Genetic Etiology of Reading Difficulties in Boys and Girls

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Although it has been suggested that genetic influences on reading difficulties may differ in boys and girls, results obtained from previous analyses of data from same-sex twin pairs have failed to provide evidence for a differential genetic etiology of reading disability (RD) as a function of gender. However, results of a recent study in which data from both same-sex and opposite-sex twin pairs were analyzed indicated a higher heritability for reading difficulties in boys (Harlaar et al., 2005). Because the current sample of twin pairs tested in the Colorado Learning Disabilities Research Center is substantially larger than that analyzed for our previous report (Wadsworth et al., 2000), this hypothesis was tested more rigorously using data from both same-sex and opposite-sex twin pairs in our current augmented sample. Composite reading scores from 634 twin pairs were subjected to DeFries–Fulker sex-limitation analysis using the model-fitting approach of Purcell and Sham (2003). Analysis of data from the combined sample of male and female twins indicated that genetic influences account for more than half the proband reading deficit (h^2_g = .58). When this model was extended to test for gender differences in the magnitude of genetic influences on RD, h^2_g estimates were somewhat higher for females than for males (.63 and .53, respectively), but the difference was nonsignificant ($p > .3$). A test for qualitative gender differences was also nonsignificant. Thus, these results provide little evidence for a differential genetic etiology of RD in boys and girls.

Numerous studies have compared reading difficulties in boys and girls, noting both mean differences and differences in prevalence rates (e.g., Finucci & Childs, 1981; Harlaar et al., 2005; Katusic et al., 2001; Liederman et al., 2005; Miles et al., 1998; Rutter et al., 2004; Rutter & Yule, 1975; Shaywitz et al., 1990; Stevenson, 1992; Wadsworth et al., 2000). However, there is considerable variation among study results, depending on the ages of the subjects, ascertainment or diagnostic criteria, severity of the disorder, and measures used, with gender ratios ranging from just over 1:1 to as high as 15:1 males to females. The highest ratios have typically been obtained in referred or clinic samples, whereas studies of research-identified samples have observed gender ratios closer to 1:1 (e.g., Harlaar et al., 2005; Shaywitz et al., 1990; Stevenson, 1992; Wadsworth et al., 2000). However, in a recent review of the history of research on gender differences in reading disability (RD), Rutter et al. (2004) presented new evidence from four independent epidemiological studies of data from research-identified subjects, examining the prevalence of RD by gender, both with and without taking IQ into account. The reviewed studies included the Dunedin Multidisciplinary Health and Development Study (Silva, 1990), with data from 989 individuals assessed on measures of reading and IQ, each combined across ages 7, 9 and 11; The Christchurch Health and Development Study (Fergusson et al., 1989), in which IQ and reading data from 895 individuals were averaged across ages 8 to 10; The Office for National Statistics Study (Meltzer et al., 2000), which comprised a population sample in the United Kingdom of 5752 children with reading and IQ assessments at 9 to 15 years of age; and The Environmental Risk Longitudinal Twin Study (Moffitt, 2002), which included 2163 twins from England and Wales, and assessed reading at age 7 and IQ at age 5. In three of the four studies, children were classified as reading-disabled if their reading scores were in the lower 15% of the distribution (non-IQ-referenced), or if their reading scores were 1 standard deviation or more below that expected based on their IQ. In one study (the Office of National Statistics Study), the larger sample allowed for the testing of gender ratios in two severity groups — those in the lower 15% and those in the lower 5% of the distribution of scores on both the non-IQ-referenced reading score, and on an IQ-discrepant score. In all four studies of research-identified probands, the rates of RD were significantly higher in boys, with odds ratios ranging from 1.39 to 3.19 for non-IQ-referenced RD, and from 1.74 to 3.29 when an IQ-discrepant score was used to define RD. The authors concluded that reading disabilities are clearly more frequent in boys than in girls. Thus, although

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the magnitude of the gender ratio has differed widely among studies, an excess of males has typically been observed in both referred and research-identified samples, raising the question: 'Does the etiology of reading difficulties differ in boys and girls?'

It has been suggested that one possible explanation for the overrepresentation of boys among children with reading difficulties is that girls may be less susceptible than boys to environmental influences, such as teaching methods, differences in socioeconomic status or societal pressures (Geschwind, 1981). If this is the case, genetic influences may be more important as a cause of reading difficulties in girls than in boys.

Results of studies investigating possible differential genetic influences on group deficits in reading performance of boys and girls have been mixed. For example, using data from a population sample of 13-year-old twins, including 102 identical pairs (monozygotic [MZ]; 47 male, 55 female) and 111 same-sex fraternal pairs (dizygotic [DZ]; 52 male, 59 female), Stevenson (1992) demonstrated the effect of different methods of identification of RD on both the gender ratio obtained, and on the heritability of the group deficit in reading. Three different methods of identifying RD were employed (including regression, discrepancy and age-only definitions) and applied to either the combined sample of males and females or to 'separate-sex' samples, both with and without an IQ restriction. This resulted in 12 separate definitions of RD. Scores on a composite measure of reading (consisting of the Neale Analysis of Reading Ability Accuracy and Comprehension subtests [Neale, 1967] and the Schonell Single Word Reading and Spelling subtests [Schonell & Schonell, 1960]) were subjected to DeFries–Fulker (DF) multiple regression analysis for selected twin data (DeFries & Fulker, 1985, 1988) to assess the extent to which the deficit of the probands was due to genetic influences (h^2_g). The average h^2_g estimate across definitions with no IQ restriction was .24 for males, and .44 for females. Although separate h^2_g estimates were not provided for males and females when an IQ restriction was imposed, Stevenson (1992) noted that when IQ was taken into account, heritability estimates were higher for males. However, samples of subjects classified as reading-disabled by each definition were small, ranging from a combined sample of 15 to 70 pairs. As a result, none of the differences reported was significant.

In a previous analysis of data from the Colorado Learning Disabilities Research Center (CLDRC) in which probands were selected from 8- to 20-year-old twin pairs with a history of reading problems, there was virtually no difference in the magnitude of genetic influence for boys and girls on the group deficit in performance on a composite measure of reading (Wadsworth et al., 2000). Data from 206 MZ (99 male, 107 female) and 159 same-sex DZ (90 male, 69 female) twin pairs, in which at least one member of each pair was reading-disabled, were analyzed. Employing an IQ restriction of 90 or above on either the Verbal or Performance scale of the Wechsler Intelligence Scale for Children — Revised (WISC-R; Wechsler, 1974) or Wechsler Adult Intelligence Scale — Revised (WAIS-R; Wechsler, 1981), discriminant function scores based on the Reading Recognition, Reading Comprehension and Spelling subtests of the Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970) were subjected to DF analysis. Estimates of h^2_g were .58 for males and .59 for females, providing no evidence for the hypothesis of greater genetic influence on reading difficulties in girls than in boys.

Stevenson (1992) and Wadsworth et al. (2000) both employed DF analysis for selected twin data. DeFries and Fulker (1985, 1988) suggested that when probands are selected because of deviant scores on a continuous variable, such as reading performance, the differential regression of MZ and DZ co-twin means toward the mean of the unselected population provides a test for genetic etiology. To the extent that the deficit of the probands is due to genetic influences, the mean of DZ co-twins (who share half their segregating genes, on average, with the DZ probands) is expected to regress more toward the mean of the unselected population than is the mean of MZ co-twins (who are genetically identical to the MZ probands). Purcell and Sham (2003) recently suggested a model-fitting implementation of the DF model which, among other benefits, permits the inclusion of data from opposite-sex twin pairs in order to test both the relative balance of genetic and environmental influences, and estimate the genetic correlation between male and female co-twins, and thus also test for qualitative/gender-specific effects.

Harlaar et al. (2005) recently employed the methodology of Purcell and Sham (2003) to analyze data from a population sample of 7-year-old twins. Data on a composite of the Sight Word Efficiency and Phonemic Decoding Efficiency subtests of the Test of Word Reading Efficiency (TOWRE; Torgesen et al., 1999) from pairs in which at least one member scored at or below the 5th percentile (153 MZ, 127 same-sex DZ and 112 opposite-sex DZ pairs) and from pairs in which at least one member scored at or below the 10th percentile (308 MZ, 246 same-sex DZ and 228 opposite-sex DZ pairs) were analyzed. A significant difference in the magnitude of h^2_g estimates was obtained for subjects scoring at or below the 5th percentile, with higher heritability for males than for females (h^2_g = .72 and .37, respectively). Although the quantitative gender difference was not significant in the lower 10% of the distribution, evidence was obtained for *qualitative* gender differences in this group (i.e., the genetic correlation between members of opposite-sex pairs was less than .5, and the shared environmental correlation was less than 1.0), suggesting that different genetic and environmental factors may influence the development of reading difficulties in males and females.

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Since data from the CLDRC were last analyzed for gender differences (Wadsworth et al., 2000), the sample has increased by about 25%. Further, data from opposite-sex pairs were not used in those analyses. The purpose of the current study, therefore, was to reassess the contributions of genetic and environmental influences on the group deficits of male and female probands by subjecting data from this larger sample, including data from opposite-sex twins, to DF analysis using the model-fitting implementation of Purcell and Sham (2003), and thereby to test both for differences in the magnitude of genetic influence and for qualitative/gender-specific effects.

Materials and Methods

Subjects and Measures

The subjects were participants in either the Colorado Reading Project (DeFries, 1985; DeFries et al., 1991) or the CLDRC (DeFries et al., 1997), an ongoing study of genetic and environmental influences on RD and attention-deficit hyperactivity disorder (ADHD). In order to minimize the possibility of ascertainment bias, twin pairs are systematically ascertained through 27 cooperating school districts in the state of Colorado. First, without regard for reading status, all twin pairs in a school are identified by school personnel. Permission is then sought from parents to review the twins' records for evidence of reading problems (e.g., low reading achievement test scores, reports by classroom teachers or school psychologists, referral to resource rooms or reading therapists). Twin pairs in which at least one member has a school history of reading problems are invited to participate in the study at the University of Colorado, Boulder, and the University of Denver, where they are administered an extensive battery of psychometric tests, including the PIAT (Dunn & Markwardt, 1970) and the WISC-R (Wechsler, 1974) or the WAIS-R (Wechsler, 1981), as well as various other tests of cognitive abilities, achievement, and reading component processes. Data from the Reading Recognition, Reading Comprehension, and Spelling subtests of the PIAT are used to compute a discriminant function score for each child using discriminant weights based on data from an independent sample of 140 reading-disabled and 140 nontwin control children tested in the Colorado Reading Project (DeFries, 1985). In order for a twin pair to be included in the current proband sample, at least one member of the pair must be classified as reading-disabled by the discriminant function score. In addition he or she must have a positive school history of reading problems, a verbal or performance IQ of at least 90, no evidence of serious emotional or behavioral problems, and no uncorrected visual or auditory acuity deficit.

Zygosity of same-sex pairs was determined using selected items from the Nichols and Bilbro (1966) questionnaire, which has a reported accuracy of 95%. In questionable cases, twin pairs were genotyped using polymorphic DNA markers from blood or buccal samples. The twins were reared in primarily English-speaking, middle-class homes, and were between 8 and 20 years of age at the time of testing, with a mean age of 11.5 years. The current sample consists of 264 pairs of MZ twins (129 male, 135 female), 214 pairs of same-sex DZ twins (121 male, 93 female), and 156 pairs of opposite-sex DZ twins, and has a proband gender ratio of 1.14:1.

Analyses

To test the hypothesis of differential genetic etiology of reading difficulties as a function of gender, the data were subjected to a model-fitting implementation of the DF model for selected twin data (Purcell & Sham, 2003) using the Mx Statistical Modeling package (Neale et al., 2002). DeFries and Fulker (1985) suggested that when probands are selected because of deviant scores on a continuous variable, such as reading performance, a statistically powerful and versatile test of genetic etiology is obtained by fitting the following multiple regression model to the selected twin data:

$$C = B_1P + B_2R + K \qquad [1]$$

where C is the co-twin's score, P is the proband's score, R is the coefficient of relationship (1.0 for MZ pairs and .5 for DZ pairs), B_1 and B_2 are the regression coefficients, and K is the regression constant. When applied to data from selected twin pairs, B_2 provides a direct test of genetic etiology. Further, when each subject's score is appropriately transformed (i.e., expressed as a deviation from the mean of the unselected population and divided by the difference between the mean of the probands and that of the unselected population), $B_2 = h^2_g$, an estimate of the extent to which the deficit of the probands is due to heritable influences (DeFries & Fulker, 1988).

The model-fitting implementation of the DF model (Purcell & Sham, 2003) facilitates the inclusion of data from opposite-sex twin pairs in a sex-limitation analysis (Figure 1) that tests the relative balance of genetic and environmental influences (e.g., a_M vs. a_F, that is, *quantitative* gender differences) and estimates the genetic correlation between male and female co-twins (r_A), to test also for *qualitative* genetic gender differences. Qualitative gender differences are suggested when the correlation between opposite-sex DZ twins is significantly less than the correlation between members of same-sex DZ pairs, thereby providing evidence that different genes may be associated with the trait or deficit in males and females. If the same genetic influences are operating in males and females, the genetic correlation is expected to be .5. A genetic correlation of less than .5 suggests different sources of genetic influence on reading difficulties in males and females (i.e., qualitative gender differences). To test for gender differences in the magnitude of genetic influence, the additive genetic parameters for males and

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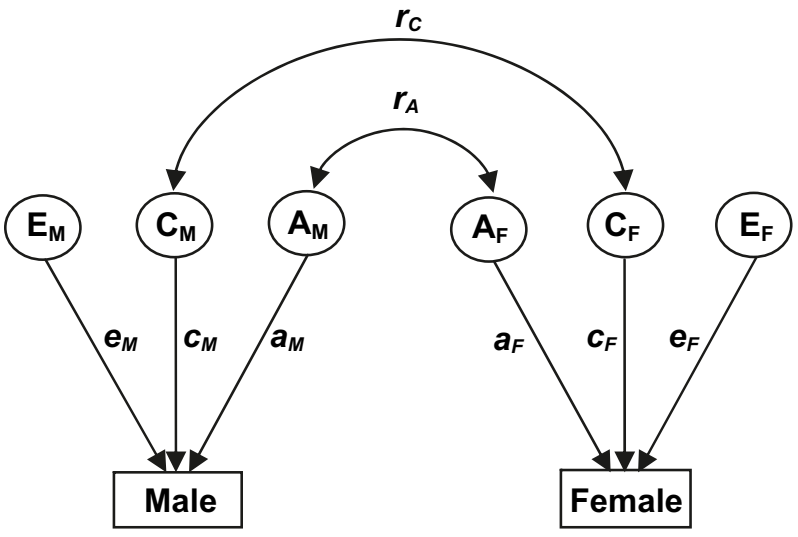

Figure 1

Sex-limitation model for opposite-sex twin pairs.

females (a_M and a_F) are constrained to be equal, and the change in chi-square evaluated. Similarly, qualitative gender differences are assessed by constraining the genetic correlation between male and female members of opposite-sex pairs to equal .5 and evaluating the change in chi-square.

Results

Table 1 presents proband and co-twin means for MZ and DZ male and female pairs as well as for DZ opposite-sex pairs for the current sample, expressed as deviations from the mean of 1097 control twins. Proband means of all groups are more than 2 standard deviations below the mean of the controls. The mean difference between MZ male and female probands is highly significant ($p \leq .001$), as is the difference between the DZ male and female probands when same-sex and opposite-sex DZ pairs are combined ($p \leq .002$). As expected, the means of the DZ co-twins have regressed more toward the mean of the controls than those of the MZ co-twins, suggesting genetic influences on reading difficulties in both males and females in this sample.

When the data were transformed and subjected to the Mx implementation of the DF model, genetic influences accounted for more than half the group deficit in reading performance in the combined sample of males and females (h^2_g = .58; 95% Confidence Intervals [CI] = .43–.71). When this model was extended to test for gender differences in the magnitude of genetic influences on RD, estimates of the heritability of the group deficit in reading (h^2_g) were somewhat higher for females (h^2_g = .63; 95% CI = .43–.79) than for males (h^2_g =.53; 95% CI = .33–.71). However, confidence intervals are broad and overlapping, suggesting that these estimates may not differ significantly. Further, the opposite-sex DZ genetic correlation was estimated at .5, suggesting that the same genes are operating in males and females.

Hypothesis testing indicated that, as expected, there is no significant difference between h^2_g estimates for males and females in this sample ($p > .3$; Table 2). Further, constraining the opposite-sex DZ genetic correlation (r_A) to equal .5 produced no deterioration of fit, confirming that there is no evidence for qualitative gender difference.

Although estimates of h^2_g do not differ significantly for males and females in this sample, they differ in

Table 1

Mean Discriminant Scores by Gender and Zygosity[a]

	Probands	Co-twins	*N* pairs
Identical			
Male	–2.81	–2.60	129
Female	–2.35	–2.13	135
Fraternal			
Male	–2.65	–1.73	121
Female	–2.46	–1.42	93
Opposite-sex	–2.55	–1.59	156

Note: a = expressed as deviations from the mean of 1097 control twins.

Table 2

Model Comparisons for Sex-Limitation

Model	–2LL	NPAR	$\Delta\chi^2$	Δdf	*p*
Full model	2321.41	7			
$a_m = a_f$	2322.00	6	.59	1	> .3
Fix r_A (older)	2321.41	6	.00	1	1.0

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direction of effect from those of Harlaar et al. (2005), the only study to report significant differential genetic etiology of reading difficulties as a function of gender. One possible reason for the disparate study findings may be the ages of the participants. The participants in the study by Harlaar et al. (2005) were all 7 years of age. In addition, although Stevenson (1992) found no significant differences in heritability estimates for males and females, the estimates across different definitions of RD suggested a trend toward higher heritability among females in the sample of 13-year-olds when no IQ restriction was imposed. In the current sample, participants ranged in age from 8 to 20 years. Therefore, given the mixed results of these three studies, and the finding that those studies with older subjects (Stevenson, 1992; Wadsworth et al., 2000) observed trends in the opposite direction to that of Harlaar et al. (2005), there may be developmental gender differences in the heritability of RD, such that genetic influences are more important for males in middle childhood, but more important for females in adolescence. In order to explore the effect of age on differential heritability of reading difficulties in males and females, the current sample was divided into younger and older age groups, based on the mean age of 11.5, and the data again subjected to DF sex-limitation analysis (Purcell & Sham, 2003). The younger and older groups had mean ages of 9.6 and 14.1 years, respectively. As indicated in Table 3, the trend of higher heritability among females persisted in the younger group, and the gender difference was more pronounced, with the heritability slightly higher among younger females than it was in the combined sample of females. Further, it appears that genetic influences on reading difficulties in the younger group are responsible for the observed trend toward higher heritability of RD among females in the combined sample, since the h^2_g estimates of males and females in the older group were almost identical. There is, however, some hint of a possible qualitative gender difference in the older group, as indicated by the estimated genetic correlation of less than .5 for members of opposite-sex DZ pairs.

Table 3

Genetic Etiology of Reading Difficulties in Males and Females as a Function of Age[a]

	h^2_g (95% CI)
Younger (≤ 11.5 years)	
Male	.53 (.28, .77)
Female	.67 (.43, .85)
r_A = .50 (.33, .50)	
Older (> 11.5 years)	
Male	.55 (.25, .84)
Female	.53 (.19, .83)
r_A = .45 (.08, .50)	

Note: a = estimated from the full sex-limitation model.

Table 4

Model Comparisons for Sex-Limitation as a Function of Age

Model	–2LL	NPAR	$\Delta\chi^2$	Δdf	p
Full model	2314.05	12			
$a_{ym} = a_{yf}$	2314.72	11	.67	1	> .3
Fix r_A (older)	2314.18	11	.13	1	> .7
Eq. *a*, fix r_A, all groups	**2314.97**	**7**	**.92**	**5**	**> .95**

As suggested by the overlapping confidence intervals, hypothesis testing indicated no significant difference between h^2_g estimates for young males and females — the estimates were equated without a significant loss of model fit ($p > .3$; Table 4). In addition, the genetic correlation between members of opposite-sex pairs in the older group was constrained to .5 with no deterioration of fit, indicating that the slight qualitative gender difference observed in this group was not significant ($p > .7$). Finally, estimates of h^2_g could be equated across all groups, and the opposite-sex DZ genetic correlation fixed at .5 with no significant deterioration of fit.

Another difference between the current study and that of Harlaar et al. (2005) is the measure of reading employed. Results of that study were based on a composite measure of word recognition alone, rather than a more general measure of reading and spelling, as was used in the current study. Similarly, the trend of higher heritability among females found by Stevenson (1992) was obtained using a composite measure of reading and spelling. Therefore, it is conceivable that the pattern of heritability for males and females may differ depending on the measure of reading used. In order to explore this possibility, data from a single measure of word recognition, the PIAT Reading Recognition subtest (Dunn & Markwardt, 1970), were subjected to the DF sex-limitation analysis (Purcell & Sham, 2003). Using this measure of word recognition, with probands defined as those scoring 1.5 standard deviations below the control mean, the results were highly similar to those obtained using our composite measure (h^2_g = .54 for males and .66 for females). However, this is not surprising as, of the three subtests included in our discriminant function score, this measure is the most highly correlated with the reading composite (r = .96).

Finally, differences in study results may be related to ascertainment criteria, such as an IQ cut-off or use of a discrepancy score. In the study of Harlaar et al. (2005) the reported results were obtained using a non-IQ-referenced definition of reading disability, although the authors noted that results were highly similar when reading difficulties were defined using a regression discrepancy method. Stevenson (1992) reported that use of an IQ restriction resulted in higher heritability in males, but heritability estimates were not reported. In

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the current study, subjects were required to have either a Verbal or Performance IQ of at least 90 to be classified as probands. In order to explore further the possibility of differences resulting from the use of an IQ-referenced definition of RD, the current analyses were performed with no IQ restriction. Results of this analysis were highly similar to the original analyses, with h^2_g estimates of .64 for females and .52 for males. Although the sample was slightly larger when no IQ restriction was imposed, this difference between h^2_g estimates for boys and girls was also not significant.

Discussion

Whereas gender ratios as high as 15:1 males to females have been reported among referred or clinic samples of children with RD, those among research-identified samples have typically been closer to 1:1. However, odds ratios in the four research-identified samples reported by Rutter et al. (2004) ranged from 1.39 to 3.29, supporting suggestions that the prevalence of RD is significantly higher among boys than girls. The gender ratio in the current sample was 1.14:1. As female identical twin pairs tend to be overrepresented in twin studies (Lykken et al., 1978), this relatively low gender ratio may have been due in part to a differential volunteer rate for male and female identical twin pairs. In agreement with this expectation, the gender ratio in our sample of identical twin probands is lower (0.98:1) than that for the DZ probands (1.29:1), which is comparable to the lower-bound estimate reported by Rutter et al. (2004).

Results of previous studies assessing the etiology of reading deficits in males and females have been mixed (Harlaar et al., 2005; Stevenson, 1992; Wadsworth et al., 2000). Only one of these studies reported evidence for significant differential heritability (Harlaar et al., 2005), citing higher heritability in males among those scoring in the lower 5% of a population sample, and qualitatively different genetic influences for males and females among those scoring in the lower 10% of the distribution. Results of the current study suggest that genetic influences account for more than half of the group deficit in reading performance in both males and females. Although there is a trend toward higher heritability of the group deficit in reading performance among females than among males, no significant quantitative or qualitative gender difference was observed in this sample. These results suggest that genetic influences on reading deficits are similarly important in boys and girls, and that the *source* of genetic influence appears to be the same in both. Similar findings were reported by Bates et al. (2004) based on the assessment of reading and spelling performance in a population sample of adolescent twins. In that sample, heritabilities for individual differences in normal-range reading performance tended to be higher in females than in males, but not significantly so, and there were no significant sex-specific effects.

There are, however, some dissimilarities between this study and previous studies of gender differences in the etiology of reading deficits, including differences in the ages of the subjects, selection criteria, and measures used. For example, Harlaar et al. (2005) analyzed data from a population sample of 7-year-old twins, Stevenson analyzed data from a population sample of 13-year-old twins, and in the current study data from 8- to 20-year-old twin pairs in which at least one member of the pair was classified as reading-disabled were analyzed. Thus, it is possible that the age differences between these samples could result in different patterns of both quantitative and qualitative genetic influences. Perhaps genetic influences are more important as a cause of reading difficulties in younger males, whereas these influences may become less important in older males, as they may gain more exposure to print and/or remediation. Similarly, the effect of genes activated in middle childhood in males may begin to wane by adolescence. Such effects would be masked in the current sample with its wide age range. However, when the sample was divided into younger and older age groups, based on the mean age of 11.5 years, there appeared to be a difference in the heritability patterns for males and females in the two age groups, but in the opposite direction to that of Harlaar et al. (2005). The trend of higher heritability of reading deficits among females was more pronounced in the younger group, but still nonsignificant, whereas there was virtually no difference between heritability estimates of older males and females. Thus, in the current sample, there was no significant difference in the magnitude of genetic influences on the group deficit in reading performance in either younger or older groups of readers. Although these findings suggest that the differing results between the current study and that of Harlaar et al. (2005) are not attributable to developmental differences, it is important to note that in the current sample, even the youngest subjects are older than the subjects of Harlaar et al. In addition, each of the two age groups included in the current study comprise a range of ages. Therefore, a more appropriate test of developmental differences might be accomplished using longitudinal assessments. To this end, follow-up data are being collected on a subset of CLDRC participants approximately 5 years after their initial participation (NIDCD grant DC-05190). Once the sample size is sufficient, it will be possible to test the hypothesis of differential genetic etiology of RD in males and females as a function of age longitudinally rather than cross-sectionally. However, this does not address the possibility that genetic influences may be more important as a cause of RD in males at ages younger than those of participants in the CLDRC. Fortunately, data are also being collected from a population sample of twins aged 4 to 7 years in order to address questions surrounding the development of reading ability and disability (Byrne et al., 2002). Once sufficient data are available it will be

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possible to revisit the question of age and its relation to gender differences in the etiology of RD.

Another difference between our study and that of Harlaar et al. (2005) is the measure of reading used. In the current study, a composite measure of global reading performance was used that included measures of word recognition, reading comprehension and spelling. In contrast, Harlaar et al. (2005) used a composite measure of word recognition alone. It is possible that the different skills required by these different tasks may be more or less heritable in males or females. Therefore, data from only the Reading Recognition subtest of the PIAT were analyzed. As expected, given that the reading composite and this subtest are highly correlated, a highly similar result was obtained. Although these results suggest that differences in study findings are not related to the measure of reading employed, it is worth noting that the measures of word recognition were different in each study. Harlaar et al. (2005) used the TOWRE (Torgesen et al., 1999), a timed test of word recognition, which was administered by phone. In the current study, the untimed PIAT (Dunn & Markwardt, 1970) was administered in person. Thus, it is possible that our differing results could be related to specific differences between the measures, including method variance. As the TOWRE is currently being administered to a subset of participants in the CLDRC, it will eventually be possible to explore the effect of the specific reading measure on estimates of the etiology of reading difficulties in males and females more rigorously.

Also, the studies of Stevenson (1992), Harlaar et al. (2005) and this study all used different definitions of RD. In particular, the use or nonuse of an IQ-referenced definition may affect estimates of heritability. Harlaar et al. (2005) reported virtually no difference between heritability estimates obtained using a non-IQ-referenced definition and a discrepancy score, whereas Stevenson (1992) reported higher heritability estimates for males when an IQ restriction was imposed. In contrast, when the IQ restriction was removed in the current study, and the analyses performed on data from this slightly larger sample, the results were highly similar to the original results, and the gender difference remained nonsignificant.

Finally, one possible difference between these studies that has not been addressed is the severity of subjects' reading deficit. The mean of probands scoring in the lower 5% of the population sample of Harlaar et al. (2005) was approximately 1.85 standard deviations below the mean of the full sample. Stevenson (1992) used several different scores, generally requiring that probands score at least 1 standard deviation below the mean of the entire sample. In the current study, probands scored an average of approximately 2.5 standard deviations below the mean of the controls. Therefore, several differences among these studies may account for their differing results. However, results of the present study continue to provide little evidence for gender differences in the etiology of reading difficulties.

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