

A Comparison of the Cognitive Deficits in Reading Disability and Attention-Deficit/Hyperactivity Disorder

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Abstract: This study used a nonreferred sample of twins to contrast the performance of individuals with reading disability (RD; $n = 93$), attention-deficit/hyperactivity disorder (ADHD; $n = 52$), RD and ADHD ($n = 48$), and neither RD nor ADHD ($n = 121$) on measures of phoneme awareness (PA) and executive functioning (EF). Exploratory factor analysis of the EF measures yielded underlying factors of working memory, inhibition, and set shifting. Results revealed that ADHD was associated with inhibition deficits, whereas RD was associated with significant deficits on measures of PA and verbal working memory. The RD + ADHD group was most impaired on virtually all measures, providing evidence against the phenocopy hypothesis as an explanation for comorbidity between RD and ADHD.

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Reading disorder (RD) and attention-deficit/hyperactivity disorder (ADHD) are two of the most common disorders of childhood, each occurring in approximately 5% of the population (American Psychiatric Association, 1994; S. E. Shaywitz, Shaywitz, Fletcher, & Escobar, 1990). ADHD and RD also co-occur significantly more frequently than expected by chance. Specifically, the rate of RD in samples selected for ADHD typically falls between 25 and 40% (e. g. , Dykman & Ackerman, 1991; Semrud-Clikeman et al. , 1992), whereas 15 to 35% of individuals with RD also meet criteria for ADHD (Gilger, Pennington, & DeFries, 1992; B. A. Shaywitz, Fletcher, & Shaywitz, 1995; Willcutt & Pennington, 2000). Moreover, this comorbidity is present in both clinical and community samples, indicating that it is not a selection artifact.

Whereas previous studies provide convincing evidence for comorbidity between RD and ADHD, the cause of this association is less clear. Results from several studies of twins suggest that the two disorders are each significantly heritable (e. g. , DeFries & Alarcón, 1996; Levy, Hay, McStephen, Wood, & Waldman, 1997) and share a small but significant common genetic cause (Light, Pennington, Gilger, & DeFries, 1995; Stevenson, Pennington, Gilger, DeFries, & Gillis, 1993; Willcutt, Pennington, & DeFries, 2000b). To understand the nature of these common genetic influences better, neuropsychological studies can be used to examine the proximal cognitive manifestation of the genetic influences on each separate disorder as well as the cognitive underpinnings of the phenotypic association between the two disorders (e. g. , Pennington, Groisser, & Welsh, 1993).

We examined the performance of individuals with RD, ADHD, or both RD and ADHD (RD + ADHD) on cognitive tests thought to be specific to either RD or ADHD. In the following section, we summarize the results of previous studies that have investigated the

cognitive profile of RD and ADHD separately. Next, we review studies that tested whether RD and ADHD are separable at the cognitive level of analysis. Finally, we review studies that used neuropsychological methods to test competing explanations for phenotypic comorbidity between RD and ADHD.

The Cognitive Profile of Each Disorder Considered Separately

Neuropsychological Profile of RD

Studies of individuals with and without reading difficulties suggested that phonological decoding, defined as the ability to translate sequences of printed letters into the corresponding sounds, has a central role in both normal and abnormal reading development (e. g. , Foorman, Francis, Novy, & Liberman, 1991; Pennington, Van Orden, Smith, Green, & Haith, 1990; Stanovich, Cunningham, & Feeman, 1984; Wagner & Torgesen, 1987). The unique contribution of phonological decoding (PD) to most cases of RD has been suggested by the presence of significant group deficits in PD when older children with RD are compared with younger normal readers at the same reading level (Olson, 1985; Rack, Snowling, & Olson, 1992). Moreover, twin studies showed that there are strong genetic influences on the group deficit in PD (Olson, Wise, Conners, Rack, & Fulker, 1989), and that these genetic influences overlap largely with genetic influences on the group deficit in word reading (Olson, Forsberg, & Wise, 1994). Deficits in PD and word reading are, in turn, linked to genetic influences on deficits in the oral language skill of phoneme awareness (PA), defined as the ability to recognize and manipulate the phonemic constituents of speech (Olson et al. , 1994; Olson et al. , 1989). Many regard problems with PA as the most proximal cause of most cases of RD (cf. Wagner, Torgesen, & Rashotte, 1994). Finally, whereas some studies suggest that individuals with RD may also exhibit mild deficits in additional areas such as visual tracking (Eden, Stern, Wood, & Wood, 1995; but see Olson, Kliegl, & Davidson, 1983), deficits on measures of PA or other measures of phonological processing consistently account for a much larger proportion of the reading deficit (e. g. , Fletcher, Foorman, Shaywitz, & Shaywitz, 1999).

Neuropsychological Profile of ADHD

A growing body of research suggests that ADHD is associated with a core deficit in executive functions (EF; Barkley, 1997; Barkley, 1998; Chelune, Ferguson, Koon, & Dickey, 1986; Heilman, Voeller, & Nadeau, 1991; Pennington & Ozonoff, 1996; Quay, 1988), defined as cognitive functions that serve to maintain an appropriate problem-solving set in order to attain a future goal (e. g. , Welsh & Pennington, 1988). These EF deficits are particularly pronounced in the domain of behavioral inhibition, as indexed by measures such as commission errors on a continuous performance test (e. g. , Barkley, Grodzinsky, & DuPaul, 1992; Halperin et al. , 1988; Nigg, Hinshaw, & Halperin, 1996; see review by Losier, McGrath, & Klein, 1996) and speed of response inhibition after an auditory stop signal (e. g. , Daugherty, Quay, & Ramos, 1993; Schachar & Logan, 1990; Schachar, Tannock, Marriott, & Logan, 1995; see meta-analyses by Oosterlaan & Sergeant, 1998; Pennington & Ozonoff, 1996). Some studies found that children with ADHD exhibit deficits in additional EF domains such as cognitive flexibility and verbal

working memory as well as slower and more variable response speed across cognitive tasks (e. g. , Brock & Knapp, 1996; Chelune et al. , 1986; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Shue & Douglas, 1992; Tannock, Martinussen, & Frijters, 2000). Other findings, however, suggested that individuals with ADHD are not significantly impaired on these measures (e. g. , Felton & Wood, 1989; Loge, Staton, & Beatty, 1990; McGee, Williams, Moffitt, & Anderson, 1989). Moreover, many of these studies did not assess important covariates such as IQ and academic achievement, leaving open the possibility that many of the EF deficits associated with ADHD could, in fact, be attributable to differences in IQ or to the association between ADHD and RD (see Pennington & Ozonoff, 1996).

The Double Dissociation Method

The finding that RD and ADHD are each associated with a primary cognitive deficit provides support for the external validity of each individual diagnosis. However, studies of either disorder alone cannot test definitively whether RD and ADHD are separable at the cognitive level of analysis, because individuals with each disorder might also be significantly impaired on measures of the core deficit associated with the other diagnosis. Instead, studies must ascertain individuals with each disorder and then test for a significant double dissociation between RD and ADHD and the two cognitive phenotypes (e. g. , PA and EF). A classic double dissociation (e. g. , Shallice, 1988) occurs when two disorders are associated with opposite patterns of impairment in two different cognitive domains.

In the first study that used a full 2×2 (RD \times ADHD) design to examine performance on measures of EF and psychological processing (PP), Pennington et al. (1993) found that individuals with ADHD were significantly impaired on EF measures but were not different from the comparison sample on PP tasks. In contrast, individuals with RD exhibited phonological processing deficits but were not impaired on the EF measures in comparison to controls. As shown in Table 1, results of subsequent studies generally replicated this cognitive double dissociation between ADHD and RD. However, several of these studies used a general learning disabled sample instead of a specific RD group (Barkley et al. , 1992; Korkman & Pesonen, 1994; Robins, 1992), and only three used the full 2×2 design (Klorman et al. , 1999; Nigg, Hinshaw, Carte, & Treuting, 1998; B. A. Shaywitz et al. , 1995). Moreover, although results of previous studies are generally consistent with a double dissociation between RD and ADHD on EF and PP tasks, several aspects of the findings outlined in Table 1 suggest that this double dissociation may not be complete. Specifically, whereas ADHD has not been associated with PP deficits in the three studies that tested this hypothesis directly, several studies suggested that children with reading or learning disabilities alone exhibit mild EF deficits. These deficits are most often present on tasks that involve sustained attention or cognitive flexibility (Barkley et al. , 1992; Kupietz, 1990; Weyandt, Rice, Linterman, Mitzlaff, & Emert, 1998), but a 1999 doctoral dissertation suggests that RD may also be associated with deficits on specific measures of inhibition (Purvis, 1999). These results call into question the specificity of the EF deficit to ADHD and suggest that further research will be useful to clarify the nature of the relation between RD and EF.

Implications of Neuropsychological Studies for the Etiology of Comorbidity Between RD and ADHD

As noted previously, in addition to significant genetic influences specific to each disorder, RD and ADHD also share a small but significant common genetic cause. The specific nature of these shared genetic underpinnings is unknown but may potentially reflect a shared predisposition toward difficulties in auditory processing (Riccio & Hynd, 1996; Riccio, Hynd, Cohen, Hall, & Molt, 1994), verbal working memory (Ashbaker & Swanson, 1996; Barkley, 1998; Denckla, 1996; Swanson & Berninger, 1995), or response speed (e. g. , Tannock et al. , 2000). The present study tested whether RD and ADHD share a common deficit in verbal working memory or PA by comparing individuals with RD, ADHD, and RD + ADHD on these measures. Because the common etiology hypothesis suggests that the same etiological influences contribute to most cases of the two disorders, it would predict that individuals with RD and ADHD exhibit the deficits associated with both RD and ADHD alone.

In contrast to the common cause hypothesis, the phenocopy hypothesis proposes that RD and ADHD co-occur because the primary disorder causes the symptoms of the other in the absence of the cognitive correlates typically associated with the secondary diagnosis in isolation (e. g. , Pennington et al. , 1993). For example, a child might appear to be inattentive or hyperactive in the classroom because of frustration elicited by difficulties with reading, or an individual might experience reading problems as a result of attentional difficulties in the classroom. If a significant double dissociation is obtained between RD and ADHD on measures of EF and PA, the profile of the comorbid group on these measures provides a direct test of the phenocopy hypothesis.

Pennington et al. (1993) found that the comorbid group exhibited significant PP deficits but were not different from the comparison group on the EF tasks. This profile mirrored the performance of the RD-only group, suggesting that primary RD had caused the phenotypic manifestation of ADHD in the comorbid group in the absence of the EF deficits typically associated with ADHD. This finding was partially replicated by B. A. Shaywitz et al. (1995), who found that the group with ADHD alone scored significantly lower than either of the RD groups on a measure of vigilance, whereas the two RD groups scored significantly lower than the group with ADHD on PP tasks. However, the majority of subsequent studies did not support the phenocopy hypothesis, instead finding that the comorbid group exhibits both the EF deficits associated with ADHD and the PP deficits associated with RD (Narhi & Ahonen, 1995; Nigg, 1999; Nigg et al. , 1998; Reader, Harris, Schuerholz, & Denckla, 1994; Robins, 1992). The discrepancy between these findings and the results of Pennington et al. (1993) may be attributable to differences in sample severity or differences in the batteries of EF measures, or these results may simply be less consistent because of the relatively small samples used in virtually all of these studies.

The Present Study

A 2 (RD vs. no RD) \times 2 (ADHD vs. no ADHD) factorial design was used to compare the performance of individuals with RD and ADHD on measures of EF and PA. Participants in this study were recruited through local schools, facilitating a direct test whether results obtained in previous studies of clinical populations replicate in a nonreferred sample. Because the number of individuals with available data does not yet provide sufficient power for behavioral genetic analyses, we describe the results of phenotypic analyses designed to expand in several ways on previous studies of the neuropsychological correlates of RD, ADHD, and their overlap.

A wide range of EF measures were included in the battery, allowing us to test for differential associations between RD and ADHD and different domains of EF. On the basis of previous research, we hypothesized that ADHD would be associated with deficits on measures of inhibition and verbal working memory but not on measures of the ability to shift cognitive set. In contrast, we predicted that RD would be associated with either no EF deficits at all or mild deficits on measures of verbal working memory.

The present sample is the largest that has been used to test for a double dissociation between RD and ADHD on measures of PA and EF. It was predicted that a double dissociation would reveal a significant association between RD and deficits in PA independent of ADHD as well as a significant relation between ADHD and inhibitory deficits independent of RD.

The performance of the four groups on the PA and EF measures was compared to test more rigorously the validity of the phenocopy hypothesis as an explanation for comorbidity between RD and ADHD.

Finally, a series of ancillary analyses were conducted to test whether the cognitive deficit associated with either disorder was mediated by general cognitive ability or comorbid psychopathology or varied as a function of gender or age.

Method

Participants

Participants between 8 and 16 years of age completed the measures described here as part of the Colorado Learning Disabilities Research Center (CLDRC) twin project, an ongoing study of the genetics of learning disabilities (DeFries et al., 1997). A two-stage process was used to ascertain the sample. First, through collaboration with school administrators and personnel, all twin pairs from 27 school districts within a 150-mile radius of the Denver/Boulder area were contacted, and parental permission was requested to review each child's academic records for evidence of learning problems. If either member of a twin pair manifested a positive history of learning difficulties (e.g., low achievement test scores, referral to a tutor, reports by classroom teachers or school

psychologists), both members of the pair were invited to complete an extensive battery of tests in the CLDRC laboratories. In addition, a comparison sample of twin pairs was recruited in which neither twin exhibited evidence of learning problems in their school records.

Individuals with pervasive developmental disorder, an early closed-head injury, or documented evidence of other more specific genetic or environmental risk factors such as maternal alcohol or substance use during pregnancy, neurofibromatosis, fragile X syndrome, or other sex chromosome anomalies were eliminated from the analyses described in this report, as were participants with Full-Scale IQ (FSIQ) scores below 70. For any participants who were taking psychostimulant medication at the time of the assessment, the parent was asked to withhold medication for 24 h before the child's participation in the study to minimize the influence of medication on the participant's performance on the cognitive measures.

Issues in the use of twins for phenotypic analyses.

The use of twins for analyses in which each twin is considered as an individual data point presents a methodological difficulty because the scores of the twins in each pair do not represent fully independent observations. Therefore, one twin was selected at random from each twin pair in which both twins met inclusion criteria for the study. Results were virtually identical when analyses were repeated in a sample in which the selected twin was replaced by the cotwin who was excluded from the first set of analyses, suggesting that the random selection of one twin from each of these pairs did not inadvertently bias the results. A dummy code for zygosity was included in all initial models to control for any differences between participants from monozygotic and dizygotic pairs, but this code was dropped from all final models because it had no significant impact on any result.

Assignment of individuals to groups.

Participants were identified as RD if they had a positive school history of reading problems and met criteria for RD on the battery of reading achievement measures described subsequently. ADHD was diagnosed based on parent ratings to subdivide the sample of individuals with RD into a group of participants with RD alone ($n = 93$) and a group with both RD and ADHD ($n = 48$). Two methods were used to identify participants for the ADHD-only group. First, individuals from the sample of twin pairs in which neither twin exhibited a school history of learning difficulties or met criteria for RD were assigned to the ADHD-only group if they met diagnostic criteria for ADHD based on parent report ($n = 28$). In addition, many of the participants in the sample selected because of a school history of learning difficulties exhibited general academic difficulties but did not meet criteria for RD. Because children with ADHD often exhibit learning problems (e. g. , poor grades, low productivity, academic underachievement) even in the absence of a specific learning disability (e. g. , Barkley, 1998), participants with ADHD who were from pairs in which at least one twin had a positive school history of learning difficulties but both twins scored within 1.0 SD of the control mean on the composite measure of reading achievement were also included in the ADHD-only group ($n = 24$). Mean scores of participants assigned to the ADHD-only group by these two procedures

were not significantly different on any of the dependent measures, so the two groups were combined for the analyses described in this report. Finally, the comparison sample ($n = 102$) included individuals from twin pairs in which neither twin had a school history of learning difficulties or met criteria for RD or ADHD.

Procedures

The reading, IQ, and PA measures were administered in an initial testing session conducted at the University of Colorado at Boulder Department of Psychology and Institute for Behavioral Genetics. The EF tasks and measures of ADHD and other disruptive behaviors were obtained during a second session scheduled approximately 2 weeks later at the University of Denver Department of Psychology. All measures at both sites were administered by trained examiners with at least a Bachelor of Arts degree who had previous experience working with children. Examiners who administered the measures of psychopathology were graduate students in the clinical child psychology doctoral program at the University of Denver. All examiners were unaware of the diagnostic status of the child and the results of the testing conducted at the other sites.

Diagnostic Measures and Operational Definition of RD and ADHD

Assessment of RD.

The description of RD in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) specifies that reading achievement scores must fall significantly below both the score typical of other children of the same age and the score that would be expected based on the individual's overall cognitive ability. However, several studies indicated that the same etiological factors and neurocognitive deficits are associated with RD with and without an IQ discrepancy, suggesting that the inclusion of an IQ discrepancy as a diagnostic criterion adds little to the external validity of the diagnosis (e. g. , Fletcher, Francis, Rourke, Shaywitz, & Shaywitz, 1993; Pennington, Gilger, Olson, & DeFries, 1992; Siegel, 1989). In contrast, Wadsworth, Olson, Pennington, and DeFries (2000), in their twin study, found that the etiology of RD varied as a linear function of IQ, suggesting that IQ differences may be relevant to the definition of RD. Because this issue has not been resolved conclusively and to ensure that the current sample met stringent criteria for RD, only those individuals with reading scores below both the achievement that would be expected based on their age and their overall intellectual functioning were included in the RD sample.

A standardized, normally distributed composite score was created based on a previous discriminant function analysis (DeFries, 1985) of reading subtests from the Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970). As recommended by Reynolds (1984), a standard score 1.65 SD below the mean of the comparison sample was used as the age-discrepancy criterion for RD. This cutoff selects approximately 5%

of the control sample, a prevalence that is consistent with estimates from epidemiological studies (e. g. , S. E. Shaywitz et al. , 1990).

The revised version of the Wechsler Intelligence Scale for Children (WISC-R; Wechsler, 1974) was used to assess the FSIQ of each participant. A standardized, normally distributed IQ discrepancy score was derived based on the child's FSIQ and reading discriminant score using the regression procedure described by Frick et al. (1991) and Pennington et al. (1992). This method takes into account the expected regression to the mean of reading scores when an individual has an IQ score higher or lower than the population mean. This avoids the overselection of children with high IQ scores and the underselection of children with low IQ scores that occurs when a simple subtraction formula is used (Kamphaus, Frick, & Lahey, 1992). A score 1.65 SD below the control mean on the standardized discrepancy score was used as the criterion for IQ-discrepant RD.

Assessment of ADHD.

The Attention Deficit Disorder with Hyperactivity module from the parent-report version of the Diagnostic Interview for Children and Adolescents, Parent Report Version (DICA-P; Reich & Herjanic, 1982) was used to assess ADHD. Individuals were diagnosed with ADHD based on the diagnostic criteria provided in the third edition of the DSM (American Psychiatric Association, 1980). The interinterview reliability of the DICA-P is reported to be .82, and diagnoses based on the DICA-P have been shown to be concordant with clinical assessments approximately 90% of the time (Welner, Reich, Herjanic, Jung, & Amado, 1987). Because of the substantially higher availability of maternal report (approximately 95% of all participants) compared with paternal report (51%), maternal report was used for the analyses reported here.

PA Measures

Pig Latin Test (Olson et al. , 1989).

This task requires the participant to transform words into their pig Latin equivalent. The participant is told the rules for transforming the words (e. g. , move the initial sound to the end of the word, and add the /a/ sound) and completes nine practice words read by the examiner. The examiner is permitted to provide assistance as necessary during the practice items to ensure that the participant understands the task. The test trials then requires the participant to transform words read by the examiner into their equivalent in pig Latin. Total number correct was used as the dependent variable.

Phoneme Deletion Task (Olson et al. , 1994).

This task is based on the Bruce (1964) phoneme deletion task and the Rosner and Simon (1971) auditory analysis task. The examiner reads aloud a pronounceable nonword (e. g. , plift) and then asks the participant to say the word after removing one phoneme (e. g. , say plift without the /p/). Total correct responses was used as the dependent variable.

Lindamood Auditory Conceptualization Test (LAC; Lindamood & Lindamood, 1971).

This task uses colored blocks to represent phonemes and requires the participant to add, remove, or transpose blocks to reflect changes in nonwords spoken by the examiner. For example, the participant might be shown three different colored blocks in a row and told "If this says aps, show me asp. " The correct response would then be to exchange the position of the second and third blocks. The LAC total score was used as the dependent variable.

Creation of a PA Composite Score

Previous exploratory and confirmatory factor analysis in the CLDRC sample indicated that the three PA measures loaded on a single factor (Olson et al. , 1994). Exploratory factor analysis in the present sample also indicated that the three PA measures all loaded above . 85 on a single factor on which none of the EF tasks loaded above . 20. Therefore, to simplify interpretation, a PA composite score was created by computing the mean of the z scores for the three PA tasks. Estimates of internal reliability were high for all three phoneme awareness measures ($\alpha = . 88-. 92$) and for the PA composite score (reliability estimate = . 94 based on procedures described by Nunnally, 1978).

EF Measures

A range of EF tasks were administered to test whether deficits in ADHD or RD were restricted to specific EF domains. The EF measures were added to the current testing battery at three different points in time. Therefore, the PA and set-shifting measures were completed by all participants (N = 314), but only a subset of the overall sample completed the measures of inhibition (n = 258) and working memory (n = 198).

Wisconsin Card Sorting Test (WCST; Heaton, 1981).

The WCST requires the individual to sort 128 cards to match either the color, form, or number of shapes on target cards. The standard examiner-administered WCST was used for the present study. After each trial the examiner verbally informs the participant whether the response is correct or incorrect. After the participant sorts 10 consecutive cards correctly (e. g. , matching to the color of the shapes on the target cards), the rule changes so that the sorting rule is based on one of the other properties of the target stimulus. Therefore, WCST performance depends on the ability to maintain a rule in memory and appropriately shift set to a new rule when presented with feedback that the previous rule is now incorrect.

Previous studies showed that WCST performance discriminates adults with damage to the prefrontal cortex from those with lesions in other brain regions (Heaton, 1981). Studies of WCST performance have been less consistent in children with ADHD, however; only 5 of the 10 studies reviewed by Pennington and Ozonoff (1996) found significant differences between individuals with and without ADHD. The primary

dependent variable for the present study was total perseverative errors, defined as errors that either are consistent with the previous sorting rule or adhere to an incorrect scoring rule generated by the participant. Ozonoff (1995) reported test-retest reliability of .90 over a period of 2 years for perseverative errors in a sample of individuals with a learning disability.

Contingency Naming Test (CNT; Taylor, 1988).

This test provides a second measure of the capacity to appropriately maintain and shift cognitive set. The CNT consists of three rows of nine different colored stimuli (pink, blue, or green). Each stimulus has a large outside shape (circle, square, triangle) and a smaller inside shape that may be congruent or incongruent with the outside shape. The first two warm-up trials of the CNT require the participant to name as rapidly as possible either the color of each stimulus or the large outside shape. The third trial then introduces a more difficult rule, wherein the participant is to name the color if the two shapes are the same and the large outside shape if the two shapes are different. This rule remains the same for the final experimental trial, with the exception of nine stimuli that are marked with a backward arrow. For these nine stimuli, the rule is reversed (e. g. , name the shape if the shapes are congruent and the color if the shapes are not congruent). Therefore, successful performance on the CNT requires the individual to maintain the primary rule in memory while inhibiting the rule and shifting to the new rule for the marked items. Before each trial nine practice stimuli are administered to be certain that the child understands the new rule. The dependent variable used for the present analyses is the total number of errors on the two experimental trials.

Continuous Performance Test (CPT; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956).

The CPT provides an index of a child's ability to sustain attention and inhibit inappropriate responses over an extended visual task. The review by Corkum and Siegel (1993) suggests that the CPT discriminates children with and without ADHD more reliably than any other cognitive measure. Indeed, although important concerns have been raised regarding the ecological validity of the CPT as a measure of ADHD (Barkley, 1991), children with ADHD exhibit impaired performance on a variety of different tasks based on the CPT paradigm (e. g. , Barkley, DuPaul, & McMurray, 1990; Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989; Halperin et al. , 1988; Loge et al. , 1990; Mariani & Barkley, 1997). Moreover, some studies suggest that CPT performance may also be associated with general academic underachievement (Aylward, Gordon, & Verhulst, 1997; Gordon, Mettelman, & Irwin, 1994), and results from one small sample indicate that individuals with RD may also exhibit significant impairment on some versions of the CPT (Kupietz, 1990).

The version of the CPT used in this study (Garfinkel & Klee, 1983) consists of two 6-min blocks of 500 letters each. An initial 500-letter warm-up sequence requires the child to press a key marked clearly with tape each time a white S is presented on the screen. For the second 500-letter sequence, the participant is instructed to press the key only when a white S is followed immediately by a blue T. Therefore, to complete this second task successfully, the child must inhibit the response that was previously correct and

implement the new rule. Fifty correct sequences appear over the course of the 500 letters presented during the task. Data for 6 participants (2 control, 1 RD only, 1 ADHD only, and 2 RD + ADHD) were not included in the analyses, because the participants responded correctly to fewer than 50% of the correct sequences, suggesting that they may have misunderstood the task. The dependent variable used for the present study was the total number of commission errors, defined as incorrect responses made after a sequence other than the target sequence. Because test-retest data are not available for the present sample, the task was subdivided into five equal blocks of stimuli to test the internal reliability of the commission error composite. The total errors of commission in each block were highly correlated, as reflected in the estimate of internal reliability for the commission error composite score ($\alpha = .92$).

Stopping Task (Logan & Cowan, 1984).

This test is a computerized measure of inhibitory control based on the dual-process "race model" of inhibition (e. g. , Logan, 1994; Logan & Cowan, 1984). Participants are seated in front of the computer and are instructed to place their left index finger on a key on the keyboard marked with an X and their right index finger on a key marked with an O. The task consists of primary task trials and stop-signal trials. On primary task trials, the letter X or O is presented in the center of the monitor, and the participant responds by pressing the corresponding key on the keyboard. For stop-signal trials the same visual stimulus appears, but an auditory tone is also presented shortly after the X or the O appears on the screen. The participant is instructed to press the X or O key as rapidly as possible for each trial but to inhibit the keypress on each of the trials on which the tone is presented. Participants are told to try to inhibit their response if possible but not to worry if they cannot stop their response (Logan, Schachar, & Tannock, 1997). Participants are also instructed not to wait to see whether the stop signal will occur but instead to respond to the primary task stimulus as quickly as possible.

Each participant completed six blocks of trials. The first block consisted of 32 primary task trials, allowing the child to become familiar with the task. The second practice block of 48 trials included 16 stop-signal trials randomly interspersed with 32 primary task trials. The remaining four blocks comprised the experimental trials. Each experimental block consisted of a randomly ordered presentation of 32 primary task trials and 16 stop-signal trials. The stop-signal trials consisted of four trials each on which the tone was presented 500 ms, 350 ms, 250 ms, and 100 ms before the child's mean reaction time (MRT) on the primary task trials of the previous block. The child's MRT on the first practice block was used to compute the delay intervals for the first experimental block. Data from 7 participants were not used because they failed to respond correctly to at least 75% of the primary task trials (2 control participants, 2 participants with RD, 1 participant with ADHD, and 2 participants with RD + ADHD).

Schachar and Logan's (1990) procedure was used to calculate stop-signal reaction time (SSRT), the primary dependent measure used in the present study. SSRT provides an index of the duration of the inhibitory process independent of the participant's MRT. This measure was chosen because it has been shown to discriminate children with ADHD

from children without ADHD (Schachar & Logan, 1990) and is sensitive to the effects of stimulant medication (Tannock, Schachar, Carr, Chajczyk, & Logan, 1989). As suggested by Logan et al. (1997), SSRT was calculated by averaging the SSRT estimates at the four delay intervals. If at any delay interval the participant's probability of responding was above . 90 (suggesting that the participant misunderstood the task or was not attempting to inhibit their response) or below . 10 (suggesting that the participant was waiting for the tone), the SSRT for that interval was not included in the summary SSRT score.

As noted, results from some studies suggest that the reaction time of individuals with ADHD is slower and more variable than that of individuals without ADHD. Although there were no significant differences in reaction time on the primary task trials among the four groups (control M = 666. 1; RD only M = 696. 1; ADHD only M = 689. 4; ADHD + RD M = 671. 5; $p > . 15$), the mean of the individual standard deviations of primary task reaction times was significantly larger in all three clinical groups (RD only M = 192. 9; ADHD only M = 202. 7; ADHD + RD M = 211. 7) than in the comparison group (M = 161. 3). Therefore, the standard deviation of response time for each individual was covaried to ensure that any significant differences in SSRT among the groups were not explainable by differences in reaction time variability. In addition, the number of errors on the primary task trials were covaried to control for marginally significant differences among the groups ($p = . 05$), but this covariate was dropped from the final analyses because it did not impact any result significantly. The SSRT composite exhibited adequate internal reliability across the four delay intervals ($\alpha = . 77$) and the four experimental blocks ($\alpha = . 81$).

Sentence Span task.

This is a working memory measure adapted by Siegel and Ryan (1989) from the procedure developed by Daneman and Carpenter (1980). This task requires the participant to process new verbal information continually while storing the results of this process for later recall. Participants are instructed to provide the last word for a set of simple sentences read by the examiner (e. g. , "I throw the ball up and then it comes ... ") and are told that they will be asked to reproduce the words that they provided after all sentences in that set have been completed. The task begins with a block of three two-sentence sets and increases in difficulty by adding one additional sentence per block up to a total of six sentences. The dependent measure is the number of sets completed correctly (α for the composite score = . 76).

Counting Span task (Case, Kurland, & Goldberg, 1982).

This is a second measure of verbal working memory that uses a procedure similar to the sentence span task. The participant is instructed to count aloud the number of yellow dots dispersed randomly on a set of $8\frac{1}{2} \times 11$ -in. ($21. 25 \times 27. 5$ -cm) cards. After all cards in a set are completed, the participant is asked to recall, in temporal order, the number of yellow dots that appeared on each of the cards in the set. Similar to the Sentence Span task, there are five blocks with three sets per block (two to six cards per set). The dependent variable is the total number of correct sets ($\alpha = . 81$).

Trailmaking Test (e. g. , Reitan & Wolfson, 1985).

Part A of this test is a warm-up task that requires the participant to use a pencil to connect, in ascending order, a series of circles containing numbers. Part B also involves connecting circles, but this time each circle contains either a number or a letter. The participant is instructed to connect the circles in ascending order, alternating between numbers and letters (i. e. , 1, A, 2, B, 3, C ...). Therefore, this task requires the individual to maintain their place in both the alphabetical and numerical series while also remembering whether a letter or number should be next in the series. Total response time on Part B was used as the primary dependent measure for the present study. Number of errors on Part B was also evaluated as a potential dependent variable but was not used because of restricted range (87% of participants made fewer than two errors). Previous studies reported high alternate-form reliability ($r = .89-.92$; Charter, Adkins, Alekoumbides, & Seacat, 1987) and adequate test-retest reliability ($r = .66-.86$; Goldstein & Watson, 1989) for Part B of the Trailmaking Test.

The Stroop Color and Word Test (Golden, 1978).

This measures the participant's ability to respond selectively to one dimension of a multidimensional stimulus. Each of the three trials of the Stroop Test uses a card containing five columns of 20 stimuli. The participant is asked to complete as many of the stimuli as possible within 45 s. The Word trial requires the participant to read as rapidly as possible the names of colors (red, blue, and green) printed on the card in black ink. The subsequent Color trial requires the participant to name consecutively the color of each stimulus on a card containing colored patches of red, blue, or green ink. Finally, the card for the Interference trial contains the words red, blue, and green printed in a different color, and the participant is asked to name the color of the ink of each stimulus. The three trials of the Stroop are hypothesized to assess reading speed, naming speed, and interference control. However, a low number of positive responses on the Interference trial could indicate true deficits in interference control or might simply reflect a deficit in naming speed. Therefore, if ADHD is associated with a specific deficit in interference control (Barkley, 1998), the main effect of ADHD on the Interference trial should remain significant when scores on the Word and Color trials are controlled. An interference control score was operationalized by subtracting each participant's mean z score on the Word and Color trials from their z score on the Interference trial.

Principal-Components Analysis of the EF Measures

Previous analyses in a subset of the present data set (Pennington, 1997) and in independent samples (e. g. , Mariani & Barkley, 1997) suggest that EF tasks tap more than one dimension of functioning. Therefore, after data-cleaning procedures were completed, a principal-components analysis (PCA) was conducted to reduce the data and simplify interpretation. The direct oblimin method was used to rotate the components to obtain the optimal solution. This oblique method of rotation permits the obtained components to correlate and, therefore, requires fewer a priori assumptions about the nature of the relations among the variables than the orthogonal method of rotation. The same number of components and similar loadings for each variable were obtained when analyses were conducted with an orthogonal method of rotation.

All eight EF measures were included in the initial PCA, but the Stroop Test Interference control score was dropped from the final PCA because it did not load above .30 on any component. PCA of the seven remaining tasks accounted for 68% of the total variance after rotation and yielded three components with eigenvalues greater than 1 (Table 2). The three putative measures of verbal working memory comprised the first component (Trailmaking Test, Counting Span, and Sentence Span). The two tests of behavioral inhibition (SSRT and CPT commission errors) loaded most strongly on the second component, and the third component included the two tasks that assess ability to shift cognitive set (WCST perseverative errors and CNT errors). Based on these results, working memory, inhibition, and set-shifting composite scores were created by summing the z scores of the tests that loaded significantly on each component. Estimates of internal reliability were adequate for the inhibition (.88) and working memory (.80) composite scores. Moreover, although the reliability of the set-shifting composite could not be computed because reliability data were not available for the CNT, both set-shifting measures correlated highly with the composite score ($r = .76-.84$). Because the Stroop Color and Word Test Interference control score did not load significantly on any of the three obtained factors, it was analyzed separately as a fourth measure of executive functioning.

Data Cleaning and Analysis

Before any statistical analysis, the distribution of each variable was assessed for outliers. Outliers were defined as scores that fell more than 3 SD from the mean of the overall sample and more than 0.5 SD beyond the next most extreme score. Each outlying score was adjusted to a score of 0.5 SD beyond the next highest score, with multiple outliers rescored to 0.1 SD apart. Adjustments were made to CPT false alarms for 2 individuals from the RD + ADHD group and 1 individual from the ADHD-only group, to SSRT for 1 individual from the RD + ADHD group and 1 individual from the RD-only group, and to Trailmaking Test time for 1 individual from the RD-only group. After these adjustments, the distribution of each variable was assessed for significant deviation from normality. A logarithmic transformation was implemented to approximate a normal distribution for variables with skewness or kurtosis greater than 1 (this included all EF tasks with the exception of Sentence Span and Counting Span).

The presence of significant IQ differences among the four groups presents a methodological dilemma. Specifically, some authors (e.g., Werry, Elkind, & Reeves, 1987) argued that IQ should be included as a covariate to ensure that deficits in clinical groups cannot be explained more parsimoniously by group differences in intelligence. In contrast, others (e.g., Barkley, 1997) suggested that ADHD may directly cause mild IQ deficits in comparison to individuals without ADHD, and that controlling for IQ removes a portion of the variance that is associated specifically with ADHD. Because this issue has not been resolved conclusively, all results are reported both without controlling FSIQ and with FSIQ covaried.

Analyses were conducted in the following order to test each specific question. To test whether either RD or ADHD was significantly associated with deficits on the PA and EF

measures independent of the other disorder, separate 2×2 (RD \times ADHD) factorial analyses of variance (ANOVAs) or covariance (ANCOVAs) were conducted for each individual cognitive measure and for the four composite scores (e. g. , phoneme awareness, inhibition, set shifting, and working memory). If either the main effects of RD or ADHD or the RD \times ADHD interaction was significant, post hoc comparisons were conducted among the four groups using the Newman-Keuls correction for multiple comparisons. To test for a significant double dissociation between RD and ADHD on measures of PA and EF, separate repeated measures multivariate ANOVAs and multivariate ANCOVAs were conducted with one of the EF composites and the PA composite as the within-participants factor. A double dissociation would be indicated by significant RD \times Domain and ADHD \times Domain interactions, such that the disorders are associated with contrasting deficits on PA and EF tasks. Finally, the phenocopy hypothesis was tested by comparing the profile of the RD + ADHD group to the groups with RD or ADHD alone. The phenocopy hypothesis would be supported if the RD + ADHD group exhibited the deficits associated with only one of the disorders.

Results

Descriptive and Diagnostic Variables

The mean age was not significantly different among the four groups (Table 3). In contrast, all three clinical groups were associated with lower socioeconomic status as measured by the Hollingshead (1975) two-factor inventory. The mean Verbal and Performance IQ and FSIQ scores of the three clinical groups also fell significantly below the mean of the comparison group.

As expected based on the way the sample was defined, the mean number of ADHD symptoms was higher for the ADHD and RD + ADHD groups than the RD and comparison groups, and the RD and RD + ADHD groups had lower means on the reading measures than the ADHD and comparison groups. Two additional findings are notable on the diagnostic measures. First, although individuals with RD alone did not score above the diagnostic cutoff for ADHD on the DICA-P, they exhibited significantly more symptoms of ADHD than the comparison group. Similarly, individuals with ADHD alone scored significantly lower on the tests of reading achievement than those in the comparison group. These findings remained significant when IQ was covaried, suggesting that individuals with either ADHD or RD alone exhibit subclinical manifestations of the other disorder even when they do not meet full criteria for the other diagnosis. The second important finding on the diagnostic measures is that individuals with RD + ADHD did not exhibit more severe reading difficulties than those with RD alone or more symptoms of ADHD than those with ADHD alone. This result suggests that the co-occurrence of RD and ADHD is not confounded with severity, simplifying the interpretation of the profiles of the three groups on the cognitive tests.

Comparison of the Means of the Four Groups

Analyses of unadjusted means.

Table 4 presents the unadjusted means of the four groups on the individual measures and composite scores. Results of 2×2 (RD \times ADHD) ANOVAs revealed a significant main effect of RD for all measures except Stroop Color and Word Test Interference control. In contrast, the ADHD main effect was significant only for the inhibition measures and the PA composite. The pattern of means of the four groups on the EF measures is generally consistent with the main effects. Planned comparisons indicated that the comorbid group was significantly more impaired than all other groups on the inhibition composite and the working memory composite.

Analyses controlling FSIQ.

FSIQ was significantly related to scores on all composites, so a series of 2×2 (RD \times ADHD) ANCOVAs were conducted to test whether RD or ADHD was significantly associated with EF or PA deficits after controlling for FSIQ differences among the groups. When FSIQ was covaried (Figure 1 - Marginal means on the phoneme awareness and executive function composites controlling Full-Scale IQ. Means with no common subscripts are significantly different, ($p < .05$). (RD = reading disability; ADHD = Attention-deficit/hyperactivity disorder.)

Figure 1), the main effects of both ADHD, $F(1,253) = 7.29$, $p < .01$, $\eta^2 = .034$, and RD, $F(1,253) = 3.96$, $p < .05$, $\eta^2 = .019$, remained significant for the inhibition composite. Similarly, the RD main effect was still significant for both the working memory composite, $F(1,194) = 7.81$, $p < .01$, $\eta^2 = .043$, and the PA composite, $F(1,309) = 121.80$, $p < .001$, $\eta^2 = .278$. In contrast, none of the main effects or interactions were significant for the set-shifting composite ($p > .25$, all $\eta^2s < .004$), suggesting that the significant RD main effect was attributable to FSIQ differences between individuals with and without RD.

Analyses controlling for subclinical elevations of the other disorder.

The analyses described previously test whether ADHD or RD are associated with PA or EF deficits independent of the influence of the other categorical diagnosis. However, a growing body of research suggests that categorical diagnostic cutoffs for RD and ADHD artificially dichotomize what is probably a continuous distribution of liability (e. g. , Barkley, 1998; DeFries & Alarcón, 1996; Levy et al. , 1997; Willcutt, Pennington, & DeFries, 2000a). Consistent with this hypothesis, individuals with RD who did not meet criteria for ADHD still exhibited a higher number of symptoms of ADHD than those in the comparison sample, and individuals with ADHD scored significantly lower than those in the comparison sample on measures of reading achievement. Moreover, zero-order correlations displayed in Table 5 reveal that scores on the neuropsychological measures were generally related to both the number of ADHD symptoms exhibited by the participant and the participant's reading/IQ discrepancy score. Therefore, the cognitive deficits putatively associated with RD or ADHD could potentially be attributable to subclinical elevations of the other disorder rather than to an association with the primary disorder per se (e. g. , Nigg et al. , 1998). Two sets of analyses were conducted to test this hypothesis. First, additional ANCOVAs were conducted with ADHD or RD as the independent variable and the dimensional measure of the other disorder included as a

covariate along with FSIQ. Second, multiple regression models were fitted in which scores on each neuropsychological composite were predicted simultaneously by scores on the continuous measures of reading achievement, ADHD symptoms, and FSIQ.

For the inhibition composite, the main effect of ADHD group membership remained significant when FSIQ, reading achievement, and the standardized IQ/reading discrepancy score were covaried, $F(1,253) = 7.46, p < .01, \eta^2 = .030$, whereas the RD main effect was no longer significant after covarying FSIQ and total symptoms of ADHD, $F(1,254) = 2.29, p > .15, \eta^2 = .010$. Similarly, results of the multiple regression analysis predicting scores on the inhibition composite revealed significant effects of ADHD symptoms ($\beta = .17, p < .001$) and FSIQ ($\beta = .14, p < .01$) but only a statistical trend toward significance for reading ($\beta = .08, p < .10$). In contrast, the main effect of RD remained significant for both the working memory composite, $F(1,194) = 7.03, p < .01, \eta^2 = .039$, and the phoneme awareness composite, $F(1,310) = 133.01, p < .001, \eta^2 = .296$, when covarying FSIQ and total symptoms of ADHD. Parallel multiple regression analyses also indicated that reading achievement was significantly associated with lower scores on the PA composite ($\beta = .49, p < .0001$) and the working memory composite ($\beta = .19, p < .001$) when FSIQ and ADHD symptoms were included in the model, whereas symptoms of ADHD were not significantly associated with deficits on either composite. Moreover, the RD main effect remained significant when the Digits Forward score from the WISC-R Digit Span subtest was also included as a covariate, suggesting that the working memory deficit associated with RD is not explainable by a deficit in verbal short-term memory. Taken together, these results suggest that inhibitory difficulties are associated primarily with ADHD, whereas deficits in PA and working memory are associated specifically with RD, providing preliminary evidence of a double dissociation between RD and ADHD.

Tests of the double dissociation and phenocopy hypotheses.

Repeated measures MANCOVAs controlling FSIQ were used to test directly for a significant double dissociation between RD and ADHD on measures of PA and EF. Results when PA and inhibition were included in the within-participants factor revealed significant interactions of RD \times Domain, $F(1,251) = 54.42, p < .001, \eta^2 = .160$, and ADHD \times Domain, $F(1,251) = 8.60, p < .01, \eta^2 = .040$. These interactions indicated that RD was more strongly associated with PA deficits than inhibition deficits, whereas ADHD was associated with greater deficits in inhibition than PA. RD \times Domain interactions were also significant for working memory and PA, $F(1,190) = 44.03, p < .001, \eta^2 = .205$, and set shifting and PA, $F(1,296) = 57.54, p < .001, \eta^2 = .165$, indicating that RD is more strongly associated with deficits in PA than deficits in any EF domain. In contrast, the ADHD \times Domain interaction was not significant when the within-participants factor included either PA and working memory, $F(1,190) = 0.46, p > .50, \eta^2 = .002$, or PA and set shifting, $F(1,296) = 1.02, p > .25, \eta^2 = .005$, suggesting that the RD \times ADHD double dissociation is restricted to measures of inhibition and PA.

The three-way RD \times ADHD \times Domain interactions were not significant when PA and any of the three EF measures were included in the within-participants factor ($p > .50, \eta^2$

< .001 for all EF measures). Moreover, the RD + ADHD group was most impaired on virtually all EF measures, providing converging evidence against the phenocopy hypothesis as an explanation for comorbidity between RD and ADHD in this sample.

The Influence of Gender and Age on the Cognitive Correlates of RD and ADHD

To understand the data more completely, additional analyses were conducted to test whether the present results differed as a function of gender or age. Dummy codes were created to subdivide the sample into males and females or individuals younger than age 12 and 12 years old and older. No significant main effects or interactions were obtained for gender (all p s > .30). In contrast, the main effect of age was significant for all composite measures, inhibition $F(1,249) = 13.13, p < .001$; set shifting $F(1,306) = 26.14, p < .001$; working memory $F(1,190) = 56.13, p < .001$; PA $F(1,306) = 49.24, p < .001$; a finding that simply reflects the development of executive functions and PA throughout childhood and adolescence. In a more interesting result, a $2 \times 2 \times 2$ (RD \times ADHD \times Age) ANCOVA controlling FSIQ revealed a significant ADHD \times Age interaction for the inhibition composite, $F(1,248) = 5.66, p < .05, \eta^2 = .027$, such that older children with ADHD were more impaired than younger children with ADHD compared with children the same age without ADHD. A closer examination of the marginal means of the four groups suggests that, whereas the inhibitory capacity of most children improves dramatically with age (Figure 2 - Marginal means on the inhibition composite in younger and older participants controlling Full-Scale IQ. Means with no common subscripts are significantly different, ($p < .05$). (RD = reading disability; ADHD = Attention-deficit/hyperactivity disorder. (Figure 2), this improvement is much more gradual for children with ADHD as a whole and is virtually null for children with RD + ADHD. No significant interactions with age were obtained for any other composite.

The Influence of Comorbid Disruptive Behavior Disorders

The DICA-P modules for oppositional defiant disorder (ODD) and conduct disorder (CD) were added to the assessment battery more recently than the other measures and were, therefore, available for only a subset of participants ($n = 157$). Because too few individuals in this subset met criteria for ODD and CD to subdivide the sample based on these categorical diagnoses, all analyses were repeated with the number of symptoms of ODD and CD included as a dimensional covariate. All previously significant findings remained significant, suggesting that the inhibition deficits associated with ADHD and the deficits in working memory and PA associated with RD are not explainable by comorbidity with other disruptive behavior disorders.

Discussion

This study used a community sample of twins to examine the cognitive profile of individuals with RD and ADHD on a battery of PA and EF measures. A principal-components analysis of the EF tasks revealed three underlying dimensions: inhibition, set shifting, and working memory. The primary goals of the study were to (a) test whether RD or ADHD was significantly associated with PA or EF deficits independent of the other diagnosis, (b) test for a significant double dissociation between RD and ADHD on measures of PA and each of the dimensions of EF, and (c) use the profile of the comorbid group on the EF and PA measures to test the validity of the phenocopy hypothesis as an explanation for comorbidity between RD and ADHD.

Executive Functions in ADHD

The present results provide clear support for theories that have proposed that ADHD is associated with a primary deficit in behavioral inhibition (e. g. , Barkley, 1997, 1998; Pennington & Ozonoff, 1996; Quay, 1988). Individuals with ADHD scored significantly lower on the inhibition composite than those without ADHD when controlling for RD, IQ, and symptoms of other disruptive behavior disorders. The inhibitory deficit was especially strong in older children with ADHD, providing further objective evidence against the historically popular notion that symptoms of ADHD and any associated impairments tend to dissipate as a child reaches adolescence (see Barkley, 1998, for a review).

In contrast to the findings for the inhibition domain, the ADHD main effect was not significant for the set-shifting or verbal working memory composites, and only the ADHD + RD group scored significantly lower than controls on these measures. This result suggests that deficits in working memory or set shifting obtained in some previous studies of ADHD may be restricted to those children who also have RD and may not be associated with ADHD per se. ADHD was also not significantly associated with deficits in interference control on the Stroop Color and Word Test. Other studies consistently found that children with ADHD exhibit mild impairment on all three trials of the Stroop Test (Pennington & Ozonoff, 1996), but none of these studies tested whether the performance of individuals with ADHD was significantly more impaired on the Interference trial than on the preceding trials assessing word reading and color naming. Several studies suggested that children with ADHD are significantly impaired on measures of rapid automatized naming (e. g. , Carte, Nigg, & Hinshaw, 1996; Nigg et al. , 1998; Tannock et al. , 2000). In combination with these studies, the present findings suggest that ADHD deficits on the Interference trial of the Stroop Test may be attributable to difficulties with speeded verbal naming and not a deficit in interference control per se.

PA in RD

The present results indicate that individuals with RD have a large deficit in PA compared with those without RD ($\eta^2 = .278$). This finding is consistent with numerous studies (e. g. , Fletcher et al. , 1999; Olson et al. , 1989; Pennington et al. , 1993; B. A. Shaywitz et al. , 1995) and provides converging evidence that deficits in PA or other measures of phonological processing represent the core deficit in RD.

Tests for a Double Dissociation Between RD and ADHD

The present results are generally consistent with a double dissociation between RD and ADHD on measures of PA and inhibition. Specifically, ADHD was associated most strongly with an inhibition deficit and was not significantly associated with a deficit in PA when FSIQ was covaried. In contrast, RD was associated with an extremely large PA deficit but was not associated with deficits in inhibition when FSIQ and symptoms of ADHD were controlled. However, it should be noted that the RD main effect was significant when either ADHD or FSIQ was controlled in isolation, and that even when both variables were covaried the marginal means for the RD groups were in the direction indicative of greater impairment. Moreover, multiple regression analyses revealed a statistical trend toward significance for the relation between the dimensional measure of reading and the inhibition composite when FSIQ and symptoms of ADHD were controlled. These results are consistent with the findings of a 1999 doctoral dissertation (Purvis, 1999), suggesting that additional research will be useful to test whether RD is significantly associated with disinhibition independent of comorbid ADHD.

Although the current findings are consistent with at least a partial double dissociation between RD and ADHD on measures of inhibition and PA, this was not the case for the other EF dimensions. Neither RD nor ADHD was associated with a significant deficit on the set-shifting measures after controlling FSIQ. In contrast, a somewhat unexpected result indicated that RD was significantly associated with impairment in verbal working memory. The RD deficit remained significant even when controlling FSIQ, Digit Span, and symptoms of ADHD, suggesting that this difference cannot be explained by differences in verbal short-term memory or overall cognitive ability. However, it is still possible that the working memory deficit associated with RD may be attributable to a deficit in auditory processing or verbal naming that is not reflected in the WISC-R, FSIQ, or Digit Span score. Therefore, to test definitively whether RD is associated with deficits in working memory, future studies of the cognitive correlates of RD should include measures of nonverbal working memory.

Implications of the Present Findings for the Etiology of Comorbidity Between RD and ADHD

The RD + ADHD group was most impaired on virtually all EF and PA measures. This finding is consistent with those of several studies that used a full 2×2 model or a subset of this design (Dykman & Ackerman, 1991; Klorman et al. , 1999; Nigg et al. , 1998; Purvis, 1999; Reader et al. , 1994), providing converging evidence against the phenocopy hypothesis. There is no clear explanation for the discrepancy between our present findings and the previous findings of our group in support of the phenocopy hypothesis (Pennington et al. , 1993). Studies differed somewhat in ascertainment procedures or sample severity, but the convergence of the present findings in a community sample with

results from independent clinical samples suggests that these variations in design may not account fully for the difference between our previous results and the present findings. Instead, it is possible that the fairly small comorbid group ($n = 16$) ascertained for the 1993 study may simply be atypical of most individuals with RD + ADHD for reasons that we are unable to determine.

Several additional competing hypotheses have also been proposed to explain the significant association between RD and ADHD. For example, comorbidity between RD and ADHD could be attributable to a sampling artifact (e. g. , Berkson, 1946), symptom overlap or shared method variance (e. g. , Caron & Rutter, 1991), common genetic influences (Willcutt et al. , 2000b), or cross-assortative mating for the two traits (Faraone et al. , 1993). The present findings converge with results from other community samples to indicate that the association between RD and ADHD is not restricted to clinical samples (e. g. , Fergusson & Horwood, 1992; McGee & Share, 1988), and is, therefore, not a sampling artifact. The significant comorbidity between RD and ADHD in the present sample also cannot be explained by symptom overlap or common method variance, because the ADHD phenotype was based on parent report whereas the RD phenotype was derived from performance on cognitive tests. Behavioral genetic analyses of the overall CLDRC data set revealed significant bivariate heritability for RD and ADHD, suggesting that common genetic influences may predispose individuals to both reading difficulties and symptoms of ADHD (Willcutt et al. , 2000b). Moreover, significant cross-assortative mating for ADHD and RD would lead to lower estimates of bivariate heritability. Therefore, although the cross-assortative mating hypothesis cannot be tested directly in the present CLDRC sample, the finding of significant bivariate heritability indicates that, even if cross-assortative mating is occurring, the overlap between RD and ADHD is also attributable to common genetic influences.

In light of the significant genetic overlap between RD and ADHD, we were somewhat surprised that the present results did not reveal a neuropsychological deficit common to the two disorders. However, several possible explanations could reconcile these two findings. First, although RD was not associated with a significant deficit in response inhibition when FSIQ and symptoms of ADHD were controlled, a statistical trend toward significance suggested that this effect might be significant in a larger sample. Therefore, additional studies of the present tasks and other measures of inhibition would be useful to test whether comorbidity between RD and ADHD is explainable by a common deficit in this cognitive domain. Alternatively, RD and ADHD may be associated with a common neuropsychological deficit that was not measured directly in this study, such as speeded verbal naming or nonverbal working memory. Finally, the influence of genes on behavior is likely to be pleiotropic, such that the same genetic influences affect more than one phenotype (e. g. , Plomin, DeFries, McClearn, & Rutter, 1997). For example, the gene for albinism in mice is also associated with significantly higher levels of emotionality (e. g. , DeFries, Hegmann, & Weir, 1966). If RD and ADHD are attributable to common genes with pleiotropic effects, these common genetic influences may cause behavioral symptoms and neuropsychological deficits that are independent at the phenotypic and cognitive levels of analysis. Future studies using molecular genetic methods will facilitate a more direct test of this hypothesis.

Potential Limitations of the Present Study and Directions for Future Research

The present results should be interpreted in light of several limitations. Although previous studies found few significant differences between twins and nontwins (e. g. , Plomin et al. , 1997), the utilization of twins for phenotypic comparisons may limit the generalization of the present findings to the population at large. In addition, because the CLDRC twin project has been ongoing for nearly 20 years, older versions of the WISC and the PIAT than those currently available have been maintained to allow comparisons to be made across the entire sample. Finally, only parental ratings of ADHD symptoms were available for the present study, and the version of the DICA-P used in the current study assessed symptoms of DSM-III ADHD.

Barkley (1998) suggested that the DSM-IV-combined subtype of ADHD is associated with inhibition deficits, whereas the predominantly inattentive subtype is related more strongly to deficits in processing speed and focused attention. Therefore, future studies of the cognitive correlates of RD and ADHD should test whether the present findings differ as a function of ADHD subtype. Studies using samples that are younger or older than the present sample would also provide a useful extension of the present findings. A younger sample could be used to test whether EF or PA deficits are present before the onset of school difficulty, whereas an older sample would provide a direct test of whether these deficits represent a developmental delay that eventually normalizes or a chronic deficit that persists into adulthood.

Summary and Conclusions

An extensive battery of measures of EF and PA was administered to one of the largest samples to date to contrast the cognitive deficits associated with RD and ADHD. Results indicate that ADHD is associated with inhibition deficits, whereas RD is associated primarily with deficits on measures of PA and verbal working memory. The RD + ADHD group was most impaired on virtually all measures, providing evidence against the phenocopy hypothesis as an explanation for comorbidity between RD and ADHD. Instead, the present results suggest that comorbidity between RD and ADHD is attributable to common etiological influences or cross-assortative mating.

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