Published in final edited form as:

Read Writ. 2007 February 1; 20(1-2): 127–146. doi:10.1007/s11145-006-9021-2.

Longitudinal genetic analysis of early reading: The Western Reserve Reading Project

STEPHEN A. PETRILL¹, KIRBY DEATER-DECKARD², LEE ANNE THOMPSON³, CHRIS SCHATSCHNEIDER⁴, LAURA S. DETHORNE⁵, and DAVID J. VANDENBERGH¹

¹ Center for Developmental and Health Genetics, The Pennsylvania State University, PA 16802, USA

² Virginia Polytechnic and State University, Blacksburg, VA, USA

³ Case Western Reserve University, Cleveland, OH, USA

⁴ Florida State University, Tallahassee, FL, USA

⁵ University of Illinois Urbana-Champaign, Urbana, IL, USA

Abstract

We examined the genetic and environmental contribution to the stability and instability of reading outcomes in early elementary school using a sample of 283 twin pairs drawn from the Western Reserve Reading Project. Twins were assessed across two measurement occasions. In Wave 1, children were either in kindergarten or first grade. Wave 2 assessments were conducted one year later. Results suggested substantial genetic stability across measurement occasions. Additionally, shared environmental influences also accounted for stability, particularly for variables more closely tied to direct instruction such as phonological awareness, letter knowledge, and word knowledge. There was also evidence for independent genetic and shared environmental effects, suggesting that new sources of variance may emerge as the demands of school change and children begin to acquire early reading skills.

Keywords

Early childhood; Environment; Genetics; Multivariate; Reading development; Twin

Introduction

Over the past two decades, studies have demonstrated four key findings concerning the role of genes in reading. First, genes are important to familial resemblance in reading ability and disability (e.g., Harlaar, Spinath, Dale, & Plomin, in press; Pennington & Smith, 1983; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, in press a; Stevenson, Graham, Fredman, & McLoughlin, 1987). Second, genetic influences are important for components of reading ability such as phonological awareness/decoding, reading comprehension, spelling, orthographic knowledge, and rapid automatized naming (see Olson, Forsberg, & Wise, 1994; Olson, Gillis, Rack, DeFries, & Fulker, 1991; Gayan & Olson, 2001; Knopik, Alarcon, & DeFries, 1998; Gayan & Olson, 2003; Compton, Davis, DeFries, Gayan, & Olson, 2001). Third, multivariate genetic techniques have been used to investigate the genetic and

environmental contributions to the covariance between different dimensions of reading. These studies have shown that genetic influences are important for the relationships among different reading skills (Gayan & Olson, 2003, Harlaar, Dale, & Plomin, in press; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, in press b) as well as the relationship between oral language and reading skills (e.g. Bishop, 2001; Hohnen & Stevenson, 1999). Fourth, there has been progress in identifying quantitative trait loci (QTLs) influencing reading difficulties and related skills (see Fisher & DeFries, 2002; Grigorenko, 2005 for a review; Fagerheim et al., 1999; Fisher et al., 2002; Nopola-Hemmi et al., 2001).

Given this consistent and growing literature, most researchers now agree that genes play an important role in reading and that genes influence the relationships among reading outcomes. However, because most studies to date have involved cross-sectional samples of children spanning a broad age range, it is less clear how genes and environments shape the development of reading skills. Currently, there are only three population-based studies that have examined more narrowly-recruited samples of young twins just learning to read: The International Longitudinal Twin Study (ILTS) involving samples in Colorado, Australia, and Scandinavia (Byrne et al., 2002, as well as Byrne et al., and Samuelsson et al. in this special issue), the Twins Early Development Study (TEDS: Trouton, Spinath, & Plomin, 2002) and the Western Reserve Reading Project (WRRP: Petrill et al., in press a, b; the dataset analyzed in the current manuscript). These studies have suggested significant genetic, but also significant shared environmental influences for content-based skills such as word knowledge (Byrne et al., 2002) and phonological awareness (Petrill et al. in press a), a finding not reported in cross-sectional studies of older children, which have instead shown strong genetic and negligible shared environmental effects.

Currently, these studies have begun to employ multivariate genetic approaches to study the relationships among reading-related outcomes at various points in the development of reading. Cross-sectional studies using children drawn from a wide age-range have shown that the component processes of reading such as phonology, fluency, and orthographic skills are correlated largely via genetic pathways (e.g., Gayan & Olson, 2001, 2003; Davis, Knopik, Olson, Wadsworth, & DeFries, 2001). However, the multivariate relationships among reading related outcomes may be different for younger readers. In particular, Petrill et al. (in press b) examined the relationships among phonological awareness, rapid naming, and reading outcomes (letter identification, word identification, and phonological decoding) in a sample of kindergarten and first grade twins, finding that shared environmental influences (in addition to genetics) contributed to the covariance among phonological awareness and reading outcomes. Similarly, Byrne et al. (in press) found evidence for substantial genetic overlap between phonological awareness, RAN, and word reading efficiency, with some evidence for shared environmental overlap.

A third approach has employed the same multivariate genetic techniques to examine the longitudinal stability of reading outcomes. A central tenet of the reading literature is that there is stability in the processes that influence emergent and developed reading skills (e.g. Catts, Hogan, & Adlof, 2005; Gough & Tunmer, 1986, Hoover & Gough, 1990). However, there is also a shift in the kinds of skills that are important for successful reading as children master reading skills (e.g. Chall, 1983; Dale & Crain-Thorenson, 1999). Behavioral genetic designs enable the examination of the etiology of the longitudinal stability and instability of reading skills in this developmental context. For example, stability may be influenced by consistent shared family environmental influences in the home or school (almost all twins attend the same school and many are in the same classroom). Stability may also be influenced by genetic effects that tap common processes across longitudinal assessments of reading. Conversely, instability over time may be a function of new genes “turning on” or new genes being tapped as the task

demands of reading change with grade level. Furthermore, instability over time may also be influenced by new environmental demands as children acquire and master reading skills.

There are only a few published reports examining the longitudinal development of reading. Wadsworth, Corley, Hewitt, Plomin, and DeFries (2002) reported substantial genetic correlations between reading skills assessed at 7, 12, and 16 years, but this study was limited to a single measure of reading (PIAT Reading Recognition) at each age. A more recent study by Byrne et al. (2005), using the International Longitudinal Twin Study sample described previously found that both genetic and environmental influences were important for the longitudinal stability of reading from preschool to kindergarten. In particular, a single genetic factor influenced the relationship between preschool print knowledge, preschool phonological awareness, and later oral reading fluency skills in kindergarten. Similarly, a single shared environmental factor also influenced the relationship among these variables.

The goal of the current study is to examine the longitudinal stability and instability of reading and related skills in the Western Reserve Reading Project. Given the results of International Longitudinal Twin Study (Byrne et al., 2005) we hypothesize that genetic influences will impact the longitudinal stability of reading outcomes. However, given the rapid development of reading skills in early elementary school, we also leave open the possibility that independent genetic influences may emerge over time. Furthermore, given the results of prior univariate and multivariate genetic studies on early reading (Byrne et al., 2002, 2005; Petrill et al., in press a, b), we also hypothesize that shared environmental influences on longitudinal stability will also be significant, particularly for outcomes such as print knowledge and phonemic awareness, as opposed to outcomes such as rapid naming. We also will examine whether additional shared environmental influences emerge as children enter and matriculate through primary school.

Method

Participants

The current sample is drawn from the Western Reserve Reading Project (WRRP), an ongoing, longitudinal twin study involving identical and same-sex fraternal twins assessed across 4 measurement occasions. Recruiting for the larger study was conducted through school nominations, Ohio State Birth Records, and media advertisements. Schools were asked to send a packet of information to parents in their school system with twins who have been enrolled for kindergarten but have not finished first grade. We secured the cooperation of 273 schools throughout the State of Ohio. Media advertisements in the Greater Cleveland Metropolitan Area were also used to recruit additional twins. Recruiting has been completed. DNA was collected on twins using a buccal swab procedure to determine zygosity (see Freeman et al., 2003).

From a total of 350 pairs of twins who have been recruited for the project, the current study is based on those twin pairs who have completed Wave 1 ($n = 119$ MZ, 164 DZ pairs) and Wave 2 ($n = 88$ MZ, 123 DZ pairs) assessments to date. In Wave 1, mean age of the twins was $M = 6.1$ ($SD = .70$, range 4.9 to 7.9 years). Mean Stanford Binet SAS was $M = 100.4$ ($SD = 13.2$, range 63–139). The Wave 2 home visit was conducted within one month of the one year anniversary of the Wave 1 home visit. Mean child age at Wave 2 was $M = 7.2$ ($SD = .67$, range 6.0 to 8.8 years). Mean Stanford Binet SAS at Wave 2 was $M = 102.4$ ($SD = 12.4$, range 66–142). Fifty-seven percent of the twin pairs were female. Although most children were white (92%) and lived in two-parent households (94%), there was a wide range of parental education that was similar for mothers and fathers. Forty-three percent of mothers and 40% of fathers had less than a 4-year college education. Eleven percent of mothers and 19% of fathers had a high school education or less.

Procedures and Measures

At each home visit, children completed a 90-min battery of cognitive and reading-related outcome measures. Separate testers assessed each child in separate rooms. The study focused on skills associated with reading: Phonological awareness, expressive vocabulary, and rapid automatized naming, as well as four reading outcome variables: Letter knowledge, word knowledge, phonological decoding, and passage comprehension. The following measures were collected at each wave of assessment. *Phonological Awareness* was assessed using Robertson and Salter's (1997) Phonological Awareness Test. It included three subtests that measure phonemic segmentation (whole word), and phonemic deletion (syllabic deletion, and phoneme deletion). Given that phonological awareness has been shown to be a unitary construct (Schatschneider, Francis, Foorman, Fletcher, & Mehta, 1999), the three subtests at each wave were summed to form a raw total score for phonological awareness that was then residualized for age and gender using a regression procedure. *Rapid Automatized Naming* was assessed using the Letter Naming and Number Naming tasks from the Comprehensive Test of Phonological Processing (Wagner, Torgesen, & Rashotte, 1999). Letter and Number Naming were highly correlated ($r = .73$) and thus were residualized for child age, gender, and, given the age of the sample, Woodcock Reading Mastery Test Letter Identification (Woodcock, 1987). Residuals were then z-scored and averaged to form a Rapid Automatized Naming Composite. This RAN composite was then reverse scored so that a high score corresponded to faster naming speed. Expressive Vocabulary was assessed using the Boston Naming Test (Goodglass & Kaplan, 2001). The total number of correct responses was residualized for child age and gender.

Reading outcomes were assessed using the Woodcock Reading Mastery Test (WRMT-R; Woodcock, 1987). We used the Letter Identification to assess *letter knowledge*, the Word Identification subtest to assess *word knowledge*, the Word Attack subtest to assess *phonological decoding* skills, and the Passage Comprehension subtest to measure *reading comprehension*. As described in Petrill et al. (in press b), the number of months of school a child had completed at the time of our home assessments correlated $r = .88$ with child age, and did not predict additional variance in reading outcomes nor did it influence univariate or multivariate genetic estimates. All outliers beyond 3 standard deviations were excluded from correlational and model-fitting analyses and all variables were standardized within zygosity prior to model fitting analyses.

Results

Descriptive statistics are shown in Table 1. The average level and range of performance are typical for samples of this kind, with mean scores slightly above the population average. Sibling intra-class correlations, also shown in Table 1, suggest that sibling similarity was greatest among MZ twins (.58 to .91), followed by DZ twins (.33 to .63). Because MZ correlations were generally less than twice the DZ correlations, these results suggest that sibling similarity was accounted for by both additive genetic and shared environmental variance. Wave 1/Wave 2 phenotypic correlations ranged from $r = .55$ to $r = .80$ (see Table 2), suggesting moderate to high longitudinal stability across annual home visits.

The major goal of the study was to examine how genes and environments influence the longitudinal stability in early reading and related outcomes. Similar to Byrne et al. (2005), a series of cholesky decomposition analyses (see Neale & Cardon, 1992) were conducted for each outcome variable. For example, as shown in Figure 1, the covariance between Wave 1 and Wave 2 Phonological Awareness (PA) was parameterized using 6 latent factors. A general genetic factor (A1) was set to load on Wave 1 and Wave 2 Phonological Awareness. If significant, genetic covariance was important for the overlap between Wave 1 and Wave 2 Phonological Awareness. A2 estimated independent genetic variance in Wave 2 Phonological

Awareness not accounted for by genes associated with Phonological Awareness at Wave 1. Similar factors were estimated for shared environment ($C1$, $C2$) and nonshared environment ($E1$, $E2$). These models were run separately for each outcome variable. All models were estimated using Mx (Neale, Boker, Xie, and Maes, 2002) using raw data.

We first employed these models to derive univariate estimates of heritability (h^2), shared environment (c^2), and nonshared environment (e^2), as well as genetic and environmental contributions to the correlations between outcomes at Wave 1 and Wave 2 (see Table 3). These estimates were calculated by multiplying standardized A, C, and E matrices (as shown in Figure 1) by their respective transposes (see Neale & Cardon, 1992). Ninety-five percent confidence intervals were estimated using Mx. Those estimates not bounded by zero were statistically significant.

The diagonals estimate the proportion of variance accounted for by genetic (h^2), shared environmental (c^2), and nonshared environmental (e^2) influences. Univariate genetic estimates were statistically significant for all outcome variables, ranging from $h^2 = .14$ for Wave 2 Phonological Awareness to $h^2 = .76$ for Wave 2 Passage Comprehension. Univariate estimates of shared environment were also significant for Phonological Awareness (Wave 1, $c^2 = .16$, Wave 2, $c^2 = .47$), Expressive Vocabulary (Wave 1, $c^2 = .40$, Wave 2, $c^2 = .40$), Letter Knowledge (Wave 1, $c^2 = .38$, Wave 2, $c^2 = .52$), Word Knowledge (Wave 1, $c^2 = .34$, Wave 2, $c^2 = .33$), and Phonological Decoding (Wave 1, $c^2 = .26$, Wave 2, $c^2 = .21$). Nonshared environmental influences (which also include error) were statistically significant for all outcome variables.

The off-diagonals in Table 3 estimate the genetic, shared environmental and nonshared environmental contributions to the correlation between Wave 1 and Wave 2 outcomes. Put another way, the estimated phenotypic correlation between Wave 1 and Wave 2 is the sum of genetic, shared environmental, and nonshared environmental covariance pathways. For example, adding genetic (.29), shared environmental (.28) and nonshared environmental (.00) pathways yields an estimated phenotypic correlation of $r = .57$ between Wave 1 and Wave 2 Phonological Awareness. This estimate was almost identical to the observed phenotypic correlation of $r = .55$ presented in Table 2. Of this estimated correlation, $r = .29$ ($P < .05$) was due to genetic stability while $r = .28$ ($P < .05$) was due to shared environmental stability. Expressive Vocabulary (estimated phenotypic $r = .84$, genetic pathway = .42, $P < .05$, shared environmental pathway = .39, $P < .05$, nonshared environmental pathway = .03, $P > .05$) and Letter Knowledge (estimated phenotypic $r = .62$, genetic pathways = .31, $P < .05$, shared environmental pathway = .28, $P < .05$, nonshared environmental pathway = .03, $P > .05$) showed similar patterns of significant genetic and shared environmental contributions to Wave 1/Wave 2 longitudinal stability. Word Knowledge, Phonological Decoding, and Passage Comprehension showed significant genetic pathways (estimates = .41, .38, and .38 respectively), nonsignificant shared environmental pathways (estimates = .17, .13, and .15 respectively), and marginally significant nonshared environmental pathways (estimates = .03, .06, and .10 respectively). Finally, only the nonshared environmental contribution to longitudinal stability was significant for Rapid Automatized Naming (estimate = .24).

By fitting submodels to the cholesky analyses described above (see model presented in Figure 1), we also examined how genes and environments contributed not only to stability, but also to instability between Wave 1 and Wave 2 assessments. These submodels were fitted to each outcome in separate analyses. The difference between $-2\log$ likelihood (-2Δ) is distributed as a χ^2 . If a submodel results in a significant decrease in model fit, it is assumed that the parameters dropped in those submodels are statistically significant.

First, we examined whether genetic and shared environmental overlap between Wave 1 and Wave 2 was significant by dropping the pathway from A1 to Wave 2 outcome (Drop W1/W2 Genetic Corr) and by dropping the pathway from C1 to Wave 2 outcome (Drop W1/W2 ShEnv Corr). These submodels (see Table 4) suggest a pattern of genetic and shared environmental overlap identical to the results presented in Table 3.

We then tested whether there was significant independent genetic and/or shared environmental variance in Wave 2. We dropped the pathway from A2 to the Wave 2 assessment (W2 Genetic Indep), to test whether there was significant independent genetic variance in Wave 2. We also tested whether there was shared environmental independence at Wave 2 (Drop W2 ShEnv Indep). Results suggested significant independent genetic effects at Wave 2 for Rapid Automatized Naming and Word Knowledge. Independent shared environmental influences were significant for Letter Knowledge and Word Knowledge (see Table 4).

Estimates of genetic and environmental overlap and independence between Waves 1 and 2 are presented in Table 5. As expected from the model fitting results, with the exception of Rapid Automatized Naming, a statistically significant proportion of the genetic variance in Wave 2 was accounted for by genetic variance in Wave 1. For example, 100% of the genetic variance and 47% of the *total* variance in Expressive Vocabulary in Wave 2 was accounted for by genetic influences in Expressive Vocabulary in Wave 1. Independent genetic effects at Wave 2 were found for Rapid Naming (35% of the total variance in Wave 2) and Word Knowledge (27% of the total variance in Wave 2). Shared environmental overlap was significant for Phonological Awareness (47%), Expressive Vocabulary (38%), and Letter Knowledge (20%). Independent shared environmental effects in Wave 2 were found for Letter Knowledge (32%) and Word Knowledge (25%).

Discussion

The results of this study suggest that, for all variables but rapid automatized naming, genetic influences accounted for a statistically significant portion of the stability among reading outcomes. Shared environmental influences were also responsible for the stability in phonological awareness, expressive vocabulary, and letter knowledge. Moreover, independent genetic effects at Wave 2 were found for rapid naming and word knowledge. Independent shared environmental effects were significant for letter knowledge and word knowledge. Finally, there was evidence for small but significant nonshared environmental effects on stability for rapid naming, word knowledge, phonological decoding, and passage comprehension.

When comparing these results to the International Longitudinal Twin Study (ILTS), it is important to note that we are limited to an examination of differential patterns of statistical significance across the two studies. As evidenced by overlapping confidence intervals, there is insufficient power to test whether point estimates are significantly different from one another. With this caveat in mind, results are largely consistent between WRRP and International Longitudinal Twin Study (ILTS: Byrne et al., 2005), particularly with respect to genetic stability. One exception was rapid automatized naming. Byrne et al. (2005) found evidence for substantial longitudinal stability whereas we did not. This may be the result of measurement differences between the two studies. Byrne et al. employed a latent factor of CTOPP color, digit, and letter naming. We formed a composite using CTOPP letter and number naming, corrected for age, gender and letter knowledge. Despite the fact that we corrected for letter knowledge, our rapid naming task may tap emerging automatized processes in Wave 2 but may function as a speeded test of letter/number identification in Wave 1. It is notable that while there was genetic instability in rapid automatized naming across measurement occasions, there was genetic stability within measurement occasion. Genetic influences were largely

responsible for the overlap among between rapid naming, phonological awareness, word knowledge, and phonological decoding within Wave 1 (Petrill et al. in press b) and Wave 2 assessments (Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, in prep).

In the case of the shared environment, a greater number of reading outcomes in WRRP show statistically significant shared environmental influences compared to the ILTS. In particular, estimates of shared environment were not only significant in WRRP, but highly similar across Wave 1 and Wave 2. The only exception was phonological awareness, but in that case Wave 1 ($c^2 = .16$) and Wave 2 ($c^2 = .47$) estimates were not significantly different from one another. In contrast, although Byrne et al. (2005) found some significant shared environmental effects; evidence for statistically significant shared environmental effects was much more limited.

There are several possible explanations for these differences. First, the ILTS recruited a sample of preschoolers in their first assessment who then matriculated through grade school, so all twins were in the same grade at each assessment. In contrast, the WRRP sample is composed of kinder-garteners and first graders at Wave 1, who mature into first and second graders at Wave 2. Prior analyses have shown that months of school is highly correlated with child age in our sample (Petrill et al. in press b, $r = .88$). Additionally, months of school did not affect genetic and environmental estimates beyond child age (we always correct for age or use age-normed variables prior to analysis). Therefore, this difference between studies is not a likely explanation for the different results. Another possibility is that differences in country of origin between ILTS and WRRP result in differential genetic and environmental effects. This topic is discussed more fully by Samuelsson et al. and Byrne et al. in this special issue, who examine country effects in ILTS. Of particular interest, when looking at the results of Byrne et al. and Samuelsson et al. in this special issue, there is evidence that point estimates are similar between WRRP and the US subsample of the ILTS. In general, there is broad overlap across WRRP and ILTS. There is consistent evidence for genetic influences on the longitudinal stability of reading skills and, unlike cross sectional samples, evidence for significant shared environmental effects in emergent readers.

It is sensible that shared environmental influences may be greater for outcomes that are more likely to be influenced by direct instruction in the home or school, such as expressive vocabulary, phonological awareness, or print knowledge. Moreover, it is also sensible that shared environmental influences for phonological awareness, expressive vocabulary, and letter knowledge are stable, given that these skills are those most likely taught (or not taught) by parents and teachers in kindergarten. It is also notable that “new” shared environmental influences in Wave 2 are significant for skills (letter knowledge and word knowledge) that are at the core of the skills taught in primary school. Given that our 283 twin pairs come from 273 different schools, it is possible that new sources of shared environmental variance are emerging as the expression of between-school differences in reading curricula.

However, despite the importance of these environmental effects, between 25% and 50% of the variance in reading skills is influenced by longitudinally stable genetic influences (with the exception of rapid naming and phonological awareness). As we continue to follow the children in the Western Reserve Reading Project sample, we will address three issues. First, there is evidence in the larger behavioral genetic literature that shared environmental influences decline with age (see Petrill, 2005 for a review). We will examine whether shared environmental influences remain significant in later elementary school or whether these influences become nonsignificant once most children have mastered decoding skills. Second, we will examine how decoding skills influence later comprehension skills. In the current study, we found substantial genetic and negligible shared environmental effects for comprehension. We also found that genetic influences in comprehension at Wave 2 are highly correlated with decoding skills (Petrill et al., in prep). However, comprehension may be more strongly tied to decoding

in a sample of first and second graders. Furthermore, the Woodcock Reading Mastery Test Comprehension measure may be highly dependent on decoding. What we will examine with subsequent data is whether the genetic overlap between decoding and comprehension gives way to genetic influences associated with language skills as most children move from “learning to read” to “reading to learn” (Chall, 1983).

Third and finally, we will turn our attention to gene-environment processes. Many studies have shown that reading success is associated with reading-related knowledge and skills children have acquired prior to coming to school (see McCardle, Scarborough, & Catts, 2001) and the quality of the home environment during school (e.g., Molfese, Molfese, Key, & Kelly, 2003). Our own prior research suggests that these measures operate largely through shared environmental pathways in early childhood (Petrill, Deater-Deckard, Schatschneider, & Davis, 2005; Seidman et al., 2005), but the effect sizes of these measures are generally small and dissipate after children master decoding skills (Scarborough, 1998).

Given the importance of genetic influences and the likely dissipation of shared environmental effects, it may be tempting to conclude that the environment is ultimately unimportant to later reading development. Such conclusions are not supported by the data. Because genetic influences are important to reading and related domains, such as oral language, it is essential to examine how genetic influences impact the probability of coming into contact with environments associated with these skills (e.g., Scarr & McCarthy, 1983). Understanding these gene-environment transactions is essential to quantifying how genetic influences related to reading ability and disability are manifested not only in the phenotype of reading but also in the environments associated with reading skills. In particular, we will examine whether measured environmental influences on early reading shift from a shared environmental to a genetic etiology as children learn to read and as the environments associated with reading become more a function of their own reading skills.

Acknowledgments

The Western Reserve Reading Project is supported by NICHD grant HD38075 and NICHD/OSERS grant HD46167.

References

- Bishop DVM. Genetic influences on language impairment and literacy problems in children: Same or different? *Journal of Child Psychology and Psychiatry, and Allied Disciplines* 2001;42(2):189–198.
- Byrne B, Delaland C, Fielding-Barnsley R, Quain P, Samuelsson S, Høien T, Corley R, DeFries JC, Wadsworth S, Willcutt E, Olson RK. Longitudinal twin study of early reading development in three countries: Preliminary results. *Annals of Dyslexia* 2002;52:49–74.
- Byrne B, Olson RK, Samuelsson S, Wadsworth S, Corley R, Quain P, DeFries JC, Willcutt E. Genetic and environmental influences on early literacy. *Journal of Reading Research*. in press
- Byrne B, Wadsworth S, Corley R, Samuelsson S, Quain P, DeFries JC, Willcutt E, Olson RK. Longitudinal twin study of early literacy development: Preschool and kindergarten phases. *Scientific Studies of Reading* 2005;9(3):219–236.
- Catts, HW.; Hogan, TP.; Adlof, SM. Developmental changes in reading and reading disabilities. In: Catts, HW.; Kamhi, AG., editors. *The connections between language and reading disabilities*. Mahwah, NJ: Erlbaum; 2005.
- Chall, JS. *Stages of reading development*. New York: McGraw-Hill; 1983.
- Compton, DL.; Davis, CJ.; DeFries, JC.; Gayan, J.; Olson, RK. Genetic and environmental influences on reading and RAN: An overview of results from the Colorado Twin Study. In: Wolf, M., editor. *Conference proceedings of the Dyslexia research foundation conference in extraordinary brain series: Time, fluency, and developmental dyslexia*. Baltimore, MD: York Press; 2001. p. 277–303.
- Dale PS, Crain-Thorenson C. Language and literacy in a developmental perspective. *Journal of Behavioral Education* 1999;9:23–33.

- Davis CJ, Knopik VS, Olson RK, Wadsworth SJ, DeFries JC. Genetic and environmental influences on rapid naming and reading ability: A twin study. *Annals of Dyslexia* 2001;51:231–247.
- Fagerheim T, Raeymaekers P, Tonnessen FE, Pedersen M, Tranebjaerg L, Lubs HA. A new gene (DYX3) for dyslexia is located on chromosome 2. *Journal of Medical Genetics* 1999;36:664–669. [PubMed: 10507721]
- Fisher SE, DeFries JC. Developmental dyslexia: Genetic dissection of a complex cognitive trait. *Nature Reviews Neuroscience* 2002;3:767–780.
- Fisher SE, Francks C, Marlow AJ, MacPhie IL, Newbury DF, Cardon LR, Ishikawa-Brush Y, Richardson AJ, Talcott JB, Gayán J, Olson RK, Pennington BF, Smith SD, DeFries JC, Stein JF, Monaco AP. Independent genome-wide scans identify a chromosome 18 quantitative-trait locus influencing dyslexia. *Nature Genetics* 2002;30:86–91. [PubMed: 11743577]
- Freeman B, Smith N, Curtis C, Hockett L, Mill J, Craig IW. DNA from buccal swabs recruited by mail: Evaluation of storage effects on long-term stability and suitability for multiplex polymerase chain reaction genotyping. *Behavior Genetics* 2003;33(1):67–72. [PubMed: 12645823]
- Gayan J, Olson RK. Genetic and environmental influences on orthographic and phonological skills in children with reading disabilities. *Developmental Neuropsychology* 2001;20(2):487–511.
- Gayan J, Olson RK. Genetic and environmental influences on individual differences in printed word recognition. *Journal of Experimental Child Psychology* 2003;84:97–123. [PubMed: 12609495]
- Goodglass, H.; Kaplan, E. The boston naming test. Vol. 2. New York: Lippincott Williams & Wilkins; 2001.
- Gough PB, Tunmer WE. Decoding, reading, and reading disability. *Remedial and Special Education* 1986;7(1):6–10.
- Grigorenko EL. A conservative meta-analysis of linkage and linkage-association studies of developmental dyslexia. *Scientific Studies of Reading* 2005;9(3):285–316.
- Harlaar N, Dale PS, Plomin R. Correspondence between telephone testing and teacher assessments of reading in 7-year-olds: II. Strong genetic overlap in a sample of 2660 twin pairs. *Reading & Writing: An Interdisciplinary Journal* 2005;18(5):401–423.
- Harlaar N, Spinath FM, Dale PS, Plomin R. Genetic influences on word recognition abilities and disabilities: A study of 7-year old twins. *Journal of Child Psychology and Psychiatry* 2005;46(4):373–384. [PubMed: 15819646]
- Hohnen B, Stevenson J. The structure of genetic influences on general cognitive, language, phonological, and reading abilities. *Developmental Psychology* 1999;35:590–603. [PubMed: 10082029]
- Hoover WA, Gough PB. The simple view of reading. *Reading and Writing* 1990;2(2):127–160.
- Knopik VS, Alarcón M, DeFries JC. Common and specific gender influences on individual differences in reading performance: A twin study. *Personality and Individual Differences* 1998;25(2):269–277.
- McCardle P, Scarborough HS, Catts HW. Predicting, explaining, and preventing children's reading difficulties. *Learning Disabilities Research and Practice* 2001;16(4):230–239.
- Molfese D, Molfese V, Key AF, Kelly S. Influence of environment on speech-sound discrimination: Findings from a longitudinal study. *Developmental Neuropsychology* 2003;24:541–558. [PubMed: 14561561]
- Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. Mx: Statistical modeling. Vol. 6. VCU Box 900125, Richmond, VA 23298: Department of Psychiatry.; 2002.
- Neale, MC.; Cardon, LR. Methodology for genetic studies of twins and families. Dordrecht, The Netherlands: Kluwer Academic Publishers; 1992.
- Nopola-Hemmi J, Myllyluoma B, Haltia T, Taipale M, Ollikainen V, Ahonen T, et al. A dominant gene for developmental dyslexia on chromosome 3. *Journal of Medical Genetics* 2001;38:658–664. [PubMed: 11584043]
- Olson, RK.; Forsberg, H.; Wise, B. Genes, environment, and the development of orthographic skills. In: Berninger, VW., editor. The varieties of orthographic knowledge I: Theoretical and developmental issues. Dordrecht, The Netherlands: Kluwer Academic Publishers; 1994. p. 27-71.
- Olson RK, Gillis JJ, Rack JP, DeFries JC, Fulker DW. Confirmatory factor analysis of word recognition and process measures in the Colorado Reading Project. *Reading and Writing* 1991;3:235–248.

- Pennington BF, Smith SD. Genetic influences on learning disabilities and speech and language disorders. *Child Development* 1983;54:369–387. [PubMed: 6347551]
- Petrill, SA. Behavioral genetics and intelligence. In: Wilhelm, O.; Engle, RW., editors. *Handbook of understanding and measuring intelligence*. Thousand Oaks, CA: Sage; 2005. p. 165-176.
- Petrill SA, Deater-Deckard K, Schatschneider C, Davis C. Measured environmental influences on early reading: Evidence from an adoption study. *Scientific Studies of Reading* 2005;9(3):237–260.
- Petrill SA, Deater-Deckard K, Thompson LA, DeThorne L, Schatschneider C. Reading skills in early readers: Genetic and shared environmental influences. *Journal of Learning Disabilities* 2006;39(1): 48–55. [PubMed: 16512082]
- Petrill SA, Deater-Deckard K, Thompson LA, DeThorne LS, Schatschneider C. Genetic and environmental effects of serial naming and phonological awareness on reading outcomes. *Journal of Educational Psychology* 2006;98(1):112–121. [PubMed: 19444324]
- Petrill, SA.; Deater-Deckard, K.; Thompson, LA.; DeThorne, L.; Schatschneider, C. The relationship between decoding and comprehension in early reading: Genetic and environmental influences. in preparation
- Robertson, C.; Salter, W. The phonological awareness test. East Moline, IL: LinguiSystems, Inc; 1997.
- Scarborough, HS. Early identification of children at risk for reading disabilities: Phonological awareness and some other promising predictors. In: Shapiro, BK.; Accardo, PJ.; Capute, AJ., editors. *Specific reading disability: A view of the spectrum*. Timonium, MD: York Press; 1998. p. 75-119.
- Scarr S, McCartney K. How people make their own environments: A theory of genotype-environment effects. *Child Development* 1983;54:424–435. [PubMed: 6683622]
- Schatschneider C, Francis DJ, Foorman BF, Fletcher JM, Mehta P. The dimensionality of phonological awareness: An application of item response theory. *Journal of Educational Psychology* 1999;91:467–478.
- Seidman, S.; Petrill, SA.; Deater-Deckard, K.; Thompson, LA.; Schatschneider, C.; DeThorne, LS. Assessing the role of home and school environments in early literacy. 2005. Manuscript submitted for publication
- Stevenson J, Graham P, Fredman G, McLoughlin V. A twin study of genetic influences on reading and spelling ability and disability. *Journal of Child Psychology and Psychiatry* 1987;28(2):229–247. [PubMed: 3584294]
- Trouton A, Spinath FM, Plomin R. Twins Early Development Study (TEDS): A multivariate, longitudinal genetic investigation of language, cognition and behaviour problems in childhood. *Twin Research* 2002;5:444–448. [PubMed: 12537874]
- Wadsworth SJ, Corley RP, Hewitt JK, Plomin R, DeFries JC. Parent-offspring resemblance for reading performance at 7, 12 and 16 years of age in the Colorado adoption project. *Journal of Child Psychology and Psychiatry and Allied Disciplines* 2002;43(6):769–774.
- Wagner, RK.; Torgesen, JK.; Rashotte, CA. Comprehensive test of phonological processing. Austin, TX: PRO-ED; 1999.
- Woodcock, RW. Woodcock reading mastery tests. Circle Pines, MN: American Guidance Service; 1987.

Bivariate cholesky model.

Figure 1.
Bivariate cholesky model.

Table 1
Means, standard deviations, and MZ/DZ intraclass correlations for Wave 1 and Wave 2 outcomes.

Variable	Mean(SD)	MZ	DZ
<i>Wave 1</i>			
Phonological awareness	13.2(6.6)	.76	.44
Rapid automatized naming	164.9(63.8)	.58	.33
Expressive vocabulary	28.2(6.9)	.75	.61
Letter knowledge	102.2(8.9)	.74	.55
Word knowledge	104.1(18.4)	.87	.61
Phonological decoding	103.2(12.3)	.81	.53
Passage comprehension	11.1(9.0)	.70	.45
<i>Wave 2</i>			
Phonological awareness	19.0(4.9)	.60	.55
Rapid automatized naming	109.9(42.2)	.61	.38
Expressive vocabulary	32.8(6.9)	.85	.62
Letter knowledge	100.6(8.6)	.76	.63
Word knowledge	111.2(12.6)	.91	.59
Phonological decoding	109.9(11.4)	.72	.44
Passage comprehension	21.7(10.7)	.85	.45

Note: Rapid Naming is expressed in number of seconds. Phonological Awareness, Expressive Vocabulary, and Passage Comprehension are expressed in number of correct items. Passage Comprehension was analyzed in this way to allow for children to receive a score that fell below standardization cutoffs. All intraclass correlations are significant at $P < .01$.

Table 2

Observed correlations between Wave 1 and Wave 2 outcomes.

Wave 1	Wave 2	N
Phonological awareness	.55*	397
Rapid automatized naming	.56*	360
Expressive vocabulary	.80*	406
Letter knowledge	.58*	392
Word knowledge	.59*	385
Phonological decoding	.57*	391
Passage comprehension	.62*	130

Note: $P < .001$. Sample sizes are lower for passage comprehension because many children could not complete the task in Wave 1.

Table 3

Heritability, shared environmental, and nonshared environmental estimates, as well as genetic, shared environmental, and nonshared environmental contributions to the phenotypic correlation between Wave 1 and Wave 2 outcomes.

Variables		Wave 1[CI]	Wave 2[CI]
<i>Phonological awareness</i>			
Estimated Wave1/Wave 2 phenotypic correlation = .57			
Genetic	Wave 1	0.59[0.34, 0.78] = h^2	0.14[0.04, 0.39] = h^2
	Wave 2	0.29[0.13, 0.48]	
Shared Env	Wave 1	0.16[0.02, 0.40] = c^2	0.47[0.23, 0.65] = c^2
	Wave 2	0.28[0.08, 0.46]	
Nonshared Env	Wave 1	0.25[0.19, 0.33] = e^2	0.39[0.31, 0.48] = e^2
	Wave 2	0.00[0.00, 0.04]	
<i>Rapid automatized naming</i>			
Estimated Wave1/Wave 2 phenotypic correlation = .59			
Genetic	Wave 1	0.42[0.04, 0.73] = h^2	0.43[0.07, 0.78] = h^2
	Wave 2	0.18[0.00, 0.48]	
Shared Env	Wave 1	0.15[0.00, 0.46] = c^2	0.20[0.00, 0.51] = c^2
	Wave 2	0.17[0.00, 0.41]	
Nonshared Env	Wave 1	0.44[0.33, 0.62] = e^2	0.37[0.28, 0.52] = e^2
	Wave 2	0.24[0.14, 0.37]	
<i>Expressive vocabulary</i>			
Estimated Wave1/Wave 2 phenotypic correlation = .84			
Genetic	Wave 1	0.38[0.20, 0.60] = h^2	0.47[0.30, 0.70] = h^2
	Wave 2	0.42[0.27, 0.61]	
Shared Env	Wave 1	0.40[0.19, 0.62] = c^2	0.40[0.18, 0.64] = c^2
	Wave 2	0.39[0.20, 0.60]	
Nonshared Env	Wave 1	0.22[0.18, 0.28] = e^2	0.13[0.11, 0.19] = e^2
	Wave 2	0.03[0.00, 0.07]	
<i>Letter Knowledge</i>			
Estimated Wave1/Wave 2 phenotypic correlation = .62			
Genetic	Wave 1	0.35[0.14, 0.61] = h^2	0.27[0.10, 0.52] = h^2
	Wave 2	0.31[0.15, 0.50]	
Shared Env	Wave 1	0.38[0.15, 0.61] = c^2	0.52[0.30, 0.79] = c^2
	Wave 2	0.28[0.09, 0.48]	
Nonshared Env	Wave 1	0.27[0.20, 0.35] = e^2	0.21[0.17, 0.29] = e^2
	Wave 2	0.03[0.00, 0.08]	
<i>Word knowledge</i>			
Estimated Wave1/Wave 2 phenotypic correlation = .61			
Genetic	Wave 1	0.55[0.39, 0.80] = h^2	0.58[0.41, 0.81] = h^2
	Wave 2	0.41[0.28, 0.61]	
Shared Env	Wave 1	0.34[0.11, 0.59] = c^2	0.33[0.10, 0.57] = c^2
	Wave 2	0.17[0.00, 0.37]	
Nonshared Env	Wave 1	0.11[0.08, 0.15] = e^2	0.09[.07, .12] = e^2
	Wave 2	0.03 [0.01, 0.05]	
<i>Phonological decoding</i>			
Estimated Wave1/Wave 2 phenotypic correlation = .57			
Genetic	Wave1	0.56[0.33, 0.81] = h^2	0.51[0.22, 0.84] = h^2
	Wave 2	0.38[0.18, 0.60]	
Shared Env	Wave 1	0.26[0.02, 0.49] = c^2	0.21[0.00, 0.49] = c^2
	Wave 2	0.13[0.00, 0.33]	
Nonshared Env	Wave 1	0.18[0.14, 0.25] = e^2	0.28[0.21, 0.38] = e^2
	Wave 2	0.06[0.01, 0.12]	
<i>Passage comprehension</i>			
Estimated Wave1/Wave 2 phenotypic correlation = .63			
Genetic	Wave 1	0.50[0.02, 1.00] = h^2	0.76[0.53, 1.00] = h^2
	Wave 2	0.38[0.08,0.70]	
Shared Env	Wave 1	0.21[0.00, 0.68] = c^2	0.11[0.00, 0.35] = c^2
	Wave 2	0.15[0.00, 0.42]	
Nonshared Env	Wave 1	0.29[0.19, 0.53] = e^2	0.13[0.10, 0.19] = e^2
	Wave 2	0.10[0.02, 0.20]	

Note: CI = lower and upper 95% confidence intervals estimated in Mx.

Table 4

Model fitting results: Wave 1 – Wave 2 bivariate genetic models.

Variable	-2ll	df	χ^2 change	df change	P change
<i>Phonological awareness</i>					
Full	2249.28	890			
Drop W1/W2 Genetic Corr	2262.29	891	13.01	1	*
Drop W1/W2 ShEnv Corr	2263.98	891	14.70	1	*
Drop W2 Genetic Indep	2249.28	891	0.00	1	NS
Drop W2 ShEnv Indep	2249.28	891	0.00	1	NS
<i>Rapid automatized naming</i>					
Full	2140.19	822			
Drop W1/W2 Genetic Corr	2141.61	823	1.42	1	NS
Drop W1/W2 ShEnv Corr	2142.05	823	1.86	1	NS
Drop W2 Genetic Indep	2145.88	823	5.69	1	*
Drop W2 ShEnv Indep	2140.19	823	0.00	1	NS
<i>Expressive vocabulary</i>					
Full	1879.77	890			
Drop W1/W2 Genetic Corr	1909.11	891	29.34	1	*
Drop W1/W2 ShEnv Corr	1894.00	891	14.23	1	*
Drop W2 Genetic Indep	1879.77	891	0.00	1	NS
Drop W2 ShEnv Indep	1880.29	891	0.52	1	NS
<i>Letter knowledge</i>					
Full	2157.60	886			
Drop W1/W2 Genetic Corr	2171.43	887	13.83	1	*
Drop W1/W2 ShEnv Corr	2165.78	887	8.18	1	*
Drop W2 Genetic Indep	2157.60	887	0.00	1	NS
Drop W2 ShEnv Indep	2174.24	887	16.64	1	*
<i>Word knowledge</i>					
Full	1982.53	876			
Drop W1/W2 Genetic Corr	2020.57	877	38.04	1	*
Drop W1/W2 ShEnv Corr	1985.50	877	2.97	1	NS
Drop W2 Genetic Indep	1994.35	877	11.82	1	*
Drop W2 ShEnv Indep	1989.99	877	7.46	1	*
<i>Phonological decoding</i>					
Full	2182.65	883			
Drop W1/W2 Genetic Corr	2196.56	884	13.91	1	*
Drop W1/W2 ShEnv Corr	2184.08	884	1.43	1	NS
Drop W2 Genetic Indep	2186.01	884	3.36	1	NS
Drop W2 ShEnv Indep	2184.34	884	1.69	1	NS
<i>Passage comprehension</i>					
Full	1326.61	523			
Drop W1/W2 Genetic Corr	1332.90	524	6.29	1	*
Drop W1/W2 ShEnv Corr	1327.62	524	1.01	1	NS
Drop W2 Genetic Indep	1329.24	524	2.63	1	NS
Drop W2 ShEnv Indep	1326.61	524	0.00	1	NS

Note:

* $P < .05$.

Table 5

Proportion of Total Variance Accounted for by Genetic and Shared Environmental Overlap and Specificity.

Wave 2 Variable	Estimate	Shared with Wave 1	Indepent in Wave 2
<i>Genetic pathway</i>	h^2	= <i>SharedWave1</i>	+ <i>IndepWave2</i>
Phonological awareness	.14 **	.14 *	.00
Rapid automatized naming	.43 **	.08	.35 *
Expressive vocabulary	.47 **	.47 *	.00
Letter knowledge	.27 **	.27 *	.00
Word knowledge	.58 **	.31 *	.27 *
Phonological decoding	.51 **	.26 *	.25
Passage comprehension	.76 **	.28 *	.48
<i>Shared environmental pathway</i>	c^2	= <i>SharedWave1</i>	+ <i>IndepWave2</i>
Phonological awareness	.47 **	.47 *	.00
Rapid automatized naming	.20	.20	.00
Expressive vocabulary	.40 **	.38 *	.02
Letter knowledge	.52 **	.20 *	.32 *
Word knowledge	.33 **	.08	.25 *
Phonological decoding	.21	.06	.15
Passage comprehension	.11	.11	.00

Note:

* $P < .05$ as described by submodels presented in Table 4,** $P < .05$ as described by confidence intervals in Table 3. Overlap and independence for nonshared environmental pathways were not calculated because the independent pathway contains random error. Despite a large point estimate for comprehension, independent genetic effects were not significant due to the smaller sample size of twins who could complete this test at both time points.