

# Word reading and reading comprehension: stability, overlap and independence

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**Abstract** Longitudinal twin data were analyzed to investigate the etiology of the stability of genetic and environmental influences on word reading and reading comprehension, as well as the stability of those influences on their relationship. Participating twin pairs were initially tested at a mean age of 10.3 years, and retested approximately five years later. Both word reading and comprehension were found to be highly stable, and genetic influences were primarily responsible for that stability. In contrast to studies with younger participants, no unique genetic influences were observed at follow-up testing in this older sample. High genetic correlations were obtained between word reading and reading comprehension at both ages, indicating common genetic influences. However, significant genetic influence on comprehension was also observed, independent of that on word reading. Although the phenotypic relation between the two measures appeared to decline across time, the genetic etiology of this relation was highly stable.

**Keywords** Behavior genetic · Etiology · Longitudinal · Reading comprehension · Stability · Word reading

## Introduction

A number of studies have assessed the stability of performance on word reading (e.g., Raskind, Gerber, Goldberg, Higgins, & Herman, 1998; Scarborough, 1998, 2005; Shaywitz et al., 1999; Sicol, 2002; Torgesen & Burgess, 1998; Torgesen, Wagner, Rashotte, Burgess, & Hecht, 1997) and reading comprehension measures

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(e.g., De Jong & van der Leij, 2002; Seigneuric & Ehrlich, 2005; Torgesen et al., 1997) with stability correlations ranging from .27 to .62 for word reading and .57 to .87 for reading comprehension. However, relatively few studies have investigated the etiologies of stability, and there is a paucity of research using data from measures of comprehension obtained from older children.

Studies with genetically informative designs, such as twin or adoption studies, facilitate an assessment of the genetic, shared environmental, and nonshared environmental contributions to etiology. The classic twin design compares the similarity within identical, or monozygotic (MZ) twin pairs who share 100% of their genes with that of fraternal, or dizygotic (DZ) twin pairs, who share on average 50% of their segregating genes (Plomin, DeFries, McClearn, & McGuffin, 2001). To the extent that individual differences in a trait are caused by genetic influences, MZ twins should be more similar than DZ twins. Shared environmental influences are those factors which make twins pairs more similar to each other, such as having a common family or school environment. To the extent that the DZ twin correlation is more than half the MZ twin correlation, shared environmental influences are implicated. In contrast, nonshared environment independently influences members of twin pairs, such as one twin having an illness or accident that the other did not have, and also typically includes measurement error. An extension of this logic facilitates assessment of the etiology of *stability* by comparing the cross-time point similarity of members of MZ and DZ twin pairs. For example, genetic influences on the stability of a measure are suggested if the cross-time point MZ correlation (between one twin's score at the initial assessment and the other twin's score at follow-up) is greater than the cross-time point DZ correlation.

There is substantial evidence for genetic influences on reading skills (see Fisher & DeFries, 2002; Olson, 2004; Pennington & Olson, 2005, for reviews). Univariate heritability estimates for word reading skill and related reading components range from .30 to .85 (e.g., Byrne et al., 2005a; DeFries et al., 1997; Gayán & Olson, 2003; Harlaar, Spinath, Dale, & Plomin, 2005; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, 2006a; Stevenson, Graham, Fredman, & McLoughlin, 1987; Tiu, Wadsworth, Olson, & DeFries, 2004; Wadsworth, Corley, Hewitt, & DeFries, 2001; Wadsworth, Corley, Plomin, Hewitt, & DeFries, 2006). While word reading and its related components are most often studied, some researchers have also assessed genetic influences on reading comprehension and have noted similar heritability estimates, in the range of .41 to .76 (e.g., Byrne et al., 2005b; Harlaar, Dale, & Plomin, 2007; Keenan, Betjemann, Wadsworth, DeFries, & Olson, 2006; Petrill, Deater-Deckard, Thompson, Schatschneider, DeThorne, & Vandenberg, 2007). Given that word reading and comprehension skills are both stable and heritable, we hypothesize that this stability may be due to genetic influences.

Previous research suggests that changes in the etiology of word reading may level off by the middle-elementary school years, but we know of only one study to assess this beyond middle childhood (Wadsworth et al., 2001, 2006). Moreover, the emphasis on comprehension in reading and reading instruction increases around the middle-elementary school years, when the demands of reading transition from “learning to read” to “reading to learn” (e.g., Catts, Hogan, & Adlof, 2005; Chall, 1983; Harlaar et al., 2007; Leach, Scarborough, & Rescorla, 2003), making it

especially important to include data from older children in the assessment of the etiology of its stability. Despite this, no previous studies have assessed longitudinal etiology of comprehension using genetically informative data from children beyond the second grade (Petrill et al., 2007). Thus, in the current study we explore the etiologies of stability of both word reading and reading comprehension in older children tested at 5–6-year intervals, and assess how the relationship between these skills may change over time.

### Etiology of the stability of reading

There have been only a few projects investigating the longitudinal stability of genetic influences on reading. The Longitudinal Twin Study of Early Reading Development (LTS; see Byrne et al., 2002 for overview) has been studying pre-reading and reading skills developmentally, so far reporting on over 600 pairs of children tested in preschool and kindergarten. The Western Reserve Reading Project (WRRP; Petrill et al., 2007) has investigated reading measures in 350 twin pairs who are slightly older, but still in the very early stages of reading development, testing children through the second grade. Both of these studies have found shared genetic influences between time points, as well as genetic influences unique to the second assessment, for component measures of reading. Byrne et al. (2005a, 2006) obtained mixed evidence for shared environmental influences, but when significant, shared environmental influences appeared to be the same over time. Petrill et al., noted significant shared environmental effects for most reading measures, with the exception of comprehension. However, in both projects, most of the continuity over time appears to be accounted for by genetic influences.

In both the LTS and WRRP projects, the children tested have been very young, in an effort to investigate the very beginning stages of reading development. Two other longitudinal studies have investigated this genetic etiology in older groups of children. Harlaar et al. (2007) assessed reading skill at ages 7, 9, and 10 among 4291 twin pairs participating in the Twins Early Development Study (TEDS; Trouton, Spinath, & Plomin, 2002). Consistent with the studies of younger children, they found common genetic influences between ages for reading scores, but also noted unique genetic effects at each age. While most of the phenotypic stability was again due to genetic influences, they did find a single shared environmental factor that was significant and consistent over time.

Finally, Wadsworth and colleagues reported on data from an even older sample, the Colorado Adoption Project (CAP; Wadsworth et al., 2001, 2006; Wadsworth, Fulker, & DeFries, 1999), assessing the stability of word recognition scores in related and unrelated sibling pairs at ages 7, 12, and 16. In contrast to the other longitudinal studies with younger participants, they found no new genetic influences at ages 12 or 16 for word reading; instead they noted a single genetic factor common to all three time points. While they also found a single shared environmental factor that was common across time points, this factor was negligible; the majority of the observed stability was due to genetic influences.

As a whole, these studies suggest that although new genetic influences for word reading may continue to be expressed through middle childhood, as children get

older, genetic influences may become more stable. However, most of these longitudinal results reported thus far have investigated word reading performance and related skills, rather than higher level comprehension.

### Etiology of the stability of reading comprehension

As reading skills develop, the demands of reading may gradually shift, requiring more complicated comprehension processes as children get older (e.g., Catts et al., 2005; Chall, 1983; Curtis, 1980; Harlaar et al., 2007; Leach et al., 2003). It is also possible that the etiology of this development could continue to change. However, to our knowledge, the etiology of the stability of comprehension has only been studied in children through the second grade (Petrill et al., 2007), so there is no prior evidence as to whether there are indeed changes in the etiology of comprehension at later ages. Thus, the current study assesses the stability of both reading comprehension and word reading in these later years of development.

### Relation between word reading and comprehension

In addition to studying stability across time within the same measure, we are also interested in investigating the etiology of the relation between the measures and whether these influences change over time. Results of several studies suggest a core set of genetic influences common to many components of reading (e.g., Davis, Knopik, Olson, Wadsworth, & DeFries, 2001; Gayán & Olson, 2001, 2003; Petrill, Deater-Deckard, Thompson, DeThorne, & Schatschneider, 2006b; Tiu et al., 2004). However, some studies have also found patterns of independent genetic effects for certain measures (Gayán & Olson, 2003; Petrill et al., 2007).

Most of these studies have examined the relations between lower-level components of word reading. Few studies have investigated common and independent genetic and environmental influences on word reading and higher-level comprehension processes. Results of phenotypic studies have indicated that reading comprehension and word reading skills are highly correlated (e.g., Catts, Fey, Zhang, & Tomblin, 1999; Gough, Hoover, & Peterson, 1996; Leach et al., 2003). But, as the “Simple View” of reading proposes (Gough et al., 1996; Gough & Tunmer, 1986; Hoover & Gough, 1990), there is evidence for their independence, as well. For example, some studies have found children who have comprehension deficits with normal word reading skills (Cain, Oakhill, & Bryant, 2000; Catts, Hogan, & Fey, 2003; Nation, 2005; Oakhill, Cain, & Bryant, 2003); others have found children with normal reading comprehension but selective word reading deficits (Leach et al., 2003). The results of a behavioral genetic study by Keenan, Betjemann, Wadsworth et al. (2006) also support the partial independence of word reading and comprehension skills. Keenan and colleagues found a genetic factor common to comprehension and word reading, but also found a second significant genetic factor for comprehension, independent from word reading.

## Stability of relationship between measures

We are interested in investigating not only the similarity of these measures within a time point, but also the stability of this relationship. As discussed above, the etiology of either measure may change across time; the etiology of their relationship may also change. Keenan, Betjemann, and Olson (2006, submitted) present cross-sectional evidence that within reading comprehension measures, the amount of phenotypic variance accounted for by performance on tests of listening comprehension and word reading tends to shift over time. They demonstrate that in younger children the reading comprehension tasks are more heavily dependent on word reading skill, while in older children the same tasks measure an increasing amount of comprehension skill that is independent of word reading (see also Johnston & Kirby, 2006; Gough et al., 1996; Hoover & Gough, 1990). This suggests that there may be a developmental shift of focus from word reading to comprehension as children get older. To our knowledge only one study has attempted to assess what might account for this shift within a genetic framework. Harlaar et al. (2007) analyzed data pertaining to general reading ability at age 7 and comprehension at age 10, and found independent genetic influences on comprehension at the latter assessment. However, since they did not use the same measures at both ages, it is impossible to determine if the unique genetic influences observed are related to differences in the tests, or to developmental differences in reading, comprehension, or both.

In the current study, we conducted a preliminary assessment of the stability of word reading and reading comprehension, as well as the stability of the relation between the two measures, among twin pairs participating in the Colorado Longitudinal Twin Study of Reading Disability (Wadsworth, DeFries, Olson, & Willcutt, submitted), a follow-up of participants in the Colorado Learning Disabilities Research Center (DeFries et al., 1997). If reading comprehension skill is more dependent on word reading in younger children, then we may see greater etiological overlap across measures at initial testing than at follow-up testing. Using data from twins allows us to partition the phenotypic variance into genetic, shared environmental, and nonshared environmental contributions. Thus, we can assess the etiologies of the stability of word reading and comprehension, as well as the etiology of the stability of the relationship between the two measures.

## Method

### Participants

Participants were tested at two time points. All participants were initially tested in the Colorado Learning Disabilities Research Center (CLDRC; see DeFries et al., 1997; Olson, 2004). Twins were identified from school records from 27 different Colorado school districts. One group of twin pairs (RD pairs) was invited to participate if one or both twins were identified by school records or parent report to have a school history of reading problems or Attention Deficit-Hyperactivity Disorder (ADHD; twin pairs who were recruited as having ADHD but no history of

reading problems in either member of the pair were not included in the current analyses.) A comparison group of twin pairs with no history of reading problems was also invited to participate (control pairs). For this initial assessment, twins completed a day-and-a-half of testing at the University of Colorado at Boulder and the University of Denver.

Twin pairs initially tested between 1996 and 2001 were then recruited for follow-up testing approximately five to six years later, as part of the ongoing Colorado Longitudinal Twin Study of Reading Disability (LTSRD; see Wadsworth et al., submitted, for details on this study). Twin pairs agreeing to participate completed a full day of testing at the Institute for Behavioral Genetics at the University of Colorado at Boulder. The test battery includes some of the most diagnostically revealing tests administered in the CLDRC; thus, subjects are retested using the same measures (or updated revisions where available) they were administered at their initial assessment.

Approximately 55% of those recontacted have participated in follow-up testing so far. Although retested participants scored better on the variables used here at initial testing than those who have not participated in follow-up testing, the variances and covariances did not differ significantly by retest status, nor did results of genetic analyses. Thus, there is no reason to believe that the current sample is biased for the genetic analyses. The current analyses include data from 59 MZ twin pairs, 57 same-sex DZ twin pairs, and 41 opposite-sex DZ pairs. The ages at initial testing ranged from 8.0 to 15.9 years (mean age = 10.3), and the ages at follow-up testing ranged from 12.9 to 23.9 (mean age = 15.8; 44.9% male, and 55.1% female). All twins spoke English as their first language.

## Procedure and measures

The full test battery at each time point included measures of general cognitive ability, reading and component skills, and behavioral measures (see Wadsworth et al., submitted, for a description of the full test battery.) The measures used in the current analyses are as follows:

### *Word reading*

Word reading performance was measured using the Reading Recognition subtest of the Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970) at initial testing, and the PIAT-R at follow-up (Markwardt, 1998). The original PIAT and the PIAT-R correlate very highly, at .88 (Markwardt, 1998). In this untimed subtest, participants read a list of increasingly difficult words until they answer five of the last seven items incorrectly. Scores are computed as the number of items correct.

### *Reading comprehension*

Reading comprehension performance was measured using the Reading Comprehension subtest of the PIAT at initial testing (Dunn & Markwardt, 1970), and the PIAT-R at follow-up (Markwardt, 1998). The versions correlate .79 on this untimed

**Table 1** PIAT published standard scores by age (with standard deviations), testing point and recruitment group

	Initial testing		Follow-up testing	
	Word reading	Comprehension	Word reading	Comprehension
RD	97.3 (12.2)	100.6 (13.4)	98.2 (14.6)	97.47 (13.4)
Control	110.7 (9.8)	112.5 (11.0)	112.5 (13.9)	110.33 (12.7)

subtest (Markwardt, 1998). Participants read a series of short passages, and after each, they choose the one of four pictures that best represents the meaning of the passage read.

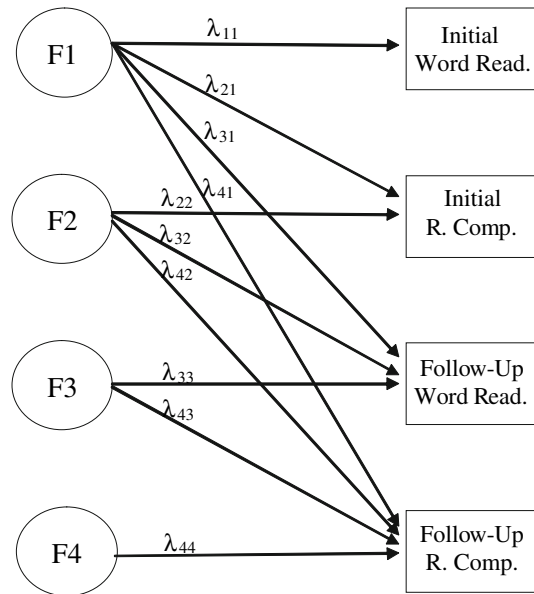
The mean age-based standard scores for each measure at initial and follow-up assessments are presented in Table 1 for RD and control groups separately. The RD group means are approximately one standard deviation below that of the control group for word reading and comprehension, at both assessments ( $z = -.96$  to  $-1.15$ ). The means of the RD pairs are close to the population average on each measure. One reason for this is that pairs were selected because one twin had a history of reading problems, but many of the co-twins did not. The control twin means are approximately one standard deviation above the population mean, but these control pairs are also selected, in that they do not include any pairs with a history of reading difficulty. For analysis of both measures, age-based standard scores were converted to  $z$ -scores standardized against the control sample at each time point, and were then re-standardized within the RD and control groups. The distribution of each variable was then assessed for outliers, defined as scores that fell more than three standard deviations from the mean of each group, and more than 0.5 standard deviations beyond the next most extreme score. No scores met these criteria.

## Analyses

### Phenotypic analyses

Phenotypic relations between variables were assessed by fitting a multivariate Cholesky decomposition model to the data using the Mx statistical modeling package (Neale, Boker, Xie, & Maes, 2002). Scores on all measures approximated a normal distribution, and raw data rather than covariance matrices were used in Mx to allow the inclusion of cases with missing data. We first fit a model with separate solutions for the RD and control groups, and then we tested whether solutions for the two groups could be equated without significant loss of fit.

The model explores the overall phenotypic stability of individual differences in both word reading and reading comprehension at the two time points, as shown in Fig. 1. The model is hierarchical, so that the paths from the second factor are assessing relationships after controlling for the paths from the first factor, etc. The phenotypic correlations between the variables are computed from the model by

**Fig. 1** Phenotypic Cholesky model

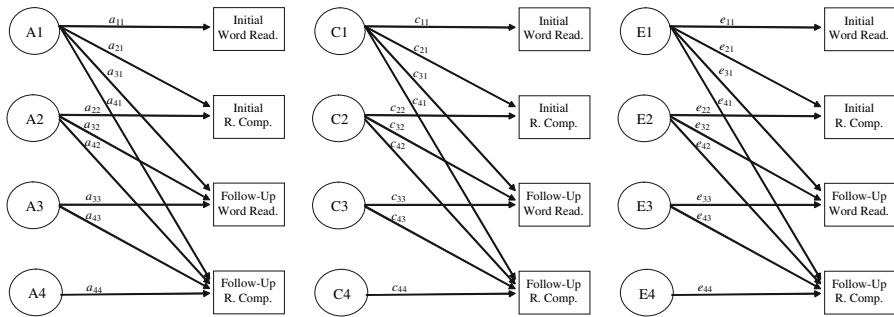
multiplying the paths in a chain connecting the two variables, then summing all of the chains involved. For example, the observed correlation ( $r_r$ ) between initial and follow-up comprehension would be computed as follows:  $(\lambda_{21} \times \lambda_{41}) + (\lambda_{22} \times \lambda_{42})$ . That portion of the correlation between initial and follow-up comprehension that is independent of initial word reading is computed as  $(\lambda_{22} \times \lambda_{42})$ .

### Genetic analyses

Using the twin design, we can determine the univariate genetic effects ( $A$  or  $a^2$ ), shared environmental effects ( $C$  or  $c^2$ ), and nonshared environmental effects ( $E$  or  $e^2$ ) for each individual measure, as described in the introduction. We can also use multivariate behavioral genetic analyses to investigate the genetic and environmental influence on the covariance between the same measure over time, or between different measures (e.g., Neale & Cardon, 1992; Plomin & DeFries, 1979). To accomplish this, the phenotypic Cholesky is further partitioned into genetic, shared environmental, and nonshared environmental factors, as shown in Fig. 2. We first fitted a full ACE model with separate solutions for the RD and control groups. Then, we tested whether solutions for the two groups could be equated without significant loss of fit.

#### *Univariate estimates of genetic and environmental components of variance*

From the standardized path coefficients indicated in Fig. 2, the proportion of variance in word reading and comprehension performance at each testing point due to genetic and environmental influences can be computed by summing the squared paths from the factors to each measure. For example, the heritability ( $a^2$ ; i.e., the proportion of



**Fig. 2** Genetic/environmental Cholesky model

phenotypic variance attributable to genetic influences) of comprehension at initial testing is the square of the path from A1 to initial comprehension ( $a_{21}$ ), plus the square of the path from A2 to initial comprehension ( $a_{22}$ ). Estimates of shared ( $c^2$ ) and nonshared ( $e^2$ ) environmental influences are obtained in an analogous fashion.

*Genetic and environmental correlations*

We can also determine the degree to which the same genetic and environmental influences affect the two different measures or the same measure at different time points. Estimates of these genetic, shared environmental, and nonshared environmental correlations are computed from the standardized paths with relative ease. For example, the genetic correlation between initial and follow-up comprehension is  $[(a_{21} \times a_{41}) + (a_{22} \times a_{42})] / (a_{\text{initial}} \times a_{\text{follow-up}})$ . From the Cholesky, we can also compute the phenotypically standardized covariances, i.e., that part of the observed correlation ( $r_p$ ) due to common genetic or environmental influences. For example, the phenotypically standardized genetic covariance between initial and follow-up comprehension can be computed from the path coefficients in Fig. 2 as  $(a_{21} \times a_{41}) + (a_{22} \times a_{42})$ . Estimates of shared and nonshared environmental correlations and phenotypically standardized correlations are obtained in a similar manner. The three phenotypically standardized covariances should sum to the standardized phenotypic covariance, i.e., the phenotypic correlation,  $r_p$ . Furthermore, the ratio of the phenotypically standardized genetic or environmental covariance to the phenotypic correlation provides an estimate of the proportion of the observed phenotypic correlation due to shared genetic or environmental influences.

**Results**

Phenotypic results

The solutions for the RD and control groups could not be equated without significant loss of fit ( $\Delta \chi^2 = 22.2, \Delta df = 10, p < .05$ ), so separate estimates are presented. However, since the patterns of results for the two groups are very similar, we primarily discuss the pattern of phenotypic results across both groups.

**Table 2** Phenotypic correlations computed from phenotypic Cholesky model: RD group estimates, with control group estimates in parentheses

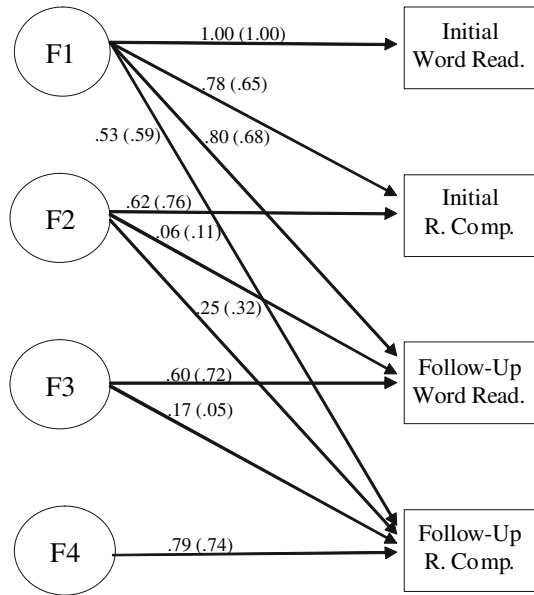
	Initial		Follow-up	
	Word reading	Comprehension	Word reading	Comprehension
Word read.—Initial				
Comp.—Initial	0.78 (0.65)			
Word read.—Follow-up	0.80 (0.68)	0.66 (0.52)		
Comp.—Follow-up	0.53 (0.59)	0.57 (0.63)	0.54 (0.48)	

The phenotypic correlations for both the RD and control groups are shown in Table 2. The measures are highly correlated with one another and are quite stable, with word reading showing somewhat greater stability than reading comprehension. Moreover, the relationship between word reading and reading comprehension appears stronger at the initial assessment than at follow-up (RD: Fisher's  $Z = 3.09$ ,  $p < .01$ ; Control: Fisher's  $Z = 1.30$ ,  $p = .09$ ). Figure 3 presents the standardized path coefficients from the phenotypic Cholesky decomposition, and here we also see high stability of covariance between measures. There is an initial phenotypic factor (F1) that accounts for substantial variance in both measures at both assessments. A second factor (F2) accounts for additional variance in comprehension only, indicated by a significant shared path for reading comprehension (RD:  $p < .001$ , Controls:  $p < .001$ ) and a non-significant path to follow-up word reading (RD:  $p = .22$ , Control:  $p = .12$ ). The remaining factors contribute variance unique to word reading and reading comprehension at the follow-up time point. From the path coefficients, we can also calculate that 27% of the phenotypic correlation between comprehension at initial testing and at follow-up is independent from word reading in the RD group. This proportion is slightly higher in the control group, where 39% of the correlation between comprehension at the two assessments is unique from word reading.

### Multivariate genetic analyses

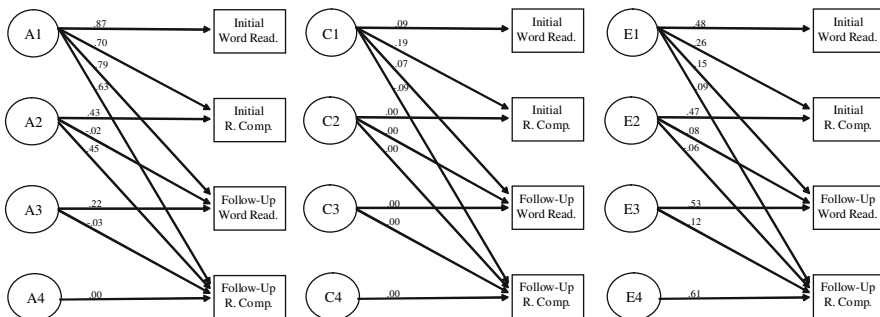
The phenotypic Cholesky was further partitioned into genetic, shared environmental, and nonshared environmental factors. In contrast to the phenotypic model, solutions for the RD and control groups could be equated without significant loss of fit. Since this is a relatively small sample for genetic analyses, we may have limited power to detect small differences between groups. Further, the ability to equate the groups in the genetic but not the phenotypic model suggests that power to detect small differences between the groups is reduced when the model is expanded to include estimates of genetic and environmental parameters. However, as with the phenotypic model, the pattern of genetic and environmental influences is highly similar for the two groups. The standardized path coefficients for the equated model are shown in Fig. 4. This is also the model against which the remaining submodels were tested. There is an initial genetic factor (A1), common to both measures at

**Fig. 3** Results of phenotypic Cholesky: path coefficients for the RD group, with estimates for the control group in parentheses



both assessments. Similar to the phenotypic results, there also appears to be a second genetic factor (A2) contributing to the covariance between reading comprehension at the two assessments, independent of word reading. The small path coefficients from the genetic factors A3 and A4 suggest that there are no new genetic influences expressed at follow-up. The paths from shared environmental factors are negligible, whereas nonshared environmental influences unique to each measure are substantial, and there appears to be a nonshared environmental common factor (E1) contributing to the covariance among the measures.

The proportions of variance in each measure due to genetic and environmental influences are presented in Table 3 with 95% confidence intervals. Heritabilities are quite high for both measures at both assessments, particularly for word reading at



**Fig. 4** Results of genetic/environmental Cholesky

**Table 3** Univariate estimates of heritability, shared environment, and nonshared environment computed from Cholesky model, with 95% confidence intervals

Measure	Heritability ( $a^2$ )	Shared environment ( $c^2$ )	Nonshared environment ( $e^2$ )
Word read.—Initial	0.76 (.58, .84)	0.01 (0, .17)	0.23 (.16, .34)
Comp.—Initial	0.67 (.45, .79)	0.04 (0, .23)	0.29 (.20, .42)
Word read.—Follow-up	0.68 (.47, .78)	0.01 (0, .17)	0.32 (.22, .45)
Comp. —Follow-up	0.60 (.42, .72)	0.01 (0, .12)	0.39 (.28, .55)

initial assessment. Each of the estimates of heritability and nonshared environment are significant, as indicated by confidence intervals above zero, whereas none of the estimates of shared environment are significant.

Estimates of the genetic, shared environmental, and nonshared environmental correlations are presented below the diagonals in Table 4. The genetic correlations ( $r_A$ ) are all high, with a correlation between the assessments of word reading at the two time points of .96. That between assessments of reading comprehension at the two time points was estimated at unity ( $r_A = 1.00$ ). These high genetic correlations indicate that the genetic influences acting within measure at the two assessments are virtually identical. We also see that at each assessment, the genetic correlations between the measures are quite high as well, i.e., the  $r_A$  between word reading and reading comprehension at initial testing is .85, and at follow-up is .75; thus, the genetic influences acting on the different measures within time point are also highly similar. Yet, they are not at unity, suggesting unique genetic effects on each measure as well.

The phenotypically standardized genetic correlations presented above the diagonals in Table 4 range from .48 to .69, indicating that 80–100% of the phenotypic correlations among the measures are due to shared genetic influences. The shared environmental correlations are estimated at unity, suggesting that the same environmental influences are acting on all measures. This can also be seen in Fig. 4, as after the common paths to C1, all other C paths are estimated at zero. However, these common shared environmental influences are negligible, accounting for only 1–4% of the total variance for each measure. As a result, shared environmental influences account for at most 3% of the phenotypic correlations. In contrast, the nonshared environmental correlations are lower, ranging from  $-.01$  to .48, but these influences account for a higher proportion of phenotypic covariance (up to 17%).

### Model comparisons

As described above, we first fitted a full ACE model (Model 1) with separate solutions for the RD and control groups. The solutions for the two groups were equated without significant loss of fit (Model 2,  $p = .17$ ). We then tested subsequent models against this equated model to determine the most parsimonious model to result in a nonsignificant decrement in fit (see Table 5).

**Table 4** Genetic, shared environmental, and nonshared environmental correlations below the diagonal, and phenotypically standardized correlations (which sum to the phenotypic correlation) above the diagonal

		Initial		Follow-up	
		Word read.	Comprehension	Word read.	Comprehension
<i>Genetic</i>					
Initial	Word reading	1.00	.61	.69	.55
	Comprehension	0.85	1.00	.55	.63
Follow-up	Word reading	0.96	0.81	1.00	.48
	Comprehension	0.81	1.00	0.75	1.00
<i>Shared environment</i>					
Initial	Word reading	1.00	.02	.01	-.01
	Comprehension	1.00	1.00	.01	-.02
Follow-up	Word reading	1.00	1.00	1.00	-.01
	Comprehension	-1.00	-1.00	-1.00	1.00
<i>Nonshared environment</i>					
Initial	Word reading	1.00	.12	.07	.04
	Comprehension	0.48	1.00	.08	-.003
Follow-up	Word reading	0.27	0.26	1.00	.07
	Comprehension	0.15	-0.01	0.21	1.00

All shared environmental (C) factors could be dropped from the model without significant loss of fit (Model 3;  $p = .999$ ), indicating that there are no significant shared environmental effects on either the variances or covariances of the measures or their stability. In contrast, we were unable to drop all genetic influences (Model 4;  $p < .001$ ), so we tested the significance of those paths which appeared to contribute little to the model. The path from A2 to Follow-up Word Reading ( $a_{32}$ ), as well as the A3 and A4 factors could be omitted from the model without loss of fit (Models 6 & 7;  $p = .976$  and  $.791$ , respectively). This leaves a genetic factor (A1) that contributes significantly to the variation and covariation of the measures at both time points, as evidence for continuity of genetic influences over time and shared genetic influence between word reading and reading comprehension tasks. There is also a significant second genetic factor (A2; Model 5,  $p = .008$ ) that is specific to the comprehension measures, suggesting that there are unique genetic influences on reading comprehension, independent of word reading, and that these influences are relatively stable. The failure to obtain significant genetic factors specific to the second time point, however, suggests that all genetic influences are common across time points, and no new genetic effects emerge later in development for either measure.

Non-shared environment also contributes significantly to continuity in word reading. Although we can drop the path from E1 to follow-up reading comprehension ( $e_{41}$ ; Model 10;  $p = .198$ ), the remaining common effects of this first factor could not be dropped. This leaves a nonshared environmental path that is common

**Table 5** Model fit statistics

Model	Model comparison <sup>a</sup>				
	$-2LL$	NPAR	$\Delta \chi^2$	$\Delta df$	$p (\Delta)$
1. Full ACE	2670.51	92			
2. ACE: Aff. and Control equated	2707.83	62	37.32	30	0.168
3. Drop C	2709.38	52	1.55	10	0.999
4. Drop A	2753.25	52	45.42	10	<.001
5. Drop A2	2719.67	59	11.84	3	0.008
6. Drop $a_{32}$ path	2707.90	61	0.07	1	0.791
7. Drop A3 and A4	2708.04	59	0.21	3	0.976
8. Drop all E common factors	2738.04	56	30.21	6	<.001
9. Drop $e_{21}$ , $e_{31}$ , $e_{41}$	2733.90	59	26.07	3	<.001
10. Drop $e_{41}$	2709.49	61	1.66	1	0.198
11. Drop $e_{32}$ , $e_{42}$ , $e_{43}$	2712.79	59	4.96	3	0.175
12. Drop all E but E1 & Specifics	2713.72	58	5.89	4	0.207
13. Final Model (A1, A2, E1 + Sp)	2717.93	44	10.10	18	0.929

<sup>a</sup> Model 2 is compared to Model 1. All other submodels are compared to Model 2

to word reading at both time points and comprehension at initial testing. All other shared paths were dropped (Model 11;  $p = .175$ ), leaving one nonshared environmental common factor and nonshared environmental specifics. Thus, the most parsimonious model to result in a non-significant deterioration in fit includes the A1 common factor, the A2 factor common to reading comprehension at initial and follow-up assessments, no shared environment, and a nonshared environmental common factor plus nonshared environmental specifics.

## Discussion

The purpose of the current study was to investigate the stability of genetic and environmental influences on word reading and reading comprehension, as well as the stability of those influences on their relationship. We used data from the Colorado Longitudinal Twin Study of Reading Disability (LTSRD; Wadsworth et al., submitted), the first longitudinal study of reading disability using genetically informative data, to assess stability of both reading comprehension and word reading among children with and without a history of reading difficulties.

Phenotypically, although the solutions for the RD and control groups could not be equated, the overall pattern of results was very similar. Both word reading and reading comprehension were highly stable, with stability correlations of .80 and .57, respectively, in the RD group, and .68 and .63 in the control group. Most of the variance in both measures at both assessments was accounted for by a single factor. However, results suggested a second factor common to reading comprehension at

both time points, independent of word reading in both groups. Furthermore, the phenotypic correlations between word reading and comprehension were higher at initial testing (RD  $r = .78$ ; control  $r = .65$ ) than at follow-up (RD  $r = .54$ ; control  $r = .48$ ), suggesting that the similarity between them decreases over time. This finding supports results from previous phenotypic studies which suggest that word reading ability is a stronger factor in reading comprehension in younger children (Catts et al., 2005; Curtis, 1980; Johnston & Kirby, 2006; Keenan, Betjemann, & Olson, 2006, submitted).

### Continuity of genetic effects

We found that across time, genetic influences were virtually identical within measure, suggesting that the genetic effects on individual differences in performance on these measures are highly stable in this sample. This finding is consistent with our predictions for word reading, based on patterns of results from previous research. The studies that have found new genetic influences over time for word reading component skills assessed reading performance among younger children, when reading skills are rapidly developing (e.g., Byrne et al., 2005a, 2006; Harlaar et al., 2007; Petrill et al., 2007). Similarly, Harlaar et al. (2007) observed new genetic influences at age 10, independent of age 9, for their reading measure, which included comprehension. However, genetic influences on our measure of reading comprehension appear to be highly stable. It may be that our wide age ranges contributed to our negative result. However, Wadsworth et al. (2001, 2006) analyzed data from same-aged subjects, ages 7, 12, and 16, and also found no new genetic influences for word reading at ages 12 and 16. Likewise, Gayán and Olson (2003) assessed genetic etiology of word reading across a wide age range, and found the interaction with age to be small and nonsignificant. Although a portion of our sample were poor readers who might have been more likely to show developmental changes in their word reading skills at later ages, the patterns of genetic influences were not significantly different for the RD and control groups. Thus, while new genetic influences may be manifested in the early stages of reading development, our results suggest that genetic influences on word reading and reading comprehension skills are generally stable by early adolescence.

Between measures, a high proportion of the covariance of reading comprehension and word reading was due to shared genetic factors, but we also noted a significant genetic factor unique to reading comprehension at both time points, independent of word reading. This supports the recent findings of Keenan and colleagues (Keenan, Betjemann, Wadsworth et al., 2006), who also observed a unique genetic factor for reading and listening comprehension. It suggests that while many of the genetic influences on reading comprehension and word reading are overlapping, there are unique genetic influences on comprehension processes, supporting the argument that reading comprehension skills may be unique and separable from word reading skills (e.g., Cain et al., 2000; Catts et al., 2003; Gough et al., 1996; Gough & Tunmer, 1986; Hoover & Gough, 1990; Keenan et al., 2006; Leach et al., 2003; Nation, 2005; Oakhill et al., 2003). Interestingly, we did not find a large difference in the genetic relation between word reading and comprehension

over time, despite finding a difference in the phenotypic correlations. The phenotypic difference appears to be due to a decrease across time in the nonshared environmental correlations, rather than in the genetic correlations. There is some suggestion of a higher genetic correlation between the two measures at initial testing than at follow-up, indicating that there may be some divergence as children age, but the difference is fairly small. The overall pattern, both within and across measures, seems to be that the etiologies at initial and follow-up assessments are nearly the same.

However, comprehension tests vary in what they measure, so it is important to interpret results in the context of the specific test used. In particular, Keenan, Betjemann, & Olson (2006, submitted) and Nation and Snowling (1997) have demonstrated that the relation between word reading and reading comprehension can be test dependent. If tests vary in their sensitivity for measuring comprehension ability independent from word reading, this could extend to differences in sensitivity to changes in the etiology of that comprehension ability over time. Thus, it will be important to replicate this result with other measures of comprehension, and with a larger sample as our sample grows.

### Environmental effects

Significant environmental influences were limited to one nonshared environmental common factor and specifics. The common factor could be due to different influences such as the children having different teachers, or random accidents affecting only one twin. One additional source of nonshared environmental influences that could possibly contribute to a nonshared environmental common factor might be individual differences in motivational level that are independent for members of twin pairs but that influence performance on measures of both word reading and reading comprehension. Since nonshared environmental estimates also include measurement error, this may account, in part, for the specific paths for each measure.

We found no evidence of significant shared environmental influences on either measure at either time point. While we have limited power to detect significance of small effects due to our relatively small sample size, the estimates for the C paths were all very small, accounting for only 1–4% of the variance in each measure. This finding is in contrast to the results of Harlaar et al. (2007) who found significant shared environmental influences on reading performance through age 10, as well as to studies of younger children that reported significant shared environmental influences on word reading and related components (e.g., Byrne et al., 2005a, 2006; Petrill, Deater-Deckard, Schatschneider, & Davis, 2005; Petrill et al., 2007; Petrill et al., 2006b; Samuelsson et al., 2005).

However, our results are consistent with those of Keenan, Betjemann, Wadsworth et al. (2006) and Wadsworth et al. (2001, 2006), who found negligible shared environmental influences in older children. Moreover, even in much younger children, Byrne and colleagues also report that shared environmental effects found in kindergarten were no longer significant by the end of first grade (Byrne et al., 2007). It may be that larger shared environmental influences are seen in the early stages of reading development, but as children age, shared environment becomes

less important to individual differences in reading performance (e.g., Scarborough & Dobrich, 1994). Our inability to find significant shared environmental influences could also be due in part to restricted environmental range of the sample (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), as our subjects are typically from middle-class homes and, in contrast to the study by Harlaar et al. (2007), only included children for whom English was the primary language spoken in the home. We additionally note that Gayán and Olson (2003) did not find significant shared environment with any reading measures, using a larger group of participants from the same overall initial testing sample reported here, with a similar age range. However, they suggest this may be because of the influence of non-additive genetic effects; if dominance or epistasis influence a measure, variance due to additive genetic effects may be overestimated and that due to shared environment may be underestimated using the standard ACE model. While we do find some evidence in our data of DZ correlations that are less than half of the MZ correlations, indicating possible non-additive genetic effects, these differences are not statistically significant. Therefore this will be an important direction to investigate as the sample size increases.

## Conclusion

Overall, our measures of word reading and comprehension were found to be both highly stable and heritable in this adolescent sample. Phenotypically, our measures of word reading and reading comprehension were highly correlated at both assessments, though less so at follow-up. However, although the phenotypic relation between the measures may decline over time, the genetic etiology of this relation is quite stable. The vast majority of covariance across time and between measures in this sample was due to shared genetic factors, with a smaller proportion due to nonshared environmental influences.

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## References

- Byrne, B., Delaland, C., Fielding-Barnsley, R., Quain, P., Samuelsson, S., Høien, T., Corley, R., DeFries, J. C., Wadsworth, S. J., Willcutt, E. G., & Olson, R. K. (2002). Longitudinal twin study of early reading development in three countries: Preliminary results. *Annals of Dyslexia*, *52*, 49–73.
- Byrne, B., Olson, R. K., Wadsworth, S., Corley, R., Samuelsson, S., & Quain, P. (2005b). *Longitudinal twin study of literacy and language: The first three years*. Paper presented at the meeting of the Society for the Scientific Study of Reading, Toronto, Canada, June 26, 2005.
- Byrne, B., Olson, R. K., Samuelsson, S., Wadsworth, S., Corley, R., DeFries, J. C., & Willcutt, E. G. (2006). Genetic and environmental influences on early literacy. *Journal of Research in Reading, Special Issue: Reading and Genetics*, *29*, 33–49.

- Byrne, B., Samuelsson, S., Wadsworth, S., Hulslander, J., Corley, R., DeFries, J. C., Quain, P., Willcutt, E. G., & Olson, R. K. (2007). Longitudinal twin study of early literacy development: Preschool through Grade 1. *Reading and Writing: An Interdisciplinary Journal*, *20*, 77–102.
- Byrne, B., Wadsworth, S., Corley, R., Samuelsson, S., Quain, P., DeFries, J. C., Willcutt, E., & Olson, R. K. (2005a). Longitudinal twin study of early literacy development: Preschool and kindergarten phases. *Scientific Studies of Reading*, *9*, 219–235.
- Cain, K., Oakhill, J., & Bryant (2000). Investigating the causes of reading comprehension failure: A test of the phonological processing deficit hypothesis. *Reading and Writing: An Interdisciplinary Journal*, *13*, 31–56.
- Catts, H. W., Fey, T. P., Zhang, X., & Tomblin, J. B. (1999). Language basis of reading and reading disabilities: Evidence from a longitudinal investigation. *Scientific Studies of Reading*, *3*, 331–361.
- Catts, H. W., Hogan, T. P., & Adlof, S. M. (2005). Developmental changes in reading, reading disabilities. In H. W. Catts & A. G. Kamhi (Eds.), *The connections between language and reading disabilities* (pp. 25–40). Mahwah, NJ: Erlbaum.
- Catts, H. W., Hogan, T. P., & Fey, M. E. (2003). Subgrouping poor readers on the basis of individual differences in reading-related abilities. *Journal of Learning Disabilities*, *36*, 151–164.
- Chall, J. S. (1983). *Stages of reading development*. New York: McGraw-Hill.
- Curtis, M. E. (1980). Development of components of reading skill. *Journal of Educational Psychology*, *72*, 656–669.
- Davis, C. J., Knopik, V. S., Olson, R. K., Wadsworth, S. J., & DeFries, J. C. (2001). Genetic and environmental influences on rapid naming and reading ability: A twin study. *Annals of Dyslexia*, *51*, 231–247.
- DeFries, J. C., Filipek, P. A., Fulker, D. W., Olson, R. K., Pennington, B. F., & Smith, S. D. (1997). Colorado Learning Disabilities Research Center. *Learning Disabilities*, *8*, 7–19.
- de Jong, P. F., & van der Leij, A. (2002). Effects of phonological abilities and linguistic comprehension on the development of reading. *Scientific Studies of Reading*, *6*, 51–77.
- Dunn, L. M., & Markwardt, F. C. (1970). *Examiner's manual: Peabody individual achievement test*. Circle Pines, MN: American Guidance Service.
- Fisher, S. E., & DeFries, J. C. (2002). Developmental dyslexia: Genetic dissection of a complex cognitive trait. *Nature Reviews Neuroscience*, *3*, 767–780.
- Gayán, J., & Olson, R. K. (2001). Genetic and environmental influences on orthographic and phonological skills in children with reading disabilities. *Developmental Neuropsychology*, *20*, 487–511.
- Gayán, J., & Olson, R. K. (2003). Genetic and environmental influences on individual differences in printed word recognition. *Journal of Experimental Child Psychology*, *84*, 97–123.
- Gough, P. B., Hoover, W. A., & Peterson, C. L. (1996). Some observations on a simple view of reading. In C. Cornoldi & J. Oakhill (Eds.), *Reading comprehension difficulties: Processes and intervention* (pp. 1–13). Mahwah, NJ: Lawrence Erlbaum Associates.
- Gough, P. B., & Tunmer, W. E. (1986). Decoding, reading, and reading disability. *Remedial and Special Education*, *7*, 6–10.
- Harlaar, N., Dale, P. S., & Plomin, R. (2007). From learning to read to reading to learn: Substantial and stable genetic influence. *Child Development*, *78*, 116–131.
- Harlaar, N., Spinath, F. M., Dale, P. S., & Plomin, R. (2005). Genetic influences on early word recognition abilities and disabilities: A study of 7-year-old twins. *Journal of Child Psychology and Psychiatry*, *46*, 373–384.
- Hoover, W. A., & Gough, P. B. (1990). The simple view of reading. *Reading & Writing: An Interdisciplinary Journal*, *2*, 127–160.
- Johnston, T. C., & Kirby, J. R. (2006). The contribution of naming speed to the simple view of reading. *Reading and Writing: An Interdisciplinary Journal*, *19*, 339–361.
- Keenan, J. M., Betjemann, R. S., Wadsworth, S. J., DeFries, J. C., & Olson, R. K. (2006). Genetic and environmental influences on reading and listening comprehension. *Journal of Research in Reading*, *29*, 79–91.
- Keenan, J. M., Betjemann, R. S., & Olson, R. K. (submitted). *A comparison of reading comprehension tests*. Manuscript submitted for publication.
- Keenan, J. M., Betjemann, R. S., & Olson, R. K. (2006). *How comparable are reading comprehension tests?* Talk presented at the meeting of the Society for the Scientific Study of Reading, Vancouver, Canada.
- Leach, J. M., Scarborough, H. S., & Rescorla, L. (2003). Late-emerging reading disabilities. *Journal of Educational Psychology*, *95*, 211–224.

- Markwardt, F. C. (1998). *Peabody individual achievement test – revised*. Bloomington, MN: Pearson Assessments.
- Nation, K. (2005). Children's reading comprehension difficulties. In M. Snowling & C. Hulme, (Eds.), *The science of reading: A handbook* (pp. 248–266). Oxford, England: Blackwell.
- Nation, K., & Snowling, M. (1997). Assessing reading difficulties: The validity and utility of current measures of reading skill. *British Journal of Educational Psychology*, *67*, 359–370.
- Neale, M. C., Boker, S. M., Xie, G., Maes, H. H. (2002). *Mx: Statistical modeling*. (6th ed.) Richmond, VA: Department of Psychiatry.
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Oakhill, J., Cain, K. E., & Bryant, P. E. (2003). The dissociation of word reading and text comprehension: Evidence from component skills. *Language and Cognitive Processes*, *18*, 443–468.
- Olson, R. K. (2004). SSSR, environment, and genes. *Scientific Studies of Reading*, *8*, 111–124.
- Pennington, B. F., & Olson, R. K. (2005). Genetics of dyslexia. In M. Snowling & C. Hulme (Eds.), *The science of reading: A handbook* (pp. 453–472). Oxford, England: Blackwells.
- Petrill, S. A., Deater-Deckard, K., Schatschneider, C., & Davis, C. (2005). Measured environmental influences on early reading: Evidence from an adoption study. *Scientific Studies of Reading*, *9*, 237–260.
- Petrill, S. A., Deater-Deckard, K., Thompson, L. A., DeThorne L., & Schatschneider, C. (2006a). Reading skills in early readers: Genetic and shared environmental influences. *Journal of Learning Disabilities*, *39*, 48–55.
- Petrill, S. A., Deater-Deckard, K., Thompson, L. A., DeThorne, L. S., & Schatschneider, C. (2006b). Genetic and environmental effects of serial naming and phonological awareness on reading outcomes. *Journal of Educational Psychology*, *98*, 112–121.
- Petrill, S. A., Deater-Deckard, K., Thompson, L. A., Schatschneider, C., DeThorne, L. S., & Vandenberg, D. J. (2007). Longitudinal genetic analysis of early reading: The Western Reserve Reading Project. *Reading & Writing: An Interdisciplinary Journal*, *20*, 127–146.
- Plomin, R., & DeFries, J. C. (1979). Multivariate behavioural genetic analysis of twin data on scholastic abilities. *Behavior Genetics*, *9*, 505–517.
- Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2001). *Behavioral genetics* (4th ed.). New York: Worth Publishers.
- Raskind, M. H., Gerber, P. J., Goldberg, R. J., Higgins, E. L., & Herman, K. L. (1998). Longitudinal research in learning disabilities: Report on an international symposium. *Journal of Learning Disabilities*, *31*, 266–277.
- Samuelsson, S., Byrne, B., Quain, P., Wadsworth, S., Corley, R., DeFries, J. C., Willcutt, E., & Olson, R. K. (2005) Environmental and genetic influences on preschool skills in Australia, Scandinavia, and the United States. *Journal of Educational Psychology*, *97*, 705–722.
- Scarborough, H. S. (1998). Predicting the future achievement of second graders with reading disabilities: Contributions of phonemic awareness, verbal memory, rapid naming, and IQ. *Annals of Dyslexia*, *48*, 115–136.
- Scarborough, H. S. (2005). Developmental relationships between language and reading: Reconciling a beautiful hypothesis with some ugly facts. In H. Catts & A. Kamhi (Eds.), *The connections between language and reading disabilities* (pp. 3–24). Mahwah, NJ: Lawrence Erlbaum Associates.
- Scarborough, H. S., & Dobrich, W. (1994) On the efficacy of reading to preschoolers. *Developmental Review*, *14*, 245–302.
- Seigneuric, A., & Ehrlich, M. (2005). Contribution of working memory capacity to children's reading comprehension: A longitudinal investigation. *Reading & Writing: An Interdisciplinary Journal*, *18*, 617–656.
- Shaywitz, S. E., Fletcher, J. M., Holahan, J. M., Shneider, A. E., Marchione, K. E., Stuebing, K. K., Francis, D. J., Pugh, K. R., Shaywitz, B. A. (1999). Persistence of dyslexia: The Connecticut Longitudinal Study at adolescence. *Pediatrics*, *104*, 1351–1359.
- Sicoli, F. (2002). Stability of school-level scores from large-scale student assessments. *Applied Measurement in Education*, *15*, 173–185.
- Stevenson, J., Graham, P., Fredman, G., & McLoughlin, V. (1987). A twin study of genetic influences on reading and spelling ability and disability. *Journal of Child Psychology and Psychiatry*, *28*, 229–247.
- Tiu, R. D., Jr., Wadsworth, S. J., Olson, R. K., DeFries, J. C. (2004) Causal models of reading disability: A twin study. *Twin Research*, *7*, 275–283.

- Torgesen, J. K., & Burgess, S. (1998). Consistency of reading-related phonological processes throughout early childhood: Evidence from the longitudinal – correctional and instructional studies. In J. L. Metsala & L. C. Ehri (Eds.), *Word recognition in beginning literacy* (pp. 161–188). Mahwah, NJ: Lawrence Erlbaum.
- Torgesen, J. K., Wagner, R. K., Rashotte, C. A., Burgess, S., & Hecht, S. (1997). Contributions of phonological awareness and rapid automatic naming ability to the growth of word-reading skills in second- to fifth-grade children. *Scientific Studies of Reading, 1*, 161–185.
- Trouton, A., Spinath, F. M., & Plomin, R. (2002). Twins Early Development Study (TEDS): A multivariate, longitudinal genetic investigation of language, cognition and behaviour problems in childhood. *Twin Research, 5*, 444–448.
- Turkheimer, E., Haley, A., Waldron, M., D’Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science, 14*, 623–628.
- Wadsworth, S. J., Corley, R. P., Hewitt, J. K., & DeFries, J. C. (2001). Stability of genetic and environmental influences on reading performance at 7, 12, and 16 years of age in the Colorado Adoption Project. *Behavior Genetics, 31*, 353–359.
- Wadsworth, S. J., Corley, R. P., Plomin, R., Hewitt, J. K., & DeFries, J. C. (2006). Genetic and environmental influences on continuity and change in reading achievement in the Colorado Adoption Project. In A. Huston & M. Ripke (Eds.), *Developmental contexts of middle childhood: Bridges to adolescence and adulthood* (pp. 87–106). New York: Cambridge University Press.
- Wadsworth, S. J., DeFries, J. C., Olson, R. A., & Willcutt, E. G. (submitted). Colorado Longitudinal Twin Study of Reading Disability.
- Wadsworth, S. J., Fulker, D. W., & DeFries (1999). Stability of genetic and environmental influences on reading performance at 7 and 12 years of age in the Colorado Adoption Project. *International Journal of Behavioral Development, 23*, 319–332.