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Parental Education Moderates Genetic Influences on Reading Disability

Angela Friend[1], **John C. DeFries**[1,2], and **Richard K. Olson**[1,2]

[1]Department of Psychology, University of Colorado at Boulder, Boulder, CO 80309

[2]Institute for Behavioral Genetics, University of Colorado at Boulder, Boulder, CO 80309

Abstract

Environmental moderation of the level of genetic influence on children's reading disabilities (RD) was explored in a sample of 545 identical and fraternal twins (mean age = 11.5 years). Parents' years of education, which are correlated with a broad range of environmental factors related to reading development, were significantly related to the level of genetic influence on reading disability (t = 3.23, P_{rep} = .99). Genetic influence was higher and environmental influence was lower among children with higher compared to children with lower parent education. We discuss the implications of these results for behavior- and molecular-genetic research, for the diagnosis and remediation of RD, and for policy in public education.

Keywords

Behavior Genetics; Dyslexia

Interactions between genetic and environmental influences (G × E) on behavior have been gaining considerable attention in recent years (Rutter, 2006). Molecular-genetic G × E studies have reported that specific-gene influences on maladaptive behaviors such as conduct disorder and alcoholism may depend on the environment (Caspi, 2002). Also, several behavior-genetic G × E studies with population samples of identical and fraternal twins have reported that the degree of genetic influence or heritability of individual differences in cognitive and academic abilities across the normal range (i.e., the “bell curve”) varies with family socioeconomic status (SES) (Turkheimer, Haley, Waldron, D'Onofrio, Gottesman, 2003). The present study of a selected twin sample from the Colorado Learning Disabilities Research Center (CLDRC, DeFries et al., 1997) is the first to explore G × E interactions for group deficits (i.e., the low tail of the normal distribution) in reading, or reading disability (RD), the most commonly identified learning disability.

In the present study, we investigate whether there are G × E interactions between parental education (our proxy measure for SES and related environmental influences) and the heritability of group deficits in a composite measure of word recognition, spelling, and reading comprehension. Parental education, as well as other measures of SES, has been shown to be a strong predictor for a variety of child and adult health and cognitive outcomes (Bradley & Corwyn, 2002). Moreover, parental education may be indicative of level of investment in children's performance in school and educational attainment (Craig, 2006).

Address for correspondence: Angela Friend 345 UCB Department of Psychology University of Colorado at Boulder Boulder, Colorado 80309 Phone: 303 735−6595 Fax: 303 492−2967 e-mail: angela.friend@colorado.edu.

Evidence for a G × E interaction in behavior-genetic studies is indicated by a significant difference in heritability that is moderated by a measured environmental factor. The direction of change in heritability may support one of two theoretical models of G × E interactions. The ***bioecological model*** first proposed by Bronfenbrenner and Ceci (1994) suggests that genetic influences on behavior should be most evident when the environment is supportive because there is greater actualization of genetic potential in supportive environments than in poor environments. The ***diathesis-stress model*** (Zubin & Spring, 1977; Scarr, 1992) suggests that heritability for a particular behavior should be greater in poorer environments, where stressors may lead to the expression of deleterious genes on behavior that would otherwise not be observed in more supportive environments. This model has been proposed to explain why certain behavioral disorders have a greater association with specific genes in environments where individuals have been exposed to a large number of stressful life events (c.f. Caspi et al., 2002; Caspi et al., 2003).

Both the diathesis-stress and the bioecological models of G × E interactions are plausible when considering genetic influences on RD. For example, the heritability of RD might be greater in poorer educational environments than in supportive environments (diathesis-stress model), because the negative consequences of any genetic susceptibilities for RD can be avoided in better educational environments in the school and home that might promote good reading for all children, the goal of the "No Child Left Behind" legislation (107th Congress, 2002). On the other hand, if the educational environment for reading acquisition is relatively poor for some children with RD, that environment may be the main reason for their failure, while genetic influences may tend to be stronger among children who fail in spite of a relatively good educational environment. Such a result would be consistent with the predictions of the bioecological model.

Reading ability is normally distributed in the population (Rodgers, 1983). Therefore, behavior-genetic analyses of data from identical and fraternal twins can be used to assess and compare the degree of genetic influence and G × E interactions for reading ability within the population as a whole as well as within samples of twins with RD that have been selected from the low tail of the reading-ability distribution. Heritability estimates for individual differences and group deficits appear to be quite similar for a variety of academic and cognitive aptitude measures (Kovas & Plomin, 2007; Plomin & Kovas, 2005), but it is unknown whether the pattern of G × E effects previously reported for individual differences in reading and general cognitive ability will be similar within samples selected for RD.

Although no previous studies have tested the diathesis-stress and bioecological model predictions for G × E interactions related to RD, one study by Kremen et al. (2005) investigated whether the heritability of individual differences in word recognition across the normal range varied as a function of parental education in a sample of 347 middle-aged male twin pairs. Kremen et al. reported that the heritability of individual differences in word recognition increased as a function of parental education, thus supporting the interaction predicted by the bioecological model. The results of Kremen et al. are consistent with three other studies of G × E interactions for individual differences in general cognitive ability (Harden, Turkheimer, and Loehlin, 2006; Rowe, Jacobson, and Van den Oord, 1999; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). However, two studies have reported no significant G × E interactions for general cognitive ability (Nagoshi and Johnson 2005; Van den Oord & Rowe, 1998).

In summary, the majority of previous behavior-genetic analyses of individual differences in reading and general cognitive abilities have provided evidence for G × E interactions that are consistent with the bioecological model, such that heritability increases with increasing environmental support when that support is indexed by parental education, composite SES

measures that include parental education, or parental income. However, no studies to date have investigated whether a bioecological model or a diathesis-stress model pattern of G × E interaction exists for group deficits in reading or other cognitive abilities. In the present study, we investigate potential G × E interactions for reading disability in a sample of identical and fraternal twins selected for a school history of reading disability by the Colorado Learning Disabilities Research Center (CLDRC).

Method

Participants

Twin pairs were ascertained from school records in 27 Colorado school districts for participation in the CLDRC studies of reading disabilities. All of the twins included in the present study had verbal or performance IQ (Wechsler, 1974; Wechsler, 1981) equal to or greater than 85, normal or corrected vision and hearing, no history of neurological problems, and English as a first language. 757 of the twin pairs who met these criteria had a positive school history for reading problems in one or both members. We also ascertained a comparison sample of 673 twin pairs who did not have a school history of reading problems for either member. The mean age for both the school-history and no-school-history samples was 11.4 years and ranged from 8 to 20 years. A sample of 545 twin pairs with a positive school history for RD were selected for low performance on a discriminant function score based on measures of word recognition, spelling, and reading comprehension in at least one member of each pair.

Measures

Reading ability was assessed in the present study with measures of word recognition, spelling and reading comprehension from the Peabody Individual Achievement Test (Dunn & Markwardt, 1970).

DISCR—A discriminant function score for reading ability (DISCR) was created using an independent sample of reading problems. performance in word non-twin individuals with and without a history of significant This yielded a normally distributed composite score based on each subject's performance in word recognition, reading comprehension, and spelling (DeFries, 1985).

Parental Education—Parents were administered a questionnaire, which included their years of education. A mean score was created from parental education for both parents when available. Approximately 96% of twin pairs had information for both parents. Otherwise, the score of the available parent, which was typically that of the mother, was used.

Analyses and Results

Distribution of Parents' Education

Descriptive information for parents' educational attainment is presented in Table 1. Percentages in educational attainment categories for adults over the age of 25 in the state of Colorado (US Census, 2000, Educational Attainment) were found to be similar to the percentages for parents of twins who had a school history for RD. However, the Colorado census data do not exclude the approximately 15 percent of families that speak a language other than English at home, while all of the twins in the present study speak English as their first language. Parents of twin pairs with no school history of RD tended to have higher levels of education compared to the positive school history parents and to individuals over 25 years of age in Colorado.

Standardization of the discriminant score (DISCR) and parent education for behavior-genetic analyses

Analyses of genetic and environmental influences on reading disability were conducted using an age-adjusted DISCR score that was standardized against the DISCR score distribution of the no-school-history group. Affected twins (called probands) were selected if their performance was 1.5 SD or more below the no-school-history group mean. To control for potential G-E correlations, the parent education variable used in the behavior genetic analysis of G × E interaction was adjusted for its correlation (.087) with probands' DISCR scores.

DF Regression analyses of average genetic influence on proband group membership

The widely employed DeFries-Fulker (DF) multiple regression method (DeFries & Fulker, 1985, 1988) provides an estimate of average genetic influence on proband group membership based on data from selected samples of identical monozygotic (MZ) twins who share all their genes, and fraternal dizygotic (DZ) twins who share half of their segregating genes on average. The present estimates of genetic influence or heritability of proband group membership are based on the differential regression of transformed MZ and DZ probands' cotwin means to the mean of the no-school-history twin sample. The transformation of cotwin scores is accomplished simply by dividing each cotwin's z score by their proband's z score. This yields a number between 1 and 0 that demonstrates how far the cotwin has regressed from the proband mean (1) to the no-school-history mean (0). These transformed scores were then analyzed in the basic DF model shown in Equation 1 to test the average level of heritability for proband group membership regardless of parent education.

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

In this equation, the cotwin score (C) is regressed on the proband score (P), the coefficient of genetic relationship (R) coded 1 for identical twin (MZ) pairs who share all of their genes and .5 for fraternal twin (DZ) pairs who share half of their segregating genes on average. K is the regression constant. B_2 estimates the heritability for the average group deficits; this is the only term that is interpreted in this regression. In cases where the cotwin also met the −1.5 SD selection criteria for proband status, the pairs were double entered in equation 1, with each twin alternately serving as a proband and cotwin, and sample sizes for significance tests were appropriately adjusted for the number of double-entry twin pairs.

Mean transformed cotwin scores and the heritability estimate for the overall group deficits in our composite measure of reading, spelling, and comprehension (DISCR) are presented in the first row of Table 2. The DZ cotwin mean regressed further than the MZ cotwin mean toward the mean (0) of the no-school-history sample. Greater regression to the no-school-history mean for cotwins of DZ probands versus cotwins of MZ probands suggests that group deficits in RD are substantially heritable (DeFries & Fulker, 1985). When the DF basic model was fitted to the transformed cotwin scores, the group heritability (h^2_g) estimate was .61 for DISCR (P_{rep} = .99). Following the procedure described in Gayán and Olson (2001), the environmental influence on RD group membership can be parsed into environmental influences that are shared by both members of a twin pair (c^2_g = .31), and non-shared environment influences that are unique to each member of a twin pair, including measurement error (e^2_g = .09) These average estimates of genetic and environmental influences on RD group membership are consistent with the extant literature on group deficits in reading and related skills (for review see Olson, 2006;Pennington & Olson, 2005). Next, we test the hypothesis that the genetic and environmental etiologies of reading difficulties vary as a function of parental education..

DF regression analyses of linear G × E interactions

Equation 2 shows the extended DF regression model, which adds a main effect and two interaction terms with parents' years of education to the basic model in equation 1.

$$C=B_1P+B_2R+B_3ED+B_4P^*ED+B_5\,R^*ED+K \quad (2)$$

The cotwin's score (C), is regressed on the proband's score (P), the coefficient of relationship (R), parental education (ED), and two interaction terms (P*ED and R*ED). The B_5 partial regression coefficient tests for the differential linear change in heritability as a function of parental education.

The result of the extended DF regression test for linear G × E interaction between parent education and genetic influences on probands' deficits in reading demonstrated that genetic influences on reading disability increased significantly with increasing levels of parental education (β= .272, t = 3.23, $\underline{P_{rep}}$ = .99). The standardized beta coefficient for the interaction provides a measure of the effect size, and a post hoc analysis using G*power3 (Faul, Erdfelder, Lang, & Buchner, 2007) indicated that the power to find this effect was .74. The positive direction of the interaction demonstrated that the heritability of deficits in reading tended to be higher for children whose parents were more highly educated than for children whose parents were less educated.

Genetic and environmental influences in low and high education groups

To illustrate the pattern of differences in genetic and environmental influences on RD depending on parents' years of education, our third analysis employed a median (13.2 years) split on parents' years of education to divide the sample into a lower parent education group and a higher parent education group. We then estimated the genetic, shared environment, and non-shared environment influences on RD separately within the higher and lower parent education groups. The resulting estimates presented in Table 2 and are illustrated in Figure 1. We tested the significance of the group differences in genetic and environmental influences using the method described by Purcell and Sham (2003). Although this analysis did not demonstrate a significant two-tailed ($p < .05$) G × E interaction due to the linear variance within groups that was lost by the median split, the group contrasts in genetic and shared environment influences were both significant in one-tailed tests ($p < .05$). These results are consistent with the direction of the significant linear G × E interaction within the full RD group: genetic influences were greater in the higher education group (h^2_g = .71) than in the lower education group (h^2_g = .49), and shared environment estimates were greater in the lower education group (c^2_g = .42) than in the higher education group (c^2_g = .22). Estimates of non-shared environment influence were similar in the lower education (e^2_g = .10) and higher education (e^2_g = .07) groups.

Discussion

The present study investigated whether level of parental education moderates the heritability of reading disability (RD) in a selected sample of 545 identical and fraternal twin pairs. Previous studies have explored G x E interactions for individual differences in reading and general cognitive abilities across the normal range, but this is the first study to explore possible G × E interactions within a sample specifically selected for RD.

We found that the heritability of RD, assessed by low performance on a composite measure of printed word recognition, spelling, and reading comprehension, was .61 averaged across the whole sample. However, there was a significant linear interaction between parents' years of education and the heritability of RD within the selected sample: Children whose parents

had higher levels of education tended to have stronger genetic influence on their RD compared to children with RD whose parents had lower levels of education. The present results based on the composite measure of word recognition, spelling and reading comprehension are consistent with unpublished results for each skill analyzed separately that are available from the authors.

Our results support a gene-by-environment interaction for RD because parental education has been shown to predict a variety of child health and educational outcomes, greater investment in children's educational development, and socioeconomic status (Craig, 2006). In addition, parental education has been shown to correlate at $r = .4$ with the average third-grade school score on the Colorado State reading assessment in a separate longitudinal twin study of individual differences in early reading development (Olson, Byrne, & Samuelsson, in press). That study included 488 twin families drawn from the same sampling area as the present RD sample that was mostly assessed before mean school scores became available . The correlation between mean school performance and parents' years of education might be due to poor early reading instruction during the early grades, on average, in schools with lower SES families However, Olson et al. also found that the correlations between parents' years of education and their twins' reading performance of about $r = .3$ across several measures were not significantly moderated by mean school score.

We recognize that parental education may also be influenced by parents' genes related to reading ability that may be transmitted to their children, and it is also possible that these parental genes could influence children's family and school environment for reading development, resulting in a gene by environment correlation (Plomin, DeFries, McClearn, & McGuffin, 2008). Although it is not possible to assess this correlation directly with the data from the twins in the present study, we controlled for its potential influence on the G × E interaction by using a residual parent education variable adjusted for its correlation with probands' reading scores. We also included the main effect of parent education in the DF regression test of the G × E interaction to control for the influence of any G × E correlation on the G × E interaction (Purcell, 2002)

In the introduction, we presented the bioecological and diathesis-stress models which offer competing hypotheses for the direction of G × E interaction effects. The bioecological model states that heritability will be greater when there is environmental support for the actualization of genetic potential, whereas the diathesis-stress model states that heritability will be greater in stressful environments, which exacerbate genetic susceptibility. Our results clearly support the bioecological model of G × E interaction for the heritability of RD. On average, children who failed in reading in spite of good environmental support for learning to read tended to have stronger genetic influences on their RD. Environmental influences on RD tended to be stronger among children learning to read in less supportive environments. Of course these results leave many questions open about the exact nature of the genetic and environmental influences on RD that are associated with parent education.

It is important to keep in mind that when genetic and environmental influences are estimated in behavior-genetic studies of RD, we are estimating their average influences on variance in RD group membership. The moderate correlation of .4 between parents' years of education and mean school performance indicates that some families with low parent education may have relatively supportive environments for learning to read. It is also possible that the range of environmental support for reading development is greater across families with lower parent education. This greater range would tend to increase estimates of shared environment and decrease estimates of genetic influence.

Conclusions

The present results have important implications for genetic research on RD, the diagnosis of RD, and current federally mandated public education policies in the U.S.

Implications for behavior- and molecular-genetic research on RD

Deviant-group and unselected-population estimates of genetic and environmental influences are average estimates that do not specify the level of these influences for any individual. The present results clarify that behavior-genetic estimates of genetic and environmental influences on RD and other learning disabilities may depend on the level of relevant environmental support within the population studied. Whenever direct information about the relevant environment for a behavior is available, it may be useful to include that information and test for potential G × E interactions. Of course it is important to recognize that these interactions will not account for all of the individual variation in genetic and environmental influence. In the context of the present study, this means that some children of parents with low education may have very strong genetic influence on their RD, while some children whose parents have very high levels of education may have little or no genetic influence on their RD.

The present behavior-genetic results also are potentially useful for future molecular-genetic studies of RD and other learning disabilities. The identification of specific genes is a considerable challenge for complex cognitive disorders such as RD. Current evidence suggests that there may be many different genes involved, each with small average effects on RD in the population (Meaburn, Harlaar, Craig, Schalwyk, & Plomin, 2007). Information about environmental factors such as parent education could be used to select samples for molecular-genetic studies that are most likely to have strong genetic influences on their RD.

Implications for the diagnosis and remediation of RD

Much current research on the diagnosis of RD is focused on assessing children's response to instruction (Fuchs & Fuchs, 2006). The four Learning Disabilities Research Centers currently funded by NIH, including the Center in Colorado (Olson, 2006), are tracking children's response to systematic and intensive interventions for RD. The basic rationale for this focus on response to instruction is that reading failure in many children may result from poor instruction and/or lack of reading practice. The goal is to identify and correct these instructional failures, and to provide much more intensive intervention for children who do not respond to good instruction. Results from the present study of G × E interactions support the idea that poor instruction or lack of reading practice may often be the main cause of RD in low SES families, while genes may be the main influence on RD among most children in higher SES families who may already be receiving good instruction.

Implications for public education policy

The “No Child Left Behind” legislation (107th Congress, 2002) has the laudable goal of improving literacy by improving the educational environment. The relatively strong influence of the environment that we found on RD in low SES families certainly supports the value of this effort. On the other hand, there is still some significant average genetic influence on RD among low SES families, and the relatively strong average genetic influence on RD in high SES families indicates that many cases of RD, particularly those expressed in a supportive educational environment, are likely to have a primarily genetic origin.

A genetic basis for some cases of RD does not imply that intense and systematic remedial interventions for these cases will have no benefit. However, recent evidence from a

longitudinal twin study of early reading development has shown that genes have a strong influence on individual differences in young children's experimentally assessed learning rates for reading and related skills (Byrne et al., 2008). Genetic constraints on learning rate are not recognized in the “No Child Left Behind” legislation, with its requirement that all children reach “grade level” (i.e., average) performance in reading and other academic skills by 2014, and its assumption that this lofty goal can be met for all children with appropriate education. A more beneficial policy would acknowledge genetic constraints on meeting a “grade-level” standard for some children with RD. It would also recognize and honor the extraordinary effort that these children, their parents, and their teachers may have to expend to make functionally important gains in reading and other academic skills, even if they do not reach “grade level” (Olson, 2006; Olson et al., in press).

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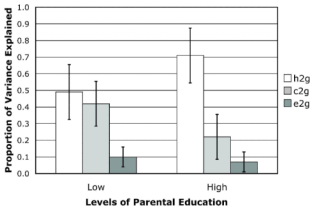

Figure 1.

Heritability (h2g), shared environment (c2g), and non-shared environment (e2g) in RD for lower and higher levels of parental education. Error bars indicate confidence intervals around the point estimates.

Table 1

Percent of Families and Levels of Educational Achievement for the State of Colorado, Proband Group, and No School History Group.

Educational Achievement	Colorado	Probands	No School History
Less than 9th grade	4.8	1.0	0.5
Less than H. S. diploma	8.2	11.9	2.1
H. S. diploma	23.2	16.1	6.2
1 year of College	24.0	21.0	12.0
2 years of college/A. A.	7.0	21.6	17.5
4 years of college/B. A	21.6	18.1	33.9
5+ years of college	11.1	9.2	26.6

Note: Colorado information based on year 2000 Census.

Table 2

Descriptive Statistics for Transformed Cotwin Scores by Zygosity and Additive Genetic, Shared Environment, and Nonshared Environment Estimates by Parental Education.

Parent Ed.	MZ Cotwins			DZ Cotwins								
	M	SD	N	M	SD	N	h^2_g	C. I.	c^2_g	C. I.	e^2_g	C. I.
All	.91	.38	236	.61	.50	309	.61	(.50 − .72)	.30	(.21 − .40)	.09	(.04 − .13)
Low	.92	.36	122	.66	.47	131	.49	(.32 − .66)	.42	(.28− .55)	.09	(.04 − .15)
High	.91	.40	114	.55	.52	178	.71	(.55 − .88)	.22	(.09 − .34)	.07	(.01 − .13)

Note. Cotwin DISCR scores were transformed such that cotwin scores vary between 0 and 1 where one indicates a perfect group correlation with probands and zero indicates no group correlation with probands. C.I. = 95% confidence interval.