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Is Executive Dysfunction a Potential Contributor to the Comorbidity between Basic Reading Disability and Attention-Deficit/Hyperactivity Disorder?

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Abstract

Our study is one of the few to analyze executive functioning (EF) in a *comprehensive, multi-modal fashion* as a potential contributor to the comorbidity between attention-deficit/hyperactivity disorder (ADHD) and basic reading disability (RD).We included multiple, traditional, neuropsychological measures of EF, along with the Behavior Rating Inventory of Executive Function (BRIEF) questionnaire, to assess inhibit, shift, working memory (WM), planning, generation fluency, and problem-solving. Participants included 263 children, ages 8-12 years, with RD, ADHD, RD/ADHD, and typically developing controls. When using the traditional measures in a 2 X 2 MANCOVA, we found both RD and ADHD had poor cognitive EF in most areas at the group level, with phonological loop deficits being more specific to RD and behavioral regulation deficits being more specific to ADHD. Children with RD/ADHD performed comparably to those with RD and ADHD alone. Results were similar on the BRIEF. In contrast, only WM predicted both basic reading and inattention when the data was assessed in a continuous fashion. It also explained the correlation between basic reading and inattention, being worthy of longitudinal research to determine if it is a shared contributor to RD/ADHD. When comparing hypotheses as to the nature of RD/ADHD, we found the multiple deficit hypothesis was better supported by our EF data than the phenocopy hypothesis or the cognitive subtype hypothesis.

Keywords

ADHD; Reading Disorder; Reading Disability; Executive Functioning; Comorbidity

Of interest in the literature over the past couple of decades is the source(s) of the comorbidity between basic reading disorders (RD) and Attention-deficit/hyperactivity disorder (ADHD). ADHD and RD are two of the most common neurodevelopmental disorders, and their comorbidity is much greater than expected based upon the base rate of either disorder alone (~20-40%; Willcutt and Pennington 2000a). Nevertheless, the extant

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Declarations

The authors have no conflicts of interest to report.

The measures included in this study are part of a larger dataset. The larger dataset is being used as part of other ongoing projects, and, hence, it is not deposited in a repository. This data is available for data checking upon request to the corresponding author.

literature lacks consensus as to the contributors to this comorbidity. In addition, the literature lacks consensus as to which hypothesis regarding the nature of comorbid RD/ADHD (RD/ADHD) is correct. Given this lack of agreement, it is imperative that researchers continue to investigate the possible reasons for this comorbidity. Therefore, as both disorders present with executive functioning (EF) deficits at a group level, the purposes of this study were to determine which EF deficits are shared versus specific to RD and ADHD and to determine which hypothesis regarding the nature of RD/ADHD is best supported by the data. It should be noted that this study is focused upon basic reading ability, rather than fluency or comprehension, as the latter abilities have differing contributors along with those that overlap with basic reading. Hence, it is worth assessing their inter-relationships with EF and ADHD in a separate study. To date, processing speed has been shown to be a shared deficit in RD and ADHD, contributing to their comorbidity (McGrath et al.,2011; Rucklidge and Tannock 2002; Willcutt et al. 2010), and EF deficits may be another. Our study is particularly suited to address the debate on executive dysfunction in RD/ADHD because it incorporates several areas and measures of EF, rather than just including a few measures or just focusing on one or two executive functions, and because it includes traditional measures typically administered in a clinic along with a commonly used questionnaire (Behavior Rating Inventory of Executive Functioning; BRIEF) to assess EF in daily life. Prior research typically has used either the traditional measures or the BRIEF instead of including both methods.

There are multiple hypotheses regarding the nature of RD/ADHD (see Germano et al. 2010; Willcutt et al. 2005). Of those published, three tend to be the most commonly studied: phenocopy hypothesis (Pennington et al. 1993), cognitive subtype hypothesis (Rucklidge and Tannock 2002), and common deficit/multiple deficit hypothesis (Pennington 2006; Willcutt et al. 2005b; 2010). The phenocopy hypothesis was among the first published on RD/ADHD. It purported that the comorbid condition is due to a “copy” of the symptoms of one of the conditions; one condition is driving the symptoms of the other rather than the child having the deficits associated with both conditions. For example, children with RD/ADHD could look inattentive due to their reading deficits and frustration in the classroom but not have the deficits associated with ADHD on neuropsychological testing. For this hypothesis, when using a 2 (RD or not) by 2 (ADHD or not) design, there would be a double dissociation between RD and ADHD alone in their neuropsychological deficits, but the comorbid group would be characterized by the deficits of one of the disorders rather than both. This hypothesis has had limited support, and it failed to be replicated by Pennington and colleagues when they gathered additional data (Willcutt, Pennington et al., 2001). As a result, Pennington, Willcutt and colleagues later developed the common deficit (Willcutt et al. 2005b) and then the multiple deficit hypothesis (Pennington 2006).

The common deficit hypothesis states that ADHD and RD are separate disorders that share certain neurobiological, genetic and/or cognitive contributors. The multiple deficit hypothesis builds upon this, acknowledging the heterogeneity of causes contributing to each disorder. It suggests that both RD and ADHD have multiple causes/deficits associated with them and that the shared deficit(s) contributes to their comorbidity. For this hypothesis, when using the 2 by 2 design, there would be a double dissociation between RD and ADHD in the deficits that are not shared, similar to the phenocopy hypothesis, but the comorbid

group would have deficits consistent with both conditions, in contrast to the phenocopy hypothesis. Further, for the shared deficit(s),all three clinical groups would perform worse on it than controls. This may result in an ‘under-additive’ interaction, as found by Shanahan and colleagues (2006) when studying processing speed in RD and ADHD. Because neuropsychological measures are not ‘pure’ and assess more than one function, it also may yield a non-significant interaction term if the comorbid group showed some simple additive effects due to the additional functions the test is measuring that are affected more substantially in RD or ADHD. It also could yield a non-significant interaction term given the substantial heterogeneity within groups or due to low power. For example, Willcutt and colleagues (2005b) did not find significant interactions when putting forward the common deficit hypothesis. As a shared predictor accounts for at least a portion of the relationship between reading and inattention when taking a dimensional approach, the (significant) correlation between reading and inattention symptoms should be at least partially accounted for by the affected cognition when using partial correlation. This hypothesis has support from the neuropsychological (Cheung et al. 2014; McGrath et al. 2011; Moura et al. 2017; Tiffin-Richards et al. 2007; Willcutt et al. 2005b) and genetic (Willcutt and Pennington 2000b; Willcutt et al. 2010) literature.

The cognitive subtype hypothesis states the comorbid condition is a different, more severe form of the disorder than RD or ADHD alone. When using a 2 by 2 design, the comorbid group would have significantly worse deficits than RD and ADHD alone on at least some tasks, and/or they would have additional deficits that RD and ADHD alone do not have. Hence, the interaction term would be significant for these deficits, being due to a deficit that is specific to this group or due to ‘over-additive’ effects. At least a few studies’ data have supported this hypothesis (McGee et al. 2004; Rucklidge and Tannock 2002; Willcutt et al. 2001).

Based upon the cognitive and neuropsychological literature, EF is an ‘umbrella term’ that is hard to define, but it is believed to be a multi-faceted/multi-factorial set of abilities that are involved in goal-directed behavior (Castellanos, Sonuga-Burke, Milham, &Tannock, 2006; Lyons et al., 2016). Currently, there is no widely agreed upon definition or model of EF. Nevertheless, most models of EF, and many researchers studying EF, include shift/flexibility, updating/working memory, and inhibition as core components, consistent with the work of Miyake and colleagues (Miyake, Friedman, Emerson, Witzki, Howerter, & Wager, 2000), who called them ‘lower-order’ executive functions. Nevertheless, some researchers view inhibition as being a single entity, similar to Miyake et al.’s model, whereas others separate it into response inhibition and interference control. Miyake et al. noted that ‘higher-order’ executive functions build upon these three ‘lower-order’ functions, frequently relying on two or more of the ‘lower-order’ functions during their execution. Others have supported this notion (Collins & Koechlin, 2012; for a review, see Diamond, 2013; Lunt, Bramham, Morris, Bullock, Selway, Xenitidis, & David, 2012). The ‘higher-order’ functions may include problem-solving, planning/organization, monitoring, initiation, attention control (especially the challenging aspects such as divided attention),and generation fluency (verbal and nonverbal fluency; Alvarez & Emory, 2006; Cirino, Ahmed, Miciak, Taylor, Gerst, & Barnes, 2018; Levin, Culhane, Hartmann, Evankovich, Mattson, Harward, … Fletcher, 1991; Lezak, Howieson, Bigler, & Tranel, 2012; Lyon & Krasnegor, 1996; Pennington,

2009), depending upon the author. Executive functioning also has been broken down into cognitive, emotional, and behavioral regulation domains (e.g., Egeland &Fallmyr, 2010;Gioia et al., 2015). Our study assessed all the components of EF mentioned above via measures commonly used in clinical practice except divided attention, verbal fluency, and interference control, as we did not have measures of these functions. Our study also assessed all three regulation domains via its inclusion of the BRIEF.

In contrast to EF as a whole, there is a model of working memory (WM) that is widely accepted and used in the neuropsychological literature: Baddeley's WM model (2012). The original version included a phonological loop component that temporarily stores verbal information (consistent with verbal short-term memory), a visuospatial sketchpad that temporarily stores visual-spatial information (consistent with visual short-term memory), and a central executive (CE) that is involved with attention control, allocation of (limited capacity) resources to the stores, mental manipulation of material/updating, and potentially other executive functions depending upon the author cited. Based upon Baddeley's(2000)revision, the model now includes the episodic buffer as well, which functions include binding the various aspects of a memory (including facets from both WM and long-term memory) to create a coherent memory trace. However, the existence and nature of the episodic buffer have been more debated in the cognitive literature than the other components(Cortis et al., 2015; Spurgeon et al., 2014), and it is not commonly studied in neuropsychology. Thus, it will not be discussed further.

In regard to EF in individuals with RD, they display deficits in verbal fluency (Moura et al., 2017; Varvara et al., 2014), organization and set-shifting (Moura et al.,2015, 2017), and problem-solving (McLeskey 1980) as compared to controls. Moreover, children with RD frequently display poor working memory (Cheung et al., 2014; Kibby and Cohen, 2008; Kibby, 2009; Moura et al., 2015, 2017). In fact, impairment in WM, especially verbal WM, constitutes one of the most severe EF impairments in children with RD (Booth et al., 2010; Rose and Rouhani, 2012).The WM deficit in RD may be due to dysfunction within the phonological loop (Kibby 2009; 2012; Kibby and Cohen 2008; Kibby et al. 2004), although deficits in the CE (e.g., Moura et al. 2015) and visuospatial sketchpad (e.g., Howes, Bigler, Burlingame, & Lawson, 2003)have been found. Despite some research suggesting that individuals with RD exhibit deficits in planning (e.g., Klorman et al. 1999), the literature is not consistent in this regard (e.g.,Reither et al. 2005). Although the study conducted by Reiter and colleagues (2005) did not find differences in accuracy on the Tower of London task, children with RD tended to take longer to plan, which the researchers hypothesized was due to slow processing speed. Research also is disparate regarding inhibition (Cheung et al. 2014; de Jong et al. 2009; McGrath et al. 2011; Reither et al. 2005; Rucklidge and Tannock 2002; Willcutt et al. 2005a; Willcutt et al. 2010), but it appears that when inhibition is affected, it typically is on cognitive tasks and/or those requiring processing speed or WM (e.g., Stop Signal reaction time), rather than problems with impulsivity in daily life or commission errors on laboratory-administered measures. It also tends to be in a milder form than that found in ADHD (Narhi and Ahonen, 1995; Willcutt et al., 2001). In contrast to cognitive regulation/control, children with RD do not tend to display poor emotional or behavioral regulation as a group (Gioia et al. 2002; Puris and Tannock 2000; Smith-Spark et al. 2016).

The most widely accepted hypothes is regarding the etiology of ADHD is the frontal-striatal theory (Castellanos and Proal 2012; Faraone et al. 2015), which postulates that the prefrontal cortex and basal ganglia are not functioning effectively in individuals with ADHD. Relatedly, they frequently display cognitive EF deficits in WM, generation fluency, inhibition, planning, and/or shift (de Jong et al. 2009; McGrath et al. 2011; Rucklidge and Tannock 2002; Takacs et al. 2014; Willcutt et al. 2005a). Nevertheless, not all individuals with ADHD have EF deficits, and most who have EF deficits are only affected in one to a few areas rather than all of them (Nigg et al. 2004; Wahlstedt et al. 2008). In terms of WM, deficits have been found in all three components when using Baddeley's model, although deficits in the visual-spatial sketchpad and on visual-spatial CE tasks are more commonly found (Cheung et al. 2014; Kibby 2012; Kibby and Cohen 2008; Martinussen et al. 2005; Moura et al. 2017). Research on emotional/behavioral regulation in ADHD is mixed, with some studies finding a deficit and others finding typical functioning (Antonini et al., 2016; Barkley, 2015; Corbisiero et al., 2017). However, it has been suggested that this may be due to investigating ADHD as a whole rather than focusing on each subtype (Toplak et al. 2005), as emotional and behavioral regulation problems are more strongly associated with the hyperactive/impulsive dimension (Chang et al. 2014;Gioria et al. 2002;Pratt 2000).

Thus far,at least three potential sources of shared etiology for RD and ADHD have been proposed: poor (cognitive) EF (Cheung et al. 2014; de Jong et al. 2006), poor phonological loop/focused auditory attention functioning (Kibby and Cohen 2008) and slow processing speed (e.g., McGrath et al. 2011). When comparing RD/ADHD to RD and/or ADHD on EF measures, RD/ADHD does not tend to differ from RD or ADHD (Moura et al., 2017; Pratt 2000; Tiffin-Richards et al. 2007; Willcutt et al. 2001), manifesting deficits of both disorders. Within EF, the most commonly found shared deficit is WM, particularly on tasks assessing the CE. Nonetheless, some suggest that EF, including WM, is not a shared contributor when controlling for non-EF abilities (McGrath et al. 2011; Willcutt et al. 2010), which may be due to the tasks used to measure EF and non-EF cognitions (e.g., some of their non-EF tasks like pig-Latin likely require WM functioning). In terms of the phonological loop, Kibby and Cohen (2008) showed children with RD and ADHD had impaired performance on forward digit span, and the RD/ADHD interaction term was not significant. All three clinical groups (RD, ADHD, and the comorbid condition) performed worse than controls on forward digit span, consistent with the common/multiple deficit hypothesis. Only children with ADHD (ADHD, RD/ADHD) had impairments in the visuospatial sketchpad. In favor of the cognitive subtype hypothesis, Rucklidge and Tannock (2002) found the comorbid group had worse impairment than RD and ADHD on Stroop color/word naming and the stop-signal task, suggesting greater problems with inhibition and naming in RD/ADHD.

Reading, attention, and motor/impulse control function on a continuum (AKA "dimension"), rather than being categorical in nature (e.g., reading disorder or not). Few studies have used a continuous data approach, but when studied in this fashion EF, particularly WM, shows some promise as a shared deficit (Cheung et al., 2014; Tiffin-Richards et al., 2007). Nevertheless, other studies taking a dimensional approach showed verbal WMis related to reading but not ADHD symptoms (Rucklidge and Tannock 2002; Willcutt et al. 2010) or found verbal WM is not related to reading nor ADHD symptoms when controlling other

cognitions (McGrath et al. 2011); again, some of the cognitive measures controlled likely included a verbal WM component. In terms of the phonological loop, a study demonstrated that its functioning predicted both reading and inattention symptoms (Cheung et al. 2014), although it may be a better predictor of reading than inattention given the findings on RD and ADHD presented above. In general, reading performance is better associated with the inattention dimension than the hyperactivity/impulsivity dimension neuropsychologically (e.g., Cheung et al. 2014) and genetically (Cheung et al. 2014; Willcutt et al.2010).

As noted in the opening paragraph, one aim of this study was to determine which aspects of EF are affected in both RD and ADHD at a group level when using a comprehensive, multi-modal approach, being potential shared deficits, and which are specific to either disorder. Consistent with this aim, we also sought to determine whether the various aspects of EF may be shared statistical predictors of basic reading, inattention, and/or hyperactivity/impulsivity when taking a dimensional approach. Using both types of approaches aids generalization to the existing literature and provides better support for any potential shared predictors found. The second aim was to determine which of the three hypotheses regarding the nature of RD/ADHD is best supported by our data. Based upon the literature reviewed, we hypothesized that children with RD would display worse cognitive EF than those without RD, and that children with ADHD would display worse functioning on all EF domains (cognitive, behavioral, and emotional regulation) versus those without ADHD when using a 2 (RD vs. no-RD) by 2 (ADHD vs. no-ADHD) design. Consistent with the multiple deficit hypothesis, children with RD/ADHD were expected to have deficits consistent with both disorders (simple additive effects), with a shared deficit(s) in cognitive EF, particularly WM. For the shared deficit(s), all three clinical groups (RD, ADHD, and RD/ADHD)were expected to perform worse than controls. When taking a dimension approach, it was hypothesized that cognitive EF measures, particularly WM, would statistically predict both basic reading and inattention when using multiple regression, being a potential shared contributor. For the shared predictor(s),the correlation between reading and inattention symptoms would be at least partially accounted for by the shared deficit(s) when using partial correlation. In terms of WM, it was hypothesized that both the CE and phonological loop would be shared statistical predictors.

Methods

Participants

Participants included 263 children, ages 8-12 years, who were tested in a university laboratory. The participants were recruited via larger, NIH-funded projects that included children with RD, ADHD, RD/ADHD, and controls (RD $n = 49$, ADHD $n = 91$, RD/ADHD $n = 49$, controls $n = 74$).Various studies have been published using this data, but none focused on this topic. The groups did not differ in age, maternal education, assigned sex at birth, or race/ethnicity, ps > .05.These four measures were gathered from parent report. Unfortunately, data related to gender-orientation were not gathered. Groups did differ in IQ as measured with the TONI-3, $F(3, 262) = 11.14$, $p < .001$. Games-Howell post-hoc testing revealed that controls scored better than the three clinical groups, who were comparable to each other. Descriptive data are displayed in Table 1.

ADHD was diagnosed by a child clinical neuropsychologist based upon DSM-IV criteria, as the DSM-5 was not available at the time of data collection. Interview data, questionnaires, and behavioral observations were used to determine diagnosis and subtype. Along with the interview, the Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds and Kamphaus 2004) parent and teacher questionnaires were used to determine the severity of inattention and hyperactivity/impulsivity symptoms in the school and home settings, as well as DSM-IV symptom count from parent report. For children being treated with medication, they were not allowed to be on medication during testing. If the medication was short-acting, they were not allowed to take it the evening before, or the day of, testing. If it were sustained release, they were not allowed to take it for an entire day or two before the evaluation, along with the day of testing, based upon the medication's half-life.

RD was diagnosed using a twofold approach, keeping with both the discrepancy and low achievement/'poor reader' models of diagnosis, following the guidelines outlined by Pennington, Peterson, and McGrath (2008) and Pennington, McGrath, and Peterson (2019). They advocated for this approach as both poor readers and discrepant readers have phonological processing deficits, the core deficit in basic RD. In addition, using both definitions aids generalization. Following this twofold approach, children were classified with RD using the 'poor reader' approach if his/her performance was at least one standard deviation below the mean in basic reading. For the discrepancy approach, a child was classified with RD if his/her basic reading ability was significantly lower than expected based upon his/her IQ. The regression formula from the State of Washington was used to determine the cutoff amount. With the use of it, the cutoff amount varied based upon the IQ score (the higher the correlation, the larger the cutoff required).Both definitions required the child to have difficulty with reading that affects school performance. A child only needed to meet one definition to meet inclusionary criteria for RD. Fourteen percent of those diagnosed with RD met the 'poor reader' definition, 19% met the discrepancy definition, and 67% met both.

Children meeting the criteria for both RD and ADHD were placed in the comorbid RD/ADHD group. The ADHD and RD/ADHD groups did not differ in the proportion of ADHD subtypes, $X^2_{1,\ N=140} = .55$, $p = .59$. In the ADHD group, there were 51 children with ADHD-PI and 40 children with ADHD-C. In the RD/ADHD group, there were 30 children with ADHD-PI and 19 children with ADHD-C. The two groups also were comparable in ADHD severity; see Table 2 for information on inattention and hyperactivity/impulsivity by group.

Controls did not meet inclusionary criteria for ADHD or RD. Exclusionary criteria were applied across all four groups. They included history of medical or neurological disorder excluding allergies or asthma, significant perinatal complications, substantial environmental problems (e.g., suspected neglect or abuse, recent parental divorce or separation), and psychiatric disorders (e.g., anxiety, depression, conduct disorder). All participants had a measured IQ > 79.

To verify the four groups differed where expected in basic reading ability, inattention, and hyperactivity/impulsivity, a MANOVA was used. The omnibus test was significant, Wilk's Λ

= .18, $F(12, 664.38) = 51.14$, $p < .001$. Groups differed in symptom levels where expected (see Table 2 for univariate data and means/SDs). The comorbid group was comparable to the RD group in reading ability and to the ADHD group in inattention and hyperactivity/impulsivity.

Measures

All of the following measures have good or excellent psychometric properties in reliability and validity according to their respective manuals.

Intelligence.—The Test of Nonverbal Intelligence, Third Edition (TONI-3; Brown et al. 1997) was used to measure intelligence. It is an untimed, nonverbal measure, so it is a fairer measure of IQ for RD and ADHD than the WISC, which requires WM, processing speed, and linguistic ability.

Attention and Hyperactivity.—The Behavior Assessment System for Children, Second Edition (BASC-2; Reynolds and Kamphaus 2004) was used to assess the severity of inattention and hyperactivity/impulsivity symptoms. The Attention Problems and Hyperactivity scales' T scores were used to measure these symptoms, respectively.

Basic Reading Ability.—The Woodcock-Johnson Test of Achievement, 3rd Edition (WJ-III; Woodcock et al. 2001) was used to measure basic reading ability for the purposes of this study. The Letter-Word Identification subtest measures the ability to identify single words. The Word Attack subtest measures the ability to decode pronounceable non-words. Both measures are untimed.

EF Measures.—See Appendix for a summary of the EF tests utilized and what cognitions they were used to assess, based upon their respective manuals.

The Children's Memory Scale (CMS; Cohen 1997) measures learning and memory. The Sequences subtest was used to measure WM functioning. On this subtest, the participant is asked to say known sequences backward (e.g., days of the week), as well as perform mental counting (e.g., count by odd numbers). The sequences manipulated increase in complexity throughout the subtest, and it is timed. Numbers Forward was used as a measure of phonological loop functioning; it is a measure of forward digit span. Picture locations was used as a measure of the visuospatial sketchpad; it is a measure of forward spatial span.

The Wisconsin Card Sorting Test – 64 Card Version (WCST-64; Kongs et al. 2000) was used as a measure of problem-solving skills and shift. The Categories Completed standard score was used as the measure of overall problem-solving ability. The Preservative Errors standard score was used as the measure of shift.

The Developmental Neuropsychological Assessment (NEPSY; Korkman et al. 1998) Tower subtest standard score was used as the measure of planning ability. On this task, participants are given a board with three pegs of varying heights that hold a different number of balls. The child is asked to make their board look like the one in the stimulus book in the requested number of moves. Each trial has a time limit. The Rule Violations raw score measures response inhibition. Before the subtest begins, the child is given specific rules to follow.

When the child breaks one of these rules, a rule violation is tallied. Rule violations are not included in their Tower standard score, making it an independent score. As it is a raw score, its data were skewed. Using a square root transformation resulted in a normal distribution, so this measure was used instead.

Design Fluency from the NEPSY was used as a measure of nonverbal generation fluency. On this task, participants are asked to create novel designs on structured and unstructured templates of five dots within a box. They have one minute to complete each part of the task (i.e., structured and unstructured). The standard score was used.

The Behavior Rating Inventory of Executive Functioning (BRIEF; Gioia et al. 2000) is a questionnaire that measures EF in everyday life. It contains eight subscales (i.e., Inhibit, Shift, Emotional Control, Initiate, WM, Plan/Organize, Organization of Materials, and Monitor) that comprise two Index scores: Behavioral Rating Index (BRI) and Metacognition Index (MI).Inhibit, Shift, and Emotional Control contribute to the BRI, with the remaining subscales contributing to the MI. Parent and teacher reports were used.

Procedure

The Human Subjects Committee of Southern Illinois University - Carbondale's Institutional Review Board approved the larger project from which this data was obtained. The materials and procedures used in this study adhere to the tenets of the Declaration of Helsinki. Participants were recruited via letters sent through schools, referrals from physicians and psychologists, and advertisements through local radio and television. Informed consent for participation was obtained from all participants' parents, and informed assent was attained from the participants before data collection commenced. The sample was recruited from the communities in the larger (rural) region and tested in a research laboratory rather than being from an outpatient clinic. As a result, our cases of RD and ADHD tended to be milder than those seen in a clinic.

All children participated in a neuropsychological evaluation that included the measures described previously, in addition to other measures. Parents and teachers completed multiple questionnaires including the BASC-2 and BRIEF, and parents participated in a semi-structured clinical interview. Some respondents did not complete all measures, but the vast majority did so. More specifically,3 participants were missing parent-report questionnaires, and 11 were missing teacher-report questionnaires. When missing data occurred, the analysis removed the participant from the equation. For participating in the study, parents received a free, written neuropsychological report, and the children received a free T-shirt along with an image of their brain (MRI data was gathered as part of the larger study).

Results

Two 2 (ADHD versus no-ADHD) by 2 (RD versus no-RD) MANCOVAs were run to examine group differences in EF. The comorbid group was assessed via the interaction term. The TONI-3 IQ was used as the covariate. In the first MANCOVA, groups were compared on all the traditional measures of EF. The overall model was significant for the main effects:RD $\lambda = .71$, $F(8, 232) = 11.76$, $p<.001$, partial $\eta^2 = .29$; ADHD $\lambda = .87$, $F(8, 232) =$

4.47, $p < .001$, partial $\eta^2 = .13$. The RD x ADHD interaction term was not significant: $\lambda = .97$, $F(8, 232) = .89$, $p = .52$, partial $\eta^2 = .03$.See Table 3for univariate results.

For the BRIEF analysis, all of the subscales were entered into the MANCOVA from both parent and teacher report. The overall equation was significant for the main effects of RD,$\lambda = .86$, $F(16, 222) = 2.21$, $p = .006$, partial $\eta^2 = .14$, and ADHD,$\lambda = .45$, $F(16, 222) = 17.06$, $p < .001$, partial $\eta^2 = .55$, but the interaction term was not significant, $\lambda = .93$, $F(16, 222) = .97$, $p = .49$, partial $\eta^2 = .07$. See Table 4 for univariate results.

Next, four hierarchical regression analyses were run to determine whether there were shared predictors for the basic reading and ADHD dimensions. TONI-3 IQ was entered into Step 1, and the EF variables were entered into Step 2,including all the traditional measures as well as the parent- and teacher-rated BRI and MI. Word Attack was the dependent variable in the first regression. Step 1 was significant, adjusted R^2=.09, $F(1, 222) = 21.63$, p<.001, as was the addition of Step 2, adjusted R^2=.38; R^2 change = .33, $F(13, 210) = 11.40$, p<.001. Similarly, when Letter-Word Identification was the dependent variable, both Step 1, adjusted R^2=.13, $F(1, 221) = 33.15$, p<.001, and Step 2, adjusted R^2=.42; R^2 change = .32, $F(13, 209) = 13.15$, p<.001, were significant. Beta values are displayed in Table 5 for all regressions.

In terms of the ADHD dimensions, when predicting parent-rated Attention Problems, Step 1 of the equation was significant, adjusted R^2=.02, $F(1, 221) = 5.97$, $p = .02$, as was Step 2, adjusted R^2=.61, R^2 change = .61, $F(13, 209) = 27.66$, p<.001. Similarly, when predicting Teacher Attention Problems, both Step 1, adjusted R^2=06, $F(1, 221) = 14.14$, p<.001, and Step 2, adjusted R^2=.62, R^2 change = .58, $F(13, 209) = 28.96$, p<.001, were significant.When predicting Parent Hyperactivity, Step 1 of the model was not significant, adjusted R^2= −.004, $F(1, 221) = .07$, $p = .79$. However, the addition of Step 2 produced a significant model, adjusted R^2=.58, R^2 change = .60, $F(13, 209) = 24.37$, p<.001. Similarly, when predicting Teacher Hyperactivity, Step 1 of the model was not significant, adjusted R^2= −.004, $F(1, 221) = .006$, $p = .94$, but the addition of Step 2 was significant, adjusted R^2=.55, R^2 change = .58, $F(13, 209) = 22.24$, p<.001.

When analyzing the data from both sets of analyses (categorical and continuous), only WM, as measured by Sequences, remains as a potential shared contributor of the EF variables. Although many cognitive EF variables were significant in the two MANCOVAs for both main effects, only Sequences predicted both reading and inattention (teacher-rated) in the regression analyses. Hence, it was tested further to see if it may be a shared contributor, as outlined in the hypotheses. All three clinical groups performed worse on Sequences than controls, with (ps < .05) and without (ps < .001) controlling for IQ. When assessing the relationships dimensionally, both aspects of basic reading were correlated with teacher-rated Attention Problems: Letter-Word Identification ($r = -.22$, $p = .001$) and Word Attack ($r = -.16$, $p = .02$). However, neither was correlated with Attention Problems when Sequences was controlled in a partial correlation (Letter-Word Identification: $r = .01$, $p = .87$; Word Attack: $r = .08$, $p = .24$).

The multiple deficit model acknowledges the heterogeneity of disorders, recognizing a substantial proportion of individuals with a disorder may have a given deficit but not all will.

Hence, we sought to determine the prevalence of WM impairments across groups. Using Sequences, standard scores of 90 or greater were classified as 'not impaired'; standard scores 1 standard deviation below the mean (75-85) were classified as 'mildly impaired'; and those 2 standard deviations or more below the mean (70 and lower) were classified as 'severely impaired.' Analyses with chi square revealed significant differences in frequency between the groups, χ^2 (1, N = 263) = 67.13, p< .001. See Table 6 for frequency counts.

Discussion

The aims of this study were two-fold: first, we sought to determine which aspects of EF are shared deficits and which are specific to RD or ADHD; second, we sought to determine which hypothesis regarding the nature of RD/ADHD is better supported by our data: multiple deficit, cognitive subtype, or phenocopy .In terms of the first aim, when comparing groups, we found most aspects of cognitive EF were affected in both RD and ADHD at the group level: shift, verbal WM, problem-solving, and nonverbal fluency. In contrast, poor inhibition/behavioral regulation was more specific to ADHD, and phonological loop deficits were more specific to RD. Children with RD/ADHD displayed deficits consistent with both RD and ADHD alone, without having additional deficits or worse deficits. Moreover, verbal WM may be a shared deficit, as described below. Thus, in terms of the second aim, our data are most consistent with the multiple deficit hypothesis of RD/ADHD.

Some of the executive functions were assessed with both traditional measures and BRIEF questionnaires (shift, inhibit, WM, planning), being a unique contribution of our study; the rest of EF was assessed using one of the two approaches. Consistent with hypotheses, shift was affected on both the WCST and BRIEF parent- and teacher-reports in RD, suggesting this deficit is not dependent upon measurement method or setting. Moreover, planning was significant for both BRIEF reporters and there was a trend on the Tower, and WM was significant on Sequences and displayed a trend on parent-report. These findings are consistent with the limited prior research that has investigated EF in RD using both traditional and ecological measures (Moura et al., 2017; Smith-Spark et al., 2016). Children with RD also displayed deficits in nonverbal fluency, problem-solving, all three components of WM, initiative across settings, and self-monitoring and emotional control at school, consistent with prior research (Cheun et al., 2014; Kibby 2009; McLeskey 1980; Moura et al. 2017). These results are consistent with our hypothesis of finding cognitive executive dysfunction in RD, except emotional control at school (which was still average overall based upon its norms). Limited research has been conducted on emotional regulation in RD; however, some suggest individuals with RD have worse internalizing symptoms than their typically developing counterparts (Eissa, 2010; Willcutt and Pennington 2000b).Further, the school setting is likely stressful for children with RD .It should be noted that each EF variable's effect size was small (≤7% of variance explained) except for verbal WM (24%), but, taken together, the EF variables accounted for 32-33% of the variance in basic reading performance (decoding and word identification).

In terms of inhibition in RD, we did not find inhibition/behavioral regulation to be affected on Tower Rule Violations or on the BRIEF, contrary to some prior research that found deficits in inhibition (de Jong et al. 2009; Purvis and Tannock 2000; Willcutt et al. 2005). As

noted in the literature review, when inhibition deficits occur in RD, they tend to be on cognitive tasks (e.g., stop-signal) and do not tend to result in commission errors (de Jong et al. 2009; Cheung et al. 2014) or impulsivity in daily life (Gioia et al. 2002; Pratt 2000). Hence, the inhibition deficits found in prior work could be due to slower processing speed and/or worse WM. Thus, when studying inhibition in RD, it is important to control for other factors that may contribute to task performance, such as WM and processing speed, as well as comorbid ADHD. Based upon our results and others', behavioral regulation may be spared in RD when these factors are controlled.

Children with ADHD (ADHD and RD/ADHD) displayed worse performance than those without it on nearly all EF tasks for both the traditional measures and questionnaires, consistent with hypotheses. Planning was not affected as measured by the Tower, but it was significant for both parent- and teacher-report on the BRIEF. Thus, our EF results are commensurate with prior work showing all domains of EF, cognitive, behavioral, and emotional, to be affected in ADHD at the group level(de Jong et al. 2009; Takacs et al. 2014; Willcutt et al. 2005a), as well with the frontostriatal model of ADHD (Castellanos and Proal 2012; Faraone et al. 2015). In terms of WM components, both the visuospatial sketchpad and the CE/verbal WM were affected, but the phonological loop was not. This finding is consistent with prior research finding the phonological loop to be spared in ADHD when the child is focused upon the task at hand (see Kibby, 2012).It should be noted that each cognitive effect size was small (<7% of variance explained), but, taken together, they explained 13% of the variance in ADHD diagnosis. In contrast, each emotional/behavioral effect size tended to be larger (5-45% of variance explained), with 55% of the variance in ADHD diagnosis being explained when taken together. This difference in explained variance for traditional measures versus questionnaires is consistent with ADHD being a behaviorally-based diagnosis.

In terms of our second aim, our findings are consistent with the multiple deficit hypothesis, commensurate with our hypotheses. Based upon this view, children with RD and those with ADHD share some deficits, contributing to their comorbidity, but differ in other areas, contributing to their being separate disorders. We found both RD and ADHD performed similarly on many areas of cognitive EF when taking a categorical approach, with behavioral inhibition deficits being more specific to ADHD and phonological loop deficits being more specific to RD. According to the model, when taking a categorical approach, there should be a double dissociation for deficits that are *not* shared, with simple additive effects (null interaction) in individuals with RD/ADHD. This is what we found, as children with RD/ADHD displayed deficits consistent with both RD and ADHD alone without having additional or worse deficits. Further, a shared deficit should be manifested by all three clinical groups performing worse than controls on it. Verbal WM, as measured by Sequences, met this criterion. The lack of an 'under-additive' interaction on Sequences likely is related to the heterogeneity of deficits found in RD and ADHD, as demonstrated by our chi-square analysis, as well as reduced power (.69). When taking a continuous approach to thedata, only verbal WM predicted both basic reading and inattention (teacher-rated) in the regressions. Furthermore, verbal WM at least partially accounted for the relationships between basic reading and inattention when using partial correlations. Taken together, our findings go against the phenocopy hypothesis, as the comorbid group showed deficits

consistent with both disorders rather than one or the other. Our findings do not support the cognitive subtype hypothesis either: RD/ADHD was not significantly more affected than RD or ADHD in any area assessed. Moreover, there was no area that was uniquely affected in RD/ADHD as compared to RD or ADHD. Our findings supporting the multiple deficit hypothesis is consistent with prior work that analyzed the various hypotheses on RD/ADHD (e.g., Moura et al., 2017; Tiffran-Richards et al., 2007; Willcutt et al., 2010). Worthy of note is that the inattention dimension correlated with basic reading but not hyperactivity/impulsivity, consistent with prior research suggesting the comorbidity likely is due to contributors associated with the inattention dimension rather than the hyperactive/impulsive dimension (Cheung et al. 2014; Willcutt and Pennington 2000a; Willcutt et al. 2010).

Prior research has shown WM is affected in both RD and ADHD (Cheung et al., 2014; Moura et al., 2017; Tiffin-Richards et al.,2007). This research, along with our findings, suggests WM may be a potential shared contributor to RD and ADHD. Nevertheless, other researchers who share a dataset have found differing results from ours, with verbal WM being affected in RD but not ADHD (Willcutt et al. 2010) or not in either disorder (McGrath et al. 2011), depending upon the method used, arguing against WM being a shared contributor. Others have found verbal WM to be affected in RD but not ADHD also (Rucklidge and Tannock 2002; Tiffin-Richards et al. 2007). One reason for these disparate findings may be the heterogeneity in the disorders as predicted by the multiple deficit model, given we found 33% of children with RD and 31% with RD/ADHD had intact WM as measured by Sequences, versus 65% of children with ADHD versus 87% of controls. Thus, the deficits found may depend upon sample composition. It also may be that the WM is affected in ADHD, but more mildly so when using verbal tasks rather than visual-spatial ones, with RD having the opposite effect (Kibby, 2012; Martinussen and Tannock 2006), being an issue of severity of deficit and modality of presentation. A third reason may be the varying tasks used to measure WM, along with the control variables used, as noted in the literature review. In our study, we included three measures of WM, one for each component (phonological loop, visuospatial sketchpad, and CE), although the CE task (Sequences) also required use of the phonological loop given it was verbal in nature, which likely is one of the reasons why the prevalence of the Sequences deficit was greater in RD (RD and RD/ADHD).

The CE specifically may be the shared deficit aspect of WM, as all three groups performed worse than controls on Sequences and it explained the correlations between basic reading and inattention. Moreover, research on the genetic basis of RD and ADHD has found CE functioning to be heritable. For example, Cheung and colleagues (2014) found verbal WM (involving the CE), as well as phonological loop functioning, to have significant phenotype and genetic associations with both inattention and reading performance. In contrast to the CE, we found the phonological loop was affected in RD but not ADHD. The visuospatial sketchpad was affected in both RD and ADHD at the group level, but it did not significantly predict basic reading ability nor inattention.

In our structural MRI work on the comorbidity between RD and ADHD, we found atypicalities in frontal volume for both RD and ADHD, consistent with the multiple deficit hypothesis and this study's findings. More specifically, when tracing each frontal gyrus on a

large subset of participants from this study (those who had MRI scans; Kibby et al., 2020), we found the right inferior frontal gyrus to be smaller in children with ADHD (including RD/ADHD) and its size related to behavioral regulation, a deficit we found to be specific to ADHD in the current study. Further, left superior volume was larger in RD than ADHD, with RD/ADHD having an intermediary value. The relationship between left superior volume and basic reading ability was accounted for by verbal WM, a deficit we found to be prevalent in RD in the current study. In our VBM study on RD/ADHD comorbidity (Jagger-Rickels et al., 2018), we also found right inferior frontal volume to be reduced in ADHD but not RD, along with additional right frontal regions. We found a frontal area that was specific to RD as well (L precentral). In addition, we found shared areas between RD, ADHD, and RD/ADHD: right superior frontal and right anterior caudate. Hence, we found shared and unique frontal areas between RD and ADHD across varying methods using this sample, consistent with the multiple deficit model and the EF deficits found in this study.

Nevertheless, we also found results consistent with the cognitive subtype hypothesis in both structural MRI studies. For both studies, the left middle frontal gyrus was implicated in comorbid RD/ADHD but not RD nor ADHD. Its size was related to attention control in the tracing study. Other research has suggested the left dorsolateral prefrontal area is involved with color naming (MacDonald et al. 2000; Stuss et al. 2001), an area we did not assess. Work by Rucklidge and Tannock (2002) showed the comorbid group was specifically affected in color naming, performing worse than both RD and ADHD, which is part of the reason why the authors developed the cognitive subtype hypothesis. They also found RAN to be differentially affected in the comorbid group. Hence, future research should continue to investigate the cognitive subtype hypothesis, particularly in the area of rapid naming including color naming.

Although our current study yielded a number of interesting findings, it has multiple limitations. First, we did not have a longitudinal dataset, which is needed to determine etiology. Hence, future research should analyze EF as a contributor to RD and ADHD using longitudinal methods. Second, we only assessed EF as a possible contributor. Future research should assess not only working memory, but other areas as well, such as processing speed and rapid naming, in order to more comprehensively test hypotheses regarding the nature of RD/ADHD. Researchers also should include the Stroop task given the findings of Rucklidge and Tannock (2002) and to represent interference control in the study of EF. Third, we had a mild sample in terms of RD and ADHD severity, as it was not a clinic sample. Findings may change when using a clinic sample. Fourth, we had small cell sizes, especially for the RD cells. Because of this, we were unable to analyze subtypes of either disorder. Fifth, due to sample size limitations, we were unable to use modeling techniques analyzing covariance such as SEM. We ran multiple regression analyses instead, which risks family-wise error. Nonetheless, most of our multiple regression findings were highly significant (ps < .001). Finally, our study only focused on basic reading; future work is needed to assess EF and comorbidity using measures of reading fluency and reading comprehension.

In conclusion, our study is one of the few that analyzes EF in a comprehensive, multi-modal fashion as a potential contributor to the comorbidity between basic RD and ADHD. When

using neuropsychological measures, we found both RD and ADHD showed deficits in most areas of cognitive EF at a group level, with phonological loop dysfunction being more specific to RD and behavioral regulation deficits being more specific to ADHD. Results related to cognitive and behavioral EF were similar on the BRIEF. In contrast, only WM predicted both basic reading and inattention, showing promise as a possible shared contributor to RD/ADHD. Finally, our data are consistent with the multiple deficit model of RD/ADHD as opposed to the phenocopy or cognitive subtype hypotheses.

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Appendix

Executive Functioning Measures Used

Cognitive EF measures	
CMS Sequences	Verbal working memory including the CE
CMS Numbers Forward	The phonological loop component of working memory
CMS Picture Locations	The visuospatial sketchpad component of working memory
WCST Categories Achieved	Problem-solving ability
WCST Perseverative Errors	Cognitive flexibility/shift
NEPSY Tower	Planning
NEPSY Tower Rule Violations	Response inhibition/impulsivity
NEPSY Design Fluency	Nonverbal, generation fluency
BRIEF Metacognition Index	Overall cognitive regulation, includes Initiate, WM, Plan/Organize, Organization of Materials, and Monitor
Emotional & Behavioral Regulation measures	
BRIEF Behavioral Rating Index	Behavioral and emotional regulation, includes Inhibit, Shift, and Emotional Control

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Table 1

Demographic Values Across the Four Groups

Variable	Controls	ADHD	RD	RD/ADHD
Sex (% Boys)	43.24	57.17	61.22	59.18
Ethnicity/Race	%	%	%	%
Caucasian	90.54	90.11	91.84	75.51
African/African American	2.70	3.30	2.04	8.16
Asian/Asian American	1.35	0.00	2.04	0.00
Hispanic/Spanish/Latino(a)	1.35	2.20	2.04	6.12
Other	4.05	4.40	2.04	10.20
	M (SD)	*M (SD)*	*M (SD)*	*M (SD)*
Age (years)	9.72 (1.39)	9.60 (1.38)	9.41 (1.24)	9.27 (1.37)
Maternal Education	5.65 (1.06)	5.52 (1.23)	5.40 (1.21)	5.46 (1.26)
TONI-3 IQ[a]	105.99 (13.58)	98.02 (11.60)	96.69 (9.40)	95.29 (10.53)

Note.

[a]Groups differed at $p < .001$. A 5 on Maternal Education = some college.

Table 2

MANOVA Comparing Reading, Inattention, and Hyperactivity/Impulsivity Means across Groups

Variable	Controls	ADHD	RD	RD/ADHD	F(3,254)	p
Letter-Word[a] Identification	104.9 (11.30)	102.57 (9.82)	78.34 (8.21)	81.70 (9.48)	98.90	<.001
Word Attack[a]	103.73 (10.18)	102.16 (8.81)	86.02 (6.67)	86.77 (9.90)	82.73	<.001
Parent Attention Problems[b]	48.47 (9.14)	66.93 (6.45)	52.66 (8.21)	66.96 (5.25)	55.83	<.001
Parent Hyperactivity[b]	44.89 (8.32)	60.92 (13.16)	46.98 (7.95)	59.17 (11.00)	113.93	<.001

[a]Controls and ADHD > RD and RD/ADHD.

[b]ADHD and RD/ADHD

Table 3

2x2 MANCOVA Univariate Results Comparing Means on Traditional EF Measures

Variable	Controls	ADHD	RD	RD/ADHD	Analysis	F(1,244)	p	ηp^2
WCST Categories Achieved	103.30 (1.82)	97.24 (1.55)	93.17 (2.15)	90.87 (2.14)	ADHD or not	4.65	.03	.02
					RD or not	17.73	<.001	.07
WCST Perseveration Errors	107.34 (2.74)	103.39 (2.33)	100.36 (3.23)	91.93 (3.22)	ADHD or not	4.53	.03	.02
					RD or not	9.81	.002	.04
NEPSY Tower	104.13 (1.71)	102.85 (1.46)	102.34 (2.02)	97.90 (2.01)	ADHD or not	2.47	.12	.01
					RD or not	3.33	.07	.01
NEPSY Design Fluency	96.74 (1.68)	89.00 (1.43)	90.05 (1.98)	82.78 (1.98)	ADHD or not	17.62	<.001	.07
					RD or not	12.71	<.001	.05
Numbers Forward	95.17 (1.92)	91.25 (1.63)	84.31 (2.26)	84.61 (2.25)	ADHD or not	0.79	.38	.003
					RD or not	17.92	<.001	.07
Picture Locations	100.16 (1.88)	95.62 (1.60)	94.54 (2.22)	88.31 (2.22)	ADHD or not	7.22	.01	.03
					RD or not	10.16	.002	.04
Sequences	102.08 (1.68)	94.92 (1.43)	84.73 (1.99)	80.97 (1.98)	ADHD or not	9.29	.003	.04
					RD or not	74.45	<.001	.24
Tower Rule Violations	1.14 (.12)	1.73 (.10)	1.42 (.14)	1.88 (.14)	ADHD or not	16.81	<.001	.07
					RD or not	2.85	.09	.01

Note. None of the interaction terms were significant ($ps > .10$). Marginal means controlling for TONI-3 IQ and standard error values are presented by group.

Table 4

2x2 MANCOVA Univariate Results Comparing Means on BRIEF Scales

Variable	Controls	ADHD	RD	RD/ADHD	Analysis	F(1,235)	p	ηp^2
Parent Shift	46.33 (1.46)	56.20 (1.23)	51.34 (1.75)	61.39 (1.75)	ADHD or not	40.14	<.001	.15
					RD or not	10.38	.001	.04
Parent Inhibit	45.23 (1.39)	60.92 (1.17)	46.77 (1.67)	61.41 (1.66)	ADHD or not	102.65	<.001	.30
					RD or not	.45	.50	.002
Parent WM	52.48 (1.19)	70.03 (1.00)	54.73 (1.43)	72.51 (1.42)	ADHD or not	190.15	<.001	.45
					RD or not	3.36	.07	.01
Parent Plan/Organize	50/13 (1.20)	65.29 (1.01)	53.14 (1.43)	69.37 (1.43)	ADHD or not	149.45	<.001	.39
					RD or not	7.51	.01	.03
Parent Initiate	50.14 (1.24)	63.32 (1.05)	53.06 (1.49)	66.21 (1.48)	ADHD or not	97.39	<.001	.29
					RD or not	4.67	.03	.02
Parent Organization of Materials	51.95 (1.14)	61.15 (.96)	50.91 (1.36)	62.02 (1.36)	ADHD or not	69.35	<.001	.23
					RD or not	.01	.95	.00
Parent Monitor	49.05 (1.24)	63.09 (1.04)	50.81 (1.49)	64.12 (1.48)	ADHD or not	105.28	<.001	.31
					RD or not	1.09	.30	.01
Parent Emotional Control	46.73 (1.42)	54.57 (1.20)	49.28 (1.70)	57.42 (1.70)	ADHD or not	27.36	<.001	.10
					RD or not	3.08	.08	.01
Teacher Shift	51.17 (1.68)	59.03 (1.42)	58.03 (2.02)	63.11 (2.01)	ADHD or not	12.80	<.001	.05
					RD or not	9.02	.003	.04
Teacher Inhibit	48.50 (1.65)	61.52 (1.39)	51.56 (1.98)	61.34 (1.97)	ADHD or not	41.28	<.001	.15
					RD or not	.65	.42	.003
Teacher WM	54.74 (1.71)	59.03 (1.42)	58.03 (2.02)	63.11 (2.01)	ADHD or not	53.35	<.001	.19
					RD or not	2.70	.10	.01
Teacher Plan/Organize	52.13 (1.52)	65.43 (1.28)	60.23 (1.82)	68.39 (1.81)	ADHD or not	43.49	<.001	.16
					RD or not	11.36	.001	.05
Teacher Initiate	53.42 (1.49)	66.03 (1.26)	60.24 (1.79)	68.69 (1.78)	ADHD or not	43.18	<.001	.16
					RD or not	8.60	.004	.04
Teacher Organization of Materials	52.45 (2.07)	65.67 (1.74)	56.38 (2.48)	68.69 (2.47)	ADHD or not	33.05	<.001	.12
					RD or not	2.42	.12	.01

Variable	Controls	ADHD	RD	RD/ADHD	Analysis	F(1,235)	p	ηp^2
Teacher Monitor	51.87 (1.59)	65.31 (1.34)	56.45 (1.91)	68.74 (1.90)	ADHD or not	56.66	<.001	.19
					RD or not	5.40	.02	.02
Teacher Emotional Control	50.69 (1.80)	58.49 (1.51)	55.59 (2.15)	62.21 (2.15)	ADHD or not	13.93	<.001	.06
					RD or not	4.90	.03	.02

Note. The interaction term was significant for Teacher-rated WM (p = .03). None of the other interaction terms were significant at the univariate level. Marginal means controlling for TONI-3 IQ and standard error values are presented by group.

Table 5

Standardized Beta Values for Hierarchical Regressions from Step 2

Executive Functions Predicting Reading Skills and ADHD Symptoms												
Dependent Variables	***Categories Achieved***	***Perseveration Errors***	***NEPSY Tower***	***Design Fluency***	***Rule Violations***	***Sequences***	***Numbers Forward***	***Picture Locations***	***Parent BRI***	***Parent MI***	***Teacher BRI***	***Teacher MI***
Word Attack	.03	.12[b]	−.02	−.11	−.003	.49***	.17**	.02	−.07	.11	.08	−.12
Letter-Word Identification	.06	.06	−.10	−.04	.03	.50***	.17**	.02	.06	−.001	.01	−.04
Parent Attention Problems	−.12*	−.07	.01	−.08	.06	.05	−.06	.03	.15*	.55***	−.10	.20**
Teacher Attention Problems	.03	.03	−.05	.02	.15**	−.13*	−.04	−.05	.10[c]	.04	−.03	.63***
Parent Hyperactivity	−.03	−.01	.03	−.05	.15**	.14**	−.03	.02	.49***	.19**	.18**	.03
Teacher Hyperactivity	.01	.004	.01	.02	.19***	.07	.02	−.04	.20**	−.04	.46***	.22**

*$p<.05$.

**$p<.01$.

***$p<.001$.

[a]$p = .06$.

[b]$p = .08$.

[c]$p=.07$.

Table 6

Frequency Counts for Standard Scores on Sequences

Sequences Standard Scores	90 or greater	75-85	70 and below	Total
Controls	64 (86.5%)	9 (12.2%)	1 (1.3%)	74 (100%)
Reading Disorder	16 (32.7%)	26 (53.1%)	7 (14.3%)	49 (100%)
ADHD	59 (64.8%)	27 (29.7%)	5 (5.5%)	91 (100%)
Comorbid	15 (30.6%)	19 (38.8%)	15 (30.6%)	49 (100%)
Total	*154*	*81*	*28*	*263*