

NIH Public Access

Author Manuscript

J Child Psychol Psychiatry. Author manuscript; available in PMC 2010 June 24.

Published in final edited form as:

J Child Psychol Psychiatry. 2010 June 1; 51(6): 660–667. doi:10.1111/j.1469-7610.2009.02204.x.

Genetic and environmental influences on the growth of early reading skills

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Abstract

Background—Studies have suggested genetic and environmental influences on overall level of early reading whereas the larger reading literature has shown environmental influences on the rate of growth of early reading skills. This study is the first to examine the genetic and environmental influences on both initial level of performance and rate of subsequent growth in early reading.

Methods—Participants were drawn from the Western Reserve Reading Project, a study of 314 twin pairs based in Ohio. Twins were assessed via three annual home visits during early elementary school. Assessments included word identification, letter identification, pseudoword decoding, expressive vocabulary, phoneme awareness, and rapid naming. Measures were analyzed using latent growth curve modeling.

Results—The heritability of initial performance (latent intercept) ranged from $h^2 = .38$ **for word** identification to h² = .72 for rapid naming. Shared environment ranged from c^2 = .11 for rapid naming to c^2 = .62 for word identification. The heritability of the rate of subsequent growth (latent slope) was statistically significant for rapid naming $h^2 = .58$ and phoneme awareness $h^2 = .20$. Shared environment accounted for nearly 100% of variance in rate of growth for word identification, letter identification and pseudoword decoding, and was statistically significant and large for phoneme awareness ($c^2 = .80$). Genetic variance for rapid naming and phoneme awareness latent slopes overlapped entirely with genetic variance on the intercepts. In contrast, one-third to two-thirds of the shared environmental variance on the slope was independent from the shared environmental variance on the intercept.

Conclusions—Genetic influences were related primarily to those already present at the initial level of performance. In contrast, shared environmental influences affecting rate of growth were both predicted by and independent from initial levels of performance. Results suggested that growth in early reading skills is amenable to family, school, or other environmental influences as reading skills develop.

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Keywords

Growth; reading; twin; genetics; environment

Over the past 25 years, research has led to important progress in the understanding of early reading development, establishing the importance of vocabulary, letter–sound correspondence, phonological processing, fluency, and language comprehension (see Whitehurst & Lonigan, 1998; National Reading Panel, 2000). Studies have also shown that there is considerable variability not only for initial reading level when children begin formalized instruction, but also in the rate of subsequent growth (Parrila, Aunola, Leskinen, Nurmi, & Kirby, 2005).

Researchers have gained insight into this variability by examining indices of the family and school that predict individual differences in initial reading as well as growth in performance. Children from higher-SES households tend to have higher initial reading scores and show faster rates of growth compared with children from lower-SES households (e.g., Aikens & Barbarin, 2008; Cheadle, 2008). Additionally, initial rates of reading performance and rates of subsequent growth are higher when parents show high levels of educational involvement (Cheadle, 2008) and provide rich home literacy experiences (Aikens & Barbarin, 2008). School factors such as the type and quality of teacher instruction have also shown significant relations with student reading outcomes (Connor et al., 2009; Foorman et al., 2006; National Reading Panel, 2000) and have also been implicated as significant contributors to growth in student reading performance during the school year (Foorman, Francis, Fletcher, Schatschneider, & Mehta, 1998; Nye, Konstantopoulos, & Hedges, 2004).

At the same time, quantitative genetic studies have established the importance of both genetic and environmental influences on overall reading ability and disability (e.g., DeFries, Fulker, & LaBuda, 1987; Harlaar, Dale, & Plomin, 2007; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, 2006a; Samuelsson et al., 2008; Stevenson, Graham, Fredman, & McLoughlin, 1987), as well as on the skills that support reading ability, including vocabulary, print knowledge, phoneme awareness/decoding, spelling, and orthographic coding (e.g., Bates et al., 2004; Byrne et al., 2008; Friend, DeFries, Wadsworth, & Olson, 2007; Gayán, & Olson, 2003; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, 2006b). To date, behavioral genetic studies have focused on two issues related to reading *development*. First, studies have examined how the relative magnitude of genetic and environmental effects varies as a function of child age. For example, genetic effects tend to be consistently important at all ages for fluency, and tend to become increasingly more important with age for phoneme awareness (Byrne et al., 2005; Petrill et al., 2007). At the same time, environmental influences appear to be consistently important across age for letter knowledge and word knowledge (Petrill et al., 2007) and may become more important with age for spelling (Friend et al., 2007).

Second, behavioral genetic research has also focused on the stability of reading outcomes; in particular whether the same genetic and environmental factors remain consistent over time (e.g. Byrne et al., 2007; Harlaar et al., 2007; Betjemann et al., 2008; Wadsworth, DeFries, Olson, & Willcutt, 2007; Petrill et al., 2007). These studies suggest that the stability and instability in reading skills is substantially influenced by genetic factors. However, these studies also provide evidence for environmental overlap across measurement occasions as well as the emergence of new environmental factors as reading skills develop.

However, behavioral genetic studies to date have not examined *growth* in reading performance – in particular, how genetics and environment impact individual differences in how quickly or slowly children's reading and related skills improve over time. This is unfortunate given that

much of the larger contemporary reading literature, particularly the intervention literature, has focused on growth as an outcome measure. Genetic and environmental factors may not just contribute to the variance of reading skills at a particular age or to the stability of individual differences of reading skills at different ages, but also to the rate at which children make reading gains. Another important issue is the extent to which genetic and environmental influences on the rate of reading growth are independent from genetic and environmental influences on initial performance. This is especially important in early reading where children come to formalized instruction with genetic variation related to reading and varying levels of preschool environmental supports for literacy; and are then educated in settings of varying instructional quality. Examining individual differences in growth in a genetically sensitive design may shed new light on children's reading development, in particular, how genetic and environmental influences on subsequent growth are related to or independent from those genetic and environmental factors impacting initial performance.

Thus, the purpose of the current study was to conduct the first behavioral genetic analysis on the growth of reading skills. We examined 6 reading-related skills: word identification, letter identification, pseudoword decoding, expressive vocabulary, phoneme awareness, and rapid naming. We determined the extent to which genetic and environmental influences contributed to variance in: (1) initial levels of reading, (2) rates of growth, and (3) the association between initial level and rates of growth, including the degree to which the rate of growth was correlated with or independent from initial levels of reading.

Method

Participants

Participants were drawn from the Western Reserve Reading Project, a sample of 314 pairs of identical (MZ: $N = 135$) and same-sex fraternal (DZ: $N = 179$) twins in Ohio. Twins were recruited into the study when they were in kindergarten or first grade, primarily through school nominations (see Petrill et al., 2006a). Twins were assessed in their homes when they were enrolled into the project and are in the process of annual followup home visits for an additional 6 years. Annual assessments occurred within one month of the anniversary of the previous assessment. Parental permission/informed consent for each assessment was obtained at the time of the home visit. Because WRRP is an ongoing longitudinal study, the present analyses were based upon data from the first three home visits for which data are currently available. At Assessment 1, twins were 6 years old (*M* = 6.07 yrs, SD = .68 yrs, range = 4.33–7.92 yrs). At Assessments 2 and 3, twins were 7 (age *M* = 7.16 yrs, SD = .67 yrs, range = 6.00–8.83 yrs), and 8 years old (age $M = 8.24$ yrs, SD = .79 yrs, range = 6.25–10.00 yrs) respectively.

For the majority of twin pairs, DNA was collected via buccal swabs for zygosity determination. In cases where parents did not consent to genotyping $(N = 76)$, zygosity was determined using parent questionnaire (Goldsmith, 1991). Most parents were married and cohabitating (92%) and nearly all were White (92% of mothers, 94% of fathers). In terms of parent education, 12% reported completing high school or less, 18% reported completing some college, 31% reported completing a bachelor's degree, 23% reported some postgraduate education or degree, and 5% did not specify educational attainment. WRRP is somewhat overrepresented for bachelor's degree and higher, but educational attainment of the sample is similar to the general population (US Census Bureau, 2007).

Procedures and measures

At each home visit, twins were assessed on a 90-minute battery of reading-based measures. Twins were assessed in separate rooms by different examiners. The study focused on three early reading outcomes: Word Identification, Letter Identification, and Word Attack, assessed

using the Woodcock Reading Mastery Test (WRMT; Woodcock, 1987). Three other skills associated with early reading were also examined. Expressive Vocabulary was measured using the Vocabulary subtest from the Stanford–Binet Intelligence Test (Thorndike, Hagen, & Sattler, 1986) and the Boston Naming Test (Goodglass & Kaplan, 2001). Phoneme Awareness was assessed using the Phonological Awareness Test (PAT; Robertson & Salter, 1997). The PAT included three subtests that assessed phonemic segmentation (whole word), and phonemic deletion (syllabic deletion, and phoneme deletion). Prior work in our sample (Petrill et al., 2006b) suggested that the PAT loads strongly on a single latent factor. Thus, the three subtests at each wave (scored from 0 to 10 possible items correct) were summed to form a raw total score for phoneme awareness. Finally, Rapid Automatized Naming (RAN) was measured using Number and Letter 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 combined to form a RAN composite.

Analyses

The goal of this study was to examine the genetic and environmental influences on growth of early reading skills. First, we examined indices of central tendency and variability to provide background information on the sample and to provide descriptive information on the amount of growth in measured reading outcomes. Second, we estimated initial level of performance and subsequent rate of growth in reading across three measurement occasions using a structural equation modeling framework. Third, using the same models, we simultaneously estimated the genetic and environmental contributions to initial performance and rate of growth of reading outcomes. Finally, we examined whether initial performance and the rate of growth of reading were influenced by overlapping or independent genetic and environmental factors.

Results

Descriptive statistics

Table 1 presents age-normed scores for WRMT measures as well as the Stanford–Binet. These data suggest that, in the case of WRMT, the mean was slightly higher than the population mean of $M = 100$. In both WRMT and Stanford–Binet tests, the standard deviations were lower than the population standard deviation of $SD = 15$.

Age-normed measures are not informative in the context of growth because the means and standard deviations are set to be the same value (e.g., $M = 100$, $SD = 15$) irrespective of age. In order to measure growth, values must increase as overall ability increases. Thus, in the case of WRMT Word Identification, Letter Identification, and Word Attack subtests, we employed W-scores at Assessments 1, 2, and 3. W-scores were obtained via the WRMT scoring protocol (Woodcock, 1987). Because they are based on an Item Response Theory model, W-scores are assumed to have equal intervals. No such scores were available for the Boston Naming Test, Stanford–Binet Vocabulary, Phonological Awareness Test, and Rapid Naming. Thus, raw scores were employed across Assessments 1 to 3 for these variables. Equal intervals cannot be assumed in the case of raw variables. All variables showed mean improvements in performance across measurement occasions, although the level of improvement was attenuated for Stanford–Binet Vocabulary and Boston Naming Test relative to the other measures.

Modeling of growth

Next, we employed a latent growth curve approach (Reynolds et al., 2005) using the W-score and raw score data described above. Figure 1 presents the model, using Word Identification as an example. Word Identification W-scores for Assessments 1, 2, and 3 for each twin were loaded on a latent intercept and a latent slope. All assessment points were loaded on the latent intercept (as noted by the 1's from each measure to latent intercept), but the intercept was

centered at Assessment 1, thus estimating individual differences among children in level of initial reading performance. The latent slope estimated individual differences in the rate of linear growth (as noted by 0, 1, and 2 from Assessments 1, 2, and 3 to latent slope). Taken together, the model estimated a regression equation for each child including an estimate of initial level of latent reading performance (latent intercept) and subsequent rate of growth in latent performance across measurement occasions (latent slope).

Assessment 1 Age was also modeled as a definition variable to account for age differences within assessment points (Neale, Boker, Xie, & Maes, 2002). Definition variables are similar to covariates in that they can account for the effect of age on each outcome independently. However, unlike covariates, definition variables can also simultaneously account for the effect of age on the covariance among a set of variables. As shown in Figure 1, in the case of the current study age differences are parameterized as a definition variable on the variance of, and covariance between, initial performance (latent intercept) and rate of growth (latent slope).

Models were run separately for each outcome, using W-scores for WRMT Word Identification, Letter Identification, Word Attack, and Passage Comprehension and raw scores for Boston Naming Test, SB Vocabulary, Phoneme Awareness, and RAN. Intercepts were centered at Assessment 1 for all variables. In keeping with our prior publications, RAN was multiplied by −1 prior to model fitting so that a higher score equals faster performance. Analyses were conducted with individual-level data using Mx (Neale et al., 2002).

Estimated mean latent intercept, slope, and model fitting estimates are presented in Table 2. Mean latent intercepts were all lower than the mean values for reading outcomes at Assessment 1 as presented in Table 1. This is because the intercept was a latent factor that also accounted for age differences at Assessment 1. In the case of Phoneme Awareness this resulted in a negative mean y-intercept because raw scores were relatively close to zero at Assessment 1. RAN was reverse scored so the mean is expected to be negative. In all cases, mean latent slope scores were positive, indicating positive rates of growth across assessment points.

Univariate genetic estimates

The model also simultaneously estimated genetic (heritability, or h^2), shared environment $(c², or between-family differences in environment) and nonshared environment (e², or within$ family differences in the environment) for the latent intercept and slope (Table 3). Starting with latent intercepts, estimates were obtained by squaring the loadings of latent intercept on Factors A (h²_{intercept} = Factor A²), C(c²_{intercept} = Factor C²), and E (e²_{intercept} = Factor E²). Heritability for initial performance as measured by the latent intercept varied from h^2 _{intercept} = .38 for WRMT Word Identification, Letter Identification, and Word Attack latent intercepts, to h^2 _{intercept} = .72 for RAN. Shared environmental estimates were statistically significant for all latent intercepts with the exception of RAN (c^2 _{intercept} = .11), ranging from c^2 _{intercept} = .62 for WRMT Word Identification and Letter Identification to c^2 _{intercept} = .45 for Boston Naming Test. The nonshared environmental estimate was statistically significant only for the RAN latent intercept (e^2 _{intercept} = .17).

Turning to the rate of growth, genetic and environmental effects on latent slope may be shared with latent intercept (Factors A, C, and E) or independent from latent intercept (factors a, c, and e). Thus, $h^2_{\text{slope}} = \text{Factor A}^2 + \text{Factor a}^2; c^2_{\text{slope}} = \text{Factor C}^2 + \text{Factor c}^2;$ and $e^2_{\text{slope}} = \text{Factor C}^2$ E^2 + Factor e^2 . Genetic influences were statistically significant for Phoneme Awareness $(h^{2}_{slope} = .20)$ and RAN ($h^{2}_{slope} = .58$). Shared environmental influences were statistically significant and substantial for WRMT Word identification (c^2 _{slope} = 1.00), WRMT Letter Identification (c^2 _{slope} = .99), WRMT Word Attack (c^2 _{slope} = .91) and Phoneme Awareness $(c² slope = .80)$. The nonshared environmental estimate was statistically significant for RAN

 $(e^{2}_{slope} = .17)$. Because estimates of intercept and slope were latent, Factors E and e were free from measurement error.

Bivariate genetic estimates

In addition to estimating univariate effects, we also examined whether genetic, shared environmental, and nonshared environmental sources of variation on the growth of reading (latent slope) overlapped with and/or were unique from initial performance (latent intercept). We examined the issue of overlap by estimating the genetic, shared environmental, and nonshared environmental contributions to the correlation between latent intercept and latent slope. Using Word Identification latent slope as an example (Table 4), the total estimated correlation between latent slope and latent intercept was $r = -0.61$, which is the sum of the genetic pathway $r_{\text{genetic}} = .02$ (NS), the shared environmental pathway r_{shared} environment = -0.63 (p < .05), and the nonshared environmental pathway $r_{nonhared$ environment = .00 (NS). Genetic pathways for the correlation between latent intercept and slope were statistically significant for Phoneme Awareness ($r_{\text{genetic}} = -.32$) and RAN ($r_{\text{genetic}} = -.65$). Shared environmental pathways were statistically significant for WRMT Word ID (rshared environment $=$ -.63), WRMT Letter ID ($r_{shared environment} = -0.49$), WRMT Word Attack $(r_{shared environment} = -0.58)$, and Phoneme Awareness $(r_{shared environment} = -0.41)$, while the nonshared environmental pathways were significant for RAN ($r_{nonhared$ environment = -.17). Taken together, these results suggested that rates of growth were slower in children with higher initial levels of performance.

In addition, estimates were also obtained for unique sources of genetic, shared environmental, and nonshared environmental variance on the rate of growth, independent from initial performance (Table 5). These estimates were obtained by examining the relative proportion of variance on latent slope that loaded on the independent factors (a, c, and e) versus general factors (A, C, E). Put another way, independent genetic effects were evidenced by the extent to which the loadings from factor a to the latent slope were larger than the loadings from factor A to the latent slope (see Figure 1). The same logic applied to the shared and nonshared environment. Independent genetic and environmental effects would point to unique etiological factors that influence the rate of growth (latent slope) above and beyond the genetic and environmental variance associated with initial performance (latent intercept).

Turning to Table 5, Phoneme Awareness and RAN latent slopes showed significant genetic effects (h²_{slope} = .20 and h²_{slope} = .58 respectively), but they were explained entirely by general genetic variance also shared with the latent intercept, as evidenced by h^2 _{slope} = .00 for independent slope pathways. All genetic variance for latent slope loaded on Factor A. In the case of the shared environment, there was evidence for both general (Factor C) and independent (Factor c) pathways. For example, in WRMT Word Identification, $c_{slope}^2 = 1.00$, $c_{slope}^2 = .65$ of which was shared with latent intercept (from Factor C) and c^2 _{slope} = .35 of which was independent to the latent slope (from Factor c). A similar pattern of results was obtained for WRMT Letter Identification, WRMT Word Attack, and Phoneme Awareness. Finally, nonshared environmental effects for RAN latent slope overlapped completely with RAN latent intercept.

Discussion

The current study was the first to examine the genetic and environmental influences on the rate of growth of early reading skills. This was accomplished by estimating genetic and environmental influences on initial level of performance (as estimated by the latent intercept) and rate of growth from that initial level of performance (as estimated by the latent slope). We also examined whether genetic and environmental influences on rate of growth were correlated with or independent from initial level of performance.

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Starting with initial level of reading performance, results were consistent with the larger behavioral genetic literature (e.g. Byrne et al., 2005; Petrill et al., 2007). We expected, and found, that both genetic and environmental influences were important to initial reading performance for word and letter identification, pseudoword decoding, vocabulary, and phoneme awareness. We also expected that RAN, because it is fluency-based, would be influenced primarily by genetics. Taken together, these results are consistent with the hypothesis that individual differences in content-based measures, such as word knowledge or spelling, are likely to be more sensitive to variation in instruction and environmental exposure (Petrill et al., 2006a; Snowling & Hayiou-Thomas, 2006), and with other recent work (Wanzek & Vaughn, 2008) suggesting that fluency outcomes in first grade are least affected by reading interventions.

What is unique about this study is that we examined genetic and environmental influences on differences in the rate of growth of reading skills (as estimated by the latent slope). We found that genetic influences on rate of growth were statistically significant for phoneme awareness and RAN, but these genetic influences were entirely correlated with initial level of reading performance. This is consistent with earlier work (e.g., Byrne et al., 2007; Petrill et al., 2007) showing overall genetic stability, but our data went further, suggesting that there were no additional genetic effects that affected rates of growth above and beyond those shared with initial level of performance. A similar pattern emerged in the case of the nonshared environmental influences on RAN, which overlapped completely with initial performance, suggesting that specific environmental influences on rate of change were completely correlated with those operating at initial performance.

A different picture emerged for shared environmental influences on the rate of growth. In particular, shared environmental influences on the rate of growth were statistically significant for word identification, letter identification, pseudoword decoding, and phoneme awareness. However, unlike genetic influences on rates of growth (that completely overlapped with initial performance for phoneme awareness and RAN), one-third to two-thirds of the shared environmental variance on the rate of growth was independent from initial performance. This is consistent with our previous results (Petrill et al., 2007) suggesting both overlapping and independent shared environmental influences when examining the correlation between reading performance over time. What is new about the current study is that these additional shared environmental influences appear to accelerate or decelerate the rate of reading growth, above and beyond shared environmental influences operating at initial performance (in our case, when children enter school).

Several limitations should be taken into account in interpreting these findings. First, the results were based on three measurement occasions, which precluded the examination of nonlinear growth. Second, the computational demands of twin latent growth curve modeling made it impossible to simultaneously include specific measures of the shared environment. Additionally, the latent growth analyses reported here assumed that a single growth trajectory could adequately approximate individual differences in the growth of reading in this sample (see Boscardin, Muthén, Francis, & Baker, 2008). Fourth, confidence intervals for genetic and environmental estimates were large, so although there was power to test differences from zero, there was not adequate power to compare differences between estimates. Power may also explain why it was not possible to obtain statistically significant genetic and shared environmental estimates for Boston Naming Test and Stanford–Binet Vocabulary latent slopes, despite moderate effect sizes. However, the amount of growth in these variables was small, as shown in the small amount of mean change in measured outcomes (Table 1) and in low mean latent slope estimates (Table 2).

Despite these limitations, our study suggests that genetic differences are important to reading but environmental experiences after entering school may independently impact rates of reading growth. These results suggest that better data and theory are needed to explain how genetics, non-academic environments, the home literacy environment, and the school environment impact literacy development. Educational research suggests that instruction or intervention quality (e.g., Connor et al., 2009) is important in explaining the deviation of growth from initial intercept. On the other hand, Byrne et al. (in press) compared identical twins in same versus different classrooms (and thus to more similar or dissimilar educational environments), and found that classroom placement accounted for a maximum of 8% of the variance in reading and spelling. Byrne et al. (in press) showed that even less variance is explained when change in reading/spelling is examined (defined as a reading/spelling residualized for earlier assessments). Our interpretation is that educational studies overestimate the importance of the environment by ignoring genetically sensitive designs but that rejecting the possibility of teacher effects is unfounded. Our prior work (e.g., Petrill, Deater-Deckard, Schatschneider, & Davis, 2005) suggests that individual predictors of the home environment account for relatively small amounts of variance, so we are not surprised that examining genetically identical individuals sent to the same school but differ whether they are in the same versus different classroom (e.g., Byrne et al., in press) would yield small amounts of variance explained.

More generally, there is a lack of connection between behavioral genetic studies of early reading and the larger reading literature. This dichotomy lends itself to a diminishment of the importance of the environment in the behavioral genetic literature and a diminishment of the potential moderating role of genetic factors in the reading intervention literature. Nonetheless, as illustrated by other complex genetically influenced traits such as heart disease and obesity, genetic risks can be mitigated through environmental interventions. The findings of the current study support the need for significant and sustained efforts to promote reading development, but also suggest that these efforts may be more effective if more attention is paid to genetic risk and protective factors, the home environment, and the larger community.

Key points

- **•** Previous studies have examined genetic and environmental influences on overall reading but have not the etiology of reading growth
- **•** Genetic and shared environmental influences are important to initial performance on multiple reading outcomes
- **•** Shared environmental influences are important for growth in letter- and wordreading, genetic and shared environmental influences significant for growth in phoneme awareness. Finally, genetic and nonshared environmental influences are significant for growth in rapid naming.
- **•** Genetic influences on growth overlap completely initial performance whereas onethird to two-thirds of shared environmental influences on growth are independent from initial performance.
- **•** Clinical implications: Genetic influences in early reading promote stability in the subsequent development of reading skills. However, deviation from initial performance is influenced primarily by the shared environment.

Acknowledgments

The project described was supported by Award Number R01HD038075 from the Eunice Kennedy Shriver National Institute Of Child Health & Human Development. The content is solely the responsibility of the authors and does not

necessarily represent the official views of the Eunice Kennedy Shriver National Institute Of Child Health & Human Development or the National Institutes of Health.

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Figure 1. Latent growth curve model

Standardized scores, W-scores, and raw scores at Assessments 1, 2, and 3. Standardized scores, W-scores, and raw scores at Assessments 1, 2, and 3.

Note: W-scores are presented for RAW WRMT Letter ID, Word ID, and Word Attack. Number correct is presented for RAW Boston Naming Test, Stanford Binet Vocabulary, and Phoneme Awareness. RAW
RAN is reported in seconds. Note: W-scores are presented for RAW WRMT Letter ID, Word ID, and Word Attack. Number correct is presented for RAW Boston Naming Test, Stanford Binet Vocabulary, and Phoneme Awareness. RAW λ RAN is reported in seconds.

** Note*: Standardized measures were not coded in the study.

Mean intercept, slope, −2log-likelihood (−2LL) and degrees of freedom (df) for reading measures

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Note: Statistically significant from zero using 95% confidence intervals, presented in brackets. s, pr <u>ລ້</u>ວ ug 5

Genetic, shared environmental, and nonshared environmental contributions to the correlation between latent intercept and latent slope Genetic, shared environmental, and nonshared environmental contributions to the correlation between latent intercept and latent slope

Note: Statistically significant using 95% confidence intervals, presented in brackets.

proportion of genetic (h2), shared environmental (c2), and nonshared environmental (e2) variance for latent slope shared with latent intercept and independent to latent slope

Note: *p* < .05 using confidence intervals from Mx.