Reading and Writing (2007) 20:13–25
DOI 10.1007/s11145-006-9016-z
© Springer 2006

Etiology of reading difficulties as a function of gender and severity

JESSE L. HAWKE, SALLY J. WADSWORTH,
RICHARD K. OLSON and JOHN C. DEFRIES
Institute for Behavioral Genetics, University of Colorado, Boulder, CO, USA

Abstract. To test the hypothesis that the etiology of reading difficulties may differ for males and females in more severely impaired samples, reading performance data from monozygotic (MZ), same-sex dizygotic (DZ_{ss}), and opposite-sex dizygotic (DZ_{os}) twin pairs were analyzed using a model-fitting implementation of the DeFries-Fulker (DF) model (Purcell & Sham, 2003, *Behavior genetics, 33*, 271–278). Five non-independent samples were selected using cut-offs of −1 (N = 737 pairs), −1.5 (N = 654), −2 (N = 468), −2.5 (N = 335), and −3 (N = 198) standard deviations (s) below the mean composite reading score of control twins. Male/female gender ratios for children with reading difficulties were significantly higher than 1.0 for all five samples and increased as a function of severity (viz., 1.15, 1.17, 1.40, 1.61, and 1.88, respectively). When the DF model was fit to the data, estimates of heritability (h_g^2) and shared environmental influences (c_g^2) were not significantly different for males and females in any of the groups. Consequently, the most parsimonious model that provided a good fit to the data at all five levels of severity equated the heritabilities and shared environmental influences for males and females, and fixed the DZ_{os} coefficient of genetic relatedness at 0.5. Thus, these results provide no evidence for a differential etiology of reading difficulties as a function of gender in more severely impaired samples, and suggest that the same genetic and environmental influences contribute to reading difficulties in males and females, irrespective of severity.

Key words: Gender, Genetics, Heritability, Reading Disability, Twins

Introduction

The ratio of males to females in samples of children with reading difficulties varies widely depending upon the ascertainment method employed. In studies where subjects are ascertained employing clinical or referral methods, gender ratios range from 2:1 to 15:1 males to females (e.g., Finucci & Childs, 1981; Vogel, 1990); however, in research-identified samples, gender ratios are closer to 1:1 (e.g., Harlaar, Spinath, Dale, & Plomin, 2005; Hawke, Wadsworth, & DeFries, 2006; Stevenson, 1992; Wadsworth & DeFries, 2005; Wadsworth, Knopik, & DeFries, 2000). Nevertheless, in both referred and research-identified samples, greater numbers of males with reading problems have typically been reported. For example, in a recent review of sex differences in reading disability, Rutter

et al. (2004) reported the gender ratios in four independent epidemiological studies in which the samples had been ascertained using research criteria. In 3 of the 4 studies, subjects were classified as reading-disabled if their reading score fell in the lower 15% of the distribution (non-IQ referenced), or if their reading score was 1s or more below that expected based on their IQ. Due to a substantially larger sample size in the fourth study, gender ratios were assessed at two different levels of severity, the lower 15% and the lower 5% of the distribution. In all four of the studies, significantly more males than females with reading disabilities were reported, with odds ratios ranging from 1.39 to 3.19 for non-IQ referenced reading disability and 1.74 to 3.29 when an IQ-discrepant score was used. Moreover, Olson (2002) has recently reported that this male to female ratio is greater in more severely affected samples of children with reading difficulties tested in the Colorado Learning Disabilities Research Center.

Several biological and environmental hypotheses have been proposed to account for this difference in prevalence rates between males and females including X-linked recessive inheritance (Symmes & Rapoport, 1972), differences in brain functioning due to differential exposure or sensitivity to androgens (Geschwind, 1981; Nass, 1993; Tallal & Fitch, 1993), immunological factors, sexual imprinting, perinatal complications, and differential resilience to neural insult (Liederman, Kantrowitz, & Flannery, 2005). It has also been suggested that females may be less susceptible to environmental factors such as teaching methods and socioeconomic status (Geschwind, 1981), and that genetic influences may be more important as a cause of reading difficulties in females than in males (DeFries & Gillis, 1993; Stevenson, 1992). However, recent studies that have examined the genetic influences of reading difficulties in males and females have obtained mixed results (Harlaar et al., 2005; Hawke, Wadsworth, & DeFries, 2006; Wadsworth & DeFries, 2005).

Harlaar et al. (2005) recently analyzed data from children participating in a large, longitudinal twin study in England and Wales that suggested that genetic influences might be more important as a cause of reading difficulties in males than females. In their study, analyses of individual differences were conducted in the entire sample of twin pairs; moreover, analyses of deficits in two extreme groups, twin pairs in which at least one member of the pair scored in the 10th percentile and twin pairs where at least one member of the pair scored in the 5th percentile, were also conducted using a model-fitting implementation of the DeFries-Fulker multiple regression analysis (Purcell & Sham, 2003). For their measure of reading ability, Harlaar et al. (2005) used composite scores calculated from two subtests of the Test of Word Reading Efficiency (TOWRE; Torgesen, Wagner, & Rashotte, 1999), the Sight Word Efficiency subtest,

which assesses the ability to read aloud printed real words, and the Phonemic Decoding Efficiency subtest, which assesses the ability to read aloud pronounceable printed non-words.

For the individual differences analyses, estimates of additive genetic influences on reading ability were similar for males and females, 0.65 and 0.67, respectively. Thus, no evidence for quantitative sex differences was found. However, evidence for a significant qualitative sex difference was indicated. The coefficient of genetic relatedness (r_A) between DZ_{os} twin pairs was significantly less than 0.5 (r_A = 0.38; 95% CI = [0.28, 0.47]), suggesting that genetic influences on individual differences in reading performance may differ for males and females even though the magnitudes of these influences are similar.

For the extremes analyses (Harlaar et al., 2005), estimates of additive genetic influences on reading difficulties were greater for males than for females at both the 10% (0.67 vs. 0.50) and 5% cut-offs (0.72 vs. 0.37). Although these estimates were not significantly different at the 10% cut-off, evidence was found for significant qualitative sex differences in this sample (r_A = 0.23; 95% CI = [0.00, 0.41]). Moreover, the estimate of heritability for males at the 5% cut-off was significantly larger than that for females, suggesting that genetic influences may be more important as a cause of reading difficulties in males than in females, and that this difference may be greater in more severely impaired samples.

Two recent analyses of data from the Colorado Twin Study have yielded results that differ from those of Harlaar et al. (2005). The first study tested the hypothesis of a differential genetic etiology of reading difficulties as a function of gender and age (Hawke, Wadsworth, & DeFries, 2006). Participants were same-sex twin pairs tested in the Colorado Learning Disabilities Research Center in which at least one member of the pair had a reading problem. A discriminant function score was calculated using discriminant weights estimated from an analysis of Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970) subtest data for Reading Recognition, Reading Comprehension, and Spelling obtained from an independent sample (DeFries, 1985), and used as a composite measure of reading performance. Estimates of h_g^2, an index of the extent to which the deficit of the probands is due to heritable influences (DeFries & Fulker, 1988), were obtained using DF multiple regression analyses (DeFries & Fulker, 1985, 1988).

Heritability estimates for males and females, prior to age categorization, were 0.54 and 0.65, respectively. Although the estimate of h_g^2 was somewhat greater for females, this difference was not significant ($p \geq 0.35$). When participants were grouped by gender and age, using the mean age of 11.5 years to demarcate younger and older twin pairs, estimates of h_g^2

were nearly identical for younger males (0.54), older males (0.53), and older females (0.53). The estimate of h_g^2 was greater for younger females (0.74), but the test of the two-way interaction of age and gender was not significant. Thus, although there was a trend toward higher heritability in younger females, the results did not support the hypothesis of a differential genetic etiology as a function of gender and age, and the nonsignificant gender difference that was observed was opposite to that reported by Harlaar et al. (2005).

Subsequently, Wadsworth and DeFries (2005) analyzed data from identical, same-sex fraternal, and opposite-sex fraternal twin pairs in the Colorado Twin Study using the model-fitting implementation of the DeFries-Fulker multiple regression analysis (Purcell & Sham, 2003). Similar to the results of Hawke et al. (2006), Wadsworth and DeFries (2005) observed a trend toward higher heritability for females (h_g^2 = 0.63) than for males (h_g^2 = 0.53) prior to grouping by age, but this difference also was not significant ($p > 0.3$). Moreover, the estimated opposite-sex DZ genetic correlation was 0.5, suggesting that the same genetic factors are influencing reading difficulties in boys and girls. Subjects were then grouped by gender and age again using the demarcation of 11.5 years to separate twin pairs into younger and old groups. In general, estimates of h_g^2 were similar to those reported by Hawke et al. (2006); however, with the inclusion of DZ_{os} twin pairs, the estimate of heritability for the younger females was somewhat lower (viz., 0.67 vs. 0.74). Consequently, differences between the estimates of h_g^2 in males and females were not significant in either the younger or older groups. Moreover, no evidence for qualitative sex differences was found in either age group.

Because Harlaar et al. (2005) found a significant difference between the estimates of h_g^2 for males and females at the 5% cut-off, but not at the 10% cut-off, genetic influences on reading disability in boys and girls may differ more in more highly selected samples. Therefore, the current study has two main purposes: first, to assess the gender ratio of reading disability as a function of severity; and, second, to test the hypothesis that the etiology of reading difficulties differs more between males and females in more severely impaired samples.

Methods

Sample and Measures

Subjects were participants in either the Colorado Reading Project (DeFries, 1985; DeFries, Olson, Pennington, & Smith, 1991) or the

Colorado Learning Disabilities Research Center (DeFries et al., 1997). To reduce the possibility of ascertainment bias, school personnel in 27 different school districts within the state of Colorado identify twin pairs without regard to reading performance. Once identified, parental permission is requested to review the school records of both members of the twin pair for any evidence of reading problems. If either member of a pair has a positive school history of reading difficulties, both are invited to participate in the study at the University of Colorado, Boulder, and the University of Denver. A comparison sample of twin pairs, in which both members of the pair have a negative school history of reading problems, was matched to the reading disabled sample when possible on the basis of age, gender, and zygosity.

Twin pairs are administered a battery of psychometric tests including the PIAT (Dunn & Markwardt, 1970) and the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974), or the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981). A discriminant function score is then computed for each participant using discriminant weights estimated from an analysis of PIAT Reading Recognition, Reading Comprehension, and Spelling data obtained from an independent sample of 140 reading disabled and 140 control non-twin children (DeFries, 1985). To participate in the current study, at least one member of the twin pair must have a positive school history for reading problems and be classified as affected by the discriminant function score. He or she must also have a verbal or performance IQ of at least 90, no evidence of neurological problems, no evidence of serious behavioral or emotional problems, and no uncorrected visual or auditory acuity deficits.

Participants in the current study were reared in primarily English-speaking , middle class homes. Ages ranged from 8 to 20 years at the time of testing, with a mean age of 11.5 years. Selected items from the Nichols and Bilbro (1966) questionnaire, which has a reported accuracy of 95%, were used to determine the zygosity of same-sex twin pairs. In questionable cases, blood or buccal samples were obtained and twin pairs were genotyped using DNA markers.

Five non-independent samples of twin pairs were then selected in which at least one member of the pair had a discriminant function score that was 1.0, 1.5, 2.0, 2.5, or 3.0 standard deviation units below the control population's mean composite reading score. If, for example, one member of a twin pair had a discriminant function score of −1.7, then data from that twin pair would be included in both the −1.0 and −1.5 severity groups. Table 1 lists the numbers of MZ, DZ_{ss}, and DZ_{os} twin pairs that were included in the five severity-group samples.

Table 1. Sample sizes (twin pairs) by zygosity and severity.

Cut-off	MZ	DZ_{ss}	DZ_{os}
−1	306	247	184
−1.5	273	218	163
−2	192	161	115
−2.5	141	114	80
−3	90	63	45

Analyses

Single degree of freedom chi-square tests were used to assess the significance of gender ratios at each of the five levels of severity. Preliminary evidence for the genetic etiology of reading difficulties in males and females at the different levels of severity was then obtained by comparing probandwise concordance rates for same-sex MZ and DZ twin pairs and pairwise concordance rates for DZ_{os} twin pairs. MZ and same-sex DZ concordance rates were analyzed using a 2×2 (zygosity $\times$ concordance) chi-square contingency table to test for initial evidence of quantitative genetic influences. Similarly, same-sex DZ and opposite-sex twin pair concordance rates were analyzed using a 2×2 chi-square contingency table to test for preliminary evidence of qualitative sex differences. To test for a possible differential genetic etiology of reading difficulties as a function of proband gender, loglinear analyses of the interaction between concordance, zygosity, and gender were then conducted for each severity level.

Although a comparison of MZ and DZ concordance rates may be employed to test for a genetic etiology, when deviant scores on a continuous measure like reading ability are being analyzed, the differential regression of MZ and DZ cotwin means toward the mean of the unselected population provides a more appropriate test. Because MZ twins are genetically identical, whereas DZ twins share only half of their segregating genes on average, the scores of DZ cotwins should regress more toward the mean of the unselected population if the variable under investigation is at least partially heritable. Therefore, if the means of the MZ and DZ probands are approximately equal, a *t*-test of the difference between the MZ and DZ cotwin means will provide a test of genetic etiology. However, DeFries and Fulker (1985, 1988) suggested that fitting the following regression model to the selected twin data provides a more general, statistically powerful, and versatile test:

$$C = B_1P + B_2R + K \quad (1)$$

where C is the cotwin's score, P is the proband's score, R is the coefficient of relationship (coded as 1.0 for MZ pairs and 0.5 for DZ pairs), B_1 and B_2 are the partial regression coefficients, and K is the regression constant. B_2 equals twice the difference between MZ and DZ cotwin means after covariance adjustment for differences between MZ and DZ proband means. Therefore, B_2 provides a test of statistical significance for genetic etiology. Also, when the data have been suitably transformed prior to multiple regression analysis (i.e., each score is expressed as a deviation from the mean of the control population and then divided by the difference between the proband and control means), B_2 directly estimates h_g^2.

The model-fitting implementation of the DeFries-Fulker regression analysis facilitates the inclusion of DZ_{os} twin pair data in a sex-limitation analysis (Figure 1). By incorporating DZ_{os} twin data, both quantitative gender differences (h_m vs. h_f; c_m vs. c_f) and qualitative gender differences ($r_A \leq 0.50$) can be tested. When the genetic correlation (r_A) between opposite-sex twin pairs is significantly less than the correlation between same-sex twin pairs, qualitative gender differences are suggested. That is, different genetic influences are being manifested in males and females. If the same genetic factors are influencing reading difficulties in males and females, then the expected genetic correlation is 0.50. To test for gender differences in the magnitude of genetic influences, the additive genetic parameters for males and females are constrained to be equal and the change in chi-square is evaluated. Similarly, by constraining the genetic correlation to be equal to 0.50 for DZ_{os} twin pairs and evaluating the change in chi-square, qualitative sex differences can be tested.

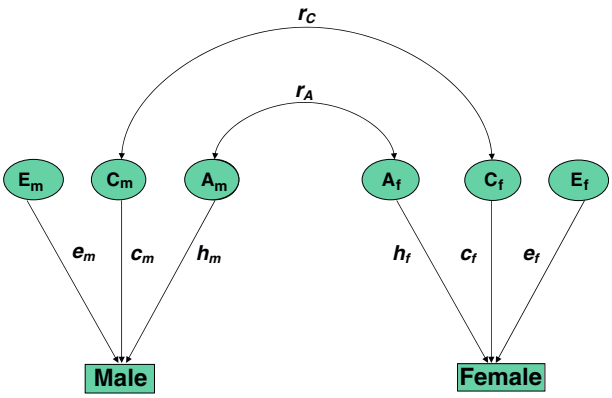

Figure 1. Sex-limitation model for opposite-sex twin pairs.

Results

Significantly more males than females were observed at all five levels of severity, and this ratio increased as a function of severity. Table 2 lists the gender ratios in each of the five cut-off samples. As indicated in Table 2, gender ratios range from 1.15:1 to 1.88:1 males to females. Except for the difference between the gender ratios in the −1 and −1.5 cut-off samples, which is only 0.02, the ratio of males to females follows an increasing linear trend as a function of greater reading impairment.

Table 3 lists the concordance rates for MZ, DZ_{ss}, and DZ_{os} twin pairs as a function of gender and severity. As can be seen, the concordance rates for both male and female MZ pairs are 66% at the −1 cut-off but decrease somewhat with increasing severity. Although both the same-sex DZ and opposite-sex twin pair concordances also decrease with increasing severity, the male and female fraternal twin pair concordance rates are lower than those for the MZ twin pairs at each severity level, and the concordance rates for the DZ_{os} twins are similar to those for DZ_{ss} twins. Because the concordance rates for the male and female MZ twin pairs are substantially greater than their fraternal twin pair counterparts at every level of severity, these results clearly suggest that reading difficulties are due in part to genetic influences. The 2×2 chi-square tests that were used to test the MZ and DZ_{ss} concordance rates at the five levels of severity further confirm the presence of genetic influences. At every level of severity, the two-way interaction of zygosity and concordance was significant, with p-values less than 0.001 for every cut-off except for the most extreme group ($p = 0.009$). When the two-way interaction of zygosity and concordance was tested using data from DZ_{ss} and DZ_{os} twin pairs, no significant interactions were found at any level of severity (−1s: $p \geq 0.46$; −1.5s: $p \geq 0.34$; −2s: $p \geq 0.47$; −2.5s: $p \geq 0.35$; −3s: $p \geq 0.74$). Thus, no evidence for qualitative sex differences was found. Tests of the three-way interaction of zygosity, gender, and concordance were also not significant for any severity group ($p \geq 0.10$ at all levels of severity), suggesting

Table 2. Gender ratios at each level of severity.

Cut-off	Gender ratio (Males:Females)	χ^2	p
−1	1.15:1	4.04	0.044
−1.5	1.17:1	4.86	0.027
−2	1.40:1	16.62	4×10^{-5}
−2.5	1.61:1	22.98	2×10^{-6}
−3	1.88:1	22.30	2×10^{-6}

Table 3. Concordance rates (%) for same-sex and opposite-sex twin pairs by gender and severity[a].

	Male			Female		
Cut-off	MZ	DZ_{ss}	DZ_{os}	MZ	DZ_{ss}	DZ_{os}
−1	66	40	44	66	28	36
−1.5	65	39	30	63	27	26
−2	68	36	29	54	22	23
−2.5	64	23	33	47	13	21
−3	49	22	32	36	17	19

[a]DZ_{os} concordance rates are given for DZ_{os} pairs in which the male or female is the proband.

no evidence for a differential genetic etiology of reading difficulties as a function of gender.

When the full sex-limitation model was fitted to the data using the model-fitting implementation of the DeFries-Fulker regression analysis, estimates of group heritability were obtained for males and females at each cut-off. Table 4 presents results of the full ACE model, including estimates of h_g^2, c_g^2, and e_g^2 for males and females as well as r_A at each of the five levels of severity. As can be seen, heritability estimates are slightly larger for females at all five cut-offs, but this difference decreases as a function of severity. Estimates of c_g^2 are correspondingly higher in males than in females, but are relatively constant throughout increasing levels of reading impairment. However, the estimate of c_g^2 in females is somewhat higher at the most extreme cut-off. Estimates of e_g^2 are similar for males and females at all levels of severity, ranging between 0.08 and 0.10 for males and 0.09 to 0.14 for females.

Table 4. h_g^2, c_g^2, e_g^2, and r_A estimates for reading difficulties in males and females as a function of severity.

	Males			Females			
Cut-off	h_g^2	c_g^2	e_g^2	h_g^2	c_g^2	e_g^2	r_A
−1	0.54	0.38	0.08	0.63	0.28	0.09	0.50
−1.5	0.55	0.36	0.09	0.64	0.24	0.12	0.49
−2	0.57	0.36	0.09	0.62	0.26	0.12	0.49
−2.5	0.57	0.36	0.09	0.61	0.25	0.14	0.50
−3	0.50	0.40	0.10	0.51	0.35	0.14	0.50

Several models were fitted to the data at each of the cut-offs. These included an ACE model which includes additive genetic, shared environmental, and non-shared environmental influences, an AE model, an ACE model with the additive genetic influences equated for males and females, an ACE model with the additive genetic and shared environmental influences equated for males and females, an ACE model with the coefficient of genetic relatedness fixed at 0.5, and an ACE model with the additive genetic and shared environmental influences equated for males and females and the coefficient of genetic relatedness fixed at 0.5.

Every model except for the AE model provided a good fit to the data at all five levels of severity. However, the most parsimonious model that provided a good fit to the data at every level of severity was the ACE model with the additive genetic influences and the shared environmental influences equated for males and females and the coefficient of genetic relatedness fixed at 0.5. Table 5 presents the goodness-of-fit information for a full ACE model with all of the parameters free, and an ACE model with the additive genetic and shared environmental influences equated for males and females and the coefficient of genetic relatedness fixed at 0.5, at all five levels of severity.

Discussion

Results obtained from previous analyses of data from the Colorado Twin Study have provided no evidence for significant gender differences in the etiology of reading difficulties. In contrast, Harlaar et al. (2005) found a

Table 5. The goodness-of-fit of a full ACE model and a sex-limitation model with the additive genetic variance and shared environmental variance equated for males and females at each level of severity.

Cut-off	Model	−2LL	df	$\Delta\chi^2$	*p*
−1	Full	2863.68	1467	–	–
	m = f	2864.82	1470	1.14	0.77
−1.5	Full	2332.42	1301	–	–
	m = f	2334.32	1304	1.90	0.59
−2	Full	1574.74	929	–	–
	m = f	1577.43	932	2.69	0.44
−2.5	Full	1059.33	663	–	–
	m = f	1062.32	666	2.99	0.39
−3	Full	586.53	389	–	–
	m = f	587.40	392	0.87	0.83

trend for a greater estimate of h_g^2 in males than in females for those falling within the lower 10% of the distribution, and evidence for a significant qualitative sex difference. In their more severely impaired sample, Harlaar et al. (2005) found a significantly greater estimate of h_g^2 for males than for females. Because of the different pattern of results that have been obtained by Harlaar et al. (2005) and Wadsworth and DeFries (2005), the current study assessed the possible differential etiology of reading difficulties in males and females as a function of severity. When the sex-limitation model was fit to the data, females tended to have somewhat higher estimates of h_g^2, but this difference was less pronounced in the more severely impaired samples. Moreover, a model with the additive genetic and shared environmental influences equated for males and females, and the coefficient of genetic relatedness fixed at 0.5 was the most parsimonious model that provided a good fit to the data at every level of severity. Thus, these results suggest that not only is the magnitude of genetic influences similar for males and females at all levels of severity, but that the same genetic and environmental influences contribute to reading difficulties in boys and girls, irrespective of severity.

Several differences between the current study and that by Harlaar et al. (2005) may account for their different results. First, Harlaar et al. (2005) used an average score obtained from two timed subtests of the TOWRE (Torgesen et al., 1999) as their measure of reading ability, whereas the current study used a composite measure of reading ability derived from the Reading Recognition, Reading Comprehension, and Spelling subtests of the PIAT (Dunn & Markwardt, 1970). Second, participants in the study by Harlaar et al. (2005) were administered timed tests over the telephone, whereas participants in the Colorado Twin Study are tested in-person by an administrator with no restriction on time. A third difference between the two studies was the age of the participants. Subjects in the study by Harlaar et al. (2005) were seven years of age, whereas those in the Colorado Twin Study ranged between eight and twenty years, with a mean age of 11.5 years. Although Hawke, Wadsworth, and DeFries (2006) and Wadsworth and DeFries (2005) examined the genetic etiology of reading difficulties as a function of gender and age, the younger groups in those two studies varied between 8.0 and 11.5 years of age. Wadsworth and DeFries (2005) also examined the genetic etiology of reading difficulties as a function of gender and age using the PIAT Reading Recognition subtest, and found similar results to those obtained when a composite measure was used.

One limitation of the present study is the relatively small sample sizes in the more severely impaired groups. The small sample sizes obviously reduce the power to detect possible gender differences in etiology.

However, the sample sizes of the three most severely affected groups in the current study are comparable to the sample size that was analyzed by Harlaar et al. (2005) at the 5% cut-off in their extremes analysis. A second limitation of the current study is the non-independence of the selected samples which limits the generalization of the findings to other samples.

In conclusion, although the ratio of males to females clearly increases as a function of severity (Olson, 2002), no significant differences in the genetic etiology of reading difficulties between males and females were found in any of the selected samples in the present study. More research is needed to clarify the greater prevalence of males in more severely impaired samples. Future research is also warranted to explain the differences between the results obtained in the Colorado Twin Study and those of Harlaar et al. (2005). However, analyses of data from the present study continue to provide no evidence of a differential genetic etiology of reading difficulties as a function of gender or severity.

References

DeFries, J. C. (1985). Colorado reading project. In D. B. Gray & J. F. Kavanagh (Eds.), *Biobehavioral measures of dyslexia* (pp. 107–122). Parkton, MD: York Press.

DeFries, J. C., Filipek, P. A., Fulker, D. W., Olson, R. K., Pennington, B. F., Smith, S. D., & Wise, B. W. (1997). Colorado learning disabilities research center. *Learning Disabilities: A Multidisciplinary Journal*, *8*, 7–19.

DeFries, J. C., & Fulker, D. W. (1985). Multiple regression analysis of twin data. *Behavior Genetics*, *15*, 467–473.

DeFries, J. C., & Fulker, D. W. (1988). Multiple regression analysis of twin data: Etiology of deviant scores versus individual differences. *Acta Geneticae Medicae et Gemellologiae*, *37*, 205–216.

DeFries, J. C., & Gillis, J. J. (1993). Genetics of reading disabilities. In R. Plomin & G. E. McClearn (Eds.), *Nature, nurture and psychology* (pp. 121–145). Washington, D.C.: American Psychological Association.

DeFries, J. C., Olson, R. K., Pennington, B. F., & Smith, S. D (1991). Colorado Reading Project: Past, present, and future. *Journal of Learning Disabilities*, *2*, 37–46.

Dunn, M. L., & Markwardt, F. C. (1970). *Examiner's manual: peabody individual achievement test*. Circle Pines, MN: American Guidance Service.

Finucci, J. M., & Childs, B. (1981). Are there really more dyslexic boys than girls? In A. Ansara, N. Geschwind, A. Galaburda, M. Albert & N. Gartrell (Eds.), *Sex differences in dyslexia* (pp. 1–9). Townson, MD: Orton Dyslexia.

Geschwind, N. (1981). A reaction to the conference on sex differences in dyslexia. In A. Ansara, N. Geschwind, A. Galaburda, M. Albert & N. Gartrell (Eds.), *Sex differences in dyslexia* (pp. xiii–xviii). Townson, MD: Orton Dyslexia.

Harlaar, N., Spinath, F. M., Dale, P. S., & Plomin, R. (2005). Genetic influences on early word recognition abilities and disabilities: A study of 7-year-old twins. *Journal of Child Psychology and Psychiatry*, *46*, 373–384.

Hawke, J. L., Wadsworth, S. J., & DeFries, J. C. (2006). Genetic influences on reading difficulties in boys and girls: The Colorado Twin Study. *Dyslexia*, *12*, 21–29.

Liederman, J., Kantrowitz, L., & Flannery, K. (2005). Male vulnerability to reading disability is not likely to be a myth: A call for new data. *Journal of Learning Disabilities*, *38*, 109–129.

Nass, R. (1993). Sex differences in learning abilities and disabilities. *Annals of Dyslexia*, *43*, 61–77.

Nichols, R. C., & Bilbro, W. C. Jr. (1966). The diagnosis of twin zygosity. *Acta Geneticae Medicae et Gemellologiae*, *16*, 265–275.

Olson, R. K. (2002). Dyslexia: Nature and nurture. *Dyslexia*, *8*, 143–159.

Purcell, S., & Sham, P. C. (2003). A model-fitting implementation of the DeFries-Fulker model for selected twin data. *Behavior Genetics*, *33*, 271–278.

Rutter, M., Caspi, A., Fergusson, D., Horwood, L. J., Goodman, R., Maughan, B., Moffitt, T. E., Meltzer, H., & Carroll, J. (2004). Sex differences in developmental reading disability: New findings from 4 epidemiological studies. *JAMA*, *291*, 2007–2012.

Stevenson, J. (1992). Identifying sex differences in reading disability: Lessons from a twin study. *Reading and Writing: An Interdisciplinary Journal*, *4*, 307–326.

Symmes, J. S., & Rapoport, J. L. (1972). Unexpected reading failure. *American Journal of Orthopsychiatry*, *42*, 82–91.

Tallal, P., & Fitch, R. H. (1993). Hormones and cerebral organization: Implications for the development and transmission of language and learning disabilities. In A. M. Galaburda (Ed.), *Dyslexia and development: Neurobiological aspects of extraordinary brains* (pp. 168–186). Cambridge, MA: Harvard University Press.

Torgesen, J. K., Wagner, R. K., & Rashotte, C. A. (1999). *Test of word reading efficiency (TOWRE)*. Austin, TX: Pro-ed.

Vogel, S. A. (1990). Gender differences in intelligence, language, visual-motor abilities, and academic achievement in students with learning disabilities: A review of the literature. *Journal of Learning Disabilities*, *23*, 44–52.

Wadsworth, S. J., & DeFries, J. C. (2005). Genetic etiology of reading difficulties in boys and girls. *Twin Research and Human Genetics*, *8*, 594–601.

Wadsworth, S. J., Knopik, V. S., & DeFries, J. C. (2000). Reading disability in boys and girls: No evidence for a differential genetic etiology. *Reading and Writing: An Interdisciplinary Journal*, *13*, 133–145.

Wechsler, D. (1974). *Examiner's manual: wechsler intelligence scale for children-revised.* New York: The Psychological Corporation.

Wechsler, D. (1981). *Examiner's manual: wechsler adult intelligence scale-revised.* New York: The Psychological Corporation.

Address for correspondence: Jesse L. Hawke, Institute for Behavioral Genetics, University of Colorado, 447 UCB, Boulder, CO, 80309-0447, USA
Phone: +1-303-735-6179; Fax: +1-303-492-8063; E-mail: hawkej@colorado.edu