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Genetic and Environmental Effects of Serial Naming and Phonological Awareness on Early Reading Outcomes

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Abstract

The current study involved 281 early-school-age twin pairs (118 monozygotic, 163 same-sex dizygotic) participating in the ongoing Western Reserve Reading Project (S. A. Petrill, K. Deater-Deckard, L. A. Thompson, & C. Schatschneider, 2006). Twins were tested in their homes by separate examiners on a battery of reading-related skills including phonological awareness, rapid automatized naming, word knowledge, and phonological decoding. Results suggested that a core genetic factor accounted for a significant portion of the covariance between phonological awareness, rapid naming, and reading outcomes. However, shared environmental influences related to phonological awareness were also associated with reading skills.

Keywords

reading; genetics; phonological awareness; development

For nearly 2 decades, researchers have argued for the primacy of phonological processing in the acquisition of early literacy skills (Bradley & Bryant, 1983; Stanovich & Siegel, 1994; Torgesen, Wagner, Rashotte, Burgess, & Hecht, 1997). More recently, others have suggested that in addition to phonology, naming speed constitutes an independent and additive source of variance in early reading skills (e.g., Wolf & Bowers, 1999). In a recent meta-analysis, Swanson, Trainin, Necoechea, and Hammill (2003) examined the association between phonological awareness, rapid automatized naming (RAN), reading, and related abilities in a set of 49 independent samples. Although their results suggested that phonological awareness and RAN were factorially distinct, particularly in their prediction of real-world reading outcomes, phonological awareness and RAN were also significantly correlated with one another (weighted r = .39). Thus, although phonological awareness and RAN contributed independently to reading outcomes, there was also significant overlap between phonological awareness and the skills that underlie RAN performance.

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One important issue is the extent to which genes and environments influence the overlap and independence among phonological awareness, serial naming, and reading outcomes. It is clear that genetic and environmental influences are significant when examining not only reading outcomes (e.g., Pennington & Smith, 1983; Stevenson, Graham, Fredman, & McLoughlin, 1987) but also reading-related measures such as phonological awareness (Gayan & Olson, 2001; Knopik, Alarcón, & DeFries, 1998; Olson, Gillis, Rack, DeFries, & Fulker, 1991) and RAN (Compton, Davis, De-Fries, Gayan, & Olson, 2001). The importance of genetic influences on reading and reading performance has been further supported by a series of independent studies that have identified and replicated quantitative trait loci for reading on the short arms of Chromosomes 2, 6, and 18 (Cardon et al., 1994; Gayan et al., 1999; Grigorenko, 2003; Grigorenko et al., 1997; Grigorenko, Wood, Meyer, & Pauls, 2000; Fagerheim et al., 1999; Fisher et al., 1999, 2002). Furthermore, studies have shown that genes are primarily responsible for the overlap among reading-related outcomes. For example, the comorbidity among orthographic and phonological kinds of reading skills is due largely to overlapping genetic influences (e.g., Gayan & Olson, 2001, 2003). Similar results have been found when comparing RAN to reading outcomes, particularly in poor readers (Davis, Knopik, Olson, Wads-worth, & DeFries, 2001).

The purpose of the current study was to examine the genetic and environmental etiology of covariance between phonological awareness, naming speed, and reading outcomes in the ongoing Western Reserve Reading Project (WRRP; Petrill, Deater-Deckard, Thompson, & Schatschneider, 2006). As suggested by Swanson et al. (2003), there is both overlap and independence between phonological awareness and RAN as they predict reading outcomes. A handful of studies have also examined the genetic and environmental etiology of the relationship between phonological awareness, RAN, and reading outcomes. Compton et al. (2001) examined this issue in a sample of 800 affected twin pairs in which one or more of the twin pairs had a history of reading problems and a related sample of 450 control twin pairs with no history of reading problems. Both samples ranged in age from 8 to 18 years. The affected group showed evidence of a core set of genes common to phonology, RAN, and reading outcomes as well as a set of genes specific to the relationship between RAN and reading outcomes. An examination of the control group revealed that although the core set of genes common to phonology, RAN, and reading outcomes was significant, independent genetic variance associated with RAN was not correlated with reading outcomes. Although there was little evidence for shared environment in the affected group, there was some evidence for shared environmental overlap between RAN and reading outcomes in the control group.

Although Compton et al.'s (2001) study was important, its generalizability to specific age groups, particularly to children who are just beginning to learn to read, is unclear. As reading skills develop, they are characterized by an early emphasis on learning to decode single words and a later emphasis on developing reading comprehension, which is the ultimate goal of reading (Catts, Hogan, & Adlof, 2005; Chall, 1983; Dale & Crain-Thoreson, 1999; Lyon, Shaywitz, & Shaywitz, 2003). Byrne et al. (2005) examined the relationship between phonological awareness, RAN, and reading outcomes in a sample of 627 preschool- and kindergarten-age twin pairs drawn from the United States (355 pairs), Australia (150 pairs), and Scandinavia (122 pairs). This study found evidence of substantial genetic overlap between phonological awareness, RAN, and word-reading efficiency. Moreover, although there was evidence for significant independent genetic contributions from RAN, the effect size was about half of the genetic variance common to phonology and rapid naming.

Thus, in the present study we explored four possibilities. First, genetics may be primarily responsible for both the overlap as well as the independence between phonological awareness and rapid naming as they predict reading outcomes, as suggested by the sample of individuals with reading disabilities reported by Compton et al. (2001) and Byrne et al. (2005). In other words, whereas rapid naming and phonological processing may share common genes, additional genes specific to rapid naming may also affect reading outcomes. A second possibility is that genes contribute to a basic set of skills common to phonological awareness and rapid naming, but environmental influences result in specificity, such as the unique effect of phonological-based instruction on phonological awareness. In the typically developing sample reported by Compton et al. (2001), shared genes influenced the overlap between rapid naming and phonological awareness, but independent environments were found for rapid naming and phonological awareness. Third, it is possible that in addition to shared genes, environmental influences are also important to the correlation between rapid naming and phonological awareness. In this case, variance in the early home literacy environment may affect phonological awareness, rapid naming, and reading outcomes. Recent behavioral genetic studies have suggested that the importance of environmental variance may be significant, particularly in early readers (Byrne et al., 2002; Petrill et al., 2006). Finally, it is possible that genes and environments are important to both overlap and independence between phonological processing and rapid naming in their prediction of reading outcomes.

Method

Participants

The WRRP (Petrill, Deater-Deckard, Thompson, & Schatschneider, 2006) is a 5-year longitudinal cohort sequential study examining gene–environment factors in the etiology of early reading and related cognitive skills. The families are located throughout Ohio and Western Pennsylvania, with most living in the greater Cleveland, Columbus, and Cincinnati metropolitan areas. Recruiting is conducted through school nominations (N = 273 schools), Ohio birth records, mothers of twins clubs, and media advertisements. Families are recruited if they have same-sex twins who have already entered kindergarten but have not yet finished first grade.

As part of the ongoing study, twins are assessed four times over a 3-year period. In the current study, we examined the 118 monozygotic (MZ) twin pairs (61% female, mean Stanford–Binet standard age scores [SAS] = 101.5, SD = 13.7) and 163 dizygotic (DZ) same-sex twin pairs (56% female, mean Stanford–Binet SAS = 99.9, SD = 12.7) who completed the first home visit. Twins were in kindergarten or first grade (mean MZ age = 6.1 years, SD = .75, range = 5.0–7.9; mean DZ age = 6.1 year, SD = .65, range = 4.9–7.7). Most children lived in two-parent households (96%), and nearly all were White (91%). Forty-three percent of mothers and 40% of fathers had less than a 4-year college education. Eleven percent of mothers and 19% of fathers had a high school education or less.

Procedure and Measures

Children completed a 90-min battery of cognitive and reading-related outcome measures as part of the first home visit. Separate testers assessed each child in separate rooms. The study focused on two predictor skills associated with reading (phonological awareness and RAN) and three outcome variables (letter knowledge, word knowledge, and phonological decoding).

Phonological awareness was assessed with Robertson and Salter's (1997) Phonological Awareness Test. It includes six subtests that measure rhyming (discrimination and production), phoneme isolation (initial), phonemic segmentation (whole word), and phonemic deletion (syllabic deletion and phoneme deletion). Given that phonological awareness has been shown to be a unitary construct (Schatschneider, Francis, Foorman, Fletcher, & Mehta, 1999), the six subtests were summed to form a raw total score for phonological awareness that was then residualized for age and gender with a regression procedure.

Rapid automatized naming was assessed with the letter-naming and number-naming tasks from the Comprehensive Test of Phonological Processing (Wagner, Torgesen, & Rashotte, 1999). Letter and number naming were highly correlated ($r = .73$) and thus were residualized for child age, gender, and, given the age of the sample, score on the Letter Identification subtest of the Woodcock Reading Mastery Tests—Revised (WRMT–R; Woodcock, 1987). Residuals were then z scored and averaged to form a RAN composite, which was then reverse scored so that a high score corresponded to faster naming speed.

Reading outcomes were assessed with the WRMT–R (Woodcock, 1987). We used the Word Identification (Word ID) subtest to assess *word knowledge* and the Word Attack subtest to assess *phonological decoding* (PD) skills. Finally, given the rapid development of reading skills from kindergarten to first grade, we also examined the data in the context of months of school. Months of school correlated .88 with child age.

Results

Descriptive statistics are shown in Table 1. The average level and range of performance are typical for community samples of this kind, with mean scores slightly above average. The average months of school was 6.8 months for identical twins and 6.7 months for DZ twins. The range for both groups was 0–22 months.

Sibling intraclass correlations, also shown in Table 1, suggest that sibling similarity was greatest among MZ twins (.74 to .89), followed by DZ twins (.37 to .57). Because MZ correlations were generally less than 2 times the DZ correlation, these results suggest that sibling similarity is accounted for by both additive genetic and shared environmental variance. Correlations among the outcome variables were generally moderate to substantial, ranging from .20 between phonological awareness and RAN to .70 between Word ID and PD (see Table 2). The months of school variable was not significantly correlated with phonological awareness, RAN, or WRMT Letter ID but was significantly correlated with Word ID and PD.

The major goals of the study were to test whether (a) phonological awareness and RAN independently predicted reading-based outcomes and (b) the overlap and/or independence between phonological awareness and RAN was influenced by genetic and/or environmental influences. Prior to conducting multivariate genetic analyses, we performed a series of multiple regressions to examine the independent prediction of reading outcomes. Phonological awareness and RAN were simultaneously entered as the independent variables. Word ID and PD were the dependent variables in separate analyses. Results suggested that phonological awareness and RAN accounted for independent variance in reading outcomes. In particular, the independent prediction of phonological awareness varied from $\beta = .47$ when predicting Word ID to $\beta = .56$ when predicting PD. Descriptively, the independent prediction of RAN was smaller in magnitude, ranging from $\beta = .14$ for Word ID to $\beta = .15$ for PD (see Table 3).

Given that phonological awareness and RAN accounted for independent sources of variance in reading outcomes, the next step was to examine the genetic and environmental etiology of these relationships. We conducted a series of Cholesky decomposition analyses (see Neale & Cardon, 1992) similar to those used in Compton et al. (2001) and Byrne et al. (2005). As shown in Figure 1, the covariance among phonological awareness, RAN, and outcomes (Word ID and PD, in separate analyses) was parameterized using nine latent factors. A general genetic factor (A1) was set to load on phonological awareness, RAN, and the outcome variable. If this factor was significant, then the overlap among phonological awareness, RAN, and the outcome was influenced by genetic covariance. A2 estimated the genetic relationship between RAN and outcome, after controlling for phonological awareness. A3 estimated the genetic variance in outcome not accounted for by phonological awareness and RAN. A core set of genetic variance common to phonology, RAN, and reading outcomes was supported if A1 was large and significant and A2 was small and nonsignificant. The genetic independence of RAN was supported if the pathway from A2 to RAN was large and significant. The independent genetic relationship between RAN and reading outcomes, separate from genes associated with phonological awareness, was supported to the extent that the pathways from A2 to RAN and reading outcomes were both significant. Similar factors were estimated for shared environment (C1, C2, C3) and non-shared environment (E1, E2, and E3). A core set of environmental influences common to all measures was supported to the extent that C1 (shared environments such as same home and same schools, etc.) and E1 (child-specific environmental experiences that influence both phonological awareness and RAN) and C2/E2 were small and nonsignificant. The role of independent shared and nonshared environmental influences associated with RAN was supported to the extent that C2 and E2 were significant. All models were estimated by means of Mx (Neale, Boker, Xie, & Maes, 1999), using raw data.

To further investigate the role of months of school, we used the model presented in Figure 2 to conduct a second set of analyses. This model was developed in our prior research (Petrill & Deater-Deckard, 2004;Petrill, Pike, Price, & Plomin, 2004) and is based on Neale et al. (1999). In this model, the months of school variable is set to load on phonology, RAN, as well as on reading outcomes. Because months of schooling was invariant within families, this variable constituted a measured shared environmental influence on early reading and related skills. Thus, to the extent that months of schooling accounted for shared environmental influences associated with phonology, rapid naming, and/or reading outcomes, the pathways from months of school should be significant. Moreover, because these pathways would describe a portion of the shared environment, the pathways from C1, C2, and/or C3 should be diminished relative to the model described in Figure 1. Because all variables were either corrected for age or involved age-standardized scores, significant effects for months of school represent residual effects of schooling not associated with the age of the child.

First, these models were used to estimate univariate estimates of heritability (h^2), shared environment (c^2), and nonshared environment (e^2), as well as genetic and environmental contributions to the correlations among phonological awareness, RAN, Word ID, and PD. These results, presented in Tables 4 and 5, decompose the estimated phenotypic correlations into genetic, shared environmental, and nonshared environmental components of variance/ covariance. The diagonals describe the proportion of variance accounted for by genetic, shared environmental, and nonshared environmental influences. The off-diagonals describe the genetic, shared environmental, and nonshared environmental contributions to the observed correlations between phonological awareness, RAN, and reading outcomes.

Table 4 presents results for phonological awareness, RAN, and Word ID. An examination of the basic Cholesky decomposition (using Figure 1: months of school not included) reveals that phonological awareness, RAN, and Word ID demonstrated significant genetic, shared environmental, and nonshared environmental effects (phonological awareness: $h^2 = .61$, $c^2 = .27$, $e^2 = .12$; RAN: $h^2 = .46$, $c^2 = .24$ $e^2 = .30$; Word ID: $h^2 = .59$, $c^2 = .31$, $e^2 = .10$). The correlation between phonological awareness and RAN was influenced significantly by genetic influences ($r = .53$) but not by shared environment ($r = .00$) or nonshared environmental ($r = .08$) overlap. The correlation between phonological awareness and Word ID was influenced by both genetic ($r = .24$) and shared environmental ($r = .27$) overlap. The correlation between RAN and Word ID was influenced significantly by genetic overlap ($r = .21$). An examination of the model in which the months of school variable was included (see Figure 2) revealed that months of school did not account for significant variance in phonological awareness or RAN, nor did this variable account for significant covariance among phonological awareness, RAN, and Word ID. However, months of school explained 15% of the variance in Word ID skills.

When the basic Cholesky model (months of school not included; see Table 5) is used to examine the relationship between phonological awareness, RAN, and PD, it is clear that phonological decoding demonstrated significant genetic, shared environmental, and nonshared environmental influences (h^2 = .46, c^2 = .35, e^2 = .19). Similar to the pattern of results found when examining the relationship between phonological awareness, RAN, and Word ID, the correlation between phonological awareness and PD was influenced by both genetic (r = .37) and shared environmental (r = .21) overlap, whereas the correlation between RAN and PD was influenced by genetic overlap (r = .33). Estimates were nearly identical for phonological awareness and RAN when the months-of-school-included model was examined. However, similar to Word ID, months of school accounted for 11% of the total variance in PD. Taken together, the results in Tables 4–5 suggest that (a) phonological awareness and RAN, (b) RAN and Word ID, and (c) RAN and PD are associated primarily through genetic influences. In contrast, phonological awareness appears to be correlated with Word ID and PD through both genetic and shared environmental pathways.

By fitting submodels to the Cholesky analyses described previously, we were able to test whether the genetic and environmental covariance found in Tables 4–5 was due to independent genetic and environmental effects related to phonological awareness and RAN. In particular, eight submodels were fit to the basic Cholesky model presented in Figure 1: four testing genetic effects and four testing shared environmental effects. First, the pathway from A1 to Word ID was dropped (Drop GenWID) to test whether Word ID was influenced by genetic effects common to both phonological awareness and RAN. Next, the pathways for A2 were dropped (Drop RAN) to test the independent genetic effects related to RAN and the independent relationship between RAN and Word ID. Next, the pathway from A2 to Word ID was dropped (Drop RANWID) to test whether the independent genetic relationship between RAN and Word ID was significant. Finally, A3 was dropped to test whether there was residual genetic variance for Word ID not accounted for by phonological awareness and RAN. Similar models were tested for shared environmental factors (C1, C2, and C2). This process was repeated for PD. The difference between −2 log likelihood is distributed as a chi-square (χ^2_{change}). If a submodel results in a significant decrease in model fit, it is assumed that the parameters dropped in those submodels are statistically significant. In particular, if the correlations found in Tables 4–5 are due to a single source of genetic or shared environmental variance, then dropping independent effects related to RAN (e.g., DROP RANWID) should not result in a significant decrease in model fit. If, however, RAN contributes independent variance to Word ID or PD, then dropping independent genetic and environmental effects related to RAN should significantly decrease model fit.

Results presented in Table 6 suggest that general genetic effects were found when examining Word ID, $\chi^2_{\text{change}}(1) = 18.37$, $p < .01$, and PD, $\chi^2_{\text{change}}(1) = 29.81$, $p < .01$. Independent genetic effects related to RAN were not significant for Word ID, $\chi^2_{\text{change}}(2) = 0.00$, $p > .05$, or PD, $\chi^2_{\text{change}}(2) = .00$, $p > .05$, suggesting that RAN does not possess genetic variance independent from PA. Finally, residual genetic effects for Word ID and PD were not significant. Thus, the genetic overlap between RAN and Word ID was influenced completely by genetic influences shared with phonological awareness. Similar effects were found for PD.

Regarding the shared environment, dropping the pathway from the general shared environmental factor led to a significant decrease in model fit for Word ID: $\chi^2_{\text{change}}(1) = 17.28$, $p < .01$, and PD, $\chi^2_{\text{change}}(1) = 8.95$, $p < .05$. Dropping all pathways from factor C2 resulted in a significant decrease in fit for Word ID and PD, $\chi^2_{\text{change}}(2) = 12.50$ and 11.57, respectively, ps < .01; dropping the pathway from factor C2 to WID, $\chi^2_{\text{change}}(1) = 0.40$, $p > .05$, and PD, $\chi^2_{\text{change}}(1) = 0.36$, $p > .05$, did not result in a significant decrease in fit. Finally, in no case was there residual shared environmental variance in Letter ID, Word ID, and PD.

Regarding the months of school model (see Table 7), results were highly similar to the basic Cholesky model when comparing the eight submodels described previously. Dropping the parameters from months of school to phonological awareness and RAN did not result in a significant decrease in model fit. Dropping the parameters from months of school to WID and PD resulted in significant decreases in model fit: $\chi^2_{change}(1) = 42.32$ and 31.48, respectively, $ps < .01$.

Discussion

The major goals of the study were to examine whether phonological processing and rapid naming independently predicted reading-based outcomes and whether the overlap and/or independence between phonological awareness and RAN was influenced by genetic and/or environmental factors. Results suggested large overlap with some specificity between phonological awareness and RAN in their prediction of reading outcomes. Pearson correlations and multiple regression analyses suggested that phonological awareness and RAN were correlated but that they also independently predicted word identification and phonological decoding. Multivariate quantitative genetic analyses suggested that a general genetic factor was responsible for the covariance between RAN, phonological processing, and reading outcomes. In contrast to Byrne et al. (2005) and the reading disabled sample described by Compton et al. (2001), there was no evidence for independent genetic effects on RAN. Furthermore, independent shared environmental covariance between phonological awareness and word identification as well as between phonological awareness and phonological decoding was also significant. Independent shared environmental influences were found for RAN, but they were not associated with word identification or phonological decoding.

These shared environmental effects are somewhat of a departure from previous studies of reading, which generally show genetic overlap with little evidence for shared environmental overlap (e.g., see Davis et al., 2001; Gayan & Olson, 2003). However, the importance of shared environment in the overlap among early reading skills is consistent with our own univariate examination of WRRP (Petrill et al., 2006) as well with univariate results reported by Byrne et al. (2002, 2005). Moreover, these results are somewhat consistent with the typically developing sample described by Compton et al. (2001).

However, despite the similarity in the age of the twins who participated in the present study and in Byrne et al.'s (2005) study, there are some discrepancies between the present results and the multivariate results presented by Byrne et al. (2005). In particular, the current study suggested shared environmental variance specific to RAN, whereas Byrne et al. (2005) suggested that shared environmental variance in RAN was related to phonological awareness. Moreover, Byrne et al. (2005) showed evidence for independent genetic effects for RAN, whereas our data do not. This inconsistency could be due to sampling and measurement differences between the two studies. In terms of sampling, Byrne et al. (2005) involved U.S., Australian, and Scandinavian samples, whereas our study examined a U.S. sample only. In addition, Byrne et al. (2005) used a composite of the Comprehensive Test of Phonological Processing's (Wagner et al., 1999) colors and shapes, whereas the current study involved a composite of letters and numbers, corrected for age, sex, and letter identification. Despite these differences, both studies converge on the general idea that shared environment may be more important in early literacy and that genetics are important for the overlap among phonological awareness, RAN, and reading outcomes.

Furthermore, although the age range of the current study is narrow relative to most behavioral genetic studies of reading, and we regressed out the effects of age prior to analysis, the children were not identical in age and grade level. Thus, the children in the current study have different levels of exposure to phonological-based instruction. Because twins are the same age and in the same grade, this constitutes a between-family effect that would be estimated as shared environmental variance. Unfortunately, we do not have detailed information about the curricula used by the twins' schools. However, months of school did not affect the etiology of the relationships among phonological awareness, RAN, Word ID, and PD skills.

Another shortcoming of the current study is that although the sample was heterogeneous in terms of the mothers' education levels and has been shown to be heterogeneous in terms of the home literacy environment (Petrill & Deater-Deckard, 2005), the sample was not heterogeneous in terms of ethnicity (91% White) or family composition (96% married or cohabiting). Thus, although our sample is more representative in terms of the range of environment than most behavioral genetic studies, the results of the current study must still be interpreted in light of the restriction of range in ethnicity and family composition.

Despite these issues, it is possible that the shared environmental influences found in the current study reflect a true developmental effect. Research examining environmental predictors of early literacy suggest that reading-related knowledge and skills that children acquire via the home environment are associated with early reading success (McCardle, Scarborough, & Catts, 2001) but that the indices of the home environment that influence early reading are no longer influential among older readers (Scarborough & Dobrich, 1994). Emerging longitudinal genetic evidence pointing to the dissipation of shared environmental influences from pre-school to kindergarten also supports this possibility (Bryne et al., 2005).

More generally, one long-standing axiom of reading development is that the main requirements of successfully learning to read in young children are phonological awareness, orthography, and visual–analytic ability (see Dale & Crain-Thoreson, 1999). As reading skills mature, children use reading to learn new words and to integrate these words into developing semantic knowledge (Chall, 1983). Therefore, it is sensible that shared environmental influences may be greater for outcomes that are more likely to be influenced by direct instruction in the home, such as expressive vocabulary, phonological awareness, or print knowledge. As longitudinal data in the WRRP become available, we will be able to address two additional issues. First, we will examine the extent to which genes and environments influence the stability and instability of reading skills over time. Second, we will examine whether measured environmental influences on early reading shift from a shared environmental to a genetic etiology as children learn to read and as the environments associated with reading become more a function of their own reading skills.

Taken together, Compton et al. (2001), Byrne et al. (2005), and the current study all converge on the importance of a core set of genes common to phonological awareness, RAN, and reading outcomes. However, these three studies also provide support for the independent effects related to RAN and phonology. In our own data, regression analyses suggested that RAN predicted significant variance in Word ID and PD. Moreover, behavioral genetic analyses suggested that phonological awareness was correlated with Word ID and PD via shared environmental pathways independent of RAN. What remains to be established is whether the important skills that are represented by naming speed performance lie outside of the phonological processing domain.

Wagner, Torgesen, Laughon, Simmons, and Rashotte (1993) identified five correlated but separable constructs that constitute the domain of phonological processing: analysis, synthesis, memory, isolated naming, and serial naming. Subsequent work by Schatschneider et al. (1999) demonstrated that the analysis and synthesis constructs should be combined into a singe factor described previously in the literature as phonological awareness. On the one hand, the significance of a core set of genes common to phonology, RAN, and reading outcomes provides support for this unitary model of phonological processing. On the other hand, in our data, naming speed predicted variance in reading above and beyond phonological awareness, and phonological awareness was correlated with reading outcomes through an additional shared environmental factor. Although this does not necessarily mean that the skills being tapped by naming speed that relate to reading lie outside the domain of phonological processing, that serial naming is phenotypically separable from phonological awareness and could constitute a second, etiologically distinct source of variance in reading skills.

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References

Bradley L, Bryant PE. Categorizing sounds and learning to read: A causal connection. Nature 1983 February 3;301:419–421.

Byrne B, Delaland C, Fielding-Barnsley R, Quain P, Samuelsson S, Hoien T, et al. Longitudinal twin study of early reading development in three countries: Preliminary results. Annals of Dyslexia 2002;52:49–74.

Byrne B, Wadsworth S, Corley R, Samuelsson S, Quain P, DeFries JC, et al. Longitudinal twin study of early literacy development: Preschool and kindergarten phases. Scientific Studies of Reading 2005;9:219–236.

Cardon LR, DeFries JC, Fulker DW, Kimberling WJ, Pennington BF, Smith SD. Quantitative trait locus for reading disability on chromosome 6. Science 1994 October 14;266:276–279. [PubMed: 7939663]

Catts, HW.; Hogan, TP.; Adlof, SM. Developmental changes in reading and reading disabilities. In: Catts, HW.; Kamhi, AG., editors. The connections between language and reading disabilities. Mahwah, NJ: Erlbaum; 2005. p. 25-40.

Chall, JS. Stages of reading development. New York: McGraw-Hill; 1983.

Compton, DL.; Davis, CJ.; DeFries, JC.; Gayan, J.; Olson, RK. Genetic and environmental influences on reading and RAN: An overview of results from the Colorado Twin Study. In: Wolf, M., editor. Conference Proceedings of the Dyslexia Research Foundation Conference in Extraordinary Brain Series: Time, fluency, and developmental dyslexia. Baltimore: York Press; 2001. p. 277-303.

Dale PS, Crain-Thoreson C. Language and literacy in a developmental perspective. Journal of Behavioral Education 1999;9:23–33.

Davis CJ, Knopik VS, Olson RK, Wadsworth SJ, DeFries JC. Genetic and environmental influences on rapid naming and reading ability: A twin study. Annals of Dyslexia 2001;51:231–247.

Fagerheim T, Raeymaekers P, Tonnessen FE, Pedersen M, Tranebjaerg L, Lubs HA. A new gene (DYX3) for dyslexia is located on chromosome 2. Journal of Medical Genetics 1999;36:664–669. [PubMed: 10507721]

Fisher SE, Francks C, Marlow AJ, MacPhie IL, Newbury DF, Cardon L, et al. Independent genome-wide scans identify a chromosome 18 quantitative-trait locus influencing dyslexia. Nature Genetics 2002;30:86–91. [PubMed: 11743577]

Fisher SE, Marlow AJ, Lamb J, Maestrini E, Williams DF, Richardson A, et al. A quantitative-trait locus on chromosome 6p influences different aspects of developmental dyslexia. American Journal of Human Genetics 1999;64:146–156. [PubMed: 9915953]

Gayan J, Olson RK. Genetic and environmental influences on orthographic and phonological skills in children with reading disabilities. Developmental Neuropsychology 2001;20:487–511.

Gayan J, Olson RK. Genetic and environmental influences on individual differences in printed word recognition. Journal of Experimental Child Psychology 2003;84:97–123. [PubMed: 12609495]

Gayan J, Smith SD, Cherny SS, Cardon LR, Fulker DW, Brower AM, et al. Quantitative-trait locus for specific language and reading deficits on chromosome 6p. American Journal of Human Genetics 1999;64:157–164. [PubMed: 9915954]

Grigorenko EL. The first candidate gene for dyslexia: Turning the page of a new chapter of research. Proceedings of the National Academy of Sciences, USA 2003;100:11190–11192.

Grigorenko EL, Wood FB, Meyer MS, Hart LA, Speed WC, Shuster A, Pauls DL. Susceptibility loci for distinct components of developmental dyslexia on chromosome 6 and 15. American Journal of Human Genetics 1997;60:27–39. [PubMed: 8981944]

Grigorenko EL, Wood FB, Meyer MS, Pauls DL. Chromosome 6p influences on different dyslexia-related cognitive processes: Further confirmation. American Journal of Human Genetics 2000;66:715–723. [PubMed: 10677331]

Knopik VS, Alarcón M, DeFries JC. Common and specific gender influences on individual differences in reading performance: A twin study. Personality and Individual Differences 1998;25:269–277.

Lyon GR, Shaywitz SE, Shaywitz BA. A definition of dyslexia. Annals of Dyslexia 2003;53:1–14.

McCardle P, Scarborough HS, Catts HW. Predicting, explaining, and preventing children's reading difficulties. Learning Disabilities Research and Practice 2001;16:230–239.

Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. Mx: Statistical modeling. Vol. 5. Department of Psychiatry; Medical College of Virginia, Box 126 MCV, Richmond, VA 23298: 1999.

Neale, M.; Cardon, LR. Methodology for genetic studies of twins and families. Dordrecht, the Netherlands: Kluwer; 1992.

Olson RK, Gillis JJ, Rack JP, DeFries JC, Fulker DW. Confirmatory factor analysis of word recognition and process measures in the Colorado Reading Project. Reading and Writing 1991;3:235–248.

Pennington BF, Smith SD. Genetic influences on learning disabilities and speech and language disorders. Child Development 1983;54:369–387. [PubMed: 6347551]

Petrill SA, Deater-Deckard K. Task orientation, parental warmth and SES account for a significant proportion of the shared environmental variance in general cognitive ability in early childhood: Evidence from a twin study. Developmental Science 2004;7:25–32. [PubMed: 15323115]

Petrill SA, Deater-Deckard K, Thompson LA, Schatschneider C. Reading skills in early readers: Genetic and shared environmental influences. Journal of Learning Disabilities 2006;39:48–55. [PubMed: 16512082]

Petrill SA, Pike A, Price T, Plomin R. Chaos in the home and socioeconomic status are associated with cognitive development in early childhood: Environmental risks identified in a genetic design. Intelligence 2004;32:445–460.

Robertson, C.; Salter, W. The Phonological Awareness Test. East Moline, IL: LinguiSystems; 1997.

Scarborough HS, Dobrich W. On the efficacy of reading to preschoolers. Developmental Review 1994;14:245–302.

Schatschneider C, Francis DJ, Foorman BF, Fletcher JM, Mehta P. The dimensionality of phonological awareness: An application of item response theory. Journal of Educational Psychology 1999;91:467–478.

Stanovich KE, Siegel LS. Phenotypic performance profile of children with reading disabilities: A regression-based test of the phonological-core variable-difference-model. Journal of Educational Psychology 1994;86:24–53.

Stevenson J, Graham P, Fredman G, McLoughlin V. A twin study of genetic influences on reading and spelling ability and disability. Journal of Child Psychology and Psychiatry 1987;28:229–247. [PubMed: 3584294]

Swanson HL, Trainin G, Necoechea DM, Hammill DD. Rapid naming, phonological awareness, and reading: A meta-analysis of the correlation evidence. Review of Educational Research 2003;73:407–440.

Torgesen JK, Wagner RK, Rashotte CA, Burgess S, Hecht S. Contributions of phonological awareness and rapid automatic naming to the growth of word-reading skills in second to fifth-grade children. Scientific Studies of Reading 1997;1:161–155.

Wagner RK, Torgesen JK, Laughon P, Simmons K, Rashotte CA. Development of young readers' phonological processing abilities. Journal of Educational Psychology 1993;85:83–103.

Wagner, RK.; Torgesen, JK.; Rashotte, CA. Comprehensive Test of Phonological Processing. Austin, TX: PRO-ED; 1999.

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Wolf M, Bowers PG. The double deficit hypothesis for the developmental dyslexias. Journal of Educational Psychology 1999;91:415–438.

Woodcock, R. Woodcock Reading Mastery Tests—Revised. Circle Pines, MN: American Guidance Service; 1987.

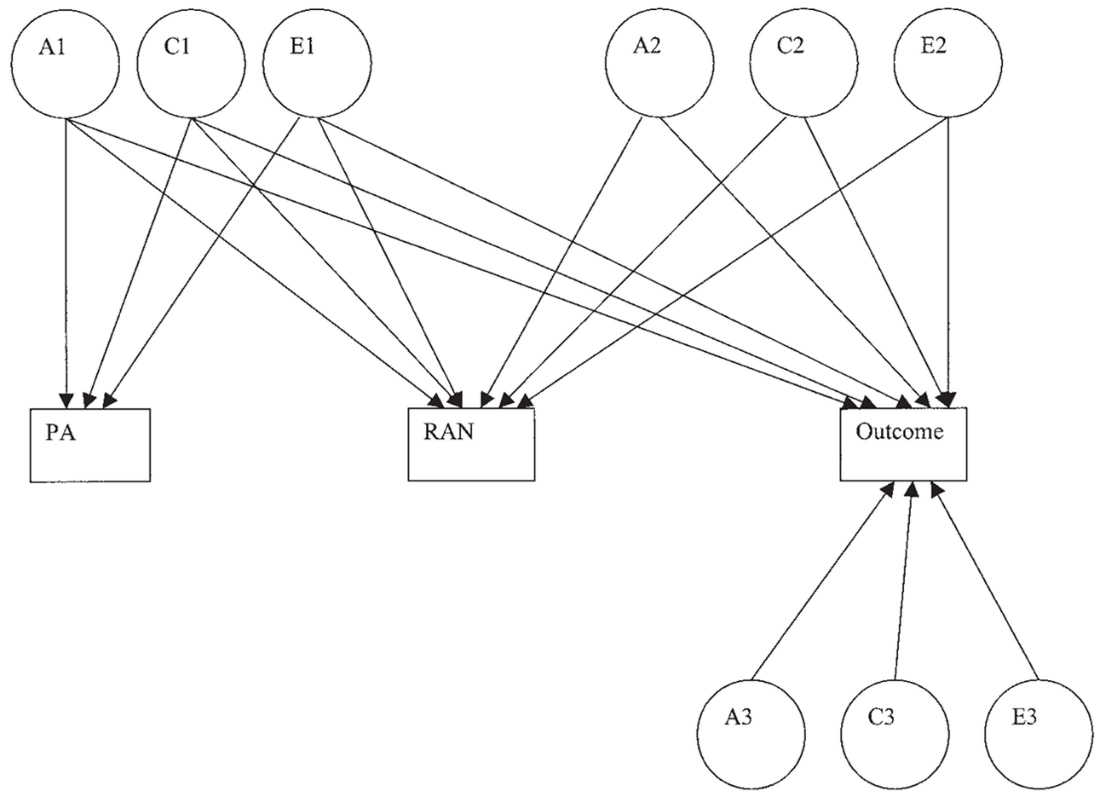

Figure 1.
General Cholesky model examining phonological awareness (PA), rapid automatized naming (RAN), and reading outcomes. Outcomes were letter identification, word identification, and phonological decoding, conducted in separate analyses. A1–A3, C1–C3, and E1–E3 refer to genetic (A), shared environmental (C), and nonshared environmental (E) factors.

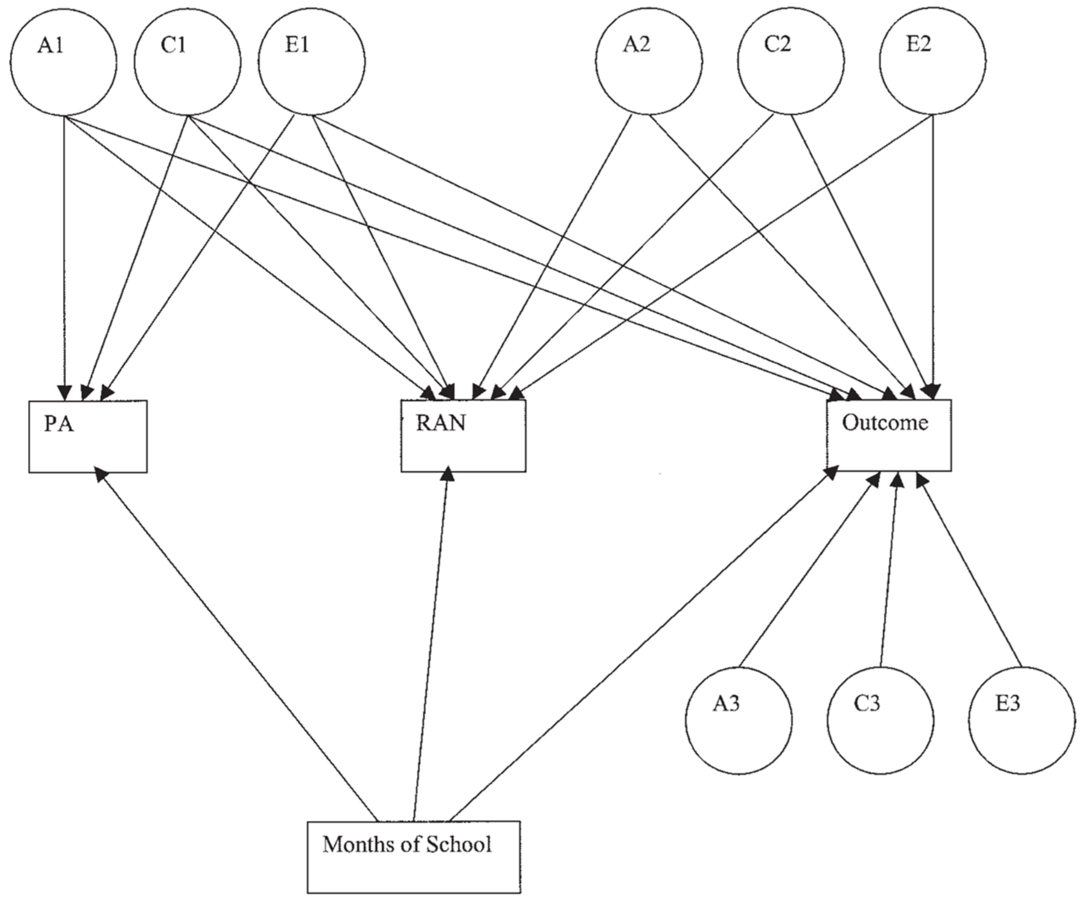

Figure 2.
General Cholesky model examining months of school, phonological awareness (PA), rapid automatized naming (RAN), and reading outcomes. Outcomes were letter identification, word identification, and phonological decoding, conducted in separate analyses. A1–A3, C1–C3, and E1–E3 refer to genetic (A), shared environmental (C), and nonshared environmental (E) factors.

Table 1

Descriptive Statistics and Sibling Intraclass Correlations Among Monozygotic (MZ) and Dizygotic (DZ) Twins

	MZ		DZ		MZ	DZ
Variable	***M***	***SD***	***M***	***SD***	***r***	***r***
Phonological awareness	36.2	13.3	34.8	13.1	.85	.51
RAN	164.2	61.4	166.1	66.2	.55	.37
Letter ID	102.9	9.0	102.2	8.7	.74	.57
Word ID	103.6	18.3	104.7	18.6	.89	.57
Phonological decoding	103.4	12.9	103.3	11.9	.82	.53
Months of school	6.8	6.7	6.7	5.5		

Note. Phonological awareness is expressed in number of correct items (total possible correct = six subtests, 10 items each = 60), whereas rapid automatized naming (RAN) is expressed in number of seconds to complete both letter and number subtests of the Comprehensive Test of Phonological Processing. Phonological awareness was residualized for age and gender and RAN for age, gender, and letter identification for all additional analyses. Letter ID and Word ID refer to the Letter Identification and Word Identification subtests of the Woodcock Reading Mastery Tests—Revised.

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Table 2

Intercorrelations Among Phonological Awareness (PA), Rapid Automatized Naming (RAN), Letter Identification (Letter ID), Word Identification (Word ID), Phonological Decoding (PD), and Months of School (MSCHOOL)

Variable	1	2	3	4	5	6
1. PA	—					
2. RAN	.20*	—				
3. Letter ID	.52*	.00	—			
4. Word ID	.52*	.23*	.56*	—		
5. PD	.59*	.26*	.42*	.70*	—	
6. MSCHOOL	.07	.06	−.07	.37*	.28*	—

Note. The correlation between RAN and Letter ID was .47, $p < .05$, when RAN was residualized for age and gender only.

* $p < .01$.

Table 3

Multiple Regression Analyses

Variable	***β***	***sr***	***t***	***df***
Word ID[a]				
Phonological awareness	.47	.46	11.70*	474
RAN	.14	.14	3.55*	474
Phonological decoding[b]				
Phonological awareness	.56	.55	15.11*	478
RAN	.15	.14	3.95*	478

Note. Word ID = the Word Identification subtest of the Woodcock Reading Mastery Tests—Revised; RAN = rapid automatized naming.

[a] $F(473) = 85.79$, $p < .01$, $R^2 = .27$.

[b] $F(477) = 139.08$, $p < .01$, $R^2 = .37$.

* $p < .01$.

Table 4

Phenotypically Standardized Genetic, Shared Environmental, and Nonshared Environmental Variance/Covariance Matrices: Phonological Awareness (PA), Rapid Automatized Naming (RAN), and Word Identification (Word ID)

	Months of school not included[a]				Months of school included[b]		
Variable	**PA**	**RAN**	**Word ID**	**Variable**	**PA**	**RAN**	**Word ID**
Genetic				**Genetic**			
PA	h^2 = .61*			PA	h^2 = .59*		
RAN	.53*	h^2 = .46*		RAN	.52*	h^2 = .46*	
Word ID	.24*	.21*	h^2 = .59*	Word ID	.26*	.22*	h^2 = .57*
Shared environment				**Shared environment**			
PA	c^2 = .27*			PA	c^2 = .27*		
RAN	.00	c^2 = .24*		RAN	.00	c^2 = .23*	
Word ID	.27*	.04	c^2 = .31*	Word ID	.22*	.04	c^2 = .18*
Nonshared environment				**Nonshared environment**			
PA	e^2 = .12*			PA	e^2 = .13*		
RAN	.08	e^2 = .30*		RAN	.08	e^2 = .30*	
Word ID	.02	.01	e^2 = .10*	Word ID	.02	.01	e^2 = .10*
Months of school				**Months of school**			
PA	—			PA	sch^2 = .01		
RAN	—	—		RAN	.01	sch^2 = .01	
Word ID	—	—	—	Word ID	.04	.03	sch^2 = .15*

Note. Asterisks indicate statistically significant values (using 95% confidence intervals in Mx). h^2 = heritability; c^2 = shared environment; e^2 = nonshared environment; *sch* = months of school.

[a]See Figure 1.

[b]See Figure 2.

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Table 5

Phenotypically Standardized Genetic, Shared Environmental, and Nonshared Environmental Variance/Covariance Matrices: Phonological Awareness (PA), Rapid Automatized Naming (RAN), and Phonological Decoding (PD)

	Months of school not included[a]				Months of school included[b]		
Variable	**PA**	**RAN**	**PD**	**Variable**	**PA**	**RAN**	**PD**
Genetic				**Genetic**			
PA	h^2 = .60*			PA	h^2 = .59*		
RAN	.53*	h^2 = .47*		RAN	.52*	h^2 = .46*	
PD	.37*	.33*	h^2 = .46*	PD	.37*	.33*	h^2 = .47*
Shared environment				**Shared environment**			
Page 1 of 1	c^2 = .27*			PA	c^2 = .27*		
RAN	.00	c^2 = .23*		RAN	.00	c^2 = .23*	
PD	.21*	.04	c^2 = .35*	PD	.18*	.00	c^2 = .23*
Nonshared environment				**Nonshared environment**			
PA	e^2 = .13*			PA	e^2 = .13*		
RAN	.08	e^2 = .30*		RAN	.08	e^2 = .30*	
PD	.03	.02	e^2 = .19*	PD	.03	.02	e^2 = .19*
Months of school				**Months of school**			
PA	—			PA	sch^2 = .01		
RAN	—	—		RAN	.01	sch^2 = .01	
PD	—	—	—	PD	.03	.03	sch^2 = .11*

Note. Asterisks indicate statistically significant values (using 95% confidence intervals in Mx). h^2 = heritability; c^2 = shared environment; e^2 = nonshared environment; *sch* = months of school.

[a] See Figure 1.

[b] See Figure 2.

Table 6

Model-Fitting Results: Cholesky Decomposition

Model	−2 log likelihood	*df*	χ^2 change	df_{change}	p_{change}
PA/RAN/Word ID					
Full	3,392.34	1450			
Genetic					
Drop GenWID (A1 to WID)	3,410.71	1451	18.37	1	<.01
Drop RAN (all A2)	3,392.34	1452	0.00	2	
Drop RANWID (A2 to WID)	3,392.34	1451	0.00	1	
Drop WIDresid (A3)	3,392.34	1451	0.00	1	
Shared environment					
Drop GenWID (C1 to WID)	3,409.62	1451	17.28	1	<.01
Drop RAN (all C2)	3,404.84	1452	12.50	2	<.01
Drop RANWID (C2 to WID)	3,392.74	1451	0.40	1	
Drop WIDresid (C3)	3,392.38	1451	0.04	1	
PA/RAN/PD					
Full	3,397.41	1454			
Genetic					
Drop GenPD (A1 to PD)	3,427.22	1455	29.81	1	<.01
Drop RAN (all A2)	3,397.41	1456	0.00	2	
Drop RANPD (A2 to PD)	3,397.41	1455	0.00	1	
Drop PDresid (A3)	3,397.41	1455	0.00	1	
Shared environment					
Drop GenPD (C1 to PD)	3,406.36	1455	8.95	1	<.01
Drop RAN (all C2)	3,408.98	1456	11.57	2	<.01
Drop RANPD (C2 to PD)	3,397.77	1455	0.36	1	
Drop PDresid (C3)	3,399.86	1455	2.45	1	

Note. Submodels were not run for nonshared environment because none of the bivariate relationships presented in Tables 4 and 5 was significant. A1, A2, A3, C1, C2, C3 refer to factors displayed in Figure 1. PA = phonological awareness; RAN = rapid automatized naming; Word ID (or WID) = word identification; Gen = genetic; PD = phonological decoding; resid = residual.

Table 7

Model-Fitting Results: Cholesky Decomposition With Months of School

Model	−2 log likelihood	*df*	χ^2 change	df_{change}	p_{change}
		PA/RAN/Word ID			
Full	4,022.65	1688			
Genetic					
Drop GenWID (A1 to WID)	4,044.24	1689	21.59	1	<.01
Drop RAN (all A2)	4,022.65	1690	0.00	2	
Drop RANWID (A2 to WID)	4,022.65	1689	0.00	1	
Drop WIDresid (A3)	4,022.65	1689	0.00	1	
Shared environment					
Drop GenWID (C1 to WID)	4,038.99	1689	16.34	1	<.01
Drop RAN (all C2)	4,034.45	1690	11.80	2	<.01
Drop RANWID (C2 to WID)	4,022.65	1689	0.00	1	
Drop WIDresid (C3)	4,022.65	1689	0.00	1	
Months of school					
Drop PA	4,025.29	1689	2.64	1	
Drop RAN	4,024.66	1689	2.01	1	
Drop WID	4,064.97	1689	42.32	1	<.01
		PA/RAN/PD			
Full	4,040.61	1692			
Genetic					
Drop GenPD (A1 to PD)	4,071.87	1694	31.26	1	<.01
Drop RAN (all A2)	4,040.61	1693	0.00	2	
Drop RANPD (A2 to PD)	4,040.61	1693	0.00	1	
Drop PDresid (A3)	4,040.61	1693	0.00	1	
Shared environment					
Drop GenPD (C1 to PD)	4,049.21	1694	8.60	1	<.01
Drop RAN (all C2)	4,052.19	1693	11.58	2	<.01
Drop RANPD (C2 to PD)	4,040.61	1693	0.00	1	
Drop PDresid (C3)	4,042.18	1693	1.57	1	
Months of school					

Model	−2 log likelihood	*df*	χ^2 change	df_{change}	p_{change}
Drop PA	4,042.91	1693	2.30	1	
Drop RAN	4,043.00	1693	2.39	1	
Drop WID	4,072.09	1693	31.48	1	<.01

Note. Submodels were not run for nonshared environment because none of the bivariate relationships presented in Tables 4 and 5 was significant. A1, A2, A3, C1, C2, C3, and months of school refer to factors/variables displayed in Figure 2. PA = phonological awareness; RAN = rapid automatized naming; Word ID (or WID) = word identification; Gen = genetic; PD = phonological decoding; resid = residual.