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## The Genetic and Environmental Etiologies of the Relations between Cognitive Skills and Components of Reading Ability

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### Abstract

While previous research has shown cognitive skills to be important predictors of reading ability in children, the respective roles for genetic and environmental influences on these relations is an open question. The present study explored the genetic and environmental etiologies underlying the relations between selected executive functions and cognitive abilities (working memory, inhibition, processing speed, and naming speed) with three components of reading ability (word reading, reading comprehension, and listening comprehension). Twin pairs drawn from the Colorado Front Range ( $n = 676$ ; 224 monozygotic pairs; 452 dizygotic pairs) between the ages of eight and 16 ( $M = 11.11$ ) were assessed on multiple measures of each cognitive and reading-related skill. Each cognitive and reading-related skill was modeled as a latent variable, and behavioral genetic analyses estimated the portions of phenotypic variance on each latent variable

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due to genetic, shared environmental, and nonshared environmental influences. The covariance between the cognitive skills and reading-related skills was driven primarily by genetic influences. The cognitive skills also shared large amounts of genetic variance, as did the reading-related skills. The common cognitive genetic variance was highly correlated with the common reading genetic variance, suggesting that genetic influences involved in general cognitive processing are also important for reading ability. Skill-specific genetic variance in working memory and processing speed also predicted components of reading ability. Taken together, the present study supports a genetic association between children's cognitive ability and reading ability.

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The significance placed on learning to read highlights the important role reading has for academic and career success. Understanding the cognitive processes that underlie the successful decoding of individual words and then combining those words into a larger semantic narrative is the goal of much research on why people vary in their reading abilities, including why some children struggle learning to read. The current study examines the predictive power of four cognitive abilities (working memory, inhibition, processing speed, and naming speed) previously associated with the three main components of reading ability (word reading, listening comprehension, and reading comprehension), and explores the genetic and environmental etiologies of the cognitive-reading relations by comparing the similarities within identical and fraternal twin pairs. Thus, the current study moves beyond identifying what cognitive skills are important for different reading skills to addressing *why* they are predictive.

Learning to read requires direct instruction from parents and teachers. We may find, therefore, that the main reason why cognitive skills are related to reading is because environmental supports important for reading also support associated cognitive abilities. Consider the relations between reading and working memory; learning to read requires having to remember what a word means, what the previous sentence said, etc. The effectiveness of the instruction provided for reading and reading practice, therefore, may also promote working memory. However, one can also make a case that the etiology between working memory and cognitive abilities may be largely genetic. As we review below, previous research has shown that most aspects of cognitive ability, as well as the main components of reading ability, are highly heritable. Just because two abilities are highly heritable, however, does not mean that they share the same genetic influences. The current study seeks to disentangle the genetic and environmental relations between cognitive and reading abilities, as well as the extent to which the different etiological influences are independent or overlapping.

The current study is an extension of a previous study by our group (Christopher et al., 2012). The main focus of Christopher et al. was exploring the extent to which different cognitive abilities predicted either word reading or reading comprehension. By also including listening comprehension, we were able to test the cognitive underpinnings of the simple view of reading, a widely-used model that posits that successful reading comprehension is the product of word reading and listening comprehension skills (Hoover & Gough, 1990). We found that word reading was more strongly tied to processing speed than was comprehension. Working memory was a significant predictor of both word reading and

comprehension. These relations were found after controlling for the influences of naming speed and inhibition, neither of which was uniquely predictive of word reading or comprehension.

Christopher et al. (2012) only included phenotypic (i.e., observed) analyses wherein one twin from each twin pair was selected at random. In addition to increasing the sample size by nearly 40%, the current study utilizes both twins in each pair, allowing for the use of behavioral genetic analyses to assess the relative influences of genes and environment on the relations between the cognitive and reading-related abilities. Understanding the genetic and environmental etiologies underlying the cognitive-reading relations is a largely unexplored question whose answer has important implications for theories of cognitive and reading development. For example, we will seek to determine whether working memory, which was the single best phenotypic predictor of both word reading and comprehension in Christopher et al., is predictive because of shared genetic factors or environmental factors such as differences in type of reading instruction. In addition, we address the question of whether the four cognitive skills all share genetic variance. If there is common cognitive genetic variance, is that variance more predictive of reading component skills than the genetic variance in the individual cognitive skills?

## **Behavioral Genetic Studies of Cognitive Ability**

While the shared etiology of reading and cognitive abilities is still largely an open question, previous behavioral genetic twin studies of executive functions and cognitive ability more generally offer strong evidence that, to the extent that there is shared variance with reading, it will likely be due to genetic influences. Previous behavioral genetic studies of individual differences in children on different aspects of executive function and cognitive ability, including working memory, inhibition, processing speed, and naming speed, have consistently found these abilities to be highly heritable, with genetic influences capturing roughly half of the variance when measured with single tasks (Ando, Ono, & Wright, 2001; Haworth et al., 2010; Lukowski et al., 2014; Plomin & Spinath, 2002; Polderman et al., 2006). When measured with latent variables, genetic influences capture up to nearly 100% of the variance (Friedman et al., 2008). This is not a fully settled question, however, as other studies found moderate significant environmental influences on both specific and common cognitive abilities in children (Alarcón et al., 1999; Harlaar et al., 2005).

Another open question is the extent to which the etiological influences on one cognitive ability are independent from other cognitive abilities. In a highly-cited phenotypic study, Miyake et al. (2000) showed that three latent variables of executive functioning (inhibition, updating, and shifting) had large amounts of overlapping phenotypic variance. Friedman et al. (2008) tested the etiology of the executive function relations and showed that genetic factors accounted for nearly all of the shared phenotypic variance, and that the genetic influences were partially independent from IQ. In addition, genetic variance specific to updating and shifting was also found. While we are using different cognitive abilities than Friedman et al. (2008), the pattern of strong genetic effects that are both shared and unique is suggestive for the current analyses. However, Friedman et al. (2008) is one of the only studies to address the extent of genetic overlap across multiple cognitive abilities in young

adults, leaving open the question of whether this finding is robust in independent samples and in children.

## Behavioral Genetic Studies of Reading

Individual differences in reading ability, both word reading and reading comprehension, are known to be highly heritable. In samples drawn from populations that receive formalized and consistent literacy education, large genetic influences emerge after the first year of formal reading instruction and continue through reading development (Christopher et al., 2013a). This finding is shown in samples from Colorado, Ohio, Florida, England, Australia, and Scandinavia (Byrne et al., 2009; Christopher et al., 2013b; Harlaar et al., 2005; Logan et al., 2013; Olson, Keenan, Byrne, & Samuelsson, 2014; Taylor & Schatschneider, 2009).

As noted earlier, both Christopher et al. (2012) and the present study include listening comprehension as a component of reading ability because it is thought that successful reading comprehension is the product of both word reading and listening comprehension (i.e., the simple view of reading; Hoover & Gough, 1990). Previous work using our sample that directly tested the etiology of the simple view (Betjemann, Keenan, Olson, & DeFries, 2011; Keenan et al., 2006) found that genetic influences on listening comprehension were largely independent from those on word reading. The combination of genetic influences from listening comprehension and word reading, however, accounted for all of the significant genetic influences on reading comprehension (see also Harlaar et al., 2010 for a similar result obtained from a different sample). While these studies offer support for important genetic overlap and independence amongst our three reading components, the etiological relations between listening comprehension and cognitive ability remain largely open questions. In addition, these results suggest that cognitive abilities genetically related to either listening comprehension or word reading will also share genetic influences with reading comprehension, but that cognitive predictors of listening comprehension may have less overlap with those of word reading.

## Previous Research into the Etiology of Cognitive-Reading Relations

Some behavioral genetic studies have included multiple measures of cognitive ability and reading ability, primarily word reading (Harlaar, Hayiou-Thomas, & Plomin, 2005; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, 2006; Thompson, Detterman, & Plomin, 1991; Wadsworth, DeFries, Fulker, Olson, & Pennington, 1995; Willcutt et al., 2010). Using a variety of cognitive measures ranging from individual component processes such as processing speed (Willcutt et al., 2010) to a single “general” cognitive construct (sometimes called *g*; Harlaar et al., 2010), these studies have consistently found that genetic influences are the main source of covariation between cognitive and reading-related abilities. The genetic link between cognitive abilities and reading-related abilities is also found in previous work focused on understanding which cognitive deficits are present in children with reading disabilities (RD), both with RD on its own (Alarcón & DeFries, 1997; Haworth et al., 2009) and in the presence of other disorders such as Attention Deficit Hyperactivity Disorder (ADHD; Willcutt et al., 2010). Using a subset of the same dataset as the present study, Willcutt et al. tested the genetic overlap between a composite measure of

word reading and composite measures of working memory, processing speed, naming speed, and inhibition. While the main focus of Willcutt et al. was exploring whether the cognitive abilities accounted for at least some of the comorbidity of RD and ADHD, they also reported significant genetic correlations between word reading and working memory, processing speed, and naming speed, but not inhibition.

Previous behavioral genetic studies of the link between reading and cognitive abilities have narrowly constrained the meaning of reading to word reading. As mentioned previously, successful reading is more than just reading isolated words out of context and will likely require additional cognitive skills. Therefore, an important aspect of the present study is that it broadens the investigation of the etiology of the relations between cognitive and reading abilities by including reading comprehension and listening comprehension along with word reading. In addition, we model the cognitive and reading abilities as latent variables, thus minimizing measurement error. Finally, we test the shared variance between the cognitive abilities as well as the reading-related abilities, which is important to answer the question of whether the genetic correlations between cognitive ability and reading ability are unique to the individual abilities, or instead reflect shared processes.

## Potential Etiological Changes Across Development

The heritability of reading can vary depending on the type of reading being assessed as well as the stage of reading development. For example, a recent study by our group using a different twin sample found that reading comprehension at the end of fourth grade had, in addition to significant genetic covariance, significant shared environmental covariance with preschool measures of vocabulary and phonological awareness (Christopher et al., 2015). In contrast, the relations between post-first-grade reading comprehension with the preschool measures were largely due to genetic influences. Word reading and spelling at the end of first grade and end of fourth grade also only had significant genetic covariance with the preschool measures. We interpret these results to reflect the changing task demands placed by reading comprehension tests across reading development: early successful reading comprehension is primarily tied to one's ability to decode individual words, which has strong genetic influences, but later reading comprehension, as passages lengthen, becomes more closely related to other abilities such as vocabulary and memory, which also have important shared environmental influences.

The present study includes children ranging in age from eight to 16. By splitting the sample into a younger and an older group, the study assesses whether reading comprehension may have stronger shared environmental covariance with some of the cognitive abilities in older children than in the younger children. In addition, the heritability of some cognitive abilities, such as IQ, increases over childhood (Haworth et al., 2009; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Given that our sample encompasses children as young as eight years old, it is possible that the genetic effects on the cognitive abilities will not be as strong in our younger sample. This leaves open the possibilities that we may see larger environmental effects in the present study or that the extent of genetic overlap between our four cognitive abilities will not be as pronounced.

## Aims of the Present Study

In summary, the present study aims to assess the etiology of the relations between working memory, inhibition, processing speed, and naming speed with word reading, reading comprehension, and listening comprehension in important and novel ways. Given that previous research has shown that most aspects of cognitive ability and reading-related abilities are highly heritable on their own, it is possible that the correlations between the cognitive and reading domains will be driven mostly by shared genetic factors. However, this is a largely untested hypothesis, especially in a study that includes multiple measures of listening comprehension, reading comprehension, and word reading. In addition, we address whether the genetic and environmental covariance between reading-related abilities and cognitive abilities are skill-specific, or instead reflect general cognitive and reading abilities. Taken together, the results of the present study will allow us to begin to address the question of why different cognitive abilities predict different reading abilities.

## Method

### Participants

The present study included data from 676 twin pairs for a total of 1322 participants (30 of the twin pairs only had data from one twin) that ranged in age from 8 to 16 years old ( $M = 11.11$ ,  $SD = 2.45$ ). Out of the 224 monozygotic (MZ) pairs, 106 were male and 118 were female. Out of the 452 dizygotic (DZ) pairs, 256 pairs were opposite sex, 95 were female, and 101 were male. As in Christopher et al. (2012), the participants were initially split into a younger age group (ages 8–10;  $n = 720$ ; 118 MZ pairs; 4 unmatched MZ twins; 235 DZ pairs; 10 unmatched DZ twins;  $M_{\text{age}} = 9.22$ ,  $SD_{\text{age}} = .88$ ) and an older age group (ages 11–16;  $n = 602$ ; 97 MZ pairs; 5 unmatched MZ twins; 196 DZ pairs; 11 unmatched DZ twins;  $M_{\text{age}} = 13.36$ ,  $SD_{\text{age}} = 1.72$ ) to test for potential developmental differences across the large age span.

The participants were part of the ongoing Colorado Learning Disabilities Research Center (CLDRC; DeFries et al., 1997; Olson, 2006). The CLDRC recruits twin pairs from across the Colorado Front Range via school records. Zygosity was confirmed with the Nichols and Bilbro (1966) questionnaire and, in borderline cases, confirmed from DNA collected via cheek swabs. Twin pairs are excluded from the study for the following reasons: learning to read English as a second language, documented brain injury, seizures, significant uncorrected hearing or visual impairment, rare genetic etiology, or sex chromosome anomalies. All twin pairs with at least one member with a school history of RD and/or ADHD are invited to participate, as are a subset of twin pairs with no school history of either disorder. The present sample included 422 (31.9%) participants with a school history of reading disability and 318 (24.1%) with a school history of attention difficulties. Out of those participants, 143 (10.8%) had a school history of both disorders. While the sample may not be fully representative of children in Colorado, the distributions on measures with standard and scaled scores were approximately normal, with means and standard deviations similar to the tests' norming populations ( $M = 100$ ,  $SD = 15$  for standard scores;  $M = 10$ ,  $SD = 3$  for scaled scores; see Table 1). In addition, Christopher et al. (2012) found little



difference in patterns of results when the children with a school history of RD and/or ADHD were excluded from the analyses.

### Procedure

Participants completed a total of four 2.5-hour testing sessions, two sessions each day at the University of Colorado and the University of Denver. For most children (80.9%), the University of Denver testing occurred within three months of the University of Colorado testing (90.2% tested within 6 months). To help control for any gap in testing, all age-controls were specific to the testing location (i.e., all tasks given by the University of Colorado were adjusted by the age at the University of Colorado testing, while all University of Denver tasks were adjusted by the age at that testing). Trained examiners administered all measures.

### Measures

The same 20 measures used in Christopher et al. (2012) were used to assess cognitive and reading abilities in the present study. Descriptive statistics split by age group are provided in Table 1 and phenotypic correlations for the individual measures are shown in supplemental Table 1. The following measures are listed under the construct they are thought to capture. Lower-bound estimates of reliability are provided by the MZ twin correlations and are shown both in parentheses following the measure and in Table 2.

#### Working memory tasks

**Digit span**—From either the WISC-R (Wechsler, 1974;  $n = 617$ ) or the WISC-III (Wechsler, 1991;  $n = 705$ ), participants repeated multiple series of numbers of increasing length either forward or backward. Raw scores for the number of series correctly recalled were used for the present analyses ( $r_{MZ} = .50$ ).

**Sentence span (Siegel & Ryan, 1989)**—Participants generated a word that completed an orally presented sentence and then had to repeat their generated words in blocks ranging from two to six sentence sets ( $r_{MZ} = .46$ ).

**Counting span (Case, Kurland, & Goldberg, 1982)**—Participants counted the number of yellow dots presented on cards and then repeated, in order, the number of dots on each card ( $r_{MZ} = .48$ ).

#### Inhibition tasks

**Gordon Diagnostic System Continuous Performance Test (CPT): Vigilance and distractibility measures**—Two different versions of the CPT (Gordon, 1983) were used. In the vigilance version, participants saw a series of digits flash on a screen and were instructed to press a button whenever “1” was followed by “9.” The distractibility version was similar, but with irrelevant digits flashing on the side of the target stimuli. The number of commission errors (i.e., responding to a nontarget) was the dependent variable used for both versions ( $r_{MZ} = .28$  for vigilance;  $r_{MZ} = .39$  for distractibility).

**Stop-signal reaction time (SSRT)**—Participants were instructed to press, as quickly as possible, either the “X” or “O” key on the keyboard with the corresponding letter flashed on the screen except if an auditory tone sounded. The dependent variable used was the lag required between the letter and tone had to be for the participant to successfully inhibit the button press (Logan, Schachar, & Tannock, 1997). Due to advances in technology, sixty percent received a newer version. Scores were standardized within versions ( $r_{MZ} = .30$  for combined SSRT variable).

### Processing speed tasks

**Identical pictures (French, Ekstrom, & Price, 1963)**—Participants were instructed to search a visual display to find which of four possible responses matched a target picture ( $r_{MZ} = .65$ ).

**Colorado Perceptual Speed (CPS; Decker, 1989; DeFries, Singer, Foch, & Lewitter, 1978)**—Similar to identical pictures but with phonetically similar letters and phonetically dissimilar letters as targets ( $r_{MZ} = .65$ ).

### Naming speed tasks

**Rapid Automatized Naming (RAN) colors and objects**—Adapted from Denckla and Rudel (1976), participants were instructed to name as many colors or objects in a display as possible in 15 s ( $r_{MZ} = .45$  for color;  $r_{MZ} = .49$  for object).

### Word reading tasks

**Time-limited oral reading of single words (word recognition; Olson, Forsberg, Wise, & Rack, 1994)**—Participants read increasingly difficult words presented individually on a screen and had to initiate their responses within a 2-s time limit ( $r_{MZ} = .84$ ).

**PIAT word recognition (Markwardt, 1970)**—Participants read unrelated words that increased in difficulty until they reached a criterion ( $r_{MZ} = .82$ ).

**PIAT spelling (Markwardt, 1970)**—Participants answered a series of multiple-choice spelling recognition questions. This is included as a word reading task as its focus is more on recognition than spelling production ( $r_{MZ} = .73$ ).

### Listening comprehension tasks

**Woodcock Johnson (WJ) oral comprehension (Woodcock, McGrew, & Mather, 2001)**—Participants listened to a short one- or two-sentence passage and supplied the correct missing word ( $r_{MZ} = .64$ ).

**Barnes KNOW-IT**—In this shortened version of the original (Barnes, Dennis, & Haefele-Kalvaitis, 1996; Barnes & Dennis, 1996), participants learned approximately 20 facts about an imaginary planet, then listened to six episodes about children visiting the planet, and finally answered 18 comprehension questions about those episodes ( $r_{MZ} = .49$ ).



**Quality Reading Inventory (QRI) listening**—Modified from Leslie and Caldwell (2001), participants first answered a question regarding the topic of the upcoming passage to assess background knowledge. Then they listened to passages and had to retell the passages as best they could. Finally, they answered six comprehension questions. Scores were made up of both the recall and comprehension questions. Passages differed in length depending on the participant's age. All scores were standardized within level ( $r_{MZ} = .48$ ).

### Reading comprehension tasks

**QRI reading**—Identical to QRI listening except that the participants read the passages aloud ( $r_{MZ} = .60$ ).

**Gray Oral Reading Test-3 (GORT; Wiederholt & Bryant, 1992)**—Participants read passages aloud and answered five multiple-choice questions for each passage ( $r_{MZ} = .50$ ).

**PIAT Individual Achievement Test (PIAT) comprehension (Markwardt, 1970)**—Participants read one or two sentences and then selected which of four pictures represented the passage ( $r_{MZ} = .75$ ).

**WJ passage comprehension (Woodcock et al., 2001)**—Participants read one or two sentences and were instructed to supply the correct missing word ( $r_{MZ} = .70$ ).

### Data Analyses

**Preliminary data analyses**—Prior to all analyses, variables were examined for skew, kurtosis, and outliers. Outliers falling more than three standard deviations beyond the mean for each age group were trimmed to three standard deviations. Outliers were minimal and accounted for less than 2% of the scores for each variable. The Barnes KNOW-IT comprehension test and the three inhibition tasks showed significant skew and were log-transformed.

Missing data was minimal: most variables were missing scores from less than two percent of the participants. The two exceptions were SSRT (missing 43 scores) and the CPT distractibility (123 scores). Because we used maximum-likelihood estimation of raw data, we were able to use data from all participants, including those with missing data.

All variables were then regressed on age, age squared, and age cubed to control for any linear and nonlinear effects of age. Because of the potential time gap between the testing at the two sites, each measure was adjusted by the age of the participant when that particular measure was given. The residuals of this age-adjustment were further standardized within age group and sex and trimmed to three standard deviations.<sup>1</sup> The resulting values were used in all analyses.

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<sup>1</sup>In addition to standardizing within sex, we tested for potential genetic and environmental etiological differences between males and females. The twin correlations separated by sex (supplemental Table 2) were highly similar for the male and female twin pairs. In addition, we tested whether the genetic and environmental estimates for each latent variable could be constrained equal between males and females. The models with males and females constrained were not significantly different in fit from the models that allowed for separate male and female estimates ( $p = .41$  for inhibition,  $p > .75$  for all other latent variables). Therefore, to increase power, males and females are constrained equal in all analyses.

**Latent multivariate analyses**—We used a combination of phenotypic (i.e., observed) and behavioral genetic models on our latent variables of interest. First, phenotypic confirmatory factor analyses (CFAs) that included one twin from each pair selected at random were conducted separately for the cognitive latent variables and the reading latent variables to assess the fit of the latent variables to the raw data as well as to test for potential age differences between the two age groups (ages 8–10 and ages 11–16). All phenotypic models were fit using AMOS (Version 22.0, Arbuckle, 2013). Four different fit indices are reported to assess how well the phenotypic models fit the raw data: chi-square ( $\chi^2$ ), chi-square difference ( $\chi^2$ ), comparative fit index (CFI), and root-mean-square error of approximation (RMSEA). In line with Hu and Bentler (1999), models with CFI values between .95 and 1.00 are considered to be good fits to the original data. Loehlin (1998) suggests that RMSEA values less than .10 show that a model has good fit.

Second, the behavioral genetic analyses decomposed the phenotypic variance and covariance in a variable into four possible components: additive genetic influences ( $a^2$ ), shared environmental influences (that make twins in a pair similar regardless of genetic factors;  $c^2$ ), non-additive genetic influences (including epistasis and dominance;  $d^2$ ), and nonshared environmental influences (that are independent for both twins in a pair, including measurement error;  $e^2$ ). Because MZ twins share 100% of their genes while DZ twins share 50% of their segregating genes on average, genetic influences are indicated if MZ twins are more similar than DZ twins. Shared family and environmental influences, however, are assumed to be equally similar regardless of zygosity (Plomin, DeFries, Knopik, & Neiderhiser, 2013). Note that, due to limited degrees of freedom, it is not possible to estimate both shared environmental and non-additive genetic influences in the same model. All behavioral genetic models were fit using the OpenMx package (Boker et al., 2011).

## Results

### Phenotypic Results

In the interest of space and because this paper is a behavioral genetic extension of previously published phenotypic results, details of the phenotypic results used to confirm the original factor structure in the expanded sample are shown in the supplemental material. Briefly, the phenotypic results from the larger sample supported the four-factor cognitive model (working memory, inhibition, processing speed, and naming speed latent variables) that was held invariant across age groups (final CFA fit:  $\chi^2(93) = 95.441, p = .411, CFI = .998, RMSEA = .006, RMSEA\ 90\% \text{ CI} = [.00, .022]$ ). A three-factor reading component model held invariant across age groups also fit the data well:  $\chi^2(94) = 185.39, p < .01, CFI = .973, RMSEA = .038, RMSEA\ 90\% \text{ CI} = [.030, .046]$ . It is important to note that the reading comprehension and listening comprehension latent variables were very highly correlated ( $r = .94$ ), but we chose to keep these two components separate in the remaining analyses, as the correlation was significantly less than 1.0 and because we wanted to directly test whether there were different etiological patterns for the different aspects of comprehension ability. The cognitive and reading-related abilities CFAs split by age groups are shown in supplemental Figures 1 and 2. The last column in Table 1 contains the standardized CFA loadings for each observed variable.

### Genetic and Environmental Influences On Individual Measures

The first step in the behavioral genetic analyses was to assess the amount of additive genetic ( $a^2$ ), shared environmental ( $c^2$ ), and nonshared environmental ( $e^2$ ) influences onto the individual measures and their respective latent variables. Table 2 shows the MZ and DZ correlations, as well as the univariate results for the measures. Surprisingly, the univariate results for the individual measures showed little evidence of shared environmental influences on any of the cognitive and reading measures, with moderate to large nonshared environmental influences. The large nonshared environmental estimates, especially for the cognitive tasks, were suggestive of large amounts of measurement error. Thus, the use of latent variables for these measures was important and, as the univariate results for the latent variables indicate, helped to reduce the amount of idiosyncratic variability. All of the latent reading-related and cognitive variables were driven primarily by genetic influences, with significant shared environmental influences only found for reading comprehension.

Whereas the majority of the MZ correlations were close to or less than twice the DZ correlations, the inhibition and naming speed measures had MZ correlations higher than twice the DZ correlations. This is indicative of non-additive genetic influences, including dominance and/or epistasis. To assess the significance of the non-additive genetic influences at the latent variable level, additional univariate analyses were run on the inhibition and naming speed latent variables. Limited degrees of freedom do not permit shared environmental influences to be estimated in the same model as non-additive genetic influences; thus, the inhibition and naming speed variances were proportioned into additive genetic ( $a^2$ ), non-additive genetic ( $d^2$ ), and nonshared environmental ( $e^2$ ) influences. The resulting estimates were as follows: for inhibition,  $a^2 = .57$ ,  $d^2 = .43$ ,  $e^2 = .00$ , and for naming speed,  $a^2 = .55$ ,  $d^2 = .38$ ,  $e^2 = .07$ . We found that we were able to drop the non-additive genetic influences ( $d^2$ ) from these models with no significant loss of fit for either the inhibition latent variable ( $\chi^2[1] = .81$ ,  $p = .37$ ) or the naming speed latent variable ( $\chi^2[1] = 1.18$ ,  $p = .28$ ).

### Genetic and Environmental Influences On the Relations Within the Cognitive and Reading-related Domains

**Phenotypically standardized covariances**—The next step in the behavioral genetic analyses was to test whether the genetic and environmental influences on individual differences in each latent trait were shared with the other latent abilities. We allowed the genetic and environmental influences on each latent variable to correlate with the genetic and environmental components of the other variables. Because the four cognitive latent traits showed no significant shared environmental or non-additive genetic variance, correlations between the cognitive traits were calculated only for the additive genetic (A) and nonshared environmental (E) variance components. For correlations between the reading-related abilities, the shared environmental correlation was also calculated, as reading comprehension had significant shared environmental influences and word reading and listening comprehension had shared environmental estimates that, although not statistically significant, were not zero. The correlations were then weighted by the genetic and environmental influences on the latent variables, resulting in “phenotypically standardized covariances” wherein the phenotypic correlation was decomposed into its genetic and

nonshared environmental (and shared environmental for reading abilities) covariances (Plomin & DeFries, 1979).

**Etiology of the relations amongst the cognitive abilities**—As shown in Table 3, the four cognitive latent variables were moderately correlated phenotypically ( $r_p = .38$  to  $.61$ ). The strongest correlation was between the two speeded latent variables, while naming speed and inhibition were least strongly correlated. The majority of the phenotypic correlations were driven by genetic influences (between 79% and 100%, calculated by dividing the phenotypically standardized genetic covariance by the phenotypic correlation). Nonshared environmental covariance was only significant for processing speed and naming speed, suggesting that participants' approaches to speeded measures were somewhat idiosyncratic but consistent across measures.

**Etiology of the relations amongst the reading-related abilities**—The phenotypically standardized covariances for the reading-related latent variables are shown in Table 4. Word reading was strongly correlated with both reading comprehension and listening comprehension ( $r_p = .65$  and  $.57$ , respectively), and the correlation between reading comprehension and listening comprehension was very high ( $r_p = .95$ ). With our sample size, this correlation was significantly different from 1.0. For all three phenotypic correlations, the genetic covariances accounted for over 70% of the shared variance. Unlike the cognitive latent variables, we found significant shared environmental covariances that accounted for between 20% and 28% of the phenotypic correlations. Nonshared environmental covariances were all not significant.

### Genetic and Environmental Influences On the Relations Between the Cognitive and Reading-related Abilities

The main focus of the paper, exploring the etiology of the relations between cognitive and reading-related abilities, was addressed by allowing the four cognitive latent traits to individually correlate with the three reading-related latent traits. Table 5 shows, as expected, that all four cognitive latent traits were significantly correlated phenotypically with listening comprehension, reading comprehension, and word reading ( $r_p$  between  $.38$  and  $.68$ ). Working memory had the strongest correlations with word reading and reading comprehension ( $r_p = .67$  and  $.68$ , respectively), with a significantly smaller correlation with listening comprehension ( $r_p = .55$ , as judged by non-overlapping 95% confidence intervals). Processing speed was more strongly correlated with word reading ( $r_p = .60$ ) than reading comprehension ( $r_p = .50$ ) and listening comprehension ( $r_p = .41$ ). Naming speed and inhibition were both moderately correlated with all three reading-related abilities ( $r_p$  between  $.38$  and  $.47$ ). Thus, in line with previous results, the individual cognitive latent traits accounted for between 14% and 46% of the variance (calculated by squaring  $r_p$ ) in the individual reading abilities.

Importantly, the phenotypically standardized covariances in Table 5 now offer insight into why the cognitive variables are correlated with the reading-related variables. Overwhelmingly, overlapping genetic influences drove the phenotypic correlations. Across

all cognitive and reading-related correlations, genetic covariance accounted for over 90% of the phenotypic relations.

**Genetic and environmental influences shared within cognitive and reading-related domains**—The results of the phenotypically standardized covariances raise the question of whether the genetic covariances between the reading and cognitive variables are unique to each cognitive construct or instead reflect more general cognitive ability. Using a behavioral genetic hierarchical model (Figure 1), the variance in the four cognitive latent variables was split between what was shared between the latent variables and what was unique. Common cognitive variance accounted for 48% of the working memory variance, 45% of the inhibition variance, 62% of the processing speed variance, and 53% of the naming speed variance. As with the univariate estimates, both the common and skill-specific variances were driven by largely genetic influences, with very small nonshared environmental influences. Thus, there is both significant shared genetic variance for the four cognitive variables, as well as significant independent genetic variance on each cognitive skill.

The same type of model was fit to the three components of reading (Figure 2). Interestingly, all of the variance in reading comprehension was shared with listening comprehension and word reading. Almost all (89%) of the variance in listening comprehension was also shared. In contrast, only 38% of the variance in word reading was shared. Although there was evidence for some small shared environmental influences in both the univariate estimates and the correlations between the reading components, we were able to drop shared environmental variance from the nested model with no significant loss in model fit ( $\chi^2[4] = 5.95, p = .20$ ). As with the cognitive variables, most of the shared and skill-specific variance was genetic, with very small nonshared environmental estimates.

The use of hierarchical behavioral genetic models allowed us to quantify common versus skill-specific cognitive and reading-related variance. The next step in the analyses was to estimate the genetic and environmental contributions to the common and unique cognitive variances with the common and unique reading variances. In order to do this, the hierarchical behavioral genetic models were first modified into nested models (see Chen, West, & Sousa, 2006). Rather than testing common and skill-specific variance at the latent variable level, in the nested model the observed variables load onto both their respective latent variable and the common latent variable. To help with model identification, factor loadings for two-indicator latent variables (processing speed and naming speed) were fixed to be equal.

The final nested cognitive model shown in Figure 3 does not include a skill-specific inhibition factor. Chen et al. (2006) noted that, when using a nested model to predict additional variables, including all of the skill-specific latent variables can cause a linear dependency among the variables. Because Christopher et al. (2012) found that inhibition did not predict reading ability once the other cognitive abilities were controlled, inhibition was a good candidate to drop from the final nested cognitive model and all subsequent analyses. As will be noted in more detail later, we tested the validity of excluding the skill-specific inhibition factor in our final models of the phenotypically standardized covariances between

the common and skill-specific cognitive factors with the common and skill-specific components of reading. In the final nested cognitive model, the cognitive variables significantly loaded onto the common cognitive latent variable (standardized loadings between .34 and .63). The three working memory variables, the two processing speed variables, and the two naming speed variables also significantly loaded onto their skill-specific factors (standardized loadings between .45 and .49).

The final nested reading model is shown in Figure 4. In this model, the unique reading comprehension latent variable was dropped because the hierarchical model showed no significant variance in reading comprehension that was not shared with listening comprehension and word reading. All of the reading component variables loaded onto the common reading latent variable (standardized loadings between .47 and .74). The five word reading variables also significantly loaded onto their skill-specific variable (standardized loadings between .28 and .69). The three listening comprehension variables significantly loaded onto the skill-specific variable, although the loadings for WJOC and QRI listening were both low (.14 and .12, respectively). Thus, what was unique to listening comprehension was largely driven by one test, the Barnes KNOW-IT. Unlike the hierarchical model, variance in skill-specific word reading had significant nonshared environmental influences. The vast majority of the variance (94%), however, was genetic, as was the variance on skill-specific listening comprehension and the shared reading latent variable.

### **Genetic and Environmental Influences on the Relations Between Common and Skill-specific Cognitive and Reading-related Abilities**

In the final set of analyses, the genetic and environmental influences from the nested cognitive model and the nested reading components model were covaried. The results of these analyses are shown in Table 6. Common cognitive variance was the best phenotypic predictor of common reading variance ( $r_p = .61$ ) and skill-specific word reading ( $r_p = .64$ ). Skill-specific listening comprehension was similarly correlated with common cognitive variance ( $r_p = .23$ ) and unique working memory ( $r_p = .29$ ). Skill-specific working memory also significantly predicted common reading ability ( $r_p = .37$ ) and unique word reading ( $r_p = .30$ ). Variance unique to processing speed was significantly correlated phenotypically with common reading variance ( $r_p = .16$ ) and unique word reading variance ( $r_p = .21$ ), but not with unique listening comprehension variance. Variance unique to naming speed was not significantly correlated phenotypically with common reading variance, skill-specific listening comprehension variance, or skill-specific word reading variance.

The significant phenotypic correlations were driven primarily by genetic covariances. Nonshared environmental covariances were only significant for common cognitive variance with common reading variance and unique word reading variance, as well as unique processing speed variance with unique word reading variance.

As noted earlier, we chose to exclude the inhibition-specific latent variable to alleviate any potential issues with linear dependency in the model. To test this decision, we reran the models including inhibition-specific variance but with skill-specific naming speed omitted. In these models, skill-specific inhibition did not significantly correlate with unique word



reading or listening comprehension. While skill-specific inhibition did have a significant phenotypic correlation with common reading variance ( $r_p = .12$ ), squaring the phenotypic correlation indicates that skill-specific inhibition only accounted for 1.4% of the variance in common reading ability.

**General cognitive ability and IQ**—We conducted an additional analysis to address the question of whether the genetic variance shared amongst the cognitive variables was independent from IQ. Full-scale IQ from the Wechsler (either Wechsler, 1974 or Wechsler, 1991 as the test administered changed halfway through data collection) was highly phenotypically correlated with the shared cognitive ability latent variable ( $r_p = .74$ ), and genetic influences accounted for 92% of this correlation. These results support that FSIQ is genetically related to the cognitive measures, but is not completely redundant with the common cognitive variance.

## Summary

There are three main findings from the above results. First, the phenotypically standardized covariances between the latent cognitive abilities and the latent reading-related abilities illustrate the important role of genetic influences on the relations between cognitive ability and reading ability regardless of the types of reading and cognitive abilities measured. Second, the hierarchical and nested behavioral genetic models found large amounts of common genetic variance for both the cognitive abilities and the reading-related abilities. Third, general cognitive ability, as defined by the common cognitive latent variable, was the best phenotypic predictor of general reading ability, as defined by the common reading latent variable, as well as skill-specific word reading ability. Skill-specific working memory was also an important predictor of general reading ability, skill-specific word reading, and skill-specific listening comprehension, with smaller contributions from processing speed for common reading ability and skill-specific word reading ability. Again, genetic influences accounted for the majority of these phenotypic relations.

## Discussion

This study examined why individual differences in certain cognitive abilities explain individual differences in reading. To what extent are the correlations due to shared environmental factors, reflecting influences such as differences amongst reading instructional programs? Or to what extent do the correlations reflect common genetic influences? We directly tested the genetic and environmental etiologies underlying the cognitive-reading relations, using not just measures of word reading but also reading comprehension and listening comprehension. The results of the present study refute a strong role for environmental factors in the correlations between cognitive ability and reading ability for the children in our sample. For our measures of working memory, inhibition, processing speed, and naming speed, genetic influences were the main source of the relations between individual differences in the cognitive and reading-related abilities. In addition, the large amount of genetic overlap within the cognitive and reading domains suggests that a majority of the genetic effects found are not specific to the individual reading and cognitive abilities, and instead are shared amongst different traits. Skill-specific genetic

influences on working memory and processing speed, however, were also found to predict reading ability.

On their own, working memory, inhibition, processing speed, and naming speed phenotypically predicted word reading, reading comprehension, and listening comprehension. However, working memory and processing speed had the largest correlations with all three reading-related abilities. In contrast the correlations between the reading-related abilities and naming speed and inhibition were somewhat lower. Importantly, these phenotypic correlations were due almost completely to genetic influences. Shared environmental and non-additive genetic factors were non-significant, and nonshared environmental covariances were very small and generally not significant. The strong genetic relationship between the individual cognitive abilities and word reading is consistent with previous behavioral genetic studies (Harlaar et al., 2005; Petrill et al., 2006; Thompson et al., 1991; Wadsworth et al., 1995). Because most previous behavioral genetic studies exploring cognitive and reading relations have limited their reading measures to word reading, the relatively high genetic covariances between the cognitive abilities and reading comprehension and listening comprehension found in the present study are novel results.

The finding that working memory and processing speed had larger correlations, and thus genetic covariances, with reading comprehension than with listening comprehension may seem surprising, given that the phenotypic correlation between the two comprehension components was very high ( $r_p = .95$ ). These results indicate that at least part of the 10% of variance in reading comprehension that was independent from listening comprehension was driven by genetic influences on working memory and processing speed, genetic influences also important for word reading.

We included listening comprehension as a reading-related skill because, per the simple view of reading (Hoover & Gough, 1990), reading comprehension relies on both listening comprehension and word reading. The nested and hierarchical reading models provide some etiological support for this model of reading ability; although word reading and listening comprehension had significant independent variance, all of the phenotypic variance, and thus genetic variance, in reading comprehension was captured by the common reading latent variable. In contrast, there was evidence of small skill-specific listening comprehension genetic variance and a moderate amount of skill-specific word reading genetic variance.

As with the reading-related models, the results of the hierarchical and nested cognitive models showed a large amount of overlapping genetic influences across all four cognitive abilities. This overlapping genetic variance, or common cognitive genetic variance, was the single best predictor of skill-specific word reading and general reading ability. The idea that cognitive measures share large amounts of variance, sometimes called *g* or general cognitive ability, has a long history in psychological literature (Spearman, 1904). Sometimes used interchangeably with intelligence (e.g., Harlaar et al., 2005), previous behavioral genetic studies have found *g* to be heritable, with heritability increasing across childhood and adolescence (Haworth et al., 2010). Note that whether or not *g* and intelligence are truly interchangeable is an open question, as other researchers found independent genetic variance

in measures of IQ after controlling for shared cognitive variance (e.g., Luciano et al., 2001). Given that full scale IQ in our sample was related, but not completely redundant with the common cognitive variance, we conclude that the genetic relations between the common cognitive variance and reading ability partially capture genetic influences shared with IQ, but there is additional genetic variance not accounted for by IQ that is also important for the cognitive-reading relations. Conversely, there is also likely additional variance in IQ linked to reading ability. For example, measures of both IQ and reading ability frequently include a vocabulary component, suggesting that the link between IQ and reading is partially due to conceptual overlap between the two abilities.

In Christopher et al. (2012) we found that working memory and processing speed phenotypically predicted reading ability after inhibition and naming speed were controlled. Although the common cognitive variance was the best single predictor of common reading ability and skill-specific word reading in the present study, significant genetic covariances between these two reading abilities and skill-specific working memory and skill-specific processing speed were also found. Genetic influences specific to actively recalling and using information in memory as well as speed of processing, therefore, are also important for reading. At the same time, there is no evidence that inhibition and naming speed predict any aspect of reading ability once common cognitive variance and variance unique to working memory and processing speed have been controlled. The genetic relations between inhibition and naming speed with reading, therefore, appear to be driven by more general genetic influences.

It is important to note that the finding of genetic links between common cognitive variance and skill-specific cognitive variance with the components of reading ability is correlational. We are unable to decipher the direction of causation in our analyses, leaving open a few possible interpretations. First, it may be that genetic influences involved in multiple aspects of children's cognitive processing are responsible for how well children learn to read. However, it is not necessarily correct to assume that reading and cognitive processing utilize the same genetic influences or have causal relations with each other. It is also possible that there are reciprocal links between reading development and cognitive development, such that the genetic influences responsible for learning to read and the genetic influences underlying cognitive ability develop alongside each other.

A recent study by our group using a different twin sample, Christopher et al. (2015), provides some evidence that cognitive genetic influences may be casually tied to the development of reading ability. Twins in Christopher et al. were tested in the year prior to starting kindergarten on measures of verbal memory and naming speed, as well as other pre-reading skills. Importantly, the vast majority of these children was unable to read a single word on a test of word reading and had not started receiving formalized literacy education. The twins were then tested on word reading, reading comprehension, and spelling in the summers following first and fourth grades. Verbal memory and naming speed measured in the preschoolers were correlated phenotypically with the reading and spelling measures at both time points, and these correlations were due primarily to genetic influences. We interpret the longitudinal genetic relations between verbal memory and naming speed, measured in nonreaders, with reading ability to be suggestive of causal pathways between

cognitive ability and learning to read. Regardless of the issue of causation, the results of the present study leave little room for potential environmental influences on the link between cognitive abilities and reading-related abilities.

The lack of environmental influences found, however, should not be interpreted to mean that the environment does not matter for either reading or cognitive abilities. Indeed, learning to read requires direct instruction from parents and teachers. It is important to remember that our analyses focus on the etiology underlying variance and covariance in cognitive and reading abilities rather than mean levels of either. Shared environmental influences that are present in much of our sample likely affect the mean levels of reading ability but not be manifested in the variance components. For example, growing up along the Front Range in Colorado, having access to formal and consistent education, and speaking English as a first language are all environmental influences present in the whole sample and, therefore, can be important influences on how well the children in our sample are reading but are not driving differences between the children. Given that the youngest children in our sample were already eight-years-old, all of the children in our study had already received at least a few years of reading instruction in school. To the extent that literacy education in schools is homogenous, variance due to school environmental factors will decrease. Under the twin model, proportions of variance in a measure are assigned to genetic or environmental factors, such that decreasing environmental variance will result in higher estimates of genetic influences. It is also possible that the high heritability estimates for the cognitive and reading-related measures reflect the presence of gene-environment correlations. Since genes express themselves through the environment, children who are genetically inclined to be better readers or excel at other cognitive measures may seek out opportunities to read or practice abilities tied to cognitive ability.

The current analytic approach does not allow us to identify which specific genetic influences are important. We cannot say for sure that, for example, the common cognitive variance is partially capturing genetic variance due to learning rate or speed of cognitive access. It is also possible that the common cognitive genetic variance reflects a third variable (or multiple variables) not measured, or that the genetic influences have a reciprocal relation. Rather the present results suggest that differences in one's environment, such as school, teacher, or peers, are not driving the relations between cognitive ability and reading.

We end with four important caveats and limitations of this and all other behavioral genetic analyses. First, it may be tempting to assume that the presence of genetic effects implies that there is little room for intervention, such as for children with reading disabilities. Estimates of heritability, however, are not estimates of immutability. Heritability estimates allow us to quantify the extent to which, in our sample, differences in DNA result in differences in phenotypic variance. They provide a snapshot of the role of genetic influences for the sample being studied at a particular time. This leads to a second caveat of behavioral genetic analyses. Our results are specific to our sample, drawn from the Colorado Front Range with English as a first language. Samples drawn from different populations could have different heritability estimates. Relatedly, the results are specific to the measures used. While the use of latent variables helped to minimize measurement error, some of the individual measures, particularly the inhibition measures, had very low MZ twin correlations, suggesting low

reliability on these measures. Finally, while we were able to drop the shared environmental variance and non-additive genetic variance components from our model without significant loss of fit, it is possible they could be significant in larger samples with more power. It is also possible that shared environmental variance could be underestimated and additive genetic variance overestimated if some of the genetic variance is truly non-additive. However, given the strength of the genetic influences found, it is likely that any potential shared environmental or non-additive genetic influences are small. Therefore, we are confident that the overall pattern of our results is robust: that the relations between different components of cognitive ability and reading ability are due primarily to genetic influences.

## Conclusion

The goal of the present study was to start to answer the question of why cognitive measures predict different aspects of reading ability. Our results suggest that genetic influences, rather than environmental influences, are responsible for the predictive links between working memory, inhibition, processing speed, and naming speed with word reading, reading comprehension, and listening comprehension. These genetic influences are largely shared across cognitive measures, with additional independent genetic variance for working memory and processing speed. Thus, our results strongly suggest that genetic influences important for many aspects of cognitive ability are also important for reading and reading-related abilities.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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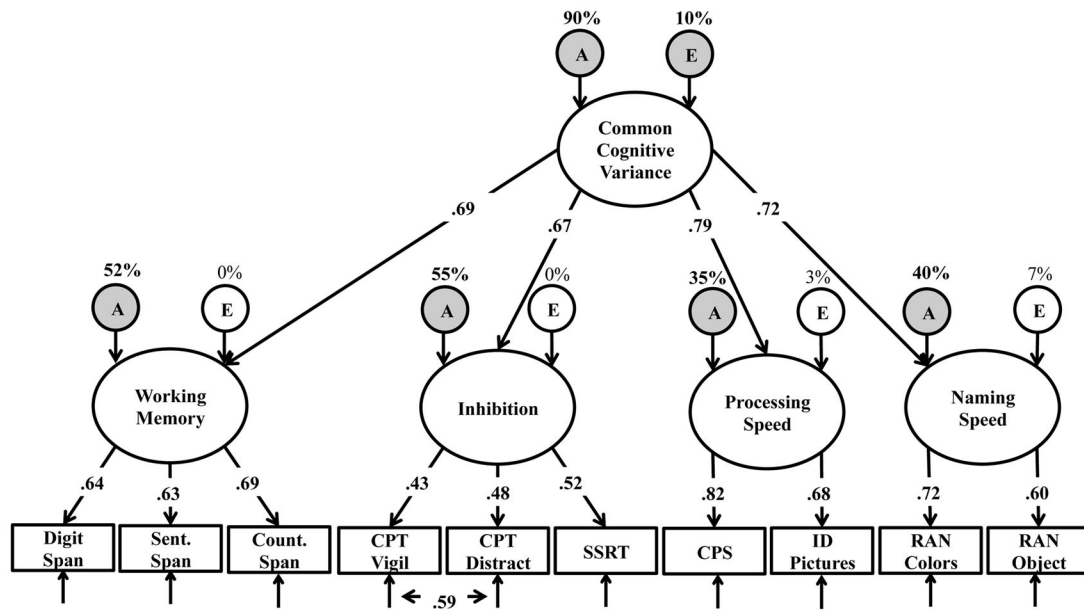
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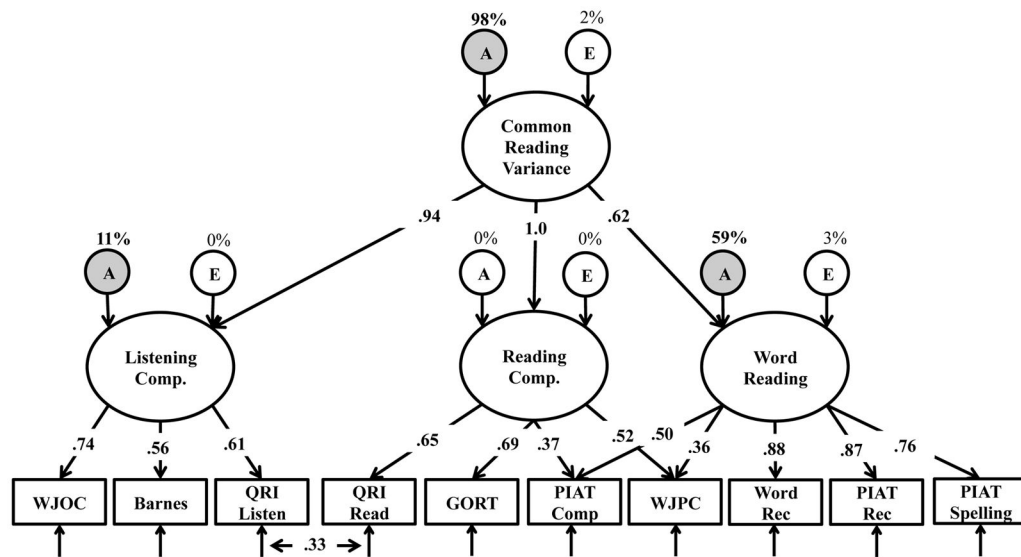


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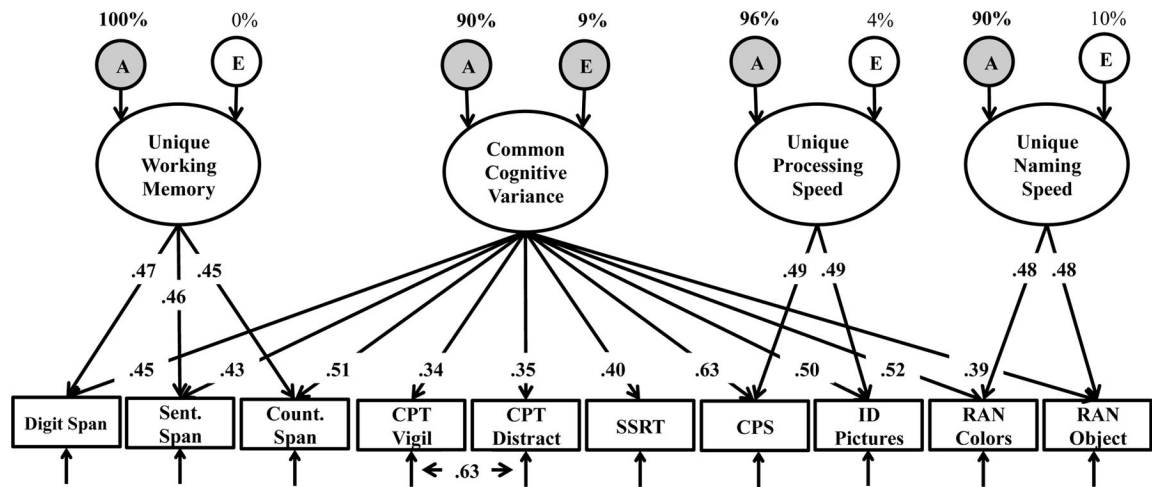
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**Figure 1.** Behavioral Genetic Hierarchical Model of the Cognitive Latent Variables. All numbers on single headed arrows are standardized regression coefficients and are significant (i.e., 95% confidence intervals do not include zero). If shaded, variance is significant (also per 95% confidence intervals). A = additive genetic variance; E = non-shared environmental variance. Residual variance (i.e., variance in the manifest variables not captured by the latent variable) =  $1 - \text{factor loading}^2$

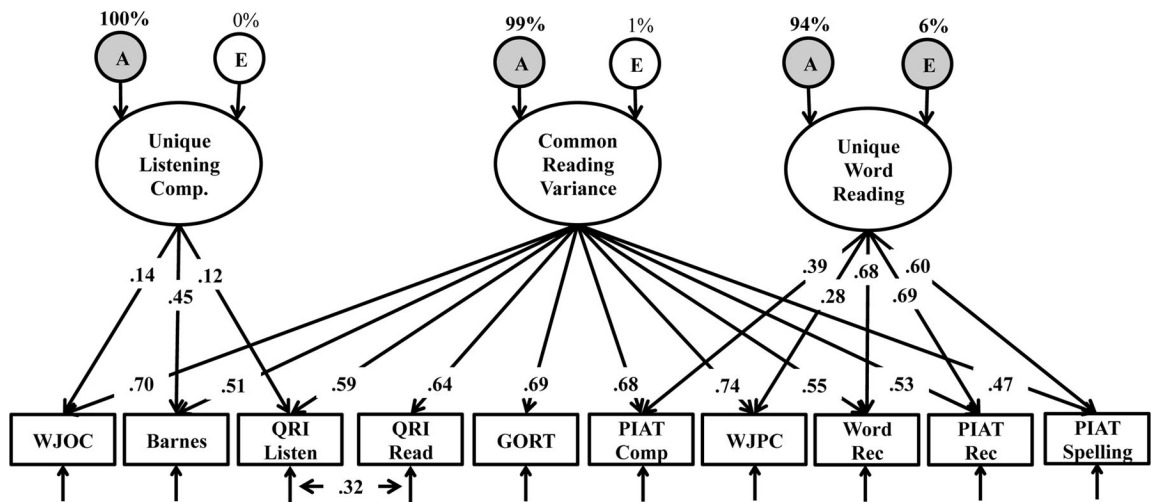


**Figure 2.** Behavioral Genetic Hierarchical Model of the Reading Ability Latent Variables. All numbers on single headed arrows are standardized regression coefficients and are significant (i.e., 95% confidence intervals do not include zero). If shaded, variance is significant (also per 95% confidence intervals). A = additive genetic variance; E = non-shared environmental variance. Residual variance (i.e., variance in a manifest variable not captured by the latent variable) =  $1 - \text{factor loading}^2$



**Figure 3.**

Behavioral Genetic Nested Model of the Cognitive Latent Variables. All numbers on single headed arrows are standardized regression coefficients and are significant (i.e., 95% confidence intervals do not include zero). If shaded, variance is significant (also per 95% confidence intervals). A = additive genetic variance; E = non-shared environmental variance. Note that total variance percentages may not equal 100% due to rounding. Residual variance (i.e., variance in the manifest variables not captured by the latent variable) =  $1 - (\text{factor loading for common latent variable}^2 + \text{factor loading for unique latent variable}^2)$



**Figure 4.** Behavioral Genetic Nested Model of the Reading Ability Latent Variables. All numbers on single headed arrows are standardized regression coefficients and are significant (i.e., 95% confidence intervals do not include zero). If shaded, variance is significant (also per 95% confidence intervals). A = additive genetic variance; E = non-shared environmental variance. Residual variance (i.e., variance in the manifest variables not captured by the latent variable) =  $1 - (\text{factor loading for common latent variable}^2 + \text{factor loading for unique latent variable}^2)$



**Table 1**

Descriptive Statistics.

	Ages 8–10 (M = 9.22, SD = .88)			Ages 11–16 (M = 13.36, SD = 1.72)			Standard. LV Loadings		
	n	Mean	SD	Skew	n	Mean	SD	Skew	
<b>Working Memory Latent Variable</b>									
Digit Span (Raw)	720	11.29	3.02	.34	601	14.58	3.53	.50	.65
Sentence Span (Raw)	718	4.32	1.70	.61	598	6.46	2.25	.57	.64
Counting Span (Raw)	719	5.77	2.23	.50	598	7.88	2.33	.29	.69
<b>Inhibition Latent Variable</b>									
CPT Vigil. Errors (Raw) <sup>~ag</sup>	713	6.81	6.27	1.68	594	2.99	4.13	3.11	.49
CPT Distract Errors (Raw) <sup>~ag</sup>	640	13.95	9.93	.74	559	6.31	6.92	1.71	.51
SSRT Version 1 (Raw) <sup>~bg</sup>	389	344.44	119.89	.75	390	262.37	86.68	1.44	.51
SSRT Version 2 (Raw) <sup>~bg</sup>	297	405.36	142.15	.46	203	275.21	102.29	1.47	
<b>Processing Speed Latent Variable</b>									
CO Perceptual Speed (Raw)	719	13.40	5.53	.66	602	26.28	8.52	.54	.85
Identical Pictures (Raw)	720	47.29	11.06	.33	602	70.03	14.00	.12	.67
<b>Naming Speed Latent Variable</b>									
RAN Color (Raw)	720	19.88	4.19	.29	602	26.23	5.19	.32	.77
RAN Object (Raw)	720	17.54	3.21	-.02	602	21.55	3.50	.00	.55
<b>Listening Comprehension Latent Variable</b>									
WJ Oral Comp. (SS)	716	107.94	11.59	-.18	602	105.23	10.14	-.23	.75
Barnes KNOW-IT- Literal (Proportion) <sup>~c</sup>	717	0.77	0.21	-.83	601	0.83	0.18	-1.28	.53
Barnes KNOW-IT- Coher. (Proportion) <sup>~c</sup>	716	0.73	0.19	-.76	600	0.82	0.16	-1.27	
Barnes KNOW-IT- Infer. (Proportion) <sup>~c</sup>	717	0.75	0.18	-.83	599	0.80	0.16	-1.40	
QRI Listen- Recall (Raw) <sup>d</sup>	713	0.34	0.16	.54	596	0.30	0.12	.37	.62
QRI Listen- Questions (Raw) <sup>d</sup>	718	4.47	1.39	-.78	602	4.38	1.13	-.90	
<b>Reading Comprehension Latent Variable</b>									
QRI Read- Recall (Raw) <sup>e</sup>	702	0.36	0.15	.16	593	0.29	.13	.87	.64
QRI Read- Questions (Raw) <sup>e</sup>	708	4.46	1.18	-.76	599	4.61	0.96	-.68	
GORT (Scaled)	706	10.58	3.06	.21	594	11.89	3.27	.31	.71

	Ages 8–10 (M = 9.22, SD = .88)		Ages 11–16 (M = 13.36, SD = 1.72)		Standard. LV Loadings		
	n	Mean	SD	Skew	Mean	SD	Skew
PIAT Comp. (SS) <sup>f</sup>	714	109.05	13.04	-.47	103.94	12.47	-.34
WJ Pass. Comp. (SS) <sup>f</sup>	715	101.10	10.67	-.17	102.75	10.40	.10
<b>Word Reading Latent Variable<sup>f</sup></b>							
Word Recognition (Raw)	720	88.98	37.37	-.01	144.93	34.83	-.63
PIAT Read. Rec. (SS)	714	106.74	12.20	-.31	102.79	11.99	-.14
PIAT Spelling (SS)	714	105.16	12.32	-.08	100.36	13.44	.15

Note: Variables that share superscripts <sup>a</sup> to <sup>e</sup>were combined together for all subsequent analyses;

<sup>f</sup>WJ Passage Comprehension and PIAT Reading Comprehension cross-loaded onto the Word Reading Latent Variable;

<sup>g</sup>all inhibition tasks were reverse-coded for all subsequent analyses, such that larger values correspond to better performance.

Standardized latent variable loadings (Stand. LV Loadings) come from phenotypic confirmatory factor analyses done on one twin per pair selected at random.

<sup>h</sup>Due to significant skew, the variable was transformed prior to all analyses.

Table 2

Twin Correlations and Univariate Estimates.

	Twin Correlations			Univariates		
	<i>r</i> MZ	<i>r</i> DZ	<i>d</i> <sup>2</sup>	<i>r</i> <sup>2</sup>	<i>d</i> <sup>2</sup>	<i>e</i> <sup>2</sup>
<b>Working Memory LV</b>	.97	.54	.86* [.57, 1.0]	.11 [.00, .34]	.03 [.00, .14]	
Digit Span	.50	.28	.43*	.06	.51*	
Sentence Span	.46	.23	.46*	.00	.54*	
Counting Span	.48	.21	.46*	.00	.54*	
<b>Inhibition LV</b>	1.00	.40	1.00* [.58, 1.0]	.00 <sup>†</sup> [.00, .31]	.00 [.00, .24]	
CPT Vigilance	.28	.07	.24*	.00	.76*	
CPT Distract	.39	.12	.36*	.00	.64*	
Stop-signal Reaction Time	.30	.15	.30	.00	.70*	
<b>Processing Speed LV</b>	.90	.51	.79* [.51, 1.0]	.11 [.00, .33]	.10* [.004, .22]	
Colorado Perceptual Speed	.65	.37	.54*	.10	.36*	
Identical Pictures	.65	.24	.60*	.00	.40*	
<b>Naming Speed LV</b>	.92	.37	.88* [.62, 1.0]	.00 <sup>†</sup> [.00, .18]	.12 [.00, .27]	
RAN Color	.45	.18	.45*	.00	.56*	
RAN Object	.49	.21	.47*	.00	.53*	
<b>Word Reading LV<sup>2</sup></b>	.97	.54	.87* [.72, .99]	.10 [.00, .25]	.03 [.00, .05]	
Word Recognition	.84	.41	.82*	.02	.17*	
PIAT Reading Recognition	.82	.41	.76*	.05	.19*	
PIAT Spelling	.73	.36	.69*	.03	.28*	
<b>Listening Comprehension LV</b>	1.00	.58	.83* [.63, 1.0]	.17 [.00, .37]	.00 [.00, .03]	
WJ Oral Comprehension	.64	.31	.58*	.04	.38*	
Bates KNOW-IT	.49	.25	.51*	.00	.49*	
QRI Listening	.48	.30	.42*	.08	.50*	
<b>Reading Comprehension LV</b>	1.00	.59	.82* [.65, .99]	.18* [.01, .34]	.00 [.00, .04]	

	Twin Correlations		Univariates	
	rMZ	rDZ	$d^2$	$c^2$
QRI Reading	.60	.29	.61*	.00
GORT	.50	.34	.42*	.12
PIAT Comprehension	.75	.39	.69*	.05
WJ Passage Comprehension	.70	.42	.49*	.19*

Note:  $d^2$  = genetic variance,  $c^2$  = shared environmental variance,  $e^2$  = nonshared environmental variance; MZ = monozygotic; DZ = dizygotic;

\*  $p < .05$  determined via 95% confidence intervals (shown for latent variables).

<sup>1</sup> Non-additive genetic influences were also tested separately from shared environmental influences, and were found to be not significant.

<sup>2</sup> Word reading latent variable includes PIAT Comprehension and WJ Passage Comprehension.

**Table 3**

Phenotypically Standardized Covariances between the Cognitive Latent Variables.

	Phenotypic Correlation ( $r_p$ )			=	Genetic (cov <sub>g</sub> )			+	Nonshared Environmental (cov <sub>e</sub> )		
	1.	2.	3.		1.	2.	3.		1.	2.	3.
1. WM	-			-				-			
2. IN	<b>.57</b> [.47, .66]	-		<b>.53</b> [.42, .65]	-			<b>.03</b> [-.06, .11]			
3. PS	<b>.51</b> [.45, .58]	<b>.52</b> [.43, .61]	-	<b>.52</b> [.44, .60]	<b>.47</b> [.35, .59]	-		<b>-.01</b> [-.06, .05]	<b>.05</b> [-.04, .14]		
4. NS	<b>.50</b> [.42, .57]	<b>.38</b> [.27, .48]	<b>.61</b> [.54, .67]	<b>.43</b> [.33, .52]	<b>.30</b> [.15, .44]	<b>.48</b> [.38, .57]		<b>.07</b> [.00, .14]	<b>.08</b> [-.04, .19]	<b>.13</b> [.07, .20]	

Note: Numbers in **bold** are significant at  $p < .05$  as judged by 95% confidence intervals. WM = Working Memory Latent Variable; IN = Inhibition Latent Variable; PS = Processing Speed Latent Variable; NS = Naming Speed Latent Variable.

**Table 4**

Phenotypically Standardized Covariances between the Reading Latent Variables.

	Phenotypic Correlation ( $r_p$ ) =		Genetic ( $cov_g$ )		+ Shared Environmental ( $cov_e$ )		+ Nonshared Environmental ( $cov_e$ )	
	1.	2.	1.	2.	1.	2.	1.	2.
<b>1. WR</b>	-		-		-		-	
<b>2. LC</b>	<b>.57</b> [.51, .63]	-	<b>.40</b> [.28, .54]	-	<b>.16</b> [.03, .27]	-	<b>.01</b> [.00, .04]	-
<b>3. RC</b>	<b>.65</b> [.59, .70]	<b>.95</b> [.92, .98]	<b>.48</b> [.36, .61]	<b>.75</b> [.60, .91]	<b>.15</b> [.02, .26]	<b>.19</b> [.04, .34]	<b>.02</b> [.00, .05]	<b>.01</b> [.00, .05]

Note: Numbers in **bold** are significant at  $p < .05$  as judged by 95% confidence intervals. WR = Word Reading Latent Variable; LC = Listening Comprehension Latent Variable; RC = Reading Comprehension Latent Variable.



**Table 5**  
Phenotypically Standardized Covariances between the Cognitive Abilities and Dimensions of Reading Ability

	Phenotypic ( $r_p$ )	Genetic ( $cov_g$ )	Nonshared Environ. ( $cov_e$ )
<b>Word Reading</b>			
Working Memory	.67 [.62, .72]	.63 [.57, .69]	.04 [.01, .07]
Inhibition	.44 [.36, .53]	.39 [.29, .49]	.05 [.01, .10]
Processing Speed	.60 [.55, .65]	.57 [.51, .62]	.03 [.00, .06]
Naming Speed	.47 [.40, .53]	.43 [.36, .51]	.03 [-.01, .08]
<b>Listening Comprehension</b>			
Working Memory	.55 [.48, .62]	.57 [.50, .64]	-.02 [-.06, .02]
Inhibition	.40 [.33, .50]	.38 [.26, .48]	.03 [-.03, .09]
Processing Speed	.41 [.33, .48]	.43 [.34, .51]	-.02 [-.06, .04]
Naming Speed	.38 [.30, .46]	.33 [.24, .43]	.05 [-.03, .10]
<b>Reading Comprehension</b>			
Working Memory	.68 [.62, .73]	.65 [.59, .72]	.02 [-.02, .06]
Inhibition	.46 [.37, .55]	.42 [.31, .52]	.05 [.00, .11]
Processing Speed	.50 [.44, .56]	.49 [.42, .55]	.02 [-.02, .06]
Naming Speed	.41 [.34, .48]	.42 [.33, .50]	-.01 [-.06, .05]

Note: Numbers in **bold** are significant at  $p < .05$  as judged by 95% confidence intervals.

**Table 6**

Phenotypically Standardized Covariances between Common and Unique Variance in the Cognitive Latent Variables with Dimensions of Reading Ability.

	Phenotypic ( $r_p$ ) = Genetic (cov <sub>a</sub> ) + Nonshared Environ. (cov <sub>e</sub> )		
<b>Common Reading</b>			
Common Cognitive Variance	<b>.61</b> [.54, .69]	<b>.57</b> [.49, .65]	<b>.05</b> [.02, .08]
Unique Working Memory	<b>.37</b> [.27, .46]	<b>.37</b> [.26, .47]	.00 [.00, .05]
Unique Processing Speed	<b>.16</b> [.06, .26]	<b>.12</b> [.01, .22]	.05 [.00, .09]
Unique Naming Speed	.03 [-.06, .10]	.07 [-.04, .16]	-.03 [-.08, .03]
<b>Unique Word Reading</b>			
Common Cognitive Variance	<b>.64</b> [.56, .71]	<b>.57</b> [.49, .65]	<b>.07</b> [.03, .10]
Unique Working Memory	<b>.30</b> [.20, .39]	<b>.29</b> [.19, .39]	.01 [.00, .06]
Unique Processing Speed	<b>.21</b> [.11, .31]	<b>.13</b> [.02, .23]	<b>.08</b> [.03, .13]
Unique Naming Speed	.01 [-.07, .10]	.05 [-.05, .15]	-.04 [-.10, .03]
<b>Unique Listening Comprehension</b>			
Common Cognitive Variance	<b>.23</b> [.19, .34]	<b>.27</b> [.14, .39]	-.03 [-.11, .04]
Unique Working Memory	<b>.29</b> [.17, .41]	<b>.29</b> [.14, .44]	.00 [-.10, .10]
Unique Processing Speed	.09 [-.04, .22]	.09 [-.05, .24]	.00 [-.12, .11]
Unique Naming Speed	.08 [-.03, .20]	.06 [-.09, .21]	.02 [-.11, .15]

Note: Numbers in **bold** are significant at  $p < .05$  as judged by 95% confidence intervals.