

Processing Speed Deficits in Attention Deficit/Hyperactivity Disorder and Reading Disability

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Abstract The goal of the current study was to test whether deficits in processing speed (PS) may be a shared cognitive risk factor in reading disability (RD) and Attention Deficit/Hyperactivity Disorder (ADHD), which are known to be comorbid. Literature on ADHD and RD suggests that deficits on tasks with a speeded component are seen in both of these disorders individually. The current study examined a wide range of speeded tasks in RD, ADHD, comorbid RD + ADHD, and a control group to test whether RD and ADHD have similar profiles of PS deficits, and whether these deficits are shared by the two disorders. The results suggest that a general PS deficit exists in both clinical groups compared to controls, although children with RD demonstrate greater PS deficits than children with ADHD. Two tests (underadditivity and partial correlations) were conducted to test whether these PS deficits are shared. Since we found that PS deficits were underadditive in the comorbid group and that partialling PS reduced the correlation between RD and ADHD, it appears that PS is a shared cognitive risk factor that may help explain the comorbidity of these two disorders.

Keywords Processing speed · ADHD · RD · Comorbidity

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Reading disability (RD) and Attention Deficit/Hyperactivity Disorder (ADHD) each occur in about 5% of the population, making them two of the most common childhood disorders (American Psychiatric Association, 1994), and they co-occur more frequently than is expected by chance. In samples of children with ADHD, the rate of RD is between 25–40% (August & Garfinkel, 1990; Semrud-Clikeman et al., 1992), whereas in samples of RD children, 15–25% also meet criteria for ADHD (Gilger, Pennington, & DeFries, 1992; Shaywitz et al., 1995; Willcutt & Pennington, 2000; Willcutt et al., 2001). Since this comorbidity is present in both clinical and community samples, it is not simply a selection artifact. Both behavioral and molecular genetic studies support a partly shared genetic etiology as an explanation for their comorbidity (Gayan et al., 2005; Willcutt, Pennington, & DeFries, 2000; Willcutt et al., 2002) and reject the three independent disorders hypothesis: that the comorbid disorder is a subtype that is etiologically distinct from either pure RD or pure ADHD (Pennington, Willcutt, & Rhee, *in press*). Given that there are shared genetic risk factors for RD and ADHD, it makes sense that there may be shared cognitive risk factors as well, even though earlier research (e.g. Pennington, Groisser, & Welsh, 1993) supported the view that RD and ADHD are cognitively distinct.

The overall goal of the current study is to test whether deficits in processing speed (PS) may be a possible shared cognitive risk factor in RD and ADHD, which would help explain their comorbidity. PS is a robust psychometric factor (Carroll, 1993) and is one of the four factors evaluated by the WISC-IV. There are both developmental (Kail & Hall, 1994; Salthouse, 1991) and individual differences in PS, but the cognitive mechanisms underlying these differences in PS are still not well understood. Unfortunately, there is no “gold standard” definition of PS as a construct. Because of this, we do not know if all speeded tasks are

measuring the same underlying cognitive process. It may be that all speeded tasks tap a common underlying process, yet also tap into more specific aspects of cognitive efficiency. Or, there may be distinct processes involved in different speeded tasks (e.g. simple RT versus naming speed versus executive speed). In addition, it may be that speeded tasks measure speed at which individuals perceive and process incoming information, they may measure output speed, or it could be that some speeded tasks require an integration of rapid perceptual, cognitive, and output processing. We will return to this issue later when we discuss our results.

While an adequate cognitive theory of PS remains elusive, there have been a series of studies attempting to isolate different aspects of information processing, breaking processing down into perceptual, cognitive processing, and output speed. In particular, a series of studies attempting to understand the relative contributions of processing load versus output on response time suggest that output speed rather than input or perceptual speed is impaired in ADHD (e.g. Sergeant, 2005). In this paper, our working definition of the construct of PS is that PS is underlying cognitive efficiency at understanding and acting upon external stimuli, which includes integrating low level perceptual, higher level cognitive, and output speed. Thus, we have included tasks that require participants to integrate higher order information in several modalities at a rapid rate. The tasks we have included cut across a wider range of higher cognitive abilities and are believed to tap various psychological and cognitive processes. Yet they are similar in that they all require participants to rapidly scan or identify stimuli, make quick associations and accurate decisions or word choices, and then rapidly reconfigure working memory in order to move quickly to the next item.

As discussed below, literature on ADHD and RD suggests that deficits on tasks with a speeded component are seen in both of these disorders individually. In RD, many of these tasks involve rapid naming of information, yet individuals with RD have also demonstrated slower reaction time on simple button press tasks and other nonlinguistic speeded matching tasks. Individuals with ADHD have been found to be slower on many tests of nonlinguistic reaction time and matching tasks, as well as naming tasks and fluency tasks. But it is not yet clear how these different types of tasks are related to each other, so one goal of the present paper is to examine their relation by means of factor analysis.

A brief review of relevant literature is presented below. First we will review the literature on rapid serial naming in RD, and then cover the studies that have looked at other PS tasks in RD. Then we will review the literature on PS in ADHD, and finally, examine the few studies that have looked at both disorders together.

Naming speed/PS in RD

It is well known that deficits in phonological awareness and phonological decoding constitute core deficits in RD (Lieberman, Shankweiler, Fischer, & Carter, 1974; Pennington, Van Orden, Smith, Green, & Haith, 1990). Additional deficits seen in individuals with RD, such as rapid serial naming (RSN), are often interpreted as stemming from their underlying phonological problems. For over 35 years many studies have shown that individuals with RD have problems with rapid naming of letters, objects, numbers, and colors (Denckla, 1972; Denckla & Rudel, 1974; Denckla & Cutting, 1999; Wolf & Bowers, 1999; Wolf et al., 2000; Ransby & Swanson, 2003; WISC-IV manual, 2004). On the rapid automatized naming task (RAN) Denckla and colleagues (1972, 1974) found that the speed at which participants could name information was correlated with their reading performance. This finding has been replicated many times. In a recent review of the rapid naming literature, it was reported that many studies found deficits in automaticity of rapid serial naming and fluency measures for children with RD (Savage, 2004). In addition, cross cultural studies provide support for rapid naming deficits in RD in both regular alphabetic orthographies and non-alphabetic logographies. (Wimmer, Mayringer, & Landerl, 2000; Suk-han Ho & Lai, 1999).

One of the important questions about RSN is how it fits with what we already know about the phonological deficits in RD. Is it due to the phonological deficit, or is it an independent deficit contributing to reading problems? Many researchers have conceptualized this naming speed deficit as stemming from an underlying difficulty with phonological representations, which cannot be called up from their long term store and provided as easily (Wagner, Torgenson, Laughon, Simmons, & Rashotte, 1993). However, if the source of the RSN deficit were solely in phonological representations, one would expect to find similar difficulties on discrete trial naming. But discrete trial naming, in which individual stimuli are presented one at a time on a computer screen, is less consistently related to reading ability than the continuous form (Perfetti, Finger, & Hogaboam, 1978).

Partly for this reason, others have suggested that the RSN deficit in RD is separate from the phonological deficit in RD. Wolf and Bowers (1993, 1999) have developed a dual deficit model of RD that includes both a phonological deficit component and a deficit in underlying naming speed. They argue that RSN and fluent reading both require the integration of precisely timed perceptual, attentional, and naming mechanisms in order to rapidly match visual stimuli to phonological codes. They propose that the automaticity problems seen in RD may be partially phonological, but are also partially due to problems in temporal processing and perceptual

speed. They offer the hypothesis that individuals with RD have an underlying impairment in their ability to process sequences of rapidly presented brief information (Wolf & Bowers, 1999).

One way to answer the independence question is to examine individuals with RD on PS tasks without a linguistic component. Are they only impaired on rapid naming tasks, or do they show a more general PS deficit? A few studies have attempted to measure non-linguistic PS in RD, and have found that individuals with RD are slower overall than controls. For instance, individuals with RD have demonstrated difficulty with tasks that are thought to be less dependent on linguistic abilities, such as WISC Coding and Symbol Search which use non-linguistic stimuli (Kail & Hall, 1994, 1999; Catts, Gillespie, Leonard, Kail, & Miller, 2002; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005). In an attempt to understand the relationship between non-linguistic PS and reading, Kail and Hall (1994) found that the link between rapid serial naming and reading is explained by global differences in PS. Others have also found that after general PS measures are entered into regression analyses, rapid naming no longer predicts additional variance in reading (Catts et al., 2002).

On the other hand, several studies have included naming and PS measures in their larger batteries for individuals with RD in order to examine how they relate to phonological and achievement measures, and they have found the linguistic versus non-linguistic distinction is meaningful. Savage and his colleagues (in press) examined children with RD on a wide variety of tasks with a speeded component as well as other phonological and reading achievement tasks. RSN tasks loaded with phonological and other nonword reading tasks, and rapid perceptual speed loaded on a separate factor. Individuals with RD were found to be impaired on both tasks, yet this finding suggests there may be an important distinction between the two in their relation to the core phonological deficit in RD. Nonetheless, all these studies support a global PS impairment in RD, but differ somewhat on whether the relation between RSN and reading skill is mediated by general processing speed.

PS in ADHD

The core cognitive deficit in ADHD is less well understood than the core cognitive deficit in RD. The executive function (EF) hypothesis is one of the most prominent neuropsychological models of ADHD (Barkley, 1997, 1983; Pennington, 1991; Pennington & Ozonoff, 1996). Barkley's (1997) model emphasizes behavioral inhibition as a core deficit of individuals with ADHD, from which all other executive problems stem. Yet a recent review indicates that about half of the individuals in ADHD samples do not have a deficit on the best replicated measure of the inhibition

deficit in ADHD, the Stop Task (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). Moreover, even across batteries of multiple EF measures, around 20% of ADHD participants are not impaired on any EF measure (Nigg et al., 2005). So at least for some children with ADHD, the EF hypothesis does not apply, leading researchers to examine other possible deficits.

There are impairments on PS measures across studies of individuals with ADHD (Willcutt et al., 2005, WISC-IV manual, 2004; Woods, Lovejoy, & Ball, 2002). In fact, several studies have found that PS measures are among the best predictors of inattentive symptoms in ADHD (Chhabildas, Pennington, & Willcutt, 2001; Rucklidge & Tannock, 2002; Weiler, Holmes Bernstein, Bellinger, & Waber, 2000). In addition, there is a significant amount of evidence that simple "go" reaction time is slower and more variable in children with ADHD on continuous performance and go no-go tasks (Kuntsi & Stevenson, 2001; Nigg, 2001; Rubia, Oosterlaan, Sergeant, Brandeis, & van Leeuwen, 1998; Rucklidge & Tannock, 2002; Scheres, Oosterlaan, & Sergeant, 2001; Slusarek, Velling, Bunk, & Eggers, 2001; van der Meere & Stermerdink, 1998; van der Meere, Stermerdink, & Gunning, 1995; Willcutt et al., 2005). One theory of ADHD, the cognitive-energetic hypothesis, proposes that children with ADHD are cognitively under-aroused and thus have much slower and more variable reaction times (van der Meere et al., 1995). A series of studies attempting to understand the relative contributions of processing load versus output on response time suggest that output speed rather than input or perceptual speed is impaired in ADHD (e.g. Sergeant, 2005).

Most of the tasks used to measure PS in individuals with ADHD, such as WISC Coding and Symbol Search, and Trails A and B, tend to be less dependent on linguistic skills than rapid serial naming. However, there are several measures commonly used in studies of ADHD that do require rapid naming. The Stroop task, for example, includes control conditions in which participants name words and colors as rapidly as possible and children with ADHD have been found to demonstrate slower naming speed in these control conditions as compared to controls (Homack & Riccio, 2004; Savitz & Jansen, 2003). Rucklidge and Tannock (2002) found that participants with childhood-onset ADHD were slower in PS, color naming, and number naming and were more variable in response times and accuracy of responses compared with the adolescent-onset participants with ADHD.

In sum, there is evidence for PS deficits on both linguistic and non-linguistic tasks in both RD and ADHD. Yet we do not know if the two disorders differ in their particular profile of PS problems, what that profile is like in children who have both disorders, or whether PS deficits are shared by the two disorders.

The current study

The present study follows up a recent study by Willcutt and his colleagues (2005) which examined a wide variety of neuropsychological and cognitive measures in search of a common deficit between RD and ADHD. They found that PS, measured by WISC Coding and Symbol Search tasks, was similarly impaired in groups with RD only, ADHD only, and both disorders (comorbid group). This finding, especially the similar level of impairment in the comorbid group, suggests that a PS deficit is a shared cognitive risk factor in RD and ADHD. If PS deficits in each disorder were independent, the deficit in the comorbid group would be the sum (or even the product) of the deficits in each pure group.

The current paper takes a more in depth look at PS in these two disorders. The first question of interest is whether there are different components of PS or whether PS is a unitary construct. To this end, we factor analyzed a battery of both linguistic and non-linguistic measures of PS. Regardless of whether PS is a unitary construct or not, the second question of interest is whether the profiles of PS deficits are similar in RD and ADHD. If the profiles are distinct, that would suggest that a PS deficit is not a shared cognitive risk factor. If the profiles are similar, that would be consistent with a PS deficit being a cognitive risk factor shared by RD and ADHD. However, because similar profiles could arise for different underlying cognitive reasons, the two disorders could still be cognitively distinct.

The third question of interest is whether the effects of each disorder on PS are independent or partially shared. To answer this question, we need a method to test whether a cognitive risk factor is shared by two disorders. The main method used here is a test for underadditivity, which uses the profile of PS deficits in the comorbid group to answer this question. If the effects are independent, then the PS deficits in the comorbid group will be greater than or equal to the sum of those in the RD- and ADHD-only groups. If the effects are at least partially shared and thus not independent, the profile in the comorbid group will be less impaired than the sum of the profiles in each pure group, thus producing an RD by ADHD interaction due to underadditivity.

We also used a second, separate test of the cognitive overlap between RD and ADHD in the domain of PS. We calculated partial correlations among ADHD and RD symptom scores and each PS factor. If the partial correlation between RD and ADHD symptoms controlling for PS is considerably less than their first order correlation, that suggests PS helps account for the relation between RD and ADHD. Similarly, if the partial correlation between either symptom dimension and PS controlling for the other symptom dimension is considerably less than the first order correlation, that also suggests sharing.

Methods

Participants

Recruitment

Participants include a total of 395 children and adolescents (203 males and 192 females) ranging in age from 8 to 18 years who completed the measures described in this paper as part of the Colorado Learning Disabilities Research Center (CLDRC) twin study. The CLDRC study is an ongoing study of the etiology of learning disabilities, ADHD, and other related disorders (e.g., DeFries et al., 1997; Willcutt, DeFries, et al., 2003). In collaboration with 27 local school districts, parents of all twins between the ages of 8 and 18 were contacted by letter and invited to participate in the study. After initial parental consent was obtained, two parallel recruitment procedures were conducted independently to identify twin pairs in which at least one of the twins met criteria for ADHD or at least one of the twins exhibited significant reading difficulties, as well as a comparison sample of twin pairs in which neither twin exhibited either ADHD or RD.

To identify twin pairs in which at least one twin exhibited significant reading difficulties, parental consent was requested to allow study staff to review each twin's academic records. If either member of a twin pair had a positive history of learning difficulties (e.g., low achievement scores, referral to tutor, reports by classroom teachers or school psychologists), both members of the pair were invited to participate in the larger study. To identify twins with ADHD, parents and teachers were asked to complete the Disruptive Behavior Rating Scale (Barkley & Murphy, 1998) to assess *DSM-IV* ADHD symptoms. If either of the twins met symptom criteria for any *DSM-IV* ADHD subtype based on parent or teacher ratings, the twin pair was invited to participate. The comparison sample included twin pairs from the same school districts in which neither twin met screening criteria for ADHD or RD. Approximately 35% of the families who were contacted agreed to participate in the initial screening procedure. If invited, 87% of the families in the screening sample agreed to participate in the larger study. Although the rate of participation was slightly higher among control twin pairs (91%) than twin pairs in which at least one twin had RD or ADHD (84%), this difference was not significant. Prior to the inception of any testing, CLDRC staff conducted a telephone screening interview. Because the focus of the overall project is on the etiology and correlates of familial RD and ADHD, potential participants with a documented brain injury, significant hearing or visual impairment, or other rare genetic or environmental etiology (e.g., Fragile \times syndrome, Down Syndrome, or other sex chromosome anomalies) were excluded from the sample.

Because including both twins in the current analyses would have violated the assumption of independence, data from one twin was chosen from each twin pair in the following way: If only one twin met criteria for RD and/or ADHD, their data was selected, and if both twins met criteria, one was chosen at random. In total, data from 395 participants were included in the study. Of that group, 105 participants met criteria for *DSM-IV* ADHD only and 95 participants met criteria for RD only. Children who met diagnostic criteria for both diagnoses ($n = 51$) were placed in a third comorbid group. The remaining children did not meet either criteria and served as our typically developing control group. Within the ADHD only group, there were 62 children who met criteria for Predominantly Inattentive Type, 15 who met criteria for Predominantly Hyperactive/Impulsive Type, and 28 who met criteria for Combined Type. Within the ADHD + RD group, there were 38 children who met criteria for Predominantly Inattentive Type, 1 who met criteria for Predominantly Hyperactive/Impulsive Type, and 12 who met criteria for Combined Type. These proportions are similar to those found in other population-based samples of ADHD, which typically find that the proportion of individuals that meets symptom criteria for the inattentive type is higher than the proportion that meets symptom criteria for the hyperactive-impulsive or combined types. Specifically, findings from a recent meta-analysis found that approximately half of all individuals who met symptom criteria for *DSM-IV* ADHD met criteria for the inattentive type (49.6% based on parent ratings, 52.6% based on teacher ratings), whereas less than one-third met symptom criteria for the combined type (29.5% based on parent ratings, 29.4% based on teacher ratings) and an even smaller percentage met criteria for the hyperactive-impulsive type (20.9% based on parent ratings, 18.0% based on teacher ratings). In contrast, samples ascertained through clinics typically include a much higher proportion of individuals with the combined type (30–93%; 58.7% of pooled clinic-based samples) in comparison to the inattentive (10–76%; 30.9% of pooled sample) or hyperactive-impulsive types (4–25%; 10.4% of pooled sample). This difference suggests that although more children and adolescents in the population at large meet symptom criteria for the inattentive type, individuals who meet symptom criteria for the combined type are more likely to be referred for clinical services (Willcutt & Pennington, 2000).

Diagnostic measures and operational definition of RD and ADHD

Assessment of RD

Academic achievement in reading and mathematics was assessed with the Peabody Individual Achievement Test (PIAT;

Dunn & Markwardt, 1970). To simplify interpretation, a normally-distributed reading composite score was created based on a previous discriminant function analysis of the PIAT Reading Recognition, Reading Comprehension, and Spelling subtests (DeFries, 1985). A standard score 1.75 standard deviations below the mean of our control sample was used as the cutoff score for RD. This cutoff selects approximately five percent of the control sample, consistent with the estimated population prevalence of RD (e.g., American Psychiatric Association, 2000). Participants were categorized as RD if they had a positive school history of reading problems and scored below this cutoff on the reading discriminant function score. In the current study, some analyses required continuous ratings of RD symptomatology. In those cases, the continuous discriminant function score was utilized.

Assessment of ADHD

The Disruptive Behavior Rating Scale (DBRS; Barkley & Murphy, 1998) was used to obtain parent and teacher ratings of the 18 symptoms of *DSM-IV* ADHD. Each symptom on the DBRS is rated on a four-point scale (*never or rarely, sometimes, often, and very often*). Items rated as *often* or *very often* were scored as positive symptoms and items rated as *never or rarely* or *sometimes* were scored as negative symptoms, consistent with the procedure used in previous studies of similar rating scales (e.g., Pelham, Gnagy, Greenslade, & Milich, 1992). Previous results from this sample and others indicate that parent and teacher ratings on the DBRS or other similar scales are internally consistent ($\alpha = .92-.96$) and have adequate to high test-retest reliability ($r = .59-.89$; e.g., DuPaul, Power, Anastopoulos, & Reid, 1998; Willcutt, Chhabildas, & Pennington, 2001). Impairment was assessed for all rating scales by asking raters to assess whether subjects were impaired in multiple domains, and age of symptom onset is also required on the parent rating scale. The algorithm from the *DSM-IV* field trials for the disruptive behavior disorders was used to combine parent and teacher ratings of ADHD symptoms (Lahey et al., 1994). This procedure codes each symptom as positive if it is endorsed by either the parent or the teacher. Consistent with *DSM-IV* criteria, children were categorized as ADHD only if symptoms were present prior to age seven and if these symptoms caused significant functional impairment. Individuals with six or more symptoms of inattention but fewer than six symptoms of hyperactivity-impulsivity were identified as Predominantly Inattentive Type, participants with six or more symptoms of hyperactivity-impulsivity but fewer than six symptoms of inattention were categorized as Predominantly Hyperactive/Impulsive Type, and individuals with six or more symptoms on both dimensions were coded as

Combined Type. Of the children meeting criteria for ADHD, 97% also met criteria for moderate to severe functional impairment. In addition, all children included in the data analyses had an age of symptom onset in childhood, and 95% had an age of onset of 7 years or younger. In the current study, continuous symptom counts based on this algorithm were used in some analyses.

Descriptive characteristics of the groups

Mean age was not different between the ADHD, RD, comorbid, and control groups (see Table 1). In contrast, the mean socioeconomic status (SES) as measured by the Hollingshead two factor inventory (Hollingshead, 1975) was significantly lower in all three clinical groups than in the comparison group. As expected, both the ADHD only and the ADHD + RD groups demonstrated significantly more symptoms of ADHD than the control or RD only group, and both RD groups had significantly lower PIAT scores and lower Reading Discriminant Function scores than the ADHD or control groups. Importantly, comorbidity was not confounded with severity since the comorbid group did not differ from the ADHD-only group on ADHD symptoms, nor from the RD-only group on the reading discriminant score. In addition, the IQ scores as measured by the *Wechsler Intelligence Scale for Children- Revised* (Wechsler, 1974) of the clinical groups fell below that of the control group.

Procedures

The WISC-R, PIAT, RAN, Colorado Perceptual Speed, and Identical Pictures tasks were administered in initial testing sessions completed at the University of Colorado Institute for Behavioral Genetics. The Stroop Test, Trailmaking Test, Gordon Diagnostic System, and Stop Task were completed during a second session scheduled approximately one month later at the University of Denver Department of Psychology. Trained examiners who had previous experience working with children administered all measures at both sites. All examiners were unaware of the diagnostic status of the child and the results of the testing conducted at other sites. Parents of participants that were taking psychostimulant medication were asked to withhold medication for 24 hours prior to each session of the study to minimize the influence of medication on the results.

Measures of PS

In order to examine the broad construct of PS and how the tasks related to each other, we included all tasks from our larger study that required quick and accurate processing of information in the initial analyses. The type of information processed on these tasks could be simple reaction time (RT) or could require integration of complex information. We later refined the set of tasks analyzed here based on the results of the factor analysis.

Table 1 Descriptive characteristics of the sample

	Comparison (N = 144) M (SD)	ADHD only (N = 105) M (SD)	RD only (N = 95) M (SD)	RD + ADHD (N = 51) M (SD)	F
Demographic variables					
Age	11.5 (2.5)	11.1 (2.7)	10.9 (2.4)	11.0 (2.5)	1.1
Socioeconomic status	3.5 _a (1.05)	2.9 _b (1.26)	2.8 _b (1.2)	2.8 _b (1.1)	8.9
Sex (M:F)	56:88	75:30	43:52	29:22	
WISC-R					
Full scale IQ	113.3 _a (11.0)	105.8 _b (11.6)	96.8 _c (11.1)	93.1 _c (11.1)	158.9
Verbal IQ	112.9 _a (11.9)	107.1 _b (13.8)	95.6 _c (12.1)	91.7 _c (12.9)	198.3
Performance IQ	111.1 _a (11.9)	103.5 _b (8.4)	99.0 _{b/c} (12.4)	93.1 _c (11.1)	145.4
Academic achievement					
PIAT math	111.2 _a (12.3)	104.7 _b (13.9)	93.6 _c (11.8)	92.0 _c (11.1)	111.4
PIAT reading recognition	109.1 _a (10.0)	103.5 _b (8.4)	86.3 _c (7.8)	85.8 _c (7.4)	62.3
PIAT reading comp	110.7 _a (10.1)	107.8 _a (11.3)	89.8 _b (8.6)	88.0 _b (9.1)	34.7
PIAT spelling	107.2 _a (11.2)	100.8 _b (10.4)	87.0 _c (9.0)	86.1 _c (9.5)	68.4
Reading discriminant score	1.51 _a (1.0)	0.78 _b (0.9)	-1.30 _c (0.7)	-1.42 _c (0.7)	299.7
ADHD symptoms					
Inattention	0.6 _a (1.2)	6.9 _b (2.3)	1.5 _a (1.7)	7.6 _b (1.6)	454.1
Hyperactivity – impulsivity	0.5 _a (1.0)	4.2 _b (3.2)	0.7 _a (1.2)	3.9 _b (3.0)	95.5
Total symptoms	1.1 _a (1.7)	11.1 _b (3.6)	2.2 _a (2.3)	11.5 _b (3.4)	449.8

Note. df = 3,391. Means with different subscripts are significantly different, *p* < .01. Sample is constructed of one twin randomly selected from each twin pair in the larger study.

WISC-R Coding subtest (Wechsler, 1974) is considered one of the “gold standards” in PS tasks. Psychometric studies for the WISC-R indicate that Coding has adequate reliability but only correlates with FSIQ moderately (Wechsler, 1974, 1991), suggesting that it taps a portion of PS that is independent of general intelligence.

The *Trailmaking test* (Reitan & Wolfson, 1985) assesses both PS (Trails A) and ability to shift cognitive set (Trails B). Previous studies have 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 these scores.

The *Rapid Automatized Naming* subtests are an adaptation of those used by Denckla and Rudel (1976). In each subtest, participants must name a set of stimuli (a set of letters, colors, or numbers) as accurately and quickly as possible in 15 s (Compton, Olson, DeFries, & Pennington, 2002). This task was thought to tap PS because it required fast and accurate naming of stimuli.

The version of the *Stroop* (Golden, 1978) task used in this study had three conditions: a word reading condition, a color naming condition, and an interference condition. This task is thought to tap into PS because it required fast and accurate recognition of stimuli in the first two conditions, and the third condition required fast and accurate recognition of stimuli while suppressing distracting information.

The *Stop Signal task* (Logan, Schachar, & Tannock, 1997; Schachar, Mota, Logan, Tannock, & Klim, 2000) is a computerized measure of inhibitory control and possibly PS that is based on the dual-process model of inhibition proposed by Logan and colleagues (Logan, 1994). On primary task trials, the letters X or O are presented in the center of the monitor, and the participant responds by pressing the corresponding key on the keyboard. On stop-signal trials, the same visual stimulus appears, but an auditory tone is also presented shortly after the X or 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 key press on each of the trials on which the tone is presented. Thus, the task requires rapid integration of auditory information to update motor output. It yields a Stop Signal Reaction Time (SSRT) which is an estimate of inhibition speed, and a mean reaction time on the primary task (GoRT).

The *Gordon Diagnostic System* (Gordon, 1983) is a standardized continuous performance test (CPT) that assesses the ability to sustain attention and inhibit inappropriate responses during an extended visual task. Because this task requires rapid integration of visual stimuli (i.e., recognition of a 1, 9 pair) and a motor response (press button on pair), this task may also tap into PS as well as sustained attention and inhibition. The mean reaction time over the course of the test was examined in this study.

The *Colorado Perceptual Speed Test (CPS): Rotatable Letters and Numbers* was specifically constructed for the Colorado Family Reading Study and was found to be one of the most reliable (DeFries, Singer, Foch, & Lewitter, 1978) and discriminating (Foch, DeFries, McClearn, & Singer, 1977) tests in that battery. In the revised form (Decker, 1989), letter strings are organized into three sections: phonetically similar letters (e.g. bceg), phonetically dissimilar letters (e.g. bhsf), and pronounceable nonwords (e.g. pelb). These three sections are highly correlated and typically combined in data analysis. This task was thought to tap PS because it required fast and accurate recognition of a target stimulus from an array of stimuli.

The *ETS Identical Pictures Test* (French, Ekstrom, & Price, 1963) is similar to the Colorado Perceptual Speed Test, using pictures rather than letters. As in the CPS, this task was thought to tap PS because it required fast and accurate recognition of a target picture from an array of pictures.

Results

Preliminary analyses

In order to avoid violating the assumption of independence between scores, the data from one twin out of each pair was randomly selected to be analyzed. The data below reflects analyses with the sample after randomly selecting one twin. The influence of age on our results was controlled by creating an age-adjusted score for each measure by regressing the variable onto age and saving the residual score. In addition, the distribution of each age-regressed score was examined for outliers prior to any analysis. An outlier was defined as any score that fell more than three standard deviations from the group mean on each variable. One individual observation was dropped from the comorbid group in Trails A. Finally, skewness and kurtosis were examined and all variables were found to be normally distributed. To understand the relationship between all variables of PS, bivariate correlations were conducted on all variables. All variables were correlated at $p < .001$ (see Table 2).

Exploratory factor analyses

Initially, we ran a Principal Axis Factor analysis with Oblimin rotation on all measures in our battery with a speeded component. This analysis yielded 2 factors with eigenvalues greater than one. Factor 1 included speeded tasks with verbal output (Eigenvalue = 5.56, 39.7% variance explained), while Factor 2 included speeded tasks with motor output (Eigenvalue = 1.36, 9.68% variance explained). In these initial analyses, mean RT from the GDS, SSRT and GoRT from the Stop Task, and Stroop Color-Word were

Table 2 Correlations among PS tasks

	Stroop words	Stroop color	Stroop interference	Trails A	Trails B	GDS RT	SSRT	GoRT	RAN colors	RAN numbers	RAN letters	RAN pictures	CP 1 & 2	CPS 3	WISC coding	Identical pictures
Stroop word	–															
Stroop color	.806	–														
Stroop interference	.718	.774	–													
Trails A	–.496	–.470	–.448	–												
Trails B	–.520	–.479	–.493	.575	–											
GDS RT	–.335	–.282	–.276	.275	.388	–										
Stop task SSRT	–.500	–.473	–.457	.251	.344	.317	–									
Stop task GoRT	–.316	–.294	–.266	.194	.188	.317	.224	–								
RAN colors	.768	.788	.673	–.439	–.431	–.287	–.394	–.321	–							
RAN numbers	.782	.691	.637	–.401	–.405	–.310	–.432	–.268	.740	–						
RAN letters	.793	.704	.635	–.443	–.429	–.330	–.440	–.288	.827	.685	–					
RAN pictures	.666	.654	.602	–.316	–.358	–.317	–.425	–.312	.675	.751	.646	–				
CPS 1 & 2	.767	.759	.759	–.486	–.501	–.354	–.490	–.321	.699	.741	.631	.924	–			
CPS 3	.750	.713	.722	–.490	–.530	–.332	–.465	–.338	.686	.698	.474	.524	.523	–		
WISC coding	.471	.443	.467	–.357	–.325	–.130	–.362	–.175	.451	.432	.362	.615	.812	.812	.527	–
Identical pictures	.687	.731	–.491	–.469	–.329	–.467	–.467	–.332	.676	.638	.674	.615	.819	.812	.527	–

Note. All correlations are significant at $p < .001$, $N = 395$.

Table 3 Task loadings for each PS factor collapsing across group

Task	Factor 1 (Eigenvalue = 5.81% of Variance = 45.94)	Factor 2 (Eigenvalue = 1.47% of Variance = 11.42)
Stroop word	.774	–
Stroop color	.601	–
RAN colors	.765	–
RAN numbers	.822	–
RAN letters	.726	–
RAN pictures	.567	–
Trails A	–	.415
Trails B	–	.486
Colorado perceptual speed – Parts 1 & 2	–	–.898
Colorado perceptual speed – Part 3	–	–.832
WISC coding	–	–.560
Identical pictures	–	–.690

Note. – indicates factor loading less than .15. Loadings in boldface indicate primary factor loading.

cross-loaded on both Factors 1 and 2. As a result, these four variables were dropped in subsequent analyses. After dropping these measures, another Principal Axis Factoring with Oblimin rotation was conducted with the remaining tasks. Consistent with the previous analysis, Factor 1 included speeded tasks with verbal output (Eigenvalue = 5.02, 45.6% variance explained), while Factor 2 included speeded tasks with motor output (Eigenvalue = 1.32, 12.0% variance explained) (see Table 3 for factor loadings). To ensure that these factors were not solely driven by the reading speed deficit in RD, we performed a follow-up analysis dropping the third subtest of the CPS (CPS III), which included speeded phonological decoding, and Stroop Word, which involves reading words. The results mirrored the results outlined above, with the same two factors. Thus for the remaining analyses, CPS III and Stroop Word were included.

Group differences by factor

In order to examine group differences between the RD, ADHD, and control groups with respect to these factors, we conducted a repeated measures analysis of variance (ANOVA) with two between subjects factors (RD or not RD) and (ADHD or not ADHD) and one within subjects factor (Verbal versus Motor PS). Means for the individual tasks within each factor score can be found in Table 4. Results indicate main effects of both RD and ADHD on PS. Children with RD differed significantly from children without RD on PS across factors ($F(1,391) = 123.3, p < .001$). Likewise, children with ADHD differed significantly from children without that disorder on PS across factors ($F(1,391) = 30.2, p < .001$). The interaction between RD and ADHD

Table 4 Group means on individual PS tasks

	Comparison		ADHD only		RD only		RD + ADHD		Main effects <i>F</i>		
	(<i>N</i> = 144) M (SD)	(<i>N</i> = 105) M (SD)	(<i>N</i> = 105) M (SD)	(<i>N</i> = 95) M (SD)	(<i>N</i> = 51) M (SD)	ADHD	RD	RD × ADHD Interaction <i>F</i>	ADHD	RD	RD × ADHD Interaction <i>F</i>
Factor 1											
Stroop word	0.4 _a (0.9)	-0.1 _b (0.8)	-0.7 _c (0.9)	-0.8 _c (1.0)	11.3***	103.0***	3.3				
Stroop color	0.4 _a (0.9)	-0.3 _b (1.1)	-0.4 _b (0.8)	-0.5 _b (1.1)	11.4***	26.2***	5.6*				
RAN colors	0.4 _a (0.9)	-0.1 _b (0.9)	-0.5 _c (1.0)	-0.6 _c (1.1)	8.7**	52.6***	5.0*				
RAN numbers	0.3 _a (0.9)	0.0 _b (1.0)	-0.5 _c (0.9)	-0.5 _c (0.9)	2.4	50.9***	1.3				
RAN letters	0.4 _a (0.9)	-0.1 _b (1.0)	-0.5 _c (0.8)	-0.8 _c (0.9)	18.7***	92.5***	0.6				
RAN pictures	0.4 _a (0.9)	-0.1 _b (1.0)	-0.3 _{b,c} (0.8)	-0.6 _c (0.9)	12.5***	30.1***	1.5				
Factor score	0.5 _a (0.8)	-0.1 _b (0.8)	-0.6 _c (0.8)	-0.8 _c (0.9)	19.2***	101.3***	6.3**				
Factor 2											
Trails A	0.2 _a (0.7)	-0.1 _b (1.1)	-0.3 _b (1.0)	-0.3 _b (1.2)	5.3*	14.7***	1.5				
Trails B	0.3 _a (0.7)	-0.1 _b (1.0)	-0.4 _b (1.3)	-0.4 _b (1.0)	3.2	18.9***	2.9				
CPS	0.5 _a (0.9)	-0.2 _b (1.1)	-0.6 _c (0.8)	-0.8 _c (0.8)	29.0***	128.2***	8.3**				
WISC – coding	0.5 _a (0.9)	-0.4 _b (0.9)	-0.3 _b (1.0)	-0.7 _c (1.0)	35.0***	32.6***	5.5*				
Identical pictures	0.4 _a (1.0)	-0.2 _b (1.1)	-0.3 _{b,c} (1.0)	-0.6 _c (1.0)	17.9***	32.5***	1.6				
Factor score	0.6 _a (0.7)	-0.2 _b (0.9)	-0.6 _c (0.8)	-0.9 _c (0.8)	32.5***	107.0***	11.2***				

Note. Scores are age corrected *z* scores to account for the influence of age on PS. *df* = 3,391. Means with different subscripts are significantly different, $p < .05$.

* $p < .05$, ** $p < .01$, *** $p < .001$.

was also significant ($F(1,391) = 10.2, p < .01$), suggesting that the effects of having both of these two disorders was not simply additive. There was no between factor main effect across the groups, and no significant interactions between group and factor. In addition, results did not change when running analyses with the factor scores that did not include CPS III and Stroop Word.

To understand the RD by ADHD interaction, the effect size for each group was calculated using Cohen's d , and results indicated that the effect size on Factor 1 for ADHD compared to controls was $d = 0.75$, for RD was 1.37, and for both RD and ADHD was 1.46. On Factor 2, the effect size for ADHD compared to controls was $d = 0.94$, for RD was 1.55, and for both RD and ADHD was 1.88. (See Fig. 1 for a graph of the effect sizes). These results indicate that the PS deficits were underadditive in the comorbid group, suggesting that the deficits are at least partially shared between RD and ADHD.

Because even the pure disorder groups exhibit higher sub-clinical levels of the other disorder than controls, we covaried symptoms of one disorder out of the repeated measures test described above in order to isolate the effects of each single disorder. This procedure also tests for whether the relation of one diagnosis with PS is mediated by the other diagnosis. The results of these ANCOVAs revealed a main effect of RD when ADHD symptoms were covaried ($F(1,390) = 106.3, p < .001$), while the main effect of ADHD was reduced to a trend and the RD by ADHD interaction remained significant ($F(1,390) = 9.09, p < .01$). When covarying symptoms of RD from the analyses, results indicated a main effect of ADHD ($F(1,390) = 17.7, p < .001$), no main effect of RD, and a significant RD by ADHD interaction ($F(1,390) = 4.21, p < .05$). In contrast, the absence of main or interaction effects of factor means that these results held for both PS factors. These results rule out the possibility that underadditivity is due to subclinical levels of each disorder in the other group.

Based on the significant associations between RD and ADHD and IQ, some researchers argue that FSIQ should always be statistically controlled to ensure that neuropsychological impairments associated with RD or ADHD cannot be explained more parsimoniously by group differences on this correlated variable (e.g., Lahey et al., 1998; Werry, Elkind, & Reeves, 1987). On the other hand, ADHD symptoms or reading difficulties may directly cause a child to perform poorly on standardized tests of intelligence (e.g., Barkley, 1997) or may precipitate the development of these disorders. In these cases, it would not be appropriate to control for this variable, as this would remove variance that is associated with ADHD or RD. These issues have not been resolved conclusively, and in the case of the current study, there is an additional concern about covarying FSIQ, namely that PS measures contribute to the overall FSIQ score. Thus, covarying FSIQ

out of the current analyses from the start is considered overly conservative. We have chosen to address the complicated issue of IQ by conducting the analyses both with and without IQ covaried. The results above were obtained without the covariate, and they did not change significantly when FSIQ was covaried from these analyses, despite the fact that PS is a component of the FSIQ score. Results with FSIQ covaried indicate main effects of both RD and ADHD on PS. Children with RD differed significantly from children without RD on PS across factors ($F(1,390) = 29.2, p < .001$). Likewise, children with ADHD differed significantly from children without that disorder on PS across factors ($F(1,390) = 14.5, p < .001$). The interaction between RD and ADHD was also significant ($F(1,390) = 6.5, p < .01$), indicating that the underadditivity found earlier is not just due to lower IQ.

Secondary analyses

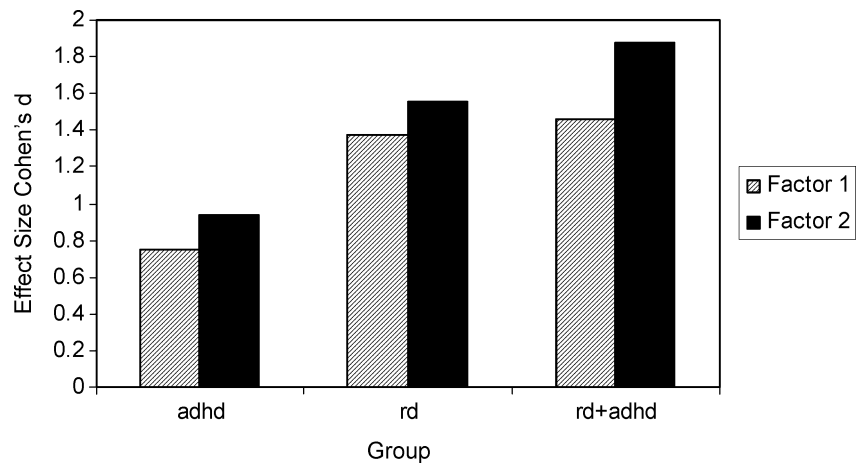
Partial correlations

As an additional test of the hypothesis that PS is a shared cognitive deficit in both RD and ADHD, we computed partial correlations using the standard formula: $pr = r_{xy} - [(r_{xz})(r_{yz})]/\sqrt{(1 - r_{xz}^2)(1 - r_{yz}^2)}$. We also examined the percent change in r^2 after a third variable is controlled for. These results are presented in Table 5. The zero-order correlation between dimensional symptoms of RD and ADHD is 0.28, whereas their partial correlation is 0.18, controlling for Factor 1. Hence, the percent change in r^2 after controlling for Factor 1 is -60% . Likewise, when Factor 2 is controlled, the partial correlation is 0.13, and the percent change in r^2 after controlling for Factor 2 is -73% . Since Factor 1 and 2 are correlated at $r = 0.75$, these results indicate that a significant portion, but not all, of the relationship between these two symptom dimensions can be accounted for by PS. In addition, the pattern of change in r^2 controlling for one symptom dimension while examining the relation of the other symptom dimension with PS mirror the results of the ANOVA presented earlier. Neither relation is entirely mediated by the other symptom dimension, although RD has a stronger relation with PS than does ADHD.

Underadditivity in other domains

Besides covarying IQ, we performed a second test to ensure that group differences found in PS factor scores are not due simply to pre-existing differences with respect to general intelligence. Specifically, we investigated whether other domains of intelligence followed the same pattern. Thus we conducted the same 2×2 analysis on the Verbal

Fig. 1 Effect size by diagnostic group



Comprehension (VC) and Perceptual Organization (PO) factors of the WISC-R, which are less confounded with PS. Results indicate significant main effects of ADHD ($F(1,394) = 10.5, p < .01$) and RD ($F(1,394) = 150.6, p < .001$) on VC, but no significant RD \times ADHD interaction. Similarly, for PO there were significant main effects of ADHD ($F(1, 394) = 23.6, p < .001$) and RD ($F(1, 394) = 62.7, p < .001$), but no significant RD \times ADHD interaction. Thus, the different results on these two other Wechsler factors argue against a general intelligence explanation of the PS results. Additionally, we can isolate other cognitive domains that do not show this underadditivity pattern. For example, running a 2×2 ANOVA with phonological awareness (PA) as the dependent variable, reveals a main effect of RD ($F(1,394) = 212.7, p < .001$), a much smaller ADHD main effect ($F(1,394) = 7.7, p < .01$), and no RD \times ADHD interaction. While there may be other cognitive domains that could also help explain the phenotypic overlap between these two disorders, not all cognitive domains related to these disorders demonstrate underadditivity.

PS in DSM-IV ADHD subtypes

Although we grouped individuals with all three *DSM-IV* subtypes of ADHD together for the main analyses discussed above, we also examined whether there are PS differences between the three ADHD subtypes. Previous research has suggested that children with ADHD-Hyperactive/Impulsive Type differ cognitively from both children with ADHD-Predominantly Inattentive Type and ADHD-Combined Type, with no cognitive deficits typically observed in children with the Hyperactive/Impulsive subtype (Gadow et al., 2004; Todd et al., 2002; Schmitz et al., 2002; Nigg, Blaskey, Huang-Pollack, & Rappley, 2002). Because of this, we dropped the 20 participants with Hyperactive/Impulsive Type and conducted another repeated measures ANOVA with two between subjects factors [(RD or not RD) and (ADHD or not ADHD)] and one within subjects factor (Motor versus Verbal PS). The results mirrored those found above for the larger sample, both with and without IQ included as a covariate.

Table 5 First order (*r*) and Partial (*pr*) correlations

	<i>r</i>	<i>r</i> ²	<i>pr</i>	<i>pr</i> ²	Change in <i>r</i> ² (%)
Factor 1					
ADHD and RD symptoms (Factor 1)	0.28	0.08	0.18	0.03	-60%
ADHD symptoms and Factor 1 (RD)	0.26	0.07	0.13	0.02	-76%
RD symptoms and Factor 1 (ADHD)	0.54	0.29	0.50	0.25	-13%
Factor 2					
ADHD and RD symptoms (Factor 2)	0.28	0.08	0.15	0.02	-73%
ADHD symptoms and Factor 2 (RD)	0.32	0.10	0.20	0.04	-60%
RD symptoms and Factor 2 (ADHD)	0.53	0.28	0.48	0.24	-17%

Note. All correlations and partial correlations are statistically significant. Formula for partial correlation: $p_{r_{xy.z}} = r_{xy} - [(r_{xz})(r_{yz})] / \sqrt{(1 - r_{xz}^2)(1 - r_{yz}^2)}$. Formula for percent change in *r*²: $\Delta r^2(\%) = [(pr^2 - r^2) / r^2] \times 100$ (Cohen & Cohen, 1983). Variable in parentheses of the row headings is the variable that is controlled in that partial correlation. So, the first column heading means the partial correlation between ADHD and RD controlling for Factor 1.

Table 6 Error analysis by task

# Errors	Comparison (<i>N</i> = 144)	ADHD only (<i>N</i> = 105) M (SD)	RD only (<i>N</i> = 95) M (SD)	RD + ADHD (<i>N</i> = 51) M (SD)	<i>F</i>
RAN					
Colors	0.0 _a (0.2)	0.1 _{a/b} (0.3)	0.1 _{a/b} (0.3)	0.2 _b (0.4)	3.4
Numbers	0.1 _a (0.4)	0.1 _a (0.4)	0.2 _a (0.7)	0.1 _a (0.4)	0.6
Letters	0.1 _a (0.4)	0.2 _a (0.5)	0.3 _a (0.6)	0.6 _b (1.1)	9.1
Pictures	0.0 _a (0.2)	0.0 _{a/b} (0.2)	0.0 _a (0.2)	0.1 _b (0.3)	2.0
CPS	1.0 _a (1.0)	1.2 _a (1.1)	1.9 _b (1.5)	1.8 _b (1.4)	12.0
Trails					
A	0.2 _a (0.4)	0.4 _a (2.0)	0.1 _a (0.4)	0.4 _a (0.8)	1.6
B	0.4 _a (0.7)	0.6 _a (1.0)	0.8 _{a/b} (1.4)	1.2 _b (2.4)	4.9
Identical pictures	0.9 _a (2.3)	0.6 _a (1.0)	1.2 _b (1.7)	0.9 _{a/b} (1.0)	1.4

Note. *df* = 3,391. Means with different subscripts are significantly different, *p* < .05.

There is also a debate in the literature about whether Predominantly Inattentive Type and Combined Type are really separate disorders, with some researchers arguing for their dissociability (Milich, Balentine, & Lynam, 2001; Carlson, Shin, & Booth, 1999) and others arguing that they are cognitively similar (Chhabildas et al., 2001; Nigg et al., 2002). To determine whether there are differences between the Predominantly Inattentive and Combined subtypes on PS, we conducted an independent samples *t* test which revealed no differences between the two groups on either Factor 1 or Factor 2. In addition, comparisons among the groups were conducted for individuals who only met criteria for Predominantly Inattentive Type, and repeated with individuals who only met criteria for Combined Type. Similar results were found for both subtypes.

Speed-accuracy trade-off

Another explanation for the slower response speed of individuals with RD and/or ADHD is that they are engaging in a speed-accuracy trade-off, slowing down in order to be more accurate in their responses. If this is the case, individuals in the pure ADHD, pure RD, and Comorbid groups would be expected to make fewer errors than individuals in the control group. To address this possibility, we analyzed the number of errors on all variables that collected error information: namely, the RAN, CPS, Identical Pictures, and Trailmaking Tests. We conducted a MANOVA with two between-subjects factors [(RD versus not RD) and (ADHD versus not ADHD)] on each dependent variable. See Table 6 for means by group. For some tasks, individuals in the clinical groups had substantial deficits, whereas for other measures, they did not differ significantly from controls. However, in no case did individuals in the clinical groups make fewer errors than individuals in the control group. As an additional test of speed-accuracy trade-off, we correlated Trails A and B reaction time with Trails A and B number of errors, as it was

the only task which generated both RT and number of errors. Correlations were significant, such that the slower participants completed each section of this measure, the more errors they made. The opposite pattern would have been found if their speed had been the result of a speed-accuracy trade-off, with faster RT's resulting in more errors. Taken together, these two pieces of evidence suggest that slow PS in these groups does not result from a speed-accuracy trade-off.

Discussion

The current study was an attempt to understand PS in RD and ADHD, and to determine whether PS is a shared cognitive risk factor for both RD and ADHD. In order to understand PS in these two disorders, it was necessary to first determine whether our battery of PS tasks tap a single cognitive construct or multiple dimensions of PS. The results from the factor analysis suggested that although all tasks believed to be tapping PS were highly correlated, they fell into two distinct but correlated factors. The first factor included tasks that required rapid naming of stimuli, and the second factor included tasks tapping speeded motor and non-linguistic abilities.

The second question of interest was whether the profiles of PS deficits are similar in RD and ADHD. Results indicate no significant group by factor interactions, suggesting that the deficits for the two factors are similar in pattern, though different in magnitude, across groups, with the PS deficit being much larger in RD than ADHD. This finding may be a true magnitude difference in the PS deficits within each clinical group. However, it may also be at least partially accounted for in the way the disorders are defined. RD is defined by the participant's performance on cognitive tasks administered in the lab while ADHD is a behaviorally defined disorder using caregiver ratings of daily behavior.

Finally, the third question of interest was whether the effects of each disorder on PS are independent or partially shared. If the deficits in the comorbid group had been the sum of the deficits of the ADHD and RD groups, this would have suggested an independent, additive relationship. In contrast, there was a significant interaction between RD and ADHD that was underadditive. The effect size of the PS deficit in the comorbid group was less than the sum of the effect sizes in the pure RD and ADHD groups. Indeed, the comorbid group did not differ significantly from the RD group on either PS factor.

Our second test of this relationship using partial correlations converged with the underadditivity method to suggest that PS accounts for a substantial part of the relationship between RD and ADHD. More specifically, however, the partial correlation analyses suggest that partialling RD significantly reduces the relationship between PS and ADHD, whereas partialling ADHD significantly but slightly reduces the relationship between PS and RD. This asymmetric relationship may be due to the greater magnitude of the PS deficit in RD than ADHD. Or, it may be more closely related to method variance in the way the disorders are defined. Since cognitive tasks are more likely to be related than information collected by two different methods, this asymmetry may be at least partially accounted for by method variance. However, this asymmetry is potentially meaningful and deserves further attention in future research.

Taken together, the varying methods used to test our third hypothesis converge on the interpretation that PS does not fully mediate or account for the relationship between RD and ADHD, but that the association between these two disorders cannot be fully explained without taking PS into account. Future research should focus on the search for additional cognitive domains that could be incorporated into a more inclusive model of shared cognitive risk between these two disorders.

Theoretically, it seems most plausible that PS is a shared cognitive risk factor that causes or at least contributes to the development of both RD and ADHD and help explains their common comorbidity. This perspective is consistent with the cognitive endophenotype explanation of psychological disorders (e.g. Doyle et al., 2005). Additionally, developmental data shows no evidence of one disorder emerging earlier and thus causing the other disorder, which eliminates the possibility of PS deficits causing one disorder which then mediates the relationship between PS and the other disorder. However, the current research does not rule out the possibility that having each disorder somehow causes PS deficits. These diagnoses could in turn lead to additional cognitive problems theoretically, and to our knowledge we have no developmental evidence of PS deficits emerging earlier than the development of these disorders.

The partial correlation tests help address this question of directionality. The common cause model suggests that PS causes symptoms of both RD and ADHD and helps explain their overlap. Thus, if PS deficits are correlated at .53 with RD (Factor 2) and .32 with ADHD, the product of those correlations is .17, which explains a good portion but not all of the .28 correlation between symptoms of RD and symptoms of ADHD. This explanation, however, explains more variance in the relationship between RD and ADHD symptoms than the mediational version of these models do (e.g. PS causes ADHD which then causes RD). Nevertheless, the question of directionality is not fully explained by the current analyses and thus is an important topic for future research.

The relationship between RD and ADHD has presented us with a paradox, since twin studies have determined the two disorders have a shared genetic etiology (Willcutt et al., 2000; Willcutt et al., 2002) whereas neuropsychological studies have suggested a pure cognitive double dissociation between the two groups (Pennington et al., 1993). Recent work looking at multiple cognitive domains across the two disorders did find some evidence for shared cognitive domains, the most prominent of which was PS (Willcutt et al., 2005). In addition, twin studies have suggested that PS is genetically related to symptoms of ADHD (Chhabildas, 2003). The current study examined a wider array of PS tasks than Willcutt and his colleagues (2005) and also did not find evidence of a double dissociation in the domain of PS. Instead, the current findings suggest that both groups have a shared deficit in PS that extends to all tasks comprising PS included in this battery, with RD having a stronger relationship with PS than ADHD.

Importantly, this study presents a new method for looking at potential shared risk factors. If two disorders are comorbid, looking at the profile of deficits in the comorbid group compared to those of each single group should help address whether the underlying risk factor is shared. The logic is fairly straightforward. If the effects are independent, then the profile in the comorbid group should be either the sum of the profiles in each pure group, with no RD by ADHD interaction, or will be greater than the sum of the profiles in the pure groups. If the effects are at least partially shared, the profile in the comorbid group should be less impaired than the sum of the profiles in each pure group, producing an RD by ADHD interaction in the direction of underadditivity. This methodology is supported by a second test of the cognitive overlap using partial correlations, which suggests it is a valid test of cognitive overlap between two disorders.

However, future research needs to explore the underadditivity methodology further. First, the logic of the underadditivity approach may be subject to some limitations. For example, in some types of tasks, floor effects could limit the range of responses and thus contribute to an underadditivity pattern unintentionally. For PS this is less of a problem, as in

speeded tasks there is a wider range of naming and response speed than in other types of tasks. Nonetheless, it is important to consider the potential confounds of floor effects and test underadditive results with converging methodologies, as this paper has done. Because our second method used continuous rather than group data, it is less susceptible to floor or range problems in a selected sample. In the future, a test of the genetic relations among RD, ADHD, and PS would extend the current findings by determining if the relation of PS to the relation of RD and ADHD is partially due to shared genes. Also, this project has attempted to explore PS as a major source of cognitive overlap based on previous work (Willcutt et al., 2005), and we believe that PS may show a more strongly underadditive pattern than deficits that may be more unique to each pure disorder. Yet in the study by Willcutt and colleagues (2005) which included a wide range of cognitive domains, there are several other cognitive domains that may trend towards underadditivity as well. It remains important for future research to explore other cognitive domains that show a similar pattern.

Implications for multiple deficit models

Finding a shared cognitive risk factor between two comorbid disorders may lend support for a multiple deficit explanation of each disorder (Pennington, 2005). Previous research has typically been focused on finding one causal cognitive deficit in each of these disorders (e.g. executive functioning in ADHD or phonological deficit in RD). Our finding that a PS deficit is shared by RD and ADHD suggests that each disorder may result from a different combination of cognitive deficits, some shared and some not shared. This multiple cognitive deficit model is more consistent with the multifactorial etiologies of these disorders.

Understanding disorders from a multiple deficit perspective also helps explain the high rates of comorbidity between different disorders. Common genes underlying two disorders may lead to a developmental change in a single pathophysiological substrate which in turn increases risk for both disorders. Then, in combination with other variants of genes related to each disorder, either the single or comorbid picture would arise (Willcutt et al., 2005; Pennington, 2005). The current work suggests that perhaps PS is one deficit that contributes to both RD and ADHD, yet this multiple deficit model needs to be tested more systematically for each of these two disorders alone as well as together.

These findings also lend support to existing multiple deficit explanations about each of these disorders. As reviewed earlier, Wolf and Bowers (1993, 1999) have developed a dual deficit model of RD that includes both a phonological deficit component and a deficit in underlying naming speed, proposing that the automaticity prob-

lems seen in RD may be partially phonological but are also partially due to problems in temporal processing and perceptual speed. While the current work does not address the underlying mechanisms for slow PS in individuals with RD, it does suggest that deficits in RSN demonstrated by many individuals with RD may be better accounted for by a general PS deficit than as a by-product of a phonological deficit.

A multiple deficit explanation of ADHD is supported by the fact that not all individuals with ADHD have an executive deficit (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2004) and that a nonexecutive deficit in delay aversion accounts for independent variance in ADHD symptoms (Solanto et al., 2001). In addition, there is another competing hypothesis in the ADHD literature which suggests that individuals with ADHD are unable to allocate enough effort to meet task demands, which results in a non-optimal energetic “state” in which they are unable to perform well on both laboratory and life tasks (Sergeant, Oosterlaan, & van der Meere, 1999). One of the main pieces of evidence for this theory stems from findings that individuals with ADHD are slower and more variable on simple RT or choice RT paradigms. The theory proposes that individuals with ADHD are unable to adjust their cognitive arousal level in order to sustain an adequate level of attention to these simple tasks, thus presenting as slower and less consistent. The current findings did not include simple RT tasks within our final battery of PS tasks, but we did find that RT cross loaded on our two PS factors, suggesting a possible relationship. So, the current findings may lend support for the cognitive energetic theory. However, we still lack a good understanding of the cognitive mechanisms of PS as a construct.

What constitutes PS?

A series of potential hypotheses could be proposed about the nature of PS. First, it may be that all speeded tasks are tapping the same construct whether they measure simple RT, naming speed, executive decision making, etc. Second, it may be that all tasks with a speeded component may tap into a common underlying processing speed function but also require more task specific aspects of cognitive efficiency (e.g. rapid serial naming may require the common speed component as well as language processing efficiency). Third, it may be that speeded tasks measure speed at which individuals perceive and process incoming information, they may measure output speed, or it could be that some speeded tasks require an integration of rapid perceptual, cognitive, and output processing. Finally, it may be that different speeded measures are actually tapping distinct processes (simple RT versus rapid naming versus rapid performance of executive tasks).

Studies using the RAN have addressed the question of level of processing demand. If the RAN deficit for children with RD was solely due to perceptual speed or output speed, the results from presenting stimuli in a discrete trial modality should also be correlated with their reading ability. However, performance in discrete trial naming is less consistently related to reading ability than when using the continuous version of this task (Perfetti et al., 1978), which suggests that something about the cognitive processing load is a specific problem for children with RD.

The results of the current study suggest that, at least in RD and ADHD, there is some common dimension that is shared across different domains in these two disorders. The fact that these two PS factors are correlated at 0.7 suggests they are highly related yet partially distinct processes in these two disorders. The similar profile suggests they are performing similarly in each group. However, there may be other aspects of PS that we have not captured in the current analyses. For example, we cannot separate out perceptual speed from cognitive or output speed, and thus cannot determine whether ADHD and RD differ on which aspect of PS presents difficulty for each disorder. Additional research is needed to understand the cognitive and neural construct of PS within these two clinical groups.

Additionally, future research should address how PS operates in different developmental disabilities. Other clinical populations may demonstrate PS deficits for different reasons. Profiles of PS strengths and weaknesses might vary in different disorders, depending on the unique cognitive profile of the disorder. Are there some disorders where individuals are impaired on all speeded tasks, from simple RT to more complex cognitive tasks, and other disorders where individuals are affected in some PS tasks and not others? Are there disorders that have difficulty with different aspects of information processing, such as perceptual speed versus cognitive speed? Research should also address the specificity of PS deficits to ADHD and RD. Perhaps PS is similar to executive function deficits in that it is a non-specific cognitive deficit occurring in many clinical populations with cognitive delays. Future research should examine this cognitive process more thoroughly.

Another possibility for future study would be to understand the directionality of the PS deficits in these two disorders. Specifically, we do not know if PS deficits are an underlying causal mechanism or a consequence of having RD or ADHD. The first step in determining causality would be to establish whether PS deficits are genetically related to symptoms of ADHD or RD. One preliminary study has examined the genetics of EF deficits in ADHD and found that PS is genetically correlated with symptoms of ADHD (Chhabildas, 2003). Careful longitudinal studies will also be necessary to address causality and determine whether PS deficits are present earlier than the onset of

ADHD and RD symptoms, or develop later, perhaps as a consequence.

Additional limitations of the current study

The present results should be interpreted in light of several additional limitations. Although previous studies have found few significant differences between twins and non-twins (Plomin, DeFries, McClearn, & McGuffin, 2001), these analyses should be replicated in an independent sample of nontwins to test if the findings generalize to the population at large. Second, because this study has been ongoing for almost 20 years, some of the measures in the current battery of tests are older versions of tasks that have remained in the battery in order to allow for comparisons across the sample (e.g. WISC-R Coding). Also, we were unable to thoroughly test the question of speed-accuracy trade-offs because we did not collect error information for many of the measures in this battery. Thus, our error analyses were not able to completely rule out this potential confound on other variables in the model. Finally, *DSM-IV* ADHD was defined by parent and teacher ratings on the DBRS rather than a full structured diagnostic interview. Although all participants who were included in the ADHD group were required to meet full *DSM-IV* criteria for ADHD, the possibility remains that the use of rating scales could influence the results. To test this possibility, the parents of 30 children with ADHD and 30 children without ADHD based on the DBRS also completed the *DSM-IV* version of the DICA (Reich, Welner, & Herjanic, 1997). The concordance between diagnoses derived from the DBRS and the DICA-IV was extremely high (97% agreement; $k = 0.93$) suggesting that these methods yield similar results.

Conclusions

This study lends support to the results of recent work by Willcutt and his colleagues (2005) suggesting that RD and ADHD may share PS as an underlying cognitive risk factor. The results suggest that a general PS deficit exists for both clinical groups compared to controls, although children with RD demonstrate greater PS deficits than children with ADHD. Two tests were conducted to test whether the similar profile of PS deficits in RD and ADHD is consistent with PS being a cognitive risk factor shared by these two disorders. Since we found that PS deficits were underadditive in the comorbid group and that partialling PS reduced the correlation between RD and ADHD, it appears that PS is a shared cognitive risk factor that may help explain the comorbidity of these two disorders. These findings also lend support to increasingly popular multiple deficit models for each of these two disorders, and raises important areas for future research about PS as a construct.

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