Published in final edited form as:
Learn Individ Differ. 2011 August 1; 21(4): 368–375. doi:10.1016/j.lindif.2011.04.008.

A Twin and Adoption Study of Reading Achievement: Exploration of Shared-Environmental and Gene-Environment-Interaction Effects

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Abstract

Existing behavior-genetic research implicates substantial influence of heredity and modest influence of shared environment on reading achievement and reading disability. Applying DeFries-Fulker analysis to a combined sample of twins and adoptees (N = 4,886, including 266 reading-disabled probands), the present study replicates prior findings of considerable heritability for both reading achievement and reading disability. A simple biometric model adequately described parent and offspring data (combined N = 9,430 parents and offspring) across differing types of families present in the sample Analyses yielded a high heritability estimate (around 0.70) and a negligible shared-environmentality estimate for both reading achievement and reading disability. No evidence of gene × environment interaction was found for parental reading ability and parental educational attainment, the two moderators analyzed.

Keywords

reading achievement; reading disability; behavior genetics; twin study; adoption study

1. Introduction

Reading is without question an important academic skill. Yet, individuals vary with respect to their level of reading ability. Further, reading disability (or “Reading Disorder”, American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders IV-TR, 2000) is not rare, affecting around 4% of school-age children in the U.S. Reading disability is characterized by “reading achievement…that falls substantially below that expected given the individual’s chronological age, measured intelligence, and age-appropriate education” (*DSM-IV-TR*, p.51). The present study is an attempt to further understand individual differences in reading achievement and the etiology of reading disability. We estimate the degree to which genetics versus the shared environment (which comprises family context, neighborhoods, schools, etc.) contribute to reading achievement and reading disability. Finally, we explore whether the reading ability and education level of parents moderates the degree to which genetics, or the shared environment, shape the reading ability of their offspring.

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There is a substantial and consistent behavior-genetic literature implicating the existence of genetic influences on reading abilities and disability. Analysis of twin samples selected for reading problems or disability (reviewed by Grigorenko, 2004) indicate that roughly 50-70% of variance in reading problems is associated with genetic factors. Analysis of twin samples unselected for reading problems have generally reported similar findings of strong heritable effects. For example, in a sample of more than 4000 pairs of British twins assessed longitudinally, Harlaar et al. (Harlaar, Dale, & Plomin, 2007) reported that genetic factors accounted for approximately 60% of the variance in teacher-rated reading achievement at ages 7, 9 and 10. Similarly, in a sample of 440 twins age 8 to 18, Gayán and Olsen (Gayan & Olson, 2003) reported heritability estimates of 80% or higher for 4 different measures of reading performance (e.g., word recognition, phonological decoding). Finally, in a community based sample of 605 pairs of twins age 10 years, Zurnberge et al. (Zurnberge, Baker, & Manis, 2007) reported a heritability estimate of 70% for reading as assessed by subtests from the Woodcock–Johnson Tests of Achievement. Importantly, not only are reading disability and reading abilities substantially heritable, but the influence of genetic risk factors for reading disability appears to be of comparable magnitude to those that contribute to individual differences in reading-related phenotypes throughout the normal range (Bates, et al., 2007; Hawke, Wadsworth, Olson, & DeFries, 2007).

Behavioral genetic research has established the heritability of reading-related phenotypes. It has additionally proved useful in providing a basis for efforts to identify the specific genetic factors that influence reading (Grigorenko, 2009). Nonetheless, several important questions about the behavioral genetics of reading remain largely unresolved. Broadly speaking, these questions concern the magnitude of shared-environmental influence, and the existence of gene-environment interaction effect.

First, even if substantial, heritability estimates for reading are typically far less than unity, implicating the contribution of environmental factors. Yet the nature of these environmental influences is unclear. Behavioral geneticists distinguish between shared environmental factors (i.e., those environmental factors that are shared by reared-together siblings and consequently contribute to their behavioral similarity), and non-shared environmental factors (i.e., those environmental factors that differ among reared-together siblings and thus do not contribute to their behavioral similarity). Most twin studies of reading suggest minimal contribution of shared environmental factors. For example, the previously mentioned twin studies by Harlaar et al. (Harlaar, et al., 2007), Gayán and Olsen (Gayan & Olson, 2003) and, Zurnberge et al. (Zurnberge, et al., 2007) all concluded that shared environmental factors accounted for 10% or less of the phenotypic variance in reading. Nonetheless, the estimation of shared environmental effects in a twin study is indirect (i.e., based on a comparison of monozygotic and dizygotic twin similarity), and estimates might differ when more direct approaches, such as an adoption study, are used.

There have been two adoption studies of reading and their results appear to conflict. In a longitudinal study in the Colorado Adoption Project, Wadsworth et al. (Wadsworth, Corley, Hewitt, & DeFries, 2001) reported small point estimates (less than .10) of shared-environmental variance at all three ages at which the children were assessed (7, 12, and 16). Further, the most parsimonious biometric model that still provided adequate fit fixed all shared-environment parameters to zero. Subsequently, Wadsworth et al. (Wadsworth, Corley, Hewitt, Plomin, & DeFries, 2002) reported that there was no significant parent-offspring resemblance in adoptive families but moderate and significant parent-offspring resemblance in non-adoptive (i.e., genetically related) families at each of the three assessments. This study thus suggests little or no shared environmental effects on reading. In contrast, in a study of elementary school aged children that included both twin and adopted sibling pairs, Petrill et al. (Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider,

2006) reported consistent and significant adopted sibling correlations (ranging 0.22-0.32) across a battery of reading measures, implicating the importance of shared environmental effects. From the combined sample of twins and adoptees, shared-environmentality estimates ranged 0.20-0.40. Petrill et al. interpret these relatively high estimates as resulting largely from the young age of the participants. In particular, they note that the skills important specifically to novice readers seem to be prone to greater influence by characteristics of the home environment (such as parents' vocabulary or availability of printed material). Among more experienced readers, such as the adolescents and older children composing the present study's sample, the influence of the shared environment on reading ability would be expected to be relatively smaller. However, it is questionable whether age difference would account for the inconsistent results reported by Wadsworth et al. (with mean participant age 7.4 years at first assessment) and Petrill et al. (with mean age of 6.1 years among twins, and 7.3 years among adoptees).

A second issue not fully resolved by the available behavioral genetic literature concerns how genetic and environmental factors combine to influence reading outcomes. A general consensus has emerged that rather than combining merely additively, as is assumed in most biometric models, genetic and environmental influences on behavioral phenotypes are likely to also act synergistically (Moffitt, Caspi, & Rutter, 2005). Unfortunately, the number of relevant gene-environment interaction studies of reading is both limited and not altogether consistent (Pennington, et al., 2009). In a study of school-aged twins, Taylor et al. (Taylor, Roehrig, Hensler, Connor, & Schatschneider, 2010) showed that heritable influences on reading increased with teacher quality, suggesting that the effect of poor teaching may be to prevent children from reaching their full potential. Friend et al. (Friend, DeFries, & Olson, 2008) further found that genetic effects on reading disability were highest among children with highly educated parents. In a subsequent publication using a different twin sample, Friend et al. (Friend, et al., 2009) reported that the heritability of high reading performance decreased with increasing level of parental education.

The present study involved the use of a large and combined sample of twins, adoptees, non-twin siblings, and their parents, who were assessed using the Wide Range Achievement Test. The present study addresses three major questions about the behavioral genetics of reading:

1. Can we confirm in a combined sample of twins and adopted individuals the strong heritable effect on reading achievement observed in previous studies?
2. What is the evidence for shared environmental influences on reading?
3. Does parent education moderate the heritability of reading?

2. Material and methods

2.1. Sample

Data were collected from participants recruited to the Minnesota Twin Family Study ("MTFS;" Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Iacono & McGue, 2002) and the Sibling Interaction and Behavior Study ("SIBS;" McGue et al., 2007). The MTFS is a longitudinal study that uses a community-based sample of parents and their same-sex twin offspring (N = 3779 twins, including five sets of triplets). All twins were born between 1972 and 1994 in the State of Minnesota. The SIBS is an adoption study of sibling pairs (N = 1232) and their parents. This community-based sample consists of families where both siblings are adopted, where both are biologically related to the parents, or where one is adopted and one is biologically related. For the present study, we excluded parents who were not the original rearing parents for the family, such as stepparents. Usually, the original

rearing parents would be the biological parents of the family's offspring, but in the case of families with only adopted offspring, the original rearing parents would be the parents with whom the children were first placed for adoption. Written informed assent or consent was obtained from all participants, and parents provided written consent for minor offspring.

Except where noted otherwise, the present report uses data only from the intake assessments of these studies. There were 2,498 families where at least one member had valid intake data for reading achievement. Of these families, 1881 were from the MTFS, comprising 3373 parents and 3712 offspring with valid data. The remaining 617 families were from the SIBS, comprising 1119 parents and 1226 offspring with valid data. Thus, the full sample consists of 9,430 individuals in 2,498 families. Descriptive statistics for the sample are provided in Table I.

The MTFS consists of two age cohorts, named for the target age of twins at the intake assessment: the eleven-year-old cohort (10-13 years old at intake; mean age = 11.78), and the seventeen-year-old cohort (16-18 years old at intake; mean age = 17.48). In SIBS families, age at intake ranged from 10-19 years (mean age = 13.75) among the younger siblings of each pair, and ranged 12-20 years (mean age = 16.09) among their older siblings. The eleven-year-old cohort of the MTFS includes a subsample, referred to as the Enrichment Study (ES) sample, recruited subsequently to the original MTFS sample. The ES sample was so named because its objective was to "enrich" the MTFS sample with younger twins at risk for externalizing psychopathology (Keyes et al., 2009). To this end, a subset of the ES sample was recruited on the basis of a screening interview for possible externalizing psychopathology[1]. As will be explained below, the intake assessment was slightly different for the ES compared to the original MTFS.

2.2. Measurements

2.2.1. Twin zygosity—Twin zygosity was assessed via three criteria: a standard zygosity questionnaire filled out by parents, staff judgment of physical similarity, and an anthropometric algorithm. In cases where these three criteria gave conflicting conclusions, zygosity was resolved by serological analysis of genetic markers.

2.2.2. Ability testing—Intake assessment for both MTFS and SIBS included evaluation of participants' academic achievement and general cognitive ability. Reading achievement was assessed using the Wide-Range Achievement Test (WRAT). WRAT-3 (Wilkinson, 1993) was used for SIBS and ES intake, whereas WRAT-R (Jestak & Wilkinson, 1984) was used for the original MTFS, using the age-appropriate form (i.e., Level 1 for twins aged eleven or younger, and Level 2 for any participant older than eleven). Scaled scores from the WRAT-3 are normed to a population distribution with mean of 100 and standard deviation of 15. However, scaled scores were not available for participants who received WRAT-R. Consequently, we formed scaled scores for the original-MTFS participants by rescaling their raw scores to the observed distribution of WRAT-3 scaled scores among comparable participants, using a linear-equating procedure. Scores from the original-MTFS 11-year-old cohort were transformed to the scale of intake WRAT-3 scores among community-sampled (i.e., not recruited for probable externalizing psychopathology) ES twins. Scores from the original-MTFS 17-year-old cohort were transformed to the scale of WRAT-3 scores among community-sampled ES twins taken at their second follow-up assessment (when seventeen

[1]Because the twins in this subset were overrepresented in our sample, we re-ran core analyses using a weighting scheme that reflected this overrepresentation. Specifically, differential weighting of the samples was used in the calculation of the Pearson correlations for reading achievement and the tetrachoric correlations for reading disability. They were also used in the full-sample DeFries-Fulker analysis for reading achievement. Estimates calculated with weighting differed, at most, by 0.005 from the unweighted results we report.

was the target age). Some 307 original-MTFS parents were tested with WRAT-3 when they returned for a follow-up assessment, and therefore had scores on both tests, which correlated r = 0.83, with approximately three years between tests[2]. All parents' WRAT-R scores from intake were transformed to the scale of WRAT-3 scores among these double-tested parents from the first follow-up. These transformed scores from WRAT-R at intake were entered into analysis.

Participants' general cognitive ability was assessed at intake by way of an abbreviated form of the Wechsler Adult Intelligence Scale-Revised (WAIS-R) or Wechsler Intelligence Scale for Children-Revised (WISC-R), as age-appropriate (that is, older than 16, and 16 or younger, respectively). The short forms consisted of two Verbal subtests (Information and Vocabulary) and two Performance subtests (Block Design and Picture Arrangement), the scaled scores of which were prorated to determine Full-Scale IQ (FSIQ). FSIQ estimates from this short form were shown to correlate 0.94 with FSIQ from the complete test (Sattler, 1974). Parents in the SIBS sample were not IQ-tested at the intake assessment, but received the WAIS-R at the first follow-up, for which only one parent per family (usually the mother) returned. Consequently, IQ data for SIBS fathers is very limited in its availability.

2.2.3. Parental Education—Parental years of education was obtained in a telephone interview with mothers completed prior to the family's in-person intake assessment. Mothers reported both their own as well as the father's level of educational attainment. Education was coded in terms of years of completed education, and when appropriate, capped at a maximum to reflect degree attainment (e.g., 12 years was the maximum allowed a participant with only a high school diploma, even if he or she reported more than 12 years). In cases where a parent's years-of-education was missing, but his or her degree attainment was known, we substituted the "cap" associated with his or her degree attainment as the (missing) number of years. When used in analysis, we treat parental education as a family-level variable by taking its midparental mean for each family. If only one parent had valid years-of-education, this midparental mean would be that one parent's value. Every family in the sample had valid education data for at least one parent.

2.2.4. Reading disability—We operationally defined reading disability as possessing a reading achievement score 1.5 standard deviations or more *below* expected reading achievement conditional on Full-Scale IQ (FSIQ). While we recognize that there is much debate about the IQ-discrepancy model of reading disability, especially in applied settings (Fletcher et al., 2004), we wanted to explore familial resemblance of reading net IQ, which the discrepancy model allows us to do. Using all individuals from MTFS and SIBS who had valid reading-achievement and IQ data from intake, we calculated the simple linear regression of reading achievement onto FSIQ. Then, using these coefficient estimates, we calculated the expected reading achievement score for each individual in the sample conditional on their FSIQ, and their residual (their conditional expected score minus their actual, observed score; hereinafter, "partial reading achievement"). We standardized these residuals, and flagged as "reading disabled" any participant with a standardized residual of −1.5 or less. There were 642 members of the sample (13 offspring, 158 mothers, and 471 fathers) who had missing IQ data. Their reading-disabled status was also treated as missing. As explained above, most of the parents who were missing IQ were SIBS parents for whom IQ testing was not included in the plan for their assessments.

[2]For the sake of comparison, the WRAT-3 manual reports an uncorrected correlation of r = 0.88 between WRAT-R and WRAT-3 Reading scores (Wilkinson, 1993).

2.3. Analysis

2.3.1. Phenotypic correlations—We began our analyses by estimating the correlational structure of reading achievement in the five distinct types of families represented in our sample (numbers of families for each type are in parentheses): monozygotic (MZ)-twin families (1,195), dizygotic (DZ)-twin families (686), SIBS families with two biological offspring (208), SIBS families with two adopted offspring (285), and "mixed" SIBS families with one biological and one adopted offspring (124). Each family type recognizes four members: "Offspring 1," "Offspring 2," the mother, and the father. The type of family determines what will distinguish Offspring 1 and Offspring 2. For instance, in mixed SIBS families, these labels serve to distinguish the adopted from the biological offspring, whereas in twin families, the distinction between twins is arbitrary. In any event, the correlation matrices for a family-type will be 4 × 4.

We fitted two models for the correlation matrices, via multivariate-normal maximum-likelihood estimation from the raw data, in Mx (Neale, Boker, Xie, & Maes, 2003). In the first model, correlations were estimated separately for each family-type, and within each family-type, offspring were considered indistinguishable, so that two parameters, an offspring-mother and an offspring-father correlation, accounted for the four observed parent-offspring correlations in each matrix. The exception was for mixed SIBS families, where all four parent-offspring correlations were free parameters. The second model imposed equality constraints across family-types, so that correlations were modeled in terms of eight free parameters, one for each distinct kind of relationship in the sample (MZ twins, DZ twins/biological siblings, adoptive siblings, spouses, biological father with offspring, biological mother with offspring, adoptive father with offspring, adoptive mother with offspring). Although this second model is not explicitly biometric, the estimates for its eight parameters are informative regarding the sort of biometric model suitable for the data, and is thus useful for model selection.

We also calculated tetrachoric correlation coefficients for reading disability, treated as a dichotomous outcome, under assumptions similar to the second model for the correlational structure of reading achievement. Namely, we assumed that only one correlation per type of familial relationship was necessary, and that estimates of the same correlation from different family types could be constrained equal. Only one parent per SIBS family was assessed for FSIQ, so only one parent per SIBS family had known reading-disability status. For this reason, we calculated one tetrachoric correlation for adoptive offspring with "parent," rather than two separate correlations for adoptive mothers and fathers.

2.3.2. DeFries-Fulker Analysis—DeFries-Fulker (DF) analysis (DeFries & Fulker, 1985) can be used in "full-range" (i.e.,unselected) samples to obtain unbiased estimates of biometric variance components of a quantitative trait, via linear regression, conditional on the assumptions of the additive-genetic model. This application of the DF model, which uses double-entered, mean-centered data from sibling pairs, is due to Rodgers & McGue (1994) and Rodgers & Kohler (2005). Kirkpatrick, McGue, and Iacono (2009) have previously described the use of DF analysis in a combined sample from the MTFS and SIBS. From Rodgers and Kohler's (2005) Equation 7, the full-range DF regression model is

$$K_1 = b_1 K_2 + b_2 (K_2 R) + e$$

where K_1 is one sibling's score, K_2 is the co-sibling's score, R is the coefficient of relationship (degree of genetic relatedness), and e is the residual; b_1 estimates shared-environmentality (c^2), and b_2 estimates additive heritability (a^2). This biometric model is sometimes referred to as the "ACE" model because it decomposes phenotypic variance into

portions due to Additive genetic, Common (or "shared") environment, and unshared Environment (which includes measurement error, and is represented in the DF model as the residual variance). Moderation of this biometric decomposition can easily be analyzed by adding a term for the hypothesized moderator variable and its interactions with K_2 and K_2R. Our full-range DF analysis used 2,443 sibling pairs; 56 participants from incomplete pairs (due to missing sibling data) were excluded.

DF analysis was originally developed for use with samples of reading-disabled probands and their co-twins (DeFries & Fulker, 1985). Siblings selected for extreme scores on a trait are referred to as "probands," and the siblings of probands are termed "co-sibs." "Differential" DF analysis in a sample of probands and co-sibs relies upon the differential regression of co-sib scores to the general population mean, as a function of degree of biological relatedness. After trait scores have been suitably transformed (DeFries & Fulker, 1988), differential DF enables estimation of the extent to which reading-disability is due to genetics and shared environment, again conditional on the assumptions of the additive-genetic model. These parameters are referred to as *group* heritability (h_g^2) and shared-environmentality (c_g^2). The modifier "group" is necessary to distinguish these parameters from a^2 and c^2, which are components of population variance. In contrast, group heritability and shared-environmentality are components not of a variance, but of a difference between the general-population mean and the mean of a subsample of probands.

Kirkpatrick et al. (2009) have previously described the use of DF analysis in a subsample of extreme-score probands from the MTFS and SIBS, including the direct estimation of group shared-environmentality (c_g^2) afforded by the inclusion of adoptive siblings. The availability of both adoptive-sibling and MZ-twin pairs in our subsample enables direct estimation of both h_g^2 and c_g^2. Group heritability is estimated by b_2 in the basic differential DF model (from Equation 1 in DeFries & Fulker, 1985),

$$C=a+b_1P+b_2R+e$$

where C is co-sib's score, a is the regression constant, P is proband score, and R is the coefficient of relationship. Group shared-environmentality can be estimated as the mean transformed score of adoptive co-sibs. Moderation analysis is easily accomplished by adding the necessary terms to the differential DF regression. For example, if M denotes the hypothesized moderator, the regression equation would be

$$C=a+b_1P+b_2R+b_3M+b_4\,(PM)+b_5\,(RM)+e$$

In samples consisting only of twins, h_g^2 is estimated as $2b_2$ in the basic DF regression, but c_g^2 is indirectly estimated as the transformed MZ co-sib mean minus h_g^2. The direct estimate of c_g^2 described previously is easily calculated and has the sampling distribution of a sample mean, but does not make use of the available twin data. The indirect estimate does not have an obvious sampling variance, which would depend upon the covariance of the transformed MZ co-sib mean with b_2. However, it makes use of the entire reading-disabled subsample, and enables straightforward testing for moderation of c_g^2 (if h_g^2 is not moderated, neither will be c_g^2).

When the sampling distribution of a statistic is difficult or even impossible to obtain analytically, it can be obtained empirically via nonparametric bootstrapping (see Johnson, 2001, for an accessible introduction to the topic; Efron, 1982, provides a detailed theoretical treatment). Provided that the sample is representative and sufficiently large, the empirical distribution of the variables of interest will approximate their true distribution. The sample can be used as a proxy for the population, and a Monte Carlo procedure can simulate the repeated sampling of independent observations from this "population." To simulate an empirical sampling distribution for both c_g^2 estimates, we drew 20,000 bootstrap samples of 2,498 families each from the full real-data sample, with replacement. For each bootstrap sample, we extracted a subsample of reading-disabled probands and obtained estimates from a differential DF analysis.

Based upon familial phenotypic correlations (reported below), we chose to restrict our biometric analysis of this phenotype to data from offspring, using parental data as a potential moderator only. We used parental data in an extended DF regression containing interaction terms (e.g., Friend et al., 2009). We used family-level midparental means of educational attainment (described above) as a moderator variable for both reading disability and for full-range reading achievement. We likewise used midparental means of available parent scores for reading achievement and partial reading achievement, using the former in an analysis of offspring reading achievement and the latter in an analysis of offspring reading disability. Thus, we conducted four moderation analyses in all.

3. Results

3.1. Model selection & phenotypic correlations

The first step of the analysis was to compare the fit of two models of the phenotypic correlational structure across family types. As described above, the first model leaves the correlational structure free to vary across family types. The implicit assumption of the second model is that the reading-achievement correlation between two family members depends upon their degree of biological relatedness and their respective roles in the family (i.e., parent or child), and not upon the type of family in which they live. If the second model fits the data poorly, that would provide evidence against this assumption, and suggest that the phenotypic correlational structure is heterogeneous across family-types. However, the second, simpler model provided adequate fit to the data while using fewer parameters than the first ($\chi^2 = 10.056$ on 14 df, $p = 0.76$; $\Delta AIC = -17.944$; $\Delta BIC = -49.735$).

Estimates for these parameters, and their likelihood-based confidence intervals (see Neale & Miller, 1997) are provided in Table II. They are quite consistent with a substantial additive heritability estimate around 0.70, and a small shared-environmentality estimate around 0.05. The parent-offspring correlations appear to be due to additive heritability only. The spousal correlation of about 0.25 indicates the possibility of a modest assortative-mating effect, which would cause twin-based estimates of heritability to be underestimated, and shared-environmentality to be commensurately overestimated. However, the consistency of the well-known Falconer estimate of shared-environmentality ($c^2 = 2r_{DZ} - r_{MZ}$) with the direct estimate from the adoptive-sibling correlation shows that the effect of assortative mating is likely negligible. A simple and easily interpretable ACE model applied to offspring data appears sufficient for biometric analysis.

Table II also reports tetrachoric correlations for reading disability as a dichotomous outcome, for each distinct familial relationship. Due to convergence problems during computation of some likelihood-based confidence intervals, we instead report confidence intervals computed from asymptotic standard errors for three tetrachorics—those for MZ-

twins and biological parents-with-offspring. As with the Pearson correlations for reading achievement, these tetrachorics are modeled with equality constraints across family-types on the correlations. Because only one parent per SIBS family had known reading-disability status, we calculated a single, pooled adoptive parent-offspring correlation, rather than two separate values for mothers and fathers. The tetrachorics for twins and siblings clearly seem to suggest a substantial additive heritability of over 0.80 for reading disability, along with essentially zero shared-environmentality. The tetrachoric correlations between parents and biological offspring also suggest substantial heritability for reading disability. However, that between adoptive parents and offspring does not point to any clear conclusion about environmentally mediated parental influence on the etiology of reading achievement: its point estimate was greater than 0.20, but was not significantly different from zero.

3.2. DeFries-Fulker analysis

We obtained standardized biometric parameter estimates for reading achievement from a full-range DF analysis, and calculated 95% confidence intervals for them using asymptotic standard errors from Kohler and Rodgers' (2001) procedure: a^2 = 0.681 (0.597, 0765), c^2 = 0.057 (−0.016, 0.0129). We repeated this analysis using *partial* reading achievement (residuals from regression of reading achievement onto FSIQ). FSIQ accounted for 27.23% of the variance in reading achievement. The additive heritability and shared-environmentality of partial reading achievement (with 95% confidence intervals) were a^2 = 0.624 (0.535, 0.712) and c^2 = 0.025 (−0.049, 0.100). Both standardized estimates were slightly smaller for partial reading achievement, suggesting that some of the reliable variance in reading achievement scores was lost when FSIQ was regressed out.

We operationally defined reading disability as having a reading achievement score 1.5 standard deviations or more below expected average given the participant's IQ. On this criterion, we identified a subsample of 266 reading-disabled probands among the twins and siblings; two MZ-twin probands were missing co-sib data, and were excluded from analysis. Differential DF analysis applied to this subsample of probands (after partial reading achievement scores have been suitably transformed; DeFries & Fulker, 1988) enables estimation of *group* heritability (h_g^2) and shared-environmentality (c_g^2). Nonparametric bootstrapping provided empirical sampling distributions for both c_g^2 estimates and the h_g^2 estimate, which we used to construct 95% confidence intervals. Table III summarizes the results of these three biometric analyses. We obtained an estimate of h_g^2=0.721 (0.585, 0.857). Both c_g^2 estimates were modest (smaller than 0.10) and neither differed significantly from zero. The direct estimate was 0.086, with a 95% confidence interval of (−0.035, 0.208) computed using bootstrap standard errors. The indirect estimate (not reported in Table III) was 0.051, with a 95% confidence interval of (−0.055, 0.161) from the 0.025 and 0.975 quantiles of the empirical sampling distribution.

Figure 1 displays the frequency distribution of partial reading achievement for the full sample, and marks the means for probands and groups of co-sibs. Consistent with a large contribution of heredity to reading disability, the mean of MZ-twin co-sibs fell closer to the proband than did that of DZ-twin co-sibs, which in turn fell closer than that of adoptive co-sibs. Consistent with a near-zero contribution of the shared environment to reading disability, the mean for adoptive co-sibs differed only slightly from the grand mean of the sample at zero.

3.3. Moderation analysis

Results of the moderation analyses are summarized in Table IV. We extended the differential DF regression to conduct two moderation analyses for group heritability. The

interaction between parental education level and biological relatedness was not significantly different from zero (p = 0.48), providing no evidence that parents' level of education moderates h_g^2. Likewise, the moderation effect of parental partial reading achievement on group heritability was not significantly different from zero (p = 0.25).

Similarly, we extended the full-range DF regression in two moderation analyses. The interaction between parental education and heritability was quite small, (0.007; 95% CI: −0.028, 0.041), amounting to less than a 1% change in the corresponding biometric component for a one-year change in parental education; it was not significantly different from zero (p = 0.71). Likewise, the interaction between parental reading achievement and heritability was quite small (0.005; 95% CI: −0.004, 0.014), and did not significantly differ from zero (p = 0.24).

4. Discussion

Our analyses were concerned with estimating the relative contribution of genetics and shared environment to reading achievement and reading disability, and whether the magnitude of these contributions depended upon parental characteristics (reading phenotype and level of education). As suggested by the phenotypic correlation matrices, we fit a simple ACE biometric model to reading-achievement and reading-disability data from a combined sample of twin and sibling pairs. For both traits, heritability was substantial (around 0.70) and shared-environmentality was quite small (around 0.05, not reliably different from zero). Our heritability estimates are of magnitude comparable to those reported in the studies we reviewed in the Background, and are consistent with the hypothesis that the effect of genetic variants underlying reading disability are of comparable magnitude to those contributing to individual variation in the full range of reading achievement. The inclusion of adoptive sibling pairs in our sample enabled us to estimate heritability with greater precision than would a sample of only twins, because the coefficient of biological relatedness in our DF regressions spanned three values (0, 0.5, and 1) rather than just the two it could take in a twin sample.

The relatively large heritability estimate for reading achievement should not be regarded as evidence that reading is "genetically determined." We have no doubt that instruction, practice, and experience with the written word are important to the development reading ability in young people. But virtually all children in the United States are taught to read, and are further given practice and experience with reading during their twelve years of compulsory education. An estimate of the heritability coefficient for a population depends upon the extent of the variation of relevant genetic and environmental factors in that population, including which of those are held constant "in the background." In a twin and adoption sample from a population lacking universal public education at the primary and secondary levels, we might well obtain a relatively smaller heritability estimate for reading achievement, as a result of relatively greater variation in relevant environmental factors.

But further, we speculate that part of the heritable variance in reading achievement found in our sample may be explained by active gene-environment correlation (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983), the proposition that environments themselves are partially heritable. Children do not simply passively receive environmental influence but actively shape, by their own volition, the environments to which they are exposed. Older children and adolescents, such as those in our sample, typically have some autonomy regarding the degree to which their environments facilitate improved reading skills. For example, young people who consistently put time and effort into their assigned reading for school, or who choose to read in their spare time, will thereby obtain additional practice and experience with reading (relative to their peers who do not make such choices). Indeed, a

recent meta-analysis (Mol & Bus, 2011) reported that amount of exposure to the printed word during leisure-time reading was associated with reading ability, even among secondary- and university-level students. Perhaps individuals make such choices due to other individual attributes that are also substantially heritable, such as Big-Five Conscientiousness (Jang & Yamagata, 2009) or recreational interests (Lykken, Bouchard, McGue, & Tellegen, 1993). Though speculative, this account demonstrates that reading achievement can depend upon environmental exposure and nonetheless be highly heritable.

Relative to heritability, there is somewhat less consistency among previously reported estimates of shared-environmentality for reading achievement and reading disability. Our results suggest that the shared environment plays a minimal role in shaping children's reading abilities. This was found despite the fact that adoption samples are ideal for detecting the effect of the shared environment. The inclusion of adoptees in our sample afforded us an additional, direct estimate of c_g^2 (Kirkpatrick et al., 2009). However, we report both the direct and indirect estimates, and both lead to the same substantive conclusions. We note that one of the advantages of a model-fitting implementation of differential DF analysis (Purcell & Sham, 2003) over our conventional regression-based implementation is that the former provides a single estimate of c_g^2 computed from all available offspring data.

The influence of the unshared environment on the phenotypes studied can be estimated by summing the heritability and shared-environmentality estimates from each row of Table III, and subtracting the sum from unity. For example, e^2 for reading achievement would be 0.262, indicating that about 26% of the observed variance in reading achievement is nonhereditary and does not reflect behavioral similarity of siblings reared together. The unshared-environmental variance is the residual of the DF model and, consequently, our results permit little interpretation thereof. However, it does comprise phenotypic variance due to measurement error. Reliability estimates for WRAT-3 reading achievement are around 0.90 (Wilkinson, 1993). If we take this as an estimate of the non-error proportion of reading-achievement variance, then approximately 10% of total variance, and approximately 38% of that due to the unshared environment, would be the result of measurement error.

We did not find evidence that parents' education level affects, or moderates, the heritability of their children's reading disability. Our study had the advantage of the availability of measurements from parents, but these data did not improve the explanatory power of a simple biometric model. The relative contributions of heredity and shared environment appeared stable across levels of parental partial reading achievement, and across levels of parental education (i.e., no moderation). Our lack of support for moderation of reading-disability h_g^2 is inconsistent with the results of Friend et al. (2008), who found that reading-disability h_g^2 was higher for children with better-educated parents. The present study differs from that of Friend et al. in its measurement of reading achievement. We measured reading achievement using one test of word recognition and pronunciation. Friend et al. measured reading achievement more thoroughly, with a weighted sum of three tests: word recognition, spelling, and reading comprehension. The weights were taken from a previously conducted discriminant function analysis intended to distinguish individuals with a history of reading difficulty from those without. This measuring instrument is longer and samples more than one component of what could be called "reading achievement;" the narrower focus of our assessment may have prevented us from observing a moderation effect. We acknowledge that the generalizability of our results is limited by the relatively narrow scope of our measure of reading achievement; our study only addresses one aspect of reading proficiency.

We also operationally define reading disability differently from Friend et al. (2008). Their criterion for reading disability is a sufficiently low reading score and a history of reading

difficulty, after ruling out low IQ, sensory deficits, neurological pathology, and language barriers. Our definition does not take account of academic history, but our sample also does not include individuals with severe physical or mental handicaps, per exclusion criteria for both MTFS and SIBS. Further, we define reading disability in terms of poor reading achievement relative to what would be predicted from FSIQ. This is akin to the clinical definition of learning disability (see *DSM-IV-TR*) and does not require that an individual's reading score be low relative to the population (i.e., a fair reader could be considered to have a disability if his or her IQ was high enough to predict that he or she should be an excellent reader). These two definitions of the reading disability phenotype differ enough that one might plausibly be prone to G×E interaction, and the other not. Again, we recognize that strong arguments exist against the IQ-discrepancy definition of reading disability, at least in the context of educational policy (e.g., Fletcher et al., 2004). But, the IQ-discrepancy definition is suitable for basic research such as the present study, and avoids confounding general cognitive deficits with weak reading proficiency. Further, our dataset does not enable many alternative operational definitions; a broader and more comprehensive assessment of reading skills might have allowed us to employ a more nuanced definition of reading disability.

We also found no evidence that parents' level of education influences the heritability of reading within the non-reading-disabled population. This result is inconsistent with that of Kremen et al. (2005), who report that standardized heritability of reading achievement was higher for twins with more highly educated parents. This discrepancy is somewhat surprising, since Kremen et al. also used word-recognition from the WRAT to measure reading achievement. However, their sample and ours differ considerably in their demographics. We used a sample of male and female adolescents, whereas Kremen et al. used a sample of middle-aged male twin pairs (mean age = 40.8 years) concordant for military service during the Vietnam era (1965-1975). Cohort effects provide one possible explanation for the differing results. Kremen et al. conclude that total phenotypic variance, and the proportion of this variance attributable to the shared environment, both decreased as parental education increased, resulting in the observed moderation of standardized heritability. It seems plausible that the amount of variation in rearing environment might have been more strongly connected to parental education in an earlier generation. On the other hand, it is difficult to explain why parental characteristics would show stronger biometric-moderation effects in middle-aged adults versus adolescents, who still reside in their families of origin. Additionally, biometric-moderation effects of SES (of which parental education is a major determinant) have been observed for cognitive abilities in samples taken from younger generations (e.g., Turkheimer et al., 2003).

5. Conclusions

On the whole, our data provide additional evidence that both reading disability and full-range reading achievement are strongly influenced by genetic factors, with little to no influence from the shared environment. Our results differ from previous studies regarding the presence of G×E interaction, by which heritability depends upon some environmental variable. We do not find evidence of such moderation effects. We identify differences in measuring instruments and in operational definitions of reading disability, as well as possible generational cohort effects, as potential explanations for the discrepancy in results. Future research can investigate the substantive matter of G×E interaction for reading achievement and disability, and help further explain existing inconsistent results concerning it.

Acknowledgments

This work was supported by U.S. Public Health Service grants # AA09367, MH066140, DA005147, DA013240, and AA011886.

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Highlights

- Twin and adoption study of reading achievement and disability.
- Familial phenotypic correlations were consistent with a simple biometric model.
- Heritability estimates ~0.70; shared-environmentality estimates < 0.10.
- Heritability not moderated by parental education or reading achievement.

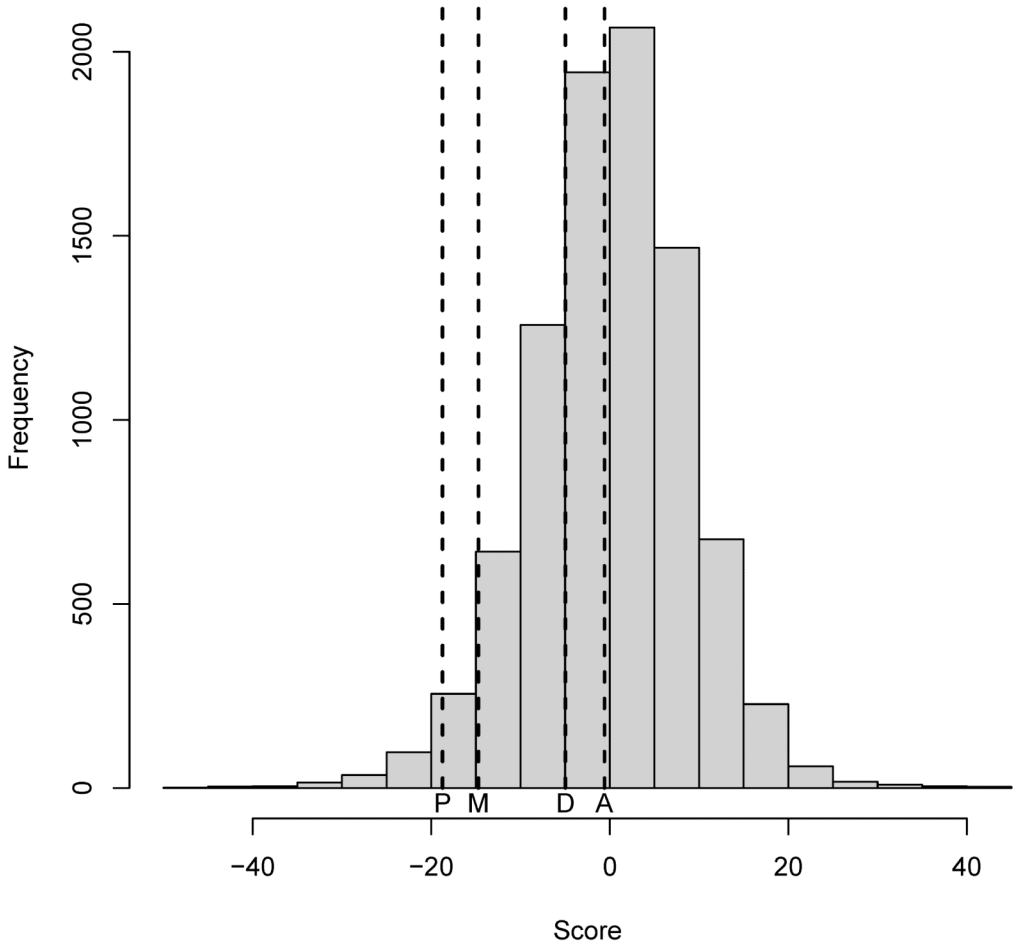

Figure 1.
Distribution of partial reading achievement
"Partial reading achievement" refers to reading achievement residualized for FSIQ. The dashed vertical line labeled "P" marks the mean score of reading-disabled probands. Those labeled "M," "D," and "A" mark the mean scores of MZ-twin, DZ-twin (including non-twin full siblings), and adoptive co-sibs of probands, respectively.

Learn Individ Differ. Author manuscript; available in PMC 2012 August 1.

Table I

Descriptive characteristics of full sample.

	MTFS MZ-twin Families			MTFS DZ-twin Families		
	Twins (older cohort)	**Twins (younger cohort)**	**Parents**	**Twins (older cohort)**	**Twins (younger cohort)**	**Parents**
N	812	1552	2168	422	926	1205
Female (%)	54.0%	49.8%	52.6%	53.0%	53.5%	54.0%
Mean Age (years)	17.45	11.77	42.44	17.52	11.80	43.12
Reading Achievement						
M	100.3	101.5	97.8	99.8	102.0	98.5
SD	10.2	11.4	9.9	9.8	10.5	9.8
Reading Disabled (%)	4.3%	6.3%	8.7%	4.0%	5.4%	7.7%

	SIBS Adoptive Families[a]		SIBS Non-adoptive Families	
	Adopted Offspring	**Adoptive Parents**	**Biological Offspring**	**Biological Parents**
N	690	754	536	365
Female (%)	55.4%	52.7%	54.1%	54.0%
Mean Age (years)	14.95	48.58	14.89	44.92
Reading Achievevent				
M	102.3	103.7	102.5	101.1
SD	9.8	9.3	10.0	10.0
Reading Disabled (%)	5.5%	3.9%	5.2%	7.0%

[a]The parents of “mixed” sibling pairs—one biological child and one adoptee—are represented as “Adoptive Parents.” The siblings themselves are represented depending upon their individual adoptive status.

Table II

Phenotypic correlations for reading achievement and reading disability.

Familial Relationship	Phenotypic Pearson correlation for reading achievement (95% confidence interval)	Phenotypic tetrachoric correlation for reading disability (95% confidence interval)
Spouses	0.255 (0.215, 0.294)	0.093 (−0.081, 0.262)
MZ twins	0.743 (0.723, 0.763)	0.858 (0.791, 0.925)
DZ twins & bio sibs	0.392 (0.339, 0.441)	0.433 (0.405, 0.618)
Unrelated sibs (in adoptive families)	0.058 (−0.038, 0.152)	−0.061 (−0.465, 0.334)
Biological offspring with mother	0.338 (0.307, 0.368)	0.301 (0.194, 0.408)
Biological offspring with father	0.36 (0.330, 0.395)	0.419 (0.326, 0.511)
Adopted offspring with mother	0.072 (−0.003, 0.146)	0.223[a] (−0.093, 0.502)
Adopted offspring with father	0.088 (0.009, 0.166)	

[a]Because only one parent per adoptive family had known reading-disability status, only one value was computed for the adoptive parent-child tetrachoric correlation, ignoring sex of parent.

Table III

Biometric analyses

Trait	Estimates Apply to…	Heritability (95% confidence interval)	Shared-environmentality (95% confidence interval)
Reading Achievement	Population Variance	$a^2 = 0.681$ (0.597, 0.765)	$c^2 = 0.057$ (−0.016, 0.129)
Partial Reading Achievement[a]	Population Variance	$a^2 = 0.624$ (0.535, 0.712)	$c^2 = 0.025$ (−0.049, 0.100)
Reading Disability	Mean Differences	$h_g^2 = 0.721$ (0.585, 0.857)	$c_g^2 = 0.086$ (−0.035, 0.208)

[a]"Partial reading achievement" refers to reading achievement scores after partialling out Full-Scale IQ. Reading disability was operationalized as having a partial reading achievement score 1.5 or more standard deviations below the mean.

Table IV

Heritability Moderation Analyses

	Parental Education		Parental Reading Achievement[a]	
	Moderation regression coefficient (SE)	**p-value**	**Moderation regression coefficient (SE)**	**p-value**
Reading Achievement	0.007 (0.018)	0.71	0.005 (0.005)	0.24
Reading Disability	−0.024 (0.033)	0.48	−0.011 (0.010)	0.25

[a]Full-Scale IQ was first regressed out of parental reading achievement when it was used as a moderator for *reading disability*.