index of twin group similarity. As explained in the text, the twin group correlation is similar to the transformed co-twin mean in DF extremes analysis (DeFries & Fulker, 1988). h^2g = group heritability; c^2g = group shared environment; e^2g = group non-shared environment. N (MZ pairs) = 287 for Maths, and 231 for PIAT; N (DZ pairs) = 481 for Maths, and 358 for PIAT.

dardized quantitative trait score of co-twins is as low as the mean standardized score of the selected extreme or diagnosed probands (see Plomin & Kovas, 2005 for detailed explanation of DF extremes analysis).

This measure of twin similarity is typically referred to in DF extremes analysis as a *transformed co-twin mean* (DeFries & Fulker, 1988). The co-twin mean is transformed to adjust for mean differences between MZ and DZ probands and standardized so that the transformed co-twin mean indicates the proportion of the difference between the proband mean and the population mean. A related index of twin group similarity (i.e., similarity of means rather than individual differences) is called a *group twin correlation* (Plomin, 1991), which is the ratio between the co-twin mean and the population mean. Genetic influence is implied if group twin correlations (or transformed co-twin means) are greater for MZ than for DZ twins. Doubling the difference between MZ and DZ group twin correlations estimates the genetic contribution to the average phenotypic difference between the probands and the population. The ratio between this genetic estimate and the phenotypic difference between the probands and the population is called *group heritability*. It should be noted that group heritability does not refer to individual differences among the probands – the question is not why one proband is slightly more disabled than another but rather why the probands as a group are so much more disabled than the rest of the population.

Although DF extremes group heritability can be estimated by doubling the difference in MZ and DZ group twin correlations (Plomin, 1991), DF extremes analysis is more properly conducted using a regression model (DeFries & Fulker, 1988). The DF extremes model fits standardized scores for MZ and DZ twins to the regression equation, $C = \beta_1 P + \beta_2 R + A$, where C is the predicted score for the co-twin, P is the proband score, R is the coefficient of genetic relatedness (1.0 for MZ twins and .5 for DZ twins), and A is the regression constant. β_1 is the partial regression of the co-twin score on the proband, an index of average MZ and DZ twin resemblance independent of β_2. The focus of DF extremes analysis is on β_2. β_2 is the partial regression of the co-twin score on R independent of β_1. It is equivalent to twice the difference between the means for MZ and DZ co-twins adjusted for differences between MZ and DZ probands (the adjustment described above as transformed co-twin data). In other words, β_2 is the genetic contribution to the phenotypic mean difference between the probands and the population. Using transformed co-twin data, β_2 is group heritability. Finding significant group heritability implies that disability and ability are both heritable and that there are genetic links between the disability and normal variation in the ability (see Plomin and Kovas, 2005 for details).

In this study, scores were standardized and transformed to adjust for proband mean differences between MZ and DZ groups so that genetic and environmental parameters could be estimated from model fitting on the basis of the regression: $C\ (M) = \beta_1 P\ (M) + \beta_2 R + A$, where *C (M)*, the co-twin's mathematics score, is predicted from *P (M)*, the proband's mathematics score, and the coefficient of relatedness (R), which is 1.0 for MZ (genetically identical) and .5 for DZ twins (who are on average 50% similar genetically). The regression weight β_2 is group heritability, the proportion of the average phenotypic difference in mathematics scores between the probands and the population that can be explained by genetic factors. The same analysis was performed for reading.

The results from the DF extremes analyses are presented in Table 2. The twin group correlations for mathematics are .64 for MZ twins and .40 for DZ twins. Doubling this difference in twin group correlations estimates group heritability as .48. The estim ate from DF extremes analysis is .47 with a standard error of .10. For reading, doubling the difference in the twin correlations for MZ (.63) and DZ (.42) twins suggests a group heritability of .42, and the DF estimate is .43 (SE = .11).

Thus, group heritabilities for mathematics and reading are significant and moderate, suggesting a genetic link between ability and disability for both reading and mathematics. Group shared environmental estimates can be conceptualized as the extent to which MZ group correlations are not explained by group heritability. In DF extremes analysis, they are estimated as the difference between the transformed co-twin mean and group heritability (β_2). Shared environment is estimated as .21 (SE = .06) for mathematics and .14 (.08) for reading. Non-shared environment, which explains the rest of the difference between the probands and the population, is .37 for both measures.

Bivariate DF extremes analysis. The logic of univariate DF extremes analysis can be extended to bivariate analysis (Light & DeFries, 1995; Plomin & Kovas, 2005). In contrast to univariate DF extremes analysis which selects probands as extreme on X and compares the quantitative scores of their MZ and DZ cotwins on X, bivariate DF extremes analysis selects probands on X and compares the quantitative scores of their cotwins on Y, a cross-trait twin group correlation. (Even univariate DF extremes analyses are best considered as bivariate analyses in the sense that the extreme score of the probands cannot be assumed to be measuring the same processes reflected in the less extreme scores of the co-twins, even when the 'same' measure is used to assess probands and co-twins.) The genetic contribution to the phenotypic difference between the means of the probands on trait X and the population on Y can be estimated by doubling the difference between the cross-trait

Table 3 Bivariate DF extremes analysis of mathematics and reading: MZ and DZ standardized means (SDs), cross-trait probandwise concordances, cross-trait twin group correlations, and bivariate DF genetic, shared environmental and non-shared environmental parameter estimates using a 15% cutoff

Bivariate DF estimates	Proband mean		Cross-trait co-twin mean (SD)		Cross-trait probandwise concordance		Cross-trait twin group correlation		Bivariate DF estimates		
	MZ	DZ	MZ	DZ	MZ	DZ	MZ	DZ	genetic (SE)	shared environmental (SE)	non-shared environmental*
Math → PIAT	−1.87 (.68)	−1.86 (.65)	−.84 (.97)	−.56 (.94)	.38	.30	.45	.30	.24 (.05)	.21 (.06)	.01
PIAT → Math	−1.65 (.50)	−1.62 (.48)	−.86 (1.16)	−.55 (1.16)	.37	.32	.52	.34	.38 (.09)	.14 (.08)	.08

Note: Bivariate DF estimates = cross-trait estimates indicating the extent to which the difference between the mean score of probands on one trait and the population mean on the other trait are explained by genetic, shared and non-shared environmental factors. *The non-shared environmental bivariate DF estimate was derived by subtracting the sum of genetic and shared environmental bivariate DF estimates from the phenotypic group correlation. *N* (mathematics → reading) MZ pairs = 300, DZ pairs = 489; *N* (reading → mathematics) MZ pairs = 308, DZ pairs = 469. The proportion of the phenotypic group correlation that is explained by the genetic, shared environmental, and non-shared environmental bivariate DF estimates can be estimated by dividing these estimates by the phenotypic group correlation. For Math → PIAT, the proportions are .52, .46, and .02 respectively. For PIAT → Math, the proportions are .63, .23, and .14 respectively.

twin group correlations for MZ and DZ twins. *Bivariate group heritability* (h^2g) is the proportion of the phenotypic difference between the probands on trait *X* and the population on *Y* that can be ascribed to genetic factors. Unlike bivariate analysis of individual differences in unselected samples, bivariate DF extremes analysis is directional in the sense that selecting probands on *X* and examining quantitative scores of cotwins on *Y* could yield different results as compared with selecting probands on *Y* and examining quantitative scores of cotwins on *X*.

Bivariate analyses yield an additional statistic called the group correlation which is an index of the extent to which genes that affect trait *X* also affect trait *Y*. In bivariate DF extremes analysis, a *group* genetic correlation can be derived from four group parameter estimates: bivariate group heritability estimated by selecting probands for *X* and assessing cotwins on *Y*, bivariate group heritability estimated by selecting probands for *Y* and assessing cotwins on *X*, and univariate group heritability estimates for *X* and for *Y* (see Knopik et al., 1997). This estimate of group genetic correlation is the central statistic directly relevant to the main question of the present study. Analogous to the more familiar genetic correlation in analyses of individual differences, the group genetic correlation indicates the extent to which genes that are responsible for the mean difference between probands and the population on *X* are also responsible for the mean difference between probands and the population on *Y*.

In this study, bivariate heritability addresses the genetic contribution to the phenotypic difference between the proband mean on reading and the population mean on mathematics as well as the genetic contribution to the phenotypic difference between the proband mean on mathematics and the population mean on reading. Two analyses need to be conducted: selecting probands for poor mathematics performance and comparing co-twin quantitative trait scores on reading (mathematics → reading) and vice versa (reading → mathematics). From these two analyses, bivariate extremes genetic correlation (r_g) can be derived using the following formula (Knopik et al., 1997):

$$r_{g(xy)} = \sqrt{\frac{(\beta_{2(xy)})(\beta_{2(yx)})}{(\beta_{2(x)})(\beta_{2(y)})}}$$

where $\beta_{2(xy)}$ is the bivariate genetic DF estimate for mathematics → reading; $\beta_{2(yx)}$ is the bivariate genetic DF estimate for reading → mathematics; $\beta_{2(x)}$ is the univariate group heritability of mathematics; and $\beta_{2(y)}$ is the univariate group heritability of reading. Similar bivariate DF estimates can be obtained for shared and non-shared environment.

As mentioned above, we selected probands for scores in the lowest 15% of reading and mathematics. For the reading → mathematics analysis, the phenotypic cross-trait group correlation was .60,

Table 4 Comparison between bivariate genetic results for the low extremes and for the entire sample for reading and mathematics test scores

	Individual differences	DF extremes Math → PIAT	DF extremes PIAT → Math
Phenotypic correlation	.50	.46	.60
Bivariate heritability	.49	.63	.52
Genetic correlation	.52	.67	
Bivariate shared environment	.41	.23	.46
Shared environmental correlation	1.00	.96	
Bivariate non-shared environment	.10	.14	.01
Non-shared environmental correlation	.16	.08	

indicating that children with the lowest reading scores also had low mathematics scores. More specifically, the reading probands had reading scores that were 1.6 SD below the population mean on reading (Table 2), and their math scores were .96 SD below the population mean on mathematics. The phenotypic group correlation is the ratio between these proband–population differences: $-.96 \div -1.6 = .60$. As shown in Table 3, the cross-twin twin group correlations are .52 for MZ and .34 for DZ twins; doubling the difference suggests that genetics contributes .36 to the phenotypic cross-trait difference between reading in the probands and mathematics in the population. Bivariate DF extremes analysis yields a similar estimate of .38 (SE = .09). Dividing .38 by the phenotypic cross-trait group correlation of .60 estimates bivariate group heritability as .63, indicating that more than half of the cross-twin covariance between reading and mathematics is mediated genetically. Results for the mathematics → reading analysis were similar. The phenotypic group correlation was .46 and bivariate β_2 estimate was .24. Bivariate group heritability was .52 (i.e., $.24 \div .46 = .52$).

Combining the results for the mathematics → reading analysis and the reading → mathematics analysis and univariate group heritabilities yielded a genetic correlation of .67 (r_g = square root of (.24*.38) / (.47*.43), using the above equation in which .47 and .43 are the univariate group heritabilities for mathematics and reading, respectively (see Table 2).

For shared environment, the bivariate DF estimate (calculated by subtracting bivariate β_2 from the transformed MZ co-twin mean) was .21 for mathematics → reading and .14 for reading → mathematics (see Table 3). Univariate group shared environment was .20 for mathematics and .17 for reading. Combining these four estimates yielded a shared environmental correlation of .96. Non-shared estimates were obtained in the same way, yielding bivariate DF estimates of .01 and .08 and univariate estimates of .37 and .37, and a non-shared environmental correlation of .08.

Table 4 compares the genetic and environmental bivariate extremes results to bivariate results for the entire sample. The results are roughly similar, suggesting general genetic effects that encompass mathematics and reading not only for abilities but also for disabilities. Shared environmental influences also contribute to both mathematics and reading for disabilities as well as abilities. However, non-shared environment largely differentiates mathematics and reading abilities and disabilities.

Discussion

The main aim of our study was to investigate the issue of genetic and environmental relationships between reading and mathematics disability. The use of web-based testing allowed us to collect individual data from a large sample of twins in order to address this issue. The group genetic correlation of .67 between mathematics and reading disabilities found in this study suggests strong genetic overlap between the two disabilities. This result is comparable to the overlap between mathematics and reading abilities found using the same sample and in previous literature and supports the Generalist Genes Hypothesis. However, this result also suggests that some genetic specificity also exists for mathematics and reading.

Consistent with findings from studies addressing the extent to which reading and mathematical abilities are influenced by the same shared environments, we found that the same shared environments influence both disabilities (group correlation of .96). However, shared environment explained very little variance in both traits and thus explained very little of the comorbidity between the two disabilities.

Also similar to previous research, we found that most environmental influences on both mathematics and reading disabilities were non-shared, although this estimate also includes measurement error. This suggests that variation between families in learning environments has limited influence on variation in mathematical and reading skills (Markowitz et al., 2005). Moreover, non-shared environmental overlap between reading and mathematics disabilities was negligible, suggesting that non-shared environment is responsible for dissociations between reading and mathematics disabilities.

The obvious limitation of this study is that no specific genes or environments were assessed. A next step for genetic research is to find genetic markers

associated with mathematical and reading disabilities, which is now made more feasible using genome-wide association strategies that test hundreds of thousands of DNA markers simultaneously (Plomin, 2005). Our results suggest that when such genetic associations are identified, to a large extent the same genes will influence both mathematics and reading. In terms of environmental influences, our results suggest that the search for dissociations between reading and mathematics disabilities should focus on non-shared environment.

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