Confirmatory Factor Analysis of Word Recognition and Process Measures in the Colorado Reading Project

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ABSTRACT: Measures of word recognition (REC) and two component skills, phonological coding (PHON) and orthographic coding (ORTH), were subjected to multivariate behavioral genetic analysis. Data were obtained from a sample of identical and fraternal twin pairs wherein at least one member of each pair was reading disabled (RD), and from a sample of twins wherein both members of each pair read in the normal range. Confirmatory factor analysis was used to fit the genetic, common environmental, and specific environmental covariance components for REC, PHON, and ORTH within the RD and normal simples. The resulting heritability estimates for REC, PHON, and ORTH were 0.59, 0.41, and 0.05 in the RD sample, and 0.35, 0.52, and 0.20 in the normal sample. After dropping the nonsignificant common environment parameters from the models, the genetic correlations between REC and PHON and between REC and ORTH were respectively 0.81 and 0.45 in the RD sample, and 0.68 and 0.45 in the normal sample. Differences between the genetic correlations were significant in the RD sample ($p < 0.005$), marginally significant in the normal sample ($p < 0.10$), and highly significant in the combined sample ($p < 0.001$), indicating that genetic influences on individual differences in REC are more strongly related to genetic variance in PHON than in ORTH. These results are consistent with previous demonstrations of substantial genetic covariance between the disabled group's deficits in REC and PHON, but not between REC and ORTH (Olson et al., 1989; Olson and Rack, 1990).

KEYWORDS: Word recognition, phonological, orthographic, genetics, dyslexia.

Primary goals of the Colorado Reading Project are to understand the genetic and environmental etiologies of reading disabilities and of individual differences in reading ability within the normal range. To achieve these goals, we compare the similarities of identical and fraternal twin pairs on a variety of reading and related process measures. If the degree of shared environment is approximately equal for the identical and fraternal pairs, greater similarity for the genetically identical twins provides evidence for genetic influence (Plomin, DeFries, and McClearn, 1990). In the present paper, confirmatory factor analysis of twin data is used to assess the etiology of variation in word recognition and two important com-

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ponent processes, as well as that of observed covariation among the three measures.

Comprehension of written text is a complex skill that depends on the integration of several component processes (Olson, Wise, Conners, and Rack, 1990). Verbal intelligence and word recognition have been identified as two major sources of independent variance in reading comprehension within both disabled and normal groups (Conners and Olson, 1990). Abilities such as oral vocabulary and general world knowledge, included under verbal intelligence, are needed to comprehend both spoken and written text, but skill in recognizing printed words is essential for fluent reading. Children with specific reading disability or dyslexia typically show deficits in word recognition that are inconsistent with their normal-range IQ and comprehension of spoken text.

Disabled readers' unique deficits in word recognition led us to examine two component processes that were initially based on the "dual route" model (Baron, 1977; Coltheart, 1978). In this model, words can be read by a slow "indirect" route that involves the sounding out of letters and letter clusters through the application of grapheme-phoneme correspondence rules. This is presumed to be the primary route for unfamiliar printed words and the most important route in the beginning stages of reading. A second rapid and "direct" or "visual" route, not dependent on phonological processes, is presumed to operate for the lexical identification of familiar printed words in skilled reading.

A number of recent criticisms have been directed toward the "dual route" model based on evidence that phonological processing is rapid and involuntary even for familiar printed words in skilled readers, and that the phonological decoding process may proceed by analogy with parts of familiar words rather than by grapheme-phoneme correspondence rules (Van Orden, Pennington, and Stone, 1990). Although this evidence suggests a primary role for phonological processes in both beginning and skilled reading, the homophonic nature of many English words requires the representation of specific orthographic patterns for lexical identification (e.g., there versus their; bear versus bare). In addition, there are many "exception" words that violate common phonological decoding rules (e.g., yacht). Therefore, both phonological coding and orthographic coding are necessary components in the recognition of printed English words. In the subsymbolic theory of Van Orden et al., word-specific orthographic knowledge is represented within the same processor that handles phonological coding, whereas in the dual process model, two independent processors are postulated.

Separate tasks were developed to assess skills in phonological coding and in the rapid identification of specific orthographic patterns (see Methods). Phonological coding required the rapid oral reading of a series of pronounceable nonwords (e.g., tegwop, framble). Orthographic coding

required subjects to decide rapidly which of two homophonic letter strings was a real word (e.g., rane or rain). Phonological processes may be automatically elicited in the orthographic task, but they are not sufficient to decide on the correct response.

Previous analyses of disabled and normal readers' phonological and orthographic coding skills revealed that both accounted for independent variance in word recognition (Olson et al., 1990). However, the disabled readers' absolute levels of performance on the two tasks were different when compared to the performance of younger normal children at the same level of word recognition. The disabled readers' level of phonological coding was nearly a full standard deviation below that of the younger normal readers.

Our initial behavior-genetic analyses of word recognition, phonological coding, and orthographic coding focused on the etiology of the reading-disabled group's *deficits* (Olson, Wise, Conners, Rack, and Fulker, 1989). A twin sample was identified wherein at least one member was reading disabled (the proband). Then we compared the differential regression of the identical and fraternal cotwins' performance toward the mean of a normal control sample. If a deficit is completely heritable and there is no test error, genetically identical cotwins would be expected to show no regression to the population mean, while fraternal twins who share half their genes on average would regress half way toward the population mean. In the other extreme, if a deficit was due only to environmental influence, the identical and fraternal cotwins would show equal regression toward the population mean. Intermediate differences in identical and fraternal cotwin regression indicate the relative balance of genetic and environmental influences on the group deficit. A multiple regression procedure developed by DeFries and Fulker (1985) was used to estimate the degree of heritability (h_g^2) and shared environmental influences (c_g^2) for the probands' group deficit. Significant levels of heritability were found for deficits in word recognition and phonological coding, but not for orthographic coding (Olson et al., 1989). The most recent analyses with a substantially larger twin sample and more appropriate selection procedures yielded h_g^2 estimates and standard errors of 0.44 $\pm$ 0.11 for word recognition, 0.75 $\pm$ 0.15 for phonological coding, and 0.31 $\pm$ 0.20 (n.s.) for orthographic coding (Olson and Rack, 1990). Estimates of shared environmental influences (c_g^2) were significant for deficits in word recognition (0.51 $\pm$ 0.11), and orthographic coding (0.48 $\pm$ 0.17), but not for phonological coding (0.12 $\pm$ 0.13, n.s.). Thus, the behavior-genetic analyses indicated significant genetic *and* shared environmental influences on group deficits in word recognition, significant genetic influences on group deficits in phonological coding, and significant shared environmental influences on group deficits in orthographic coding.

The above pattern of high h_g^2 for phonological coding and low h_g^2 for

orthographic coding has also been suggested from data reported by Stevenson (this volume). Although his sample of reading disabled twins in London was comparatively small, Stevenson found significant estimates of heritability (h_g^2 = about 0.7) for deficits in judging whether two nonwords would sound the same when read aloud. Heritability estimates for deficits in accuracy for oral reading of nonwords were also moderately high (h_g^2 = about 0.5) but not significant in his small sample. Heritability estimates were generally negative and not significant for accuracy deficits in reading a list of exception words such as "yacht", which may predominantly tap subjects' orthographic coding skills. Thus, it is encouraging that our general pattern of higher heritability for phonological coding than for orthographic coding has been replicated in the London study. Contrary to the Colorado study, the London twins' deficits on a standardized word recognition measure (Schonell 1971) were not significantly heritable, although this may be due in part to the predominance of exception words in this test.

Now we turn to the main focus of the present paper, the heritability of individual differences in the normal range. Estimates of genetic and shared environmental influence on extreme group *deficits* (h_g^2 and c_g^2) do not necessarily reflect the pattern of genetic and shared environmental influences on individual differences *within* groups (h^2 and c^2), i.e., the etiology of extreme scores may differ from that of variation within the normal range. Recall that the statistical procedure for assessing the etiology of the probands' group deficit focuses on the differential regression of the MZ and DZ cotwins' group scores toward the normal population mean (DeFries and Fulker, 1985). This approach is quite different from the assessment of heritability for individual differences *within* the disabled and normal groups, which has been traditionally based on doubling the difference between intraclass correlations for identical and fraternal twins to obtain estimates of h^2 and c^2.

For an extreme (and unlikely) example of a contrast between h_g^2 and h^2 in a disabled twin sample, the MZ and DZ cotwin groups' means could regress equally to the population mean, indicating that the probands' *group* deficit is not due to heritable influences. (Such a result might be expected if one randomly chosen member of each twin pair was deprived of reading instruction while the other was given a good education.) However, the reading disabled probands may still vary in the severity of their reading deficit, and there may be differential covariance with the MZ and DZ cotwins' performance. It is theoretically possible that a much higher correlation for MZ than for DZ pairs could indicate high heritability for *within* group individual differences, in spite of low heritability for the probands' *group* deficit. Furthermore, it is theoretically plausible that the estimate for within-group heritability derived from the disabled twin sample is a valid index of heritability for individual differences in the

normal range. In fact, we will show that estimates for h^2 within the disabled and normal twin groups are not significantly different.

DeFries and Fulker (1988) have formulated an extension of their regression procedure (DeFries and Fulker, 1985) that provides an assessment of h^2 and c^2 along with the significance of the difference between h^2 for individual differences in the population and h_g^2 for the group deficit. (DeFries and Fulker noted that the power for this test is low and very large samples would be needed to reach acceptable levels of significance for all but the most extreme differences.) If significant, differences between h^2 and h_g^2 would indicate a theoretically important difference in the relative influence of genetic and environmental factors on individual variation in the population versus the extreme-group deficits of disabled readers. However, such differences would not necessarily indicate differences in the underlying genetic mechanism. For example, it would be possible that the same gene or genes are responsible for both normal and extremely deviant variation in reading, but in the deviant cases there is a uniquely potent environmental effect, leading to a lower h_g^2.

In the present paper, we employ confirmatory factor analyses of twin data to assess genetic and shared environmental influences on individual differences within the normal range. This approach facilitates estimates of h^2 and c^2 for word recognition and the component coding skills, as well as estimates of the genetic correlations among the three measures. A genetic correlation provides a measure of the extent to which individual differences in two characters are due to the same genetic influences (Plomin et al., 1990). Through confirmatory factor analysis we will directly assess the degree to which genetic variance in phonological coding and orthographic coding is correlated with genetic variance in word recognition. In addition, we will determine whether the genetic correlations with word recognition are significantly different for phonological and orthographic coding. Data from the disabled twin group, the normal twin group, and the combined group will be subjected to separate analyses.

METHODS

Subjects

Two groups of twins between 7 and 20 years of age were ascertained from school records in 27 Colorado districts. In the disabled group, at least one member of each pair had a positive history of reading problems in their school records, and when tested in the laboratory on the word recognition, reading comprehension and spelling subtests of the Peabody Individual Achievement Test (PIAT; Dunn and Markwardt, 1970), they were below a discriminant function score previously established to discriminate

independent samples of 140 disabled and 140 normal readers (DeFries, 1985). In the normal comparison group, there was no evidence in the school records for reading problems in either twin and both twins scored above the critical discriminant function score. Additional inclusionary criteria for twins in both groups were a score of at least 90 on the Wechsler (1974) Verbal or Performance subscales, no evidence of neurological problems such as seizures, no uncorrected visual acuity or auditory deficits, and English was the primary language spoken in the home. The disabled group contained 86 identical or monozygotic (MZ) twin pairs and 73 same-sex fraternal or dizygotic (DZ) pairs. The normal group contained 92 MZ pairs and 59 same-sex DZ pairs. The twins' zygosity was determined using selected items from the Nichols and Bilbro (1966) zygosity questionnaire which has a reported accuracy of 95%.

Measures

A battery of reading and related cognitive measures, including the PIAT and Wechsler tests, was administered to the twins in DeFries' laboratory at the Institute for Behavior Genetics. Experimental measures of reading and related processes, including phonological and orthographic coding, were administered in a second session at Olson's laboratory in the Department of Psychology. Because the present analyses focus on PIAT word recognition, phonological coding, and orthographic coding, only these measures are described here. More complete descriptions are given in Olson et al. (1990).

PIAT Word Recognition (REC) (Dunn and Markwart, 1970). This standardized test consists of a series of 66 unrelated words placed in rows across several pages in order of increasing difficulty. Subjects were asked to read the words across the rows until they made five errors on the last seven items. The test-retest reliability is reported to be 0.89 across grade levels 1—12. Most of the words are regularly spelled and the subjects had unlimited time to reach each word.

Phonological Coding (PHON). This experimental task required subjects to read a block of 45 one-syllable nonwords (e.g., ter, calch, doun), followed by a block of 40 two-syllable nonwords (e.g., tegwop, stalder, framble), as quickly and accurately as possible when the nonwords appeared individually on a computer screen. Oral responses were timed with a voice key and were tape recorded for later scoring of percent correct. A response was considered correct if it followed grapheme-phoneme correspondence rules (tive pronounced to rhyme with hive), or by analogy to an orthographically similar word (tive pronounced to rhyme with give). Z scores for percent correct and mean response time on

correct trials were combined for each block, and a final score was derived by adding the combined z scores for the two blocks.

Orthographic Coding (ORTH). This experimental task required subjects to designate the word in word-pseudohomophone pairs as quickly as possible by pushing a right or left button after the pairs were presented on the computer monitor. The stimuli in each pair ranged from relatively short and frequent words (e.g., room-rume, rane-rain, sleep-sleap) in the first block of 40 trials to longer and less frequent words (e.g., sammon-salmon, explaine-explain) in the second block of 40 trials. Subjects were shown their response time in msec. after correct responses, and were shown the word "error" after incorrect responses. Z scores for percent correct and response time on correct trials were combined for each block, and a final score was derived by adding the combined z scores for the two blocks.

ANALYSES AND RESULTS

The twins' scores on each of the above measures were adjusted for their linear relation to age and standardized based on the mean and standard deviation in the normal sample. Although the mean of the disabled sample was therefore substantially lower, the variance was similar to that of the normal sample, thus reflecting a wide range of reading deficit.

In order to estimate genetic variances and covariances, the age-adjusted standardized scores for REC, PHON, and ORTH were subjected to multivariate behavioral genetic analysis (Fulker, Baker, and Bock, 1983; DeFries and Fulker, 1986; Heath, Neale, Hewitt, Eaves, and Fulker, 1989). Observed MZ and DZ cross-covariance matrices were fit to a simple factor model that included one common factor and three specific variances. The factor loadings in the full model were estimated from the phenotypic correlations using confirmatory factor analysis. An expected covariance matrix, [E], was compared to the observed phenotypic covariance matrix, [P], using the following log-likelihood function:

$$F = N[\log_e(\mathrm{DET}[E]/\mathrm{DET}[P]) + \mathrm{tr}([P][E]^{-1}) - k],$$

where N is the degrees of freedom for subjects, and k is the number of variables. The function was minimized using the MINUIT optimization package (CERN, 1977). The change in chi-square values for reduced models as compared to the full model was used to test the significance of factors in a given model.

Observed covariances between the REC, PHON, and ORTH variables

were partitioned into genetic, common environmental, and specific environmental components as follows:

$$[P] = [G] + [C] + [S],$$

where [P] is the phenotypic variance/covariance matrix, and [G], [C], and [S] are the additive genetic, common environmental, and specific environmental (including measurement error) component matricies, respectively. The genetic and environmental covariances were assumed to be uncorrelated.

Confirmatory factor analyses were first performed separately for the reading disabled and normal groups (results of these analyses were previously summarized in an abstract by Gillis, DeFries, Olson, and Rack, 1990). The phenotypic correlations for REC, PHON, and ORTH in the disabled and normal groups are presented in Table 1. In both groups, the correlation between REC and PHON is higher than that between REC and ORTH, although the difference is significant ($p < 0.025$) in only the disabled group. This result is partly due to the lower reliability of the forced-choice ORTH task, and partly due to the fact that most words in the REC measure were phonologically regular and could be phonologically decoded without memory for their specific orthographic patterns. Correlations were higher between ORTH and a timed experimental word recognition measure that included about half regular and half irregular words (Olson et al., 1990).

The full model yielded the estimates of heritability (h^2) and shared environment (c^2) presented in Table 2. It can be seen that the heritability estimates for REC and PHON range from 0.35 to 0.59 across the disabled and control groups, but the heritability for ORTH is only 0.05 in the disabled group and 0.20 in the control group. Estimates of shared environment are low except for ORTH (0.47) in the disabled group. The pattern of higher heritability for REC and PHON compared to ORTH is

Table 1. Phenotypic correlations among Word Recognition (REC), Phonological Coding (PHON), and Orthographic Coding (ORTH) measures

	Reading disabled				Controls		
	REC	*PHON*	*ORTH*		*REC*	*PHON*	*ORTH*
REC	1.00	0.65	0.30	REC	1.00	0.55	0.33
PHON		1.00	0.41	PHON		1.00	0.34
ORTH			1.00	ORTH			1.00
$N_{MZ} = 86$ pairs				$N_{MZ} = 92$ pairs			
$N_{DZ} = 73$ pairs				$N_{DZ} = 59$ pairs			

Table 2. Heritability (h^2) and Shared Environment (c^2) for Word Recognition (REC), Phonological Coding (PHON), and Orthographic Coding (ORTH)[a]

	Reading disabled				Controls		
	h^2	c^2	$(h^2)^b$		h^2	c^2	$(h^2)^b$
REC	0.59	0.00	0.58	REC	0.35	0.24	0.60
PHON	0.41	0.07	0.49	PHON	0.52	0.01	0.53
ORTH	0.05	0.47	0.52	ORTH	0.20	0.20	0.40

[a] h^2 and c^2 estimates are for the full model.
[b] (h^2) estimates are based on dropping c^2 from the model.

consistent with the results of the Olson et al. (1989) and Olson and Rack (1990) analyses for the heritability of the disabled groups' deficits (h_g^2) on these measures.

Table 3 summarizes the results of fitting alternative multivariate models of genetic and shared environmental influence to data for the three variables in the two samples. Significant changes in chi-square and deterioration of model fit occurred when genetic factors [G] were dropped for both the reading disabled and control groups. In contrast, shared environmental factors [C] could be dropped in both groups without a significant change in chi-square. Therefore, shared environment was dropped from the model to facilitate a more powerful test of the genetic correlations between the variables. The resulting correlations are presented in Table 4. In both groups, the genetic correlation between REC and PHON is greater than that between REC and ORTH. Constraining these correlations to be equal in the model led to a highly significant deterioration of fit in the disabled group ($\chi^2 = 11.36$, df $= 1$, $p < 0.005$), and a marginally significant deterioration of fit in the control group ($\chi^2 = 3.36$, df $= 1$, $p < 0.10$).

The pattern of genetic correlations was similar in the disabled and

Table 3. Model fitting results

			Reading disabled			Controls		
Model	df	Δ df	χ^2	$\Delta\chi^2$	p	χ^2	$\Delta\chi^2$	p
Full Model	24		54.14		<0.025	72.64		<0.005
Drop [G]	30	6	73.61	19.47	<0.005	87.38	14.74	<0.025
Drop [C]	30	6	63.72	9.58	>0.10	77.51	4.87	>0.50
Drop [C] and [G]	36	12	137.75	83.61	<0.005	160.61	87.97	<0.005

Table 4. Genetic correlations among Word Recognition (REC), Phonological Coding (PHON), and Orthographic Coding (ORTH) measures

	Reading disabled				Controls		
	REC	*PHON*	*ORTH*		*REC*	*PHON*	*ORTH*
REC	1.00	0.81	0.45	REC	1.00	0.68	0.45
PHON		1.00	0.56	PHON		1.00	0.52
ORTH			1.00	ORTH			1.00

control samples, as indicated by the test of homogeneity presented in Table 5. Therefore, the groups were combined for a final analysis. The genetic correlations were 0.75 for REC and PHON, 0.42 for REC and ORTH, and 0.56 for PHON and ORTH. Constraining the genetic correlations between REC and PHON (r_{g12}) and between REC an ORTH (r_{g13}) to be equal in the model yielded a highly significant deterioration in fit ($\chi^2 = 14.78, \text{df} = 1, p < 0.001$).

It should be emphasized that it was necessary to drop shared environment from the model to obtain significant contrasts between genetic correlations. This is partly due to the very low heritabilities for ORTH in the full model (see Table 2), which resulted in highly unstable estimates of genetic correlations between REC and ORTH. For example, when shared environment was included in the model for the disabled group, the genetic correlation between REC and ORTH was estimated to be 1.00, but with a standard error of 0.63! The primary effect of dropping shared environment was to increase the heritability estimate for ORTH from 0.05 in the

Table 5. Model fitting results from the combined reading disabled and control twin samples

	Combined reading disabled and control sample				
Model	df	χ^2	Δ df	$\Delta\chi^2$	*p*
Estimate [G] and [S]: RD	30	63.72			
Estimate [G] and [S]: Controls	30	77.51			
Estimate [G] and [S]: Combined	72	150.57			
Homogeneity			12	9.34	> 0.50
Constrain r_{g12}[a] = r_{g13}[b]	73	165.35			
Difference			1	14.78	< 0.001

[a] Genetic correlation between REC and PHON.
[b] Genetic correlation between REC and ORTH.

full model for disabled readers to 0.52 for the more parsimonious model. With this constraint, the genetic correlation between REC and ORTH was 0.45 with a standard error of 0.09, enabling a significant contrast with the much higher genetic correlation of 0.81 ± 0.14 between REC and PHON. Nevertheless, results of fitting both models indicate that the genetic *covariance* between ORTH and REC is substantially lower than that between PHON and REC.

DISCUSSION

The confirmatory factor analyses showed remarkable agreement in the results for the disabled and normal groups, even though the disabled group was highly selected. There was a similar degree of genetic influence on within group individual differences in word recognition and phonological coding. This result suggests that genetic factors operate in a similar way on individual differences in word recognition and phonological coding within the normal range and within the low tail of the distribution, although it is possible that the specific genes influencing individual differences could vary across the range of ability (Pennington, 1989).

Another striking result was the absence of significant shared environment influences, as these could be dropped from the model without a significant deterioration in fit. Therefore, genetic and non-shared environmental factors were the predominant etiological basis for within-group individual differences in word recognition and phonological coding. However, the results for orthographic coding were not as clear, particularly in the disabled group. Although the c^2 estimate of 0.47 for orthographic coding was not statistically significant in our small sample, it suggests the strong possibility of shared environmental influences. Such a result would be consistent with Olson and Rack's (1990) previous c_g^2 estimates for the group deficit in orthographic coding to be discussed later.

The most significant results from the confirmatory factor analyses were the contrasts in genetic correlations between word recognition and the two coding measures. Genetic influences on word recognition and phonological coding are highly correlated in both groups, and these correlations are significantly higher than those for word recognition and orthographic coding.

The above results are consistent with differences in genetic *covariance* previously reported between the disabled group's deficits in word recognition, phonological coding, and orthographic coding (Olson et al., 1989; Olson and Rack, 1990). (A genetic correlation is a function of both the genetic covariance between the two variables *and* of their heritabilities. Thus, even if the covariance between genetic influences on two variables is

low, the genetic correlation could be high if one or both variables has a low heritability.) The most recent analyses by Olson and Rack (1990) yielded evidence for significant genetic covariance ("bivariate h_g^2"), but no significant environmental covariance, between probands' deficits in word recognition and cotwins' phonological coding ($h_g^2 = 0.73 \pm 0.17$; $c_g^2 = 0.22 \pm 0.14$). In contrast, for word recognition and orthographic coding, there was no significant genetic covariance, but shared environmental covariance was significant ($h_g^2 = 0.32 \pm 0.25$, n.s.; $c_g^2 = 0.60 \pm 0.20$).

Olson and Rack's (1990) estimate of $h_g^2 = 0.75 \pm 0.15$ for the group deficit in phonological coding is somewhat higher than the h^2 estimates for within-group individual differences presented in Table 2. Our twin sample is still too small for the contrast between h_g^2 and h^2 to be statistically significant in DeFries and Fulker's (1988) augmented model, but the current trend suggests a stronger genetic influence on disabled readers' deficits in phonological coding than on individual differences in phonological coding within the normal range.

The h^2 and h_g^2 estimates for word recognition were similar at about 0.5, but the absence of c^2 (0.00) for individual differences within the disabled group contrasts sharply with our most recent estimate of shared environment for the disabled group's deficit in word recognition ($c_g^2 = 0.51 \pm 0.11$). These results suggest that shared environmental influences may be an important cause of the group deficit in word recognition, but not of individual differences within the group.

Shared environment was also significant for group deficits in orthographic coding ($c_g^2 = 0.48 \pm 0.17$), and there was substantial shared environmental covariance for probands' deficits in word recognition and cotwins' orthographic coding ($c_g^2 = 0.60 \pm 0.20$) (Olson and Rack, 1990). In agreement with Stanovich and West (1989), we have argued that exposure to print is an important factor in the development of orthographic skills in word recognition, and print exposure is likely to be influenced by the twins' shared environment (Olson et al., 1990). Consistent with this view, orthographic coding manifested comparable levels of c^2 (0.47) and c_g^2 (0.48) for the disabled group. We should add a caveat that just as similar estimates of h^2 and h_g^2 do not necessarily imply the same specific genetic mechanism, it is theoretically possible that the specific shared environmental influences are different, even if estimates of c^2 and c_g^2 are similar.

In contrast to word recognition and orthographic coding, estimates of the proportion of variance due to shared environmental influences for phonological coding have *never* been significant in either the present analyses of within-group differences or in previous analyses of the group deficit. Genetic factors and non-shared environment are consistently the predominant causes of variance in phonological coding, and most of the heritable variance in word recognition is shared with heritable variance in

phonological coding. Stanovich (1988) has emphasized the central role of phonological coding for reading disabilities in his "phonological-core variable-difference model". Results of the present study suggest that it is also central to the eitology of individual differences within the normal range.

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