

Etiology of the Comorbidity Between RD and ADHD: Exploration of the Non-Random Mating Hypothesis

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The present research sought to test the validity of the non-random mating hypothesis in a community sample in order to determine whether it contributes significantly to the comorbidity between attention-deficit/hyperactivity disorder (ADHD) and reading disability (RD) and to test whether the two disorders segregate independently. Data were gathered from 394 twin participants of the Colorado Learning Disabilities Research Center (CLDRC) and their biological parents. Parents were asked to complete retrospective questionnaires regarding their own ADHD and RD symptomatology as children, as well as rate their children on *DSM-IV* ADHD symptomatology before age 12. Twin RD was assessed by a composite score consisting of several reading measures. Chi-square and correlational analyses assessed the presence of non-random mating, as well as whether the two disorders were transmitted independently. Non-random mating between those with ADHD and those with RD did not significantly contribute to the comorbidity of the two disorders. Furthermore, there was some evidence that these two disorders did not segregate independently in this sample. These results are consistent with recent evidence that ADHD and RD are often comorbid because of significant

genetic overlap. There was little evidence to support that non-random mating also contributes to this comorbidity in the present sample. © 2003 Wiley-Liss, Inc.

KEY WORDS: behavior genetics; learning disabilities; dyslexia; assortative mating

INTRODUCTION

There is substantial literature documenting a significant comorbidity between attention-deficit/hyperactivity disorder (ADHD) and reading disability (RD). If a child has one of these disorders, the chance that he/she will also experience the second ranges from about 25–40% [Dykman and Ackerman, 1991; Biederman et al., 1992; Semrud-Clikeman et al., 1992; Willcutt and Pennington, 2000]. Recent evidence suggests that the substantial comorbidity between ADHD and RD is partially attributable to shared (pleiotropic) genetic influences. Each disorder itself is moderately to highly heritable [e.g., DeFries et al., 1987; Stevenson, 1992], and twin studies have demonstrated significant bivariate heritability between the two disorders, suggesting that some of the same genes influence both disorders [Stevenson et al., 1993; Light et al., 1995; Willcutt et al., 2000]. Moreover, recent molecular genetic findings indicate that the quantitative trait locus (QTL) for RD on chromosome 6p21.3 is also a susceptibility locus for ADHD [Willcutt et al., 2002]. Thus, there is converging evidence that the two disorders are influenced by some of the same genes, which act pleiotropically to influence the development of both disorders.

Alternative explanations for this comorbidity have been posited. For example, in a cognitive study of the etiology of comorbidity between RD and ADHD, Pennington et al. [1993] found that children with comorbid RD and ADHD did not exhibit the executive function deficits characteristic of children with ADHD alone. Thus, Pennington et al. [1993] proposed that RD and ADHD co-occur because RD causes the phenotypic manifestation (or phenocopy) of ADHD in the absence of the etiological influences typically associated with ADHD in isolation. This hypothesis was of particular

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interest for behavioral geneticists, because if having RD or ADHD causes children to exhibit a phenocopy of the other disorder, the pattern of results could mimic the pattern indicative of bivariate heritability. However, more recent findings provide strong evidence against this hypothesis. Specifically, the results of a molecular genetics study conducted by Willcutt et al. [2002] indicated that the linkage for ADHD remained significant when reading scores were regressed out of the ADHD scores prior to the analysis, suggesting that linkage for ADHD was not simply a secondary consequence of reading difficulties. In addition, most subsequent studies of the cognitive correlates of RD and ADHD have not supported the phenocopy hypothesis [e.g., Robbins, 1992; Reader et al., 1994; Nigg et al., 1998; Willcutt et al., 2001]. Instead, these studies have found that individuals with RD and ADHD exhibit both the executive function deficits associated with ADHD and the phonological processing deficits associated with RD. Thus, the phenocopy hypothesis has shown little validity as an explanation for the comorbidity of RD and ADHD.

However, there remains an alternative explanation for this comorbidity that has not been sufficiently addressed in the literature: the non-random mating hypothesis. This hypothesis for the comorbidity of ADHD and RD was posited by Faraone et al. [1993]. Their study provided evidence indicating that spouses of those with ADHD had significantly higher rates of RD than spouses of those without ADHD and that the two disorders were transmitted independently in families. Thus, they suggested that the reason for their co-occurrence was a higher than expected rate of mating between individuals with ADHD and those with RD.

There has been little attempt to assess the validity of Faraone and colleagues' [Faraone et al., 1993] findings despite the fact that they contradict the recent evidence for significant genetic overlap between the two disorders. If the two disorders were actually transmitted independently, then shared genes would not contribute significantly to both disorders. However, it is important to note that the significant bivariate heritability and molecular genetic results do not contradict the non-random mating finding. Even if common genes contribute to the overlap of the two disorders, non-random mating could still be an additional influence on this comorbidity. Furthermore, behavioral genetic research typically assumes that there is no significant non-random mating. If this assumption were incorrect, bivariate heritability estimates would *underestimate* the significant genetic overlap of the two disorders. Specifically, heritability estimates would be lowered because significant non-random mating would cause dizygotic (DZ) twins to appear more similar, while not affecting the correlation between monozygotic (MZ) twins since they are already at the point of maximal genetic resemblance. Therefore, clarification of the non-random mating finding regarding the comorbidity of ADHD and RD is critical in order to truly understand the reasons for the relationship between these two disorders. The present research sought to test the validity of the non-random mating hypothesis in a community

sample in order to determine whether it contributes significantly to this commonly observed comorbidity.

MATERIALS AND METHODS

The present research was conducted as part of the Colorado Learning Disabilities Research Center (CLDRC), which uses a population-based twin sample to investigate the etiology of RD, ADHD, and comorbid disorders [DeFries et al., 1997]. This project was conducted by the Institute for Behavioral Genetics and the Department of Psychology at the University of Colorado, Boulder, the Department of Psychology at the University of Denver, and the Center of Human Molecular Genetics at the University of Nebraska.

Participants

The CLDRC Twin Project recruited 8–18-year-old twins and their parents from 27 school districts within 150 miles of the Denver/Boulder area. Twin pairs were selected in which at least one twin has shown a school history of reading difficulty or significant ADHD symptomatology. Control pairs in which neither twin has shown evidence of reading difficulty or ADHD symptomatology were also recruited. Twin pairs were excluded from the study if at least one twin has a significant environmental or genetic risk factor, such as a history of closed head injury, a pervasive developmental disorder, prenatal exposure to drugs or alcohol, or Fragile X Syndrome. The ethnic makeup of the CLDRC sample reflected that of the Denver metropolitan area (approximately 81% Caucasian, 12% Hispanic, 5% African-American, and 2% Asian-American).

The sample utilized for the present research included 394 twin participants of the CLDRC and their biological parents (197 families); the parents responded to a follow-up mail survey designed to assess the presence of non-random mating in the CLDRC sample. We were unable to locate 123 of the 638 families who had participated in the CLDRC twin project from 1991 to 1999. Of the remaining 515 families, we received responses from 228 families, 31 of which were excluded because information from both biological parents was not available or incomplete (see Table I for demographics). Because the sample utilized for this study consisted of only those participants who responded to a follow-up mail survey, it was important to determine whether these respondents were representative of our sample at large. Therefore, we compared the sample of participants who responded to the mailing to the total sample obtained from 1991–1999 on the following dimensions: rate of twin ADHD, rate of twin RD, rate of twin comorbidity, zygosity, full-scale IQ (FSIQ), age, reading composite score, and gender. These results are summarized in Table II. Although, there were significant differences between groups ($P < 0.05$) on FSIQ and reading composite score, the mean difference between groups was quite small (about 2 IQ points and 0.1 SD on the reading composite score). The rates of diagnoses of ADHD, RD, and comorbidity were comparable across the samples, and there were no significant differences in age, gender ratio, or Zygosity between the samples. Therefore the

TABLE I. Demographics of Twins by Group

	RD	ADHD	Comorbid	None
n	116	22	42	214
Mean age (SD) ^a	10.84 (1.82)*	10.86 (2.45)***	11.36 (2.42)**	11.49 (2.50)**
% male	37.1*	77.3**	81.0**	50.5***
Mean full scale IQ (SD) ^b	98.16 (10.57)*	109.41 (11.25)**	97.08 (11.68)*	112.84 (11.66)**

Note: Values with differing superscripts (i.e., ***, **, *) are significantly different from one another ($P < 0.05$).

^aAssessed at initial testing session.

^bAssessed by either WAIS-R or WISC-R [Wechsler, 1974, 1981].

mailing sample was felt to be an adequate sample in which to determine the occurrence of non-random mating and independent assortment.

Procedures

Prior to testing, potential CLDRC participants were screened by phone interview based on the above mentioned exclusionary criteria. Following this screening, each twin was administered a psychoeducational battery at the University of Colorado, Boulder that included measures of RD and cognitive ability. In January 2000, follow-up questionnaires were mailed to participants in order to assess the presence of non-random mating in the CLDRC sample. Parents were asked to respond to the survey only if both biological parents were able to participate. Biological parents were asked to complete measures of retrospective ADHD as well as RD regarding themselves, while the parent who had spent the most time with the twins when they were young was asked to complete a measure of retrospective ADHD symptomatology for their children based on *The Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition* [DSM-IV; American Psychiatric Association, 1994] diagnostic criteria.

Measures of RD

To assess the presence of RD in twins, a discriminant function analysis was utilized that was conducted on a separate non-twin sample of individuals with and without a school history of reading problems. This

analysis produced a normally distributed, weighted composite score [DeFries, 1985] based on the reading recognition, reading comprehension, and spelling subtests of the Peabody Individual Achievement Test [Dunn and Markwardt, 1970] that best predicted a school history of reading difficulty. A cut-off score of 1.50 standard deviations below the mean in a separate sample of control twins [i.e., approximately 6.7% of controls; Willcutt et al., 2000] was used to identify the presence of RD in the present sample. Lower composite scores reflected greater levels of reading difficulty.

Parental RD was measured retrospectively using an adaptation of the Adult Reading History Questionnaire (ARHQ) originally devised by Finucci and colleagues [Finucci et al., 1984]. The ARHQ contains 23 items answered on a five-point likert scale ranging from 0 (indicating less difficulty) to 4 (indicating greater difficulty). Scores on the ARHQ were computed by dividing the total number of points obtained by the total number of possible points (i.e., 92). A conservative cut-off score of 0.40 was used to determine a positive history of RD. This cut-off score has been shown to have a sensitivity rate of 81.8%, a specificity rate of 77.5%, and an overall correct classification rate of 79% in a sample of adults with and without RD defined by test scores [Lefly and Pennington, 2000]. The ARHQ has been shown to have strong internal consistency ($\alpha = 0.92$ and 0.94), and strong test-retest reliability (3-year interval = $0.81-0.84$). Further, the ARHQ was highly correlated with adult reading ability ($r = 0.70$) as well as RD symptomatology measured in childhood [$r = 0.75$; Lefly and Pennington, 2000].

TABLE II. Comparison of Mail Survey Respondents to Sample at Large

	CLDRC sample ($N = 1,276$)	Mailing sample ($N = 394$)	Test statistic ^a
Rate of ADHD alone	8.4%	5.6%	1.83
Rate of RD alone	30.8%	29.4%	0.53
Rate of comorbidity	13.1%	10.7%	1.27
Zygoty	57.6% DZ	58.4% DZ	-0.28
Mean full scale IQ (SD)	104.25 (12.96)	106.62 (13.40)	-3.18**
Mean age (SD)	11.16 (2.32)	11.25 (2.32)	-0.68
Mean reading score ^b (SD)	0.19 (1.42)	0.36 (1.47)	-2.07*
Gender	52.3% male	51.3% male	0.35

CLDRC, Colorado Learning Disabilities Research Center; ADHD, attention-deficit/hyperactivity disorder; RD, reading disability.

^aTest statistic = t for full scale IQ, age, and reading score; z for all other comparisons.

^bLower scores indicate greater levels of reading difficulty.

* $P < 0.05$.

** $P < 0.005$.

Measures of ADHD

Twin and parental ADHD were measured retrospectively using the Disruptive Behavior Rating Scale [DBRS; Barkley and Murphy, 1998]. Ratings of *DSM-IV* ADHD were not available for the children who had participated in the study from 1991–1995. In order to have consistent *DSM-IV* ratings for all of the children in this study, parents were asked to complete a retrospective DBRS. The DBRS contains 18 items corresponding directly to the *DSM-IV*. Parents were instructed to circle the number next to each question that best described their child before age 12 using a four point scale (i.e., 0 = *no*, 1 = *just a little*, 2 = *pretty much*, 3 = *very much*). Parents completed the same measure regarding themselves, but with a three point rating scale (i.e., 0 = *no*, 1 = *sometimes*, 2 = *yes*) in order to maintain consistency with a previous parental ADHD interview in the CLDRC battery. For both parents and children, symptoms that were given a rating of 2 or 3 were considered to be present. Consistent with *DSM-IV*, participants were considered to have ADHD if they demonstrated 6 or more inattentive symptoms and/or 6 or more hyperactive/impulsive symptoms. The total ADHD score on the DBRS was compared to the twin total ADHD score for a subset of our sample whose parents had completed a *DSM-IV* rating scale at the initial time of testing, and the correlation between ratings at time of testing and ratings in the retrospective mailing was quite high ($r = 0.84, P < 0.0001, n = 135$). Further, the DBRS has been shown to have strong internal consistency [$\alpha = 0.96$; Willcutt and Pennington, 2000].

RESULTS

Analyses were designed to answer two main questions:

- (1) Is non-random mating contributing significantly to the comorbidity between ADHD and RD?
- (2) Are ADHD and RD transmitted independently in this sample?

Question 1

To answer question 1, we first wanted to determine whether there was a higher proportion of cross-assorted couples in our sample than would be expected by chance. Of the 197 couples in the sample, 15 of them (7.6%) were cross-assorted. Chi-square tests showed that there was not a significantly higher number of fathers with ADHD who paired with mothers with RD than expected by chance $\chi^2 (1, N = 197) = 1.007, P = 0.316$. Similarly, there was not a significantly higher number of mothers with ADHD who paired with fathers with RD than expected by chance $\chi^2 (1, N = 197) = 0.120, P = 0.729$.

Additionally, we wanted to determine if there was a higher than expected rate of cross-assortment among parents of comorbid children. Results indicated that even among parents of comorbid children, there was not a significantly higher number of fathers with ADHD who paired with mothers with RD than what would be

expected by chance $\chi^2 (1, N = 33) = 0.503, P = 0.639$ (Fisher’s exact test). The rate of mothers with ADHD who paired with fathers with RD was also not significantly higher than what would be expected by chance $\chi^2 (1, N = 33) = 0.002, P = 0.316$ (Fisher’s exact test).

Moreover, of 42 comorbid children in our sample, only 5 of them (11.9%) came from cross-assorted couples. Therefore, 88.1% of the comorbid children in our sample did not come from cross-assorted parents. Chi-square analyses demonstrated that the proportion of comorbid children from assorted couples was not significantly different than the proportion of comorbid children from non-assorted couples $\chi^2 (1, N = 197) = 3.201, P = 0.140$ (Fisher’s exact test). Taken together, the above analyses provide strong evidence that non-random mating does not contribute substantially to the comorbidity of RD and ADHD in this sample.

Because our measures of ADHD and RD could also be scored continuously, we decided to further analyze our data using correlational methods, since it is possible that the null results just reported were a function of the particular diagnostic cut-off utilized. In this way, we could look at the correlations between symptoms of RD and symptoms of ADHD both across and within parents. Strong within-parent correlations between ADHD and RD would suggest high levels of comorbidity, while strong across-parent correlations between ADHD and RD would suggest non-random mating. All parent RD and ADHD total scores were transformed prior to analysis to approximate normality. As Table III indicates, results strongly supported comorbidity of ADHD and RD in both mothers and fathers ($r = 0.323$ and 0.389 , respectively, $P < 0.001$). Although, we were not specifically interested in non-random mating for RD alone or ADHD alone, there was no evidence of non-random mating between parents for either RD or ADHD, as neither of these correlations were significant. These results demonstrate that even for either disorder alone, non-random mating is not occurring at significant levels. In addition, the correlation between mothers’ RD symptoms and fathers’ ADHD symptoms was not significant ($r = 0.013, P = 0.855$), nor was the correlation between mothers’ ADHD symptoms and fathers’ RD symptoms ($r = 0.156, P < 0.05$), after controlling for Type I error [Larzelere and Mulaik, 1977]. Therefore, there was no evidence of non-random mating for RD, no evidence of non-random mating for ADHD, and little evidence in support of non-random mating between one parent with RD and one parent with ADHD. These continuous analyses corroborate the categorical

TABLE III. Correlations Among Parental Phenotypes

	Father RD	Mother ADHD	Mother RD
Father ADHD	0.389**	0.085	0.013
Father RD	—	0.156 ^a	0.045
Mother ADHD	—	—	0.323**

N = 197. ADHD, attention-deficit/hyperactivity disorder; RD, reading disability.

^aCorrelation no longer reaches significance after the Larzelere and Mulaik correction for multiple correlations.

** $P < 0.0005$.

analyses, which also did not demonstrate significant non-random mating.

Question 2

We first tested for the familiarity of each disorder alone. Of the 70 families in which one or both parents had RD, 46 of their children (65.7%) had RD. This rate was significantly greater than the rate of RD (44%) in children of parents who did not have RD $\chi^2 (1, N = 197) = 8.448, P < 0.01$, providing strong evidence for the familiarity of RD. Of the 99 families in which one or both parents had ADHD, 32 (32.3%) of their children had ADHD. This rate was not significantly greater than the rate of ADHD (20.4%) in children from families where neither parent had ADHD $\chi^2 (1, N = 197) = 3.599, P = 0.058$. Although this result did not quite reach significance, it still indicated there is a strong trend for the familiarity of ADHD alone.

If two disorders (for instance, disorders A and B) are being transmitted independently, there should not be an elevated rate of disorder A among relatives of those who only have disorder B, and vice versa. For instance, if RD and ADHD are being transmitted independently, parents who have ADHD should not have children with RD at a higher rate than parents who do not have ADHD. However, if the two disorders are co-segregating due to common genetic or environmental influences, then one would expect a higher rate of the second disorder among relatives of those who only have the first disorder. To determine whether ADHD and RD were being transmitted independently in this sample, we tested whether there was a higher than expected rate of RD among children of parents with ADHD only, as well as whether there was a higher than expected rate of ADHD in children of parents with RD only. For these analyses, we used parents who had *only* ADHD or *only* RD in order to avoid artificially inflating rates of the second disorder in the children. Hence, comorbid parents or parents who were cross-assorted were not used in these analyses. Of the 55 families containing parents with ADHD only, 28 (50.9%) had children with RD. This rate was greater than the rate of RD (38.9%) in the 72 families containing parents with neither ADHD nor RD, but not significantly so $\chi^2 (1, N = 127) = 1.828, P = 0.176$. One reason this test may not reach statistical significance is that the rate of RD in children in this sample is inflated, since many of the children are recruited specifically due to a history of reading problems in school. Therefore, this is a conservative test. Of the 26 families containing parents with RD only, 9 (34.6%) had children with ADHD. This rate was significantly greater than the rate of ADHD (15.3%) in the families in which neither parent had ADHD nor RD $\chi^2 (1, N = 98) = 4.398, P < 0.05$. Thus there is evidence that the two disorders are not transmitting independently in this sample.

In order to control for inflated rates of RD and ADHD in our sample, we also utilized correlational analyses to investigate questions regarding familiarity of each disorder and their co-segregation. The relationships between parent RD and ADHD scores and the average twin RD and ADHD scores were assessed. Again, parental

data had been transformed prior to analysis due to failure to adhere to assumptions of normality. As Table IV indicates, there were significant correlations between parental RD and twin RD ($r = -0.301, -0.346, P < 0.001$), strongly supporting the familiarity of RD. Similarly, there was a significant correlation between maternal measure of ADHD and twin measure of ADHD ($r = 0.245, P < 0.001$), while the correlation between paternal ADHD and twin ADHD ($r = 0.157, P < 0.05$) failed to reach significance after controlling for Type I error [Larzelere and Mulaik, 1977]. Generally, however, this pattern of results also supported familiarity of ADHD. In addition, there was evidence for the shared familiarity of RD and ADHD. There was a significant correlation between father RD and twin ADHD ($r = 0.185, P = 0.009$), but the correlation between mother ADHD and twin RD ($r = 0.146, P < 0.05$) failed to reach significance after the Larzelere and Muliak correction for multiple correlations. Taken together, these results lend support to the evidence of shared familiarity of the disorders and corroborate the categorical analyses.

DISCUSSION

The present study sought to assess whether non-random mating contributed significantly to the comorbidity between RD and ADHD in our sample. Our findings indicated that cross-assortment of RD and ADHD was not significantly different than chance levels, and that cross-assorted couples did not account for more comorbid children than what would be expected by chance. There was also little evidence in support of the independent transmission of these disorders. The rate of ADHD in children whose parents had RD only was significantly greater than the rate of ADHD in children from parents with neither ADHD nor RD. The rate of RD in children whose parent's had ADHD only was also greater than the rate of RD in children from control parents (although this difference was not statistically significant). In addition, one parental measure of RD correlated significantly with twin measure of ADHD, lending support to the shared familiarity of the two disorders. These findings are generally inconsistent with prior research suggesting that cross-assortment of parents with RD and parents with ADHD can account for the comorbidity of the two disorders [Faraone et al., 1993]. Instead, they are consistent with the evidence of

TABLE IV. Parent and Child Correlations between ADHD and RD Measures

	Mother (N = 197)		Father (N = 197)	
	RD	ADHD	RD	ADHD
Twin RD ^a	-0.364**	-0.146 ^b	-0.301**	-0.052
Twin ADHD	0.127	0.245**	0.185**	0.157 ^b

Note: Twin scores were calculated using the average score within each twin pair. ADHD, attention-deficit/hyperactivity disorder; RD, reading disability.

^aLower scores indicate greater levels of reading difficulty.

^bCorrelation no longer reaches significance after the Larzelere and Muliak correction for multiple correlations.

**P < 0.01.

shared genetic influences on the two disorders demonstrated by both behavioral genetic [Stevenson et al., 1993; Light et al., 1995; Willcutt et al., 2000] and molecular genetic studies [Willcutt et al., 2002].

There are several factors that may contribute to the failure to replicate Faraone and colleagues' findings. To begin with, there are diagnostic differences between the two samples. Although the significant non-random mating finding in the Faraone et al. sample occurred between parents with ADHD and parents with RD, their test for independent assortment included a broader group of children with learning disabilities (LD; including both RD and math disability). The inclusion of children with math disability as well may influence the finding of independent transmission. Another important factor is that Faraone and colleagues' sample was recruited from a clinic population, while the current study utilized a community sample. Increased symptomatology is often found in clinic samples, and this increased symptomatology may affect the rate of cross-assortment. That is, perhaps only the individuals with the most severe ADHD or RD cross-assort, or perhaps families with cross-assorted parents are more likely to seek clinical services. It is also possible that the etiology of severe levels of ADHD and RD is somewhat different than the etiology of less severe cases. Perhaps the genes contributing to severe ADHD and RD are not the same genes shared between the two disorders. For example, if there were 10 genes contributing to ADHD and 10 genes contributing to RD and 5 of them contributed to both, the remaining genes contributing independently to each disorder could be the ones that account for the most severe cases. Alternatively, the etiology of comorbidity between RD and ADHD in clinic samples could be different in another way. It is possible that severe cases of ADHD are associated with other disorders, such as conduct disorder (CD). In fact, the comorbidity between ADHD and CD is highest in the *DSM-IV* combined subtype of ADHD [Morgan et al., 1996; Eiraldi et al., 1997], which is frequently found in clinic samples. Therefore, in these severe ADHD cases, it is possible that the CD diagnosis is associated with RD rather than the ADHD diagnosis. For instance, maybe children with CD are less likely to attend school and thus, are less likely to have opportunities to develop their reading abilities. This could then be an environmentally mediated association between CD and RD that impacts the comorbidity of RD and ADHD, rather than the genetic overlap between RD and ADHD that is found in other less severe samples. Thus, independent transmission could still occur in very extreme groups, and in these cases non-random mating may contribute to significant levels of comorbidity.

Another factor that may contribute to the discrepancy between the present and previous findings is the level of hyperactive/impulsive symptomatology in each sample. Recent behavior genetic analyses have shown that while many of the genetic factors contributing to RD are the same as those contributing to the symptoms of inattention in ADHD, few of the genetic influences contributing to RD also contribute to symptoms of hyperactivity and impulsivity [Willcutt et al., 2000]. Specifically, approxi-

mately 95% of the phenotypic overlap between RD and inattention could be accounted for by common genetic influences, while only 21% of the phenotypic overlap of RD and hyperactivity/impulsivity could be accounted for by genetic factors. Thus, if Faraone and colleagues' [Faraone et al., 1993] clinic sample contained a high proportion of children with the predominantly hyperactive/impulsive subtype of ADHD, findings in support of bivariate heritability would not be as likely. However, since the predominantly hyperactive impulsive subtype of ADHD is rare [Lahey et al., 1994; Eiraldi et al., 1997], this explanation is plausible, but not probable.

Limitations and Future Directions

There are some limitations in the present study. To begin with, the data analyzed consisted mainly of retrospective ratings. Although there has been much support for the reliability and validity of the retrospective measures used in this study (e.g., retrospective ratings correlated highly with ratings at time of initial testing or other childhood measures), ideally one would prefer all participants to be assessed in childhood. Another potential limitation is the fact that the study was done using a follow-up mail study, and that we could only use data when it was available from *both* parents. It is possible that the parents who responded to the mailing are somehow different from the population at large and do not accurately represent the broader population. However, the mail survey respondents did not differ from our study participants in general in terms of rate of ADHD, RD, or rate of comorbidity between the two disorders.

Despite these limitations, the present study provides compelling evidence that non-random mating does not contribute significantly to the comorbidity of RD and ADHD, and also provides some evidence for the cosegregation of these two disorders in a community sample. Recent data from the lab of Faraone et al. [Doyle et al., 2001] also did not demonstrate evidence of significant non-random mating between parents with LD and parents with ADHD in a sample of female probands with ADHD. However, they still found some results supporting independent transmission of ADHD and LD. Future research may wish to replicate our findings in groups of varying levels of severity of ADHD and RD, as well as within differing subtypes of ADHD and different definitions of LD, to determine if the contribution of non-random mating and the mode of transmission vary as a function of these sample characteristics.

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