WORKING MEMORY, RESPONSE INHIBITION, AND WITHIN-SUBJECT VARIABILITY IN CHILDREN WITH ADHD OR READING DISORDER

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This study compared children with ADHD (n=19), reading disorder (RD; n=17), ADHD+RD (n=21), and control children (n=19) on linguistic and executive function measures. We found no evidence of response inhibition problems in ADHD or RD when a baseline measure of functioning was taken into account. General working memory problems were only found in children with RD or ADHD+RD. Both children with ADHD and RD showed a highly inaccurate (more commission errors) and variable (higher within-subject standard deviation of reaction time) response style. The comorbid group made most errors, suggesting that different factors underlie the high error rate in both disorders.

ADHD is one of the most prevalent developmental disorders found in child populations all over the world (Faraone, Sergeant, Gillberg, & Biederman, 2003). Different theoretical models have been proposed to account for the observed symptoms of inattention, hyperactivity and impulsivity. Those models each point to different cognitive/neuropsychological deficits that are supposed to be the core deficits underlying ADHD (for an overview, see Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). One of the most dominant models guiding research over the past decade has been the executive function (EF) deficit model. It originated from the observation that individuals with ADHD have problems with executive functions that are similar to those observed in patients with frontal lobe lesions (Pontius, 1973). EF is currently conceptualized as a collection of higher-order cognitive control processes that are necessary to guide goal-directed behaviour (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006). Working memory (the ability to temporarily maintain and manipulate information needed for generating upcoming action) and response inhibition (the ability to inhibit inappropriate action) are sometimes viewed as the most dominant EF domains (Roberts & Pennington, 1996). Children with ADHD have been found to show significant impairments in both domains, together with deficits in vigilance and planning (see review by Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Although research based on the EF model has obviously advanced our understanding of ADHD (Castellanos & Tannock, 2002), some problems with this theory have become undeniably apparent.

First, executive dysfunction does not seem to be a necessary condition for the expression of the disorder in all children with ADHD. A recent review by Nigg and colleagues (2005) for example indicated that about 21% of children with ADHD showed no EF problem at all. In addition, a recent review by Willcutt, Doyle, et al. (2005) found that effect sizes for each of the EF measures were only moderate (d = 0.4 - 0.6), leading the authors to conclude that none of these EF weaknesses is a sufficient cause of ADHD.

Second, EF problems have also been found in other developmental disorders such as autism and Tourette's syndrome, which questions its specificity to ADHD (Pennington & Ozonoff, 1996; Sergeant, Geurts, & Oosterlaan, 2002; Weyandt, 2005). It is therefore possible that EF deficits found in ADHD are due to comorbidity with another disorder (Pennington & Ozonoff, 1996). This makes it very important to control for comorbid disorders when trying to find out which deficits are a unique feature of ADHD (Banaschewski et al., 2005; Lazar & Frank, 1998; Sergeant et al., 2002).

One important comorbidity is that of ADHD with learning disorders, and more specifically reading disorder (RD). ADHD and RD co-occur much more often than can be expected by chance, and they share some behavioural symptoms, like inattentive behaviour and poor academic performance (Hinshaw, 1992). These findings make differential diagnosis difficult and urge research into cognitive and neurobiological variables that might better distinguish between both disorders (Rashid, Morris, & Morris, 2001). Reading disorder is primarily associated with linguistic problems, like impairments in phonological processing and slower serial naming speed (Pennington, Groisser, & Welsh, 1993; Purvis & Tannock, 2000; Rucklidge & Tannock, 2002; Semrud-Clikeman, Guy, Griffin, & Hynd, 2000; Willcutt et al., 2001). When comparing these deficits in RD with those in ADHD, a few studies found a double dissociation with children with ADHD showing only EF deficits and children with RD exhibiting only linguistic problems (e.g., Nigg, Hinshaw, Carte, & Treuting, 1998; Pennington et al., 1993). However, it appears that this dissociation is not complete (Pennington, 2006) as subsequent studies also found linguistic problems, such as deficits in orthographic coding and slower naming of objects and colours, in ADHD (Semrud-Clikeman et al., 2000; Tannock, Martinussen, & Frijters, 2000; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005), and EF deficits, like inhibition and working memory problems, in children with RD (Purvis & Tannock, 2000; Swanson, Mink, & Bocian, 1999).

Unequivocal results concerning the relationship between ADHD, RD, and EF have yet not been obtained. The current study aims at enhancing insight into these relations by addressing some important issues that have hampered research into these relations and have made it difficult to draw firm conclusions.

First, one of the major problems with EF is that it is a very complex and multi-faceted construct (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000) which has given rise to different measurement problems (Weyandt, 2005). Many studies have used tasks that tap multiple EF components, making it difficult to find out which of those processes is impaired when performance on these tasks is low. Effort should be made to more stringently measure isolated EF components because several authors found that ADHD is associated with specific rather than with general EF deficits (e.g., Sergeant et al., 2002; Wu, Anderson, & Castiello, 2002). Consequently not all EF components are assumed to be equally important in the aetiology of ADHD, and theoretical models therefore focus on core deficits like Barkley's inhibition deficit model (Barkley, 1997). Sergeant et al. (2002) recommend the use of well-defined computer paradigms when trying to measure specific cognitive processes.

Another problem is that impairments in EF can also result from deficits in lower order cognitive processes as these are the building stones of the higher order cognitive operations. It is therefore unclear whether weaker performance on tasks measuring EF is caused by real EF deficits or by deficits in more basic cognitive processes such as perceptual, visuomotor, temporal or language processing (Halperin & Schulz, 2006; Rommelse et al., 2007). This is problematic when trying to differentiate ADHD from RD because it is well-established that children with RD have deficits in lower order cognitive processes such as perception and temporal processing (see reviews by Farmer & Klein, 1995; Stein, 2001; Wright, Bowen, & Zecker, 2000). It is therefore important to include some kind of baseline measure to which the EF measure can be compared. Evidence is accumulating that when a baseline or control

condition is included, children with ADHD seem to perform poorly across both the experimental and control condition (Andreou et al., 2007; Rommelse et al., 2007). This suggests that deficits in lower order cognitive processes may underlie the EF problems of children with ADHD (Halperin & Schulz, 2006; Rommelse et al., 2007), and posits a threat to the EF theory of ADHD. The main aim of the present study was to further examine the relationship between ADHD, RD, and EF by comparing both disorders on specific computerized measures of response inhibition and working memory, two well-established EF components, in relation to a baseline measure of functioning, that is, a condition of low memory or inhibition load compared to one with a high load.

A third issue is the possible moderating role of the modality of the stimulus that has to be processed in finding group differences in EF or not (Alderson, Rapport, & Kofler, 2007). With respect to working memory for example, there is still no agreement on the kind of impairment that children with ADHD or RD show (Kibby, Marks, Morgan, & Long, 2004; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005). According to Baddeley (1992), working memory can be subdivided into a verbal-auditive and a visual-spatial component. Some studies find only visual working memory deficits in ADHD, whereas others find both visual and verbal working memory deficits (for an overview, see Martinussen et al., 2005). With respect to children with RD, results have been in favour of no problems, only verbal, or both verbal and visual-spatial working memory problems (Kibby et al., 2004; Savage, Lavers, & Pillay, 2007; Swanson, Ashbaker, & Lee, 1996). With respect to response inhibition, tasks that are often employed to measure this EF component (e.g., Stop and Go/no-go tasks) typically use letters as stimuli. It is however possible that children of a particular clinical group have problems with inhibiting their responses only when certain types of stimuli are used and not with others. This could partly explain why inhibition problems are sometimes found in these disorders and sometimes not. In a recent meta-analysis of the Stop-signal

paradigm in ADHD, Alderson et al. (2007), for example, found larger effect sizes for visualspatial stimuli than for text-based stimuli. The second aim of our study was therefore to examine whether children with ADHD or RD show general or modality-specific EF deficits by exploring the influence of stimulus modality on performance in the response inhibition and working memory tasks.

Finally, when trying to differentiate between ADHD and RD on cognitive measures, it is important to study the comorbid group next to the single disorder groups in the same investigation. Comparison of both single disorder groups can clarify which deficits are unique to one disorder independently of the other disorder and which are shared. Comparison of the profile of deficits of the comorbid group with those of the single disorder groups allows for an examination of the nature of the comorbidity. The comorbid group can display the symptoms of only one of the disorders (e.g., Pennington et al., 1993), the additive combination of both disorders (e.g., Purvis & Tannock, 2000; Willcutt et al., 2005b), or more/other symptoms than the combination of those of the pure groups (e.g., Rucklidge & Tannock, 2002). When a deficit is associated with the 3 clinical groups and the level of impairment of the comorbid group is similar to that of the single disorder groups, this suggests that this deficit is a shared cognitive risk factor in RD and ADHD. If however this deficit is related to different underlying factors in both disorders, then the deficit in the comorbid group would be the sum or the product of the deficits of each single disorder group (Shanahan et al., 2006). Our third research aim was to explore the nature of the comorbidity of ADHD and RD by examining the performance of the comorbid group relative to the single disorder groups.

The last objective of the current study was to compare ADHD and RD on speed of responding when performing linguistic and neuropsychological tasks. Children with ADHD have been found to be slower and more variable when performing reaction time tasks (for a review, see Douglas, 1999). This typical response style is found across different neuropsychological tasks (Barkley, 2005; Castellanos & Tannock, 2002; Halperin & Schulz, 2006) and therefore seems to be independent of the EF measured. Greater within-subject variability or inconsistency in reaction time is currently the most consistent finding for ADHD, and has recently been put forward as a possible endophenotype, or intermediate vulnerability factor, of ADHD (Bidwell, Willcutt, DeFries, & Pennington, 2007; Castellanos et al., 2005; Kuntsi, Andreou, Ma, Börger, & van der Meere, 2005; Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Rommelse et al., 2008). However, to test its uniqueness to ADHD, we urgently need to contrast children with ADHD with children from other clinical groups on this measure. With respect to RD, little is known about within-subject variability, and few studies have contrasted both disorders on this measure (but see Williams, Strauss, Hultsch, Hunter, & Tannock, 2007). Children with RD are usually impaired on Rapid Automatized Naming (RAN) tasks, which measure naming speed of rapid serially presented visual stimuli (Rucklidge & Tannock, 2002; Semrud-Clikeman et al., 2000). No study however investigated whether children with RD are also more variable in their response speed on this type of task in addition to being slower.

In summary, the present study aimed primarily at enhancing insight into the relationship between ADHD, RD and EF by using specific computerized measures of response inhibition and working memory in relation to a baseline measure of functioning. A second aim was to examine whether ADHD and/or RD were associated with general or modality-specific EF deficits by manipulating the modality of the stimulus that was presented. A third aim was to explore the nature of the comorbidity by examining the performance of the comorbid group relative to the single disorder groups. The last objective was to compare ADHD and RD on speed of responding and within-subject variability in response speed when performing linguistic and neuropsychological tasks.

Method

Participants

Four groups of children aged 8-12 years participated: 19 children with ADHD, 17 children with RD, 21 children with ADHD+RD, and 19 typically developing controls ('control group'). All children were recruited through newspaper advertisements, through referral by speech therapists or paediatric psychologists, and through letters to parents distributed in schools. Children were selected for the screening procedure if they had a diagnosis of ADHD and/or had a history of reading problems (diagnosis of RD or referral to a speech therapist). Parents and teachers completed the following questionnaires: the Disruptive Behaviour Disorder Rating Scale (DBDRS; Pelham, Gnagy, Greenslade, & Milich, 1992), the Child Behaviour Checklist/Teacher Report Form (CBCL/TRF; Achenbach, 1991), and the Social Communication Questionnaire (SCQ; Berument, Rutter, Lord, Pickles, & Bailey, 1999). Children were included in the control group if they had no history of learning or psychiatric problems and scored in the normal range on these questionnaires. The first two questionnaires were used as selection instruments in the control group only; in the clinical groups they were used to obtain a description of possible comorbid problems. Exclusion criteria for all groups were: (1) neurological problems, uncorrected hearing or vision, or speech problems, (2) native language different from Dutch, (3) a clinical score on the SCQ (symptoms of autism), (4) presence of other diagnoses (e.g. anxiety disorder), or (5) an estimated Full Scale IQ (FSIQ) below 80, based on the Vocabulary, Similarities, Picture Arrangement and Block Design subtests of the WISC-III (Wechsler, 1991). This short version of the WISC-III is the one recommended by Grégoire (2000) and has a high correlation (r =.93) with FSIQ (Kaufman, Kaufman, Balgopal, & McLean, 1996). Sociodemographic information was obtained from the parents; The Hollingshead Index (Hollingshead & Redlich, 1958) with 5 classes of social status was used as a measure of socioeconomic status (SES).

Children's ADHD diagnosis was validated with the parent-administered Diagnostic Interview Schedule for Children for DSM-IV (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). Reading problems were evaluated with 2 standardized Dutch reading measures: the Dutch One-Minute-Test (Brus & Voeten, 1973) and the Klepel, a pseudoword reading task (van den Bos, lutje Spelberg, Scheepsma, & de Vries, 1994). The raw scores on these reading measures were converted into standard scores (SS) using grade related norms with a mean of 10 and a standard deviation (SD) of 3. Children in the control group had to obtain a SS of at least 8 on both reading measures. Assignment to one of the 3 clinical groups was based on the DISC-IV (diagnosis of ADHD) and the reading measures (SS \leq 5 on at least 1 of the 2 reading tasks). Children with a clinical diagnosis but insufficient symptom levels to meet these criteria were excluded from the study to make groups as homogeneous as possible. The ODD (Oppositional Defiant Disorder) and CD (Conduct Disorder) modules of the DISC-IV were administered to evaluate the presence of comorbid behavioural disorders. Children with ADHD were allowed to have ODD but were excluded from further participation in the study if they had a CD diagnosis.

Sample Characteristics

As can be seen in Table 1, there were no significant differences between the groups with respect to age, gender, estimated FSIQ, or SES.

The mean score of ADHD symptoms on the DBDRS was significantly higher for the ADHD groups than for the non-ADHD groups, and the RD groups had a significantly lower reading score than the non-RD groups. However, children with ADHD had a significantly lower reading score compared to the control group, although they did not meet the cut-off for RD. The comorbid group did not significantly differ from the ADHD-only group on ADHD symptoms or from the RD-only group on the reading score. Based on the DISC-IV ADHD diagnoses, we found no differences between both ADHD groups in the proportion of ADHD subtypes ($\chi^2(2) = 1.46$, p = .48). In the ADHD-only group, 4 children (21%) met criteria for the inattentive type, 3 children (15.8%) for the hyperactive-impulsive type, and 12 children (63.2%) for the combined type. In the ADHD+RD group, 6 children (28.6%) met criteria for the inattentive type, 1 child (4.8%) for the hyperactive-impulsive type, and 14 children (66.6%) for the combined type. Both ADHD groups had significantly more ODD symptoms on the DBDRS than both groups without ADHD. There were no differences between both ADHD groups in percentage of children meeting a DISC-IV ODD diagnosis ($\chi^2(1) = 0.35$, p =.56): 7 children (38.9%) in the ADHD-only and 5 children (29.4%) in the comorbid group. With regard to CD, all groups exhibited insufficient symptoms on the DBDRS to make a sound comparison, and none of the children had a DISC-IV CD diagnosis.

[Insert Table 1 about here]

Procedure

The study was approved by the Ethical Committee of Ghent University. After written consent was obtained from the parents, appointments for the 2 testing days were made. During the first session all diagnostic and linguistic measures were administered. Neuropsychological measures were administered at the second visit. Children on psychostimulant medication discontinued it at least 24 hours before testing. No medication other than methylphenidate was used.

Linguistic Measures

Phonological Tasks

Phonological processing was measured with 4 tasks that required the manipulation of phonemes. From the Dutch version of the Dyslexia Screening Test (Kort et al., 2005) we took

the Phonemic Segmentation and Letter Switching subtests. The other tasks were the Spoonerisms and Phoneme Reversal subtests of the Test for Advanced Reading and Writing (Depessemier & Andries, 2009). Phonemic Segmentation assesses the ability to break down a word into its constituent sounds and to manipulate those sounds (e.g., say *speak* without the *p*). Letter Switching and Spoonerisms both require the child to switch the first letter of two words (e.g., *big fish* becomes *fig bish*). In Phoneme Reversal children have to decide if two nonsense letter strings are the exact reverse of each other (e.g., *cam* and *mac*). We calculated a composite score for accuracy and one for tempo (i.e., mean time needed to complete the tasks). Phonological tasks were included to make sure that reading problems had the same basis in both RD groups.

Rapid Automatized Naming (RAN) Tasks

Four RAN tasks based on those of Denckla and Rudel (1976) were administered: colours, objects, letters and numbers (van den Bos & lutje Spelberg, 2007). In each of these tasks, children have to name a set of 50 stimuli as quickly and accurately as possible. The RAN administration was computer recorded and scored using the Rapid Reading Scoring Software, developed and validated by Neuhaus and colleagues (Neuhaus, Carlson, Jeng, Post, & Swank, 2001). RAN total time was split up into its 2 components, articulation and pause time, allowing us to differentiate between cognitive and motor processes. Naming speed was operationalized as total pause time, being the total time needed to complete the task minus the articulation time. Both components were measured at item level so that within-subject variability or consistency of processing could also be evaluated (SD of item pause times per subject).

Neuropsychological Measures

Go/no-go Tasks

Response inhibition was assessed using several Go/no-go tasks, programmed in Inquisit 2.0. The Go/no-go paradigm has been one of the most frequently used to investigate response inhibition (Simmonds, Pekar, & Mostofsky, 2008). In this paradigm either a Go or a No-go stimulus is presented on the screen in each trial. Children have to make a response (e.g., push the spacebar) when they see a Go-stimulus but not when they see a No-go stimulus. We included two experimental manipulations: modality (3 levels) and inhibition condition (2 levels). This resulted in 6 blocks of 100 trials (390 Go and 210 No-go trials), with stimulus duration (300 ms) and inter-trial interval (2000 ms) kept constant. Blocks were not counterbalanced across participants, but blocks were relatively short (maximum 5 minutes) and frequent breaks were provided to minimize the effects of fatigue and problems with sustaining attention.

Modality. Tasks differed in the kind of stimuli that had to be processed: meaningless symbols (symbol 1 vs. symbol 2, see Figure 1), digits (1 vs. 6), or letters (X vs. O). This simple format (only one Go and one No-go stimulus per task) is best suited to study response inhibition under minimal influence of other cognitive processes like working memory or stimulus-response conflict (Simmonds et al., 2008).

Inhibition condition. Inhibition difficulty can be manipulated by varying the No-go probability (Bruin & Wijers, 2002; Dimoska & Johnstone, 2008; Ramautar, Kok, & Ridderinkhof, 2004). When Go stimuli are presented more often than No-go stimuli, a prepotent tendency to respond is created. The more prepotent a certain response, the higher the inhibition load or the inhibitory effort that is needed to successfully refrain from responding when a rare No-go stimulus is presented. We included a 'low inhibition condition'

(50% Go trials and 50% No-go trials) and a 'high inhibition condition' (80% Go and 20% Nogo trials). In the latter, the higher frequency of the ongoing response makes it more difficult to inhibit.

Dependent measures. Percentage of commission errors (i.e., pressed the button after a No-go signal) was used as a measure of response inhibition, with more errors reflecting more deficient response inhibition. As omission errors were rare, they were not included in the analyses. Other dependent measures were mean reaction time (RT), and within-subject variability, operationalized as within-subject standard deviation of RTs (SD-RT). Mean RT and SD-RT were calculated for correct Go responses between 150 and 1500 ms.

N-back Tasks

N-back tasks have often been used in experimental studies of working memory (Parmenter, Shucard, Benedict, & Shucard, 2006). Not only do they allow for the memory load to be manipulated but they also provide a measure of RT in addition to accuracy. We used a control condition (0-back) and two memory conditions (1-back and 2-back). In the 0back task children had to push one of two buttons when a certain stimulus was presented, and the other button when any other stimulus was presented. In the memory conditions subjects had to respond by pushing one button whenever a stimulus was presented that was the same as the one presented the previous trial (1-back) or last-but-one trial (2-back), and pushing the other button when the stimulus was not the same. As the 2-back task appeared too difficult for this age group, only two levels of difficulty (0- and 1-back) were analyzed. Three different stimulus modalities were used: meaningless symbols (see Figure 2), letters, and digits. This allowed us to compare the groups on both visual and verbal-auditive working memory. The 2 levels of difficulty and the 3 levels of modality generated a choice RT task of 6 blocks of 40 trials each, with a stimulus duration of 500 ms and a self-paced inter-trial interval of maximum 4000 ms (programmed in E-Prime 1.1). This rather large response interval was chosen to enlarge the chances of 2-back success.

Dependent measures. Accuracy was calculated as percentage of commission errors in the 1-back condition. Other dependent variables were RT and SD-RT of correct responses between 150 and 1500 ms in both memory tasks.

[Insert Figure 1 and Figure 2 about here]

Results

Statistical Analyses

Prior to statistical testing, extreme scores, defined as scores falling 3 SDs from the group mean, were removed. This led to the exclusion of 2 control subjects in the analyses of the n-back variables, 1 ADHD subject in the RAN analyses, and 2 control subjects in the Go/no-go RT and SD-RT analyses. After these adjustments were made, assumptions of normality and homogeneity of variances were tested in each group separately. Whenever assumptions were not met, a logarithmic transformation (log₁₀) was performed and analyses were then conducted on these transformed data (Tabachnick & Fidell, 2007). This was the case for RAN pause times and SD of pause times, and Go/no-go and n-back commission errors.

Several 2 (ADHD vs. no ADHD) x 2 (RD vs. no RD) factorial ANOVAs were conducted. None of the ADHD by RD interactions was significant and therefore they are not reported. The performance of the comorbid group was explored by means of post hoc comparisons with a Bonferroni correction. Repeated measures ANOVAs were used to examine the effect of stimulus modality (symbols vs. digits vs. letters) on performance. Because there were some (non-significant) differences in gender composition, we reanalyzed the data with only the boys included, but this did not change the results.

Comparison of the Means

Unadjusted means for the four groups on each variable are presented in Table 2. *Phonological Tasks*

For both accuracy (F(1,72) = 43.70, p < .001, $\eta_p^2 = .38$) and tempo (F(1,72) = 13.74, p < .001, $\eta_p^2 = .16$) of phonological processing a RD main effect emerged, which indicates that

children with RD made more errors and were slower to complete phonological tasks than children without RD. Post hoc analyses revealed no significant differences between the comorbid group and the RD-only group, which implies that the reading problems have the same basis in both RD groups. Therefore, possible differences between both groups in EFs can not be attributed to differences in phonological ability.

RAN Tasks

Differences in total pause time and SD of item pause times were analyzed using an ADHD (0 vs. 1) x RD (0 vs. 1) x Modality (letters vs. digits vs. objects vs. colours) repeated measures ANOVA. No significant interaction effects emerged between the diagnostic factors and the within-subject factor Modality (p > .26), which means that the presentation of different types of stimuli had no differential effect on children with ADHD or RD compared to children without ADHD or RD. There was however a significant main effect of RD for total pause time (F(1,71) = 18.80, p < .001, $\eta^2_p = .21$) and SD of item pause times (F(1,70) = 29.62, p < .001, $\eta^2_p = .30$) for all modalities. This indicates that children with RD were slower and more variable in their speed of naming letters, digits, objects and colours than children without RD. The main effect of ADHD was not significant for any of the variables (p > .51). Both RD groups did not differ significantly from each other, indicating that the comorbid group was not more impaired than the RD-only group.

Go/no-go Tasks

Results are subdivided into response execution outcomes (associated with Go stimulus) measuring processing efficiency and response inhibition outcomes (associated with No-go stimulus) measuring executive function. The effect of the inhibition manipulation was examined by calculating the inhibition difference score of the percentage of commission errors in the high inhibition condition minus the percentage of commission errors in the low inhibition condition. Response inhibition problems are reflected by a significant main effect of the diagnostic factor(s) on this difference score. In order to investigate if inhibition problems are general or modality-specific, a repeated measures ANOVA with Modality (letters vs. digits vs. symbols) as within-subject factor was performed. If an interaction between diagnostic factor and modality is found, this means that inhibition problems in ADHD or RD are dependent on the type of stimulus that has to be processed.

Response inhibition variable: commission errors. The repeated measures analysis ADHD x RD x Modality for the difference score revealed no significant interaction or main effects of the diagnostic factors (p > .57). This means that we did not find evidence for the presence of inhibition deficits in children with ADHD nor in children with RD, and that the modality of the stimulus did not matter. Comparison of the profiles of deficits of the comorbid group and the other clinical groups, revealed no significant differences.

We also analyzed the results for percentage of errors in the low (baseline measure) and the high inhibition condition (experimental measure) separately. In the high inhibition condition, there was a main effect of both ADHD (F(1,72) = 20.43, p < .001, $\eta^2_p = .22$) and RD (F(1,72) = 18.29, p < .001, $\eta^2_p = .20$) in all modalities. But also in the low inhibition condition there was a main effect for both ADHD (F(1,72) = 16.31, p < .001, $\eta^2_p = .19$) and RD (F(1,72) = 11.10, p < .01, $\eta^2_p = .13$) for all modalities. This means that both disorders were associated with making more errors overall, independent of the level of inhibition. The profile of the comorbid group differed significantly from the single disorder groups in both inhibition conditions, such that the comorbid group made more errors than the other clinical groups (p < .02). Inspection of the means showed that the mean error rate in the comorbid group was almost equal to the sum of the means of both single disorder groups. Response execution variables: mean RT and SD-RT. An ADHD main effect was found for overall SD-RT in the low inhibition condition (F(1,71) = 4.89, p < .05, $\eta^2_p = .06$) but not in the high inhibition condition (F(1,71) = 1.34, p = .25). There were no group differences in RT (p > .21), which indicates that children with ADHD and children with RD were as fast as children without these disorders when performing simple RT tasks. Comparison of the clinical groups revealed no significant differences between the comorbid group and the single disorder groups.

N-back Tasks

The effect of the memory manipulation was examined by calculating the memory difference score of the percentage of commission errors in the 1-back condition minus the percentage of commission errors in the 0-back condition. Working memory problems are reflected by a significant main effect of the diagnostic factor(s) on this difference score. In order to investigate if memory problems are general or modality-specific, a repeated measures ANOVA with Modality (letters vs. digits vs. symbols) as within-subject factor was performed. If an interaction between diagnostic factor and modality is found, this means that working memory problems in ADHD or RD are dependent on the type of stimulus that has to be processed.

The repeated measures analysis ADHD x RD x Modality for the difference score revealed no significant interaction effects (p > .40). There was however a significant RD main effect (F(1,70) = 5.80, p < .05, $\eta^2_p = .08$), which means that children with RD had working memory problems independent of the type of material that had to be remembered.

We also analyzed the results for percentage of errors in the 0-back (baseline) and 1back condition separately. In the 0-back condition, there was a main effect of ADHD (F(1,70)= 13.59, p < .001, $\eta^2_p = .16$). In the 1-back condition there was a main effect of both ADHD $(F(1,70) = 15.79, p < .001, \eta_p^2 = .18)$ and RD $(F(1,70) = 14.64, p < .001, \eta_p^2 = .17)$. The fact that children with ADHD already differed in the baseline condition suggests that they have a general tendency to make more errors on a 2-choice RT task independent of memory load.

Again no differences emerged between groups in RT (p > .51); there was however a main effect of ADHD (F(1,70) = 6.36, p < .05, $\eta^2_p = .08$) and a main effect of RD (F(1,70) = 4.58, p < .05, $\eta^2_p = .06$) for overall SD-RT (over all modalities and over both memory conditions). Children with ADHD or RD were not slower than controls in performing memory tasks, they were however more variable in their speed of responding. The comorbid group was not more impaired than the other clinical groups on any of the variables.

[Insert Table 2 about here]

Discussion

Linguistic problems

In accordance with numerous studies (e.g., Purvis & Tannock, 2000; Semrud-Clikeman et al., 2000; Willcutt, Pennington, et al., 2005), we found that children with RD exhibited symptoms of impaired phonological processing and slower serial naming speed. In addition, we found that children with RD were also more variable in their naming speed on the RAN tasks. To our knowledge, this is the first study that investigated within-subject variability in naming speed in ADHD and RD. Implications of this new interesting finding are discussed further on. Contrary to previous studies (Semrud-Clikeman et al., 2000; Tannock et al., 2000) we did not find slower naming to be associated with ADHD. Both previous studies however did not include 4 groups.

EF problems or a general tendency to make more errors?

Response Inhibition

We found no evidence for response inhibition problems in ADHD or RD when a baseline measure of functioning was taken into account. Children with ADHD or RD were not disproportionally more inaccurate than children without ADHD or RD when inhibition load was forced up. This is in contrast to most studies on ADHD (see meta-analysis of Stop task studies by Oosterlaan, Logan, & Sergeant, 1998) and some studies on RD (e.g., Purvis & Tannock, 2000; Willcutt, Pennington, et al., 2005) where response inhibition problems were found. However, these studies did not include a control condition to which performance in the inhibition condition could be compared. In line with these studies, we found that children with ADHD made more errors than children without ADHD in the high inhibition condition, but this was also the case in the low inhibition condition, that is, the baseline measure. Apparently, children with ADHD showed a general tendency to make more commission errors in a RT task, and this could not uniquely be attributed to impairments in response inhibition. This general inaccuracy in performance has been reported by others in various neuropsychological domains (Rommelse et al., 2007) and fits well within the current belief that children with ADHD have problems with error monitoring rather than with response inhibition alone (e.g., Wiersema, van der Meere, & Roeyers, 2005). It is also in line with the conclusions of 2 meta-analytic reviews of the performance of children with ADHD on the Stop task (Alderson et al., 2007; Lijffijt et al., 2005) that the slower Stop Signal Reaction Time (SSRT) of children with ADHD may not reflect a specific problem with response inhibition as problems in response execution were also observed (slower and more variable reactions). However, this inaccurate response style does not appear to be a unique feature of ADHD as children with RD in our study also made more errors than children without RD in both inhibition conditions. This higher active error rate in children with RD has also been noted by others (e.g., Burgio-Murphy et al., 2007). The source of this general inaccuracy of performance may lie in deficiencies in the early processing of incoming signals, in deficiencies in the organization of response, and/or in deficiencies in the monitoring of errors and/or conflict (Jonkman, van Melis, Kemner, & Markus, 2007; Rommelse et al., 2007; Sergeant, 2000). Because we used a fixed instead of a variable response interval, one could argue that problems with timing, often found in ADHD and RD (Farmer & Klein, 1995; Toplak, Dockstader, & Tannock, 2006), are responsible for the poorer performance of the clinical groups. However, we believe that timing problems can not fully account for our findings since they should be reflected in both accuracy and reaction times, while we only found differences in number of errors and not in reaction times. Nevertheless, future studies should incorporate a variable response interval to prevent children from being able to anticipate the onset of the stimuli. It has also been found that children with ADHD are impaired in sustaining attention to a lengthy, rather boring task (see review by Willcutt,

Doyle, et al., 2005). To prevent this from playing a dominant role, we made sure conditions lasted no longer than five minutes and frequent breaks were provided. Although we can not fully exclude the possibility that problems with sustaining attention are responsible for the performance deficit in the Go/no-go tasks, there are two facts that argue against this. First, as was already mentioned, we did not find any differences between groups in reaction times and second, the percentage of omission errors was very low in all the groups.

It is also possible that different underlying factors cause this similar performance deficit in ADHD and RD (Tannock et al., 2000). The fact that we found that the error percentage of children in the comorbid group was nearly equal to the sum of the means of both single disorder groups, may support this possibility (Shanahan et al., 2006). It could be that a specific ADHD deficit (e.g., weak error monitoring) and a specific RD deficit (e.g., a problem of rapid processing) combine in an additive fashion to produce greater functional impairment in children with both disorders (August & Garfinkel, 1990). However, it must be noted that a single common underlying factor with additive effects could also produce the observed pattern. Future research should take a closer look at these possible (different) underlying factors, for example by means of event-related potentials (ERPs), which are very well suited to unravel the covert processes underlying overt performance deficits in the order of milliseconds (Banaschewski & Brandeis, 2007).

Working Memory

With respect to working memory, we found no evidence of deficits in children with ADHD as they were not disproportionally more inaccurate in the 1-back than in the 0-back task in comparison to children without ADHD. This is in contrast to a substantial amount of studies reporting verbal as well as visual working memory deficits in ADHD (see Martinussen et al., 2005). Comparison with other studies is however difficult because most of them used span tasks and not a computerized RT task like the n-back task. We found that children with ADHD made more errors in the working memory condition (1-back), but this inaccurate response style was again already present in the control condition (0-back). This was also found in another study that used the n-back task to study working memory in children with ADHD (Klein, Wendling, Huettner, Ruder, & Peper, 2006). These authors also found no significant increase in errors in children with ADHD compared to controls when memory load was added. They concluded that it is important to include a control condition to demonstrate that children with ADHD have specific working memory problems. However, in this study as well as in ours the 2-back task appeared too difficult for the children and consequently results were not analyzed. It is possible that the memory load in the 1-back task was too low to elicit problems in children with ADHD. Another possibility is that, in this type of task, a possible working memory deficit in children with ADHD is masked by problems with sustaining attention. However, the fact that blocks lasted no longer than 2.5 minutes each, the finding of no differences in reaction time between groups, and the very low overall omission error rate, do not fully support this possibility.

In contrast to the absence of working memory problems in ADHD, we found evidence for a deficit in this domain in children with RD. They were disproportionally more inaccurate in the 1-back than in the 0-back task in comparison to children without RD, not only for verbal material (digits and letters) but also for purely visual material (symbols). This suggests that these problems can not be uniquely related to deficits in phonological/linguistic processing. However, it must be noted that we used a rather large response interval (maximum 4000 ms). As longer inter-stimulus intervals place more demands on working memory, this task could have been more challenging for the children who were slow compared to the children who were quick in their response. However, the fact that we did not find any differences in reaction time between groups and that none of the children used this full interval, suggests that differences in task difficulty alone can not account for the group differences in performance.

There is no consensus in the literature yet about what kind of memory impairments are associated with RD (Kibby et al., 2004). Several factors can be mentioned to account for these inconsistencies. First, working memory has been measured with a wide range of different tasks, making it very hard to interpret and compare results (Savage et al., 2007). Second, short-term memory tasks (e.g. span tasks) are often used to measure working memory. This is problematic as it has been suggested that children with RD can show working memory problems independent of impairments in short-term memory (Swanson et al., 1996). Our results are in line with Swanson's finding of both verbal and visual working memory problems being present when high demands are placed on the central executive (Swanson et al., 1996). Children with RD appear to show general instead of modality-specific working memory problems. The comorbid group did not differ from the RD-only group, which means that children with ADHD did show working memory problems but only when RD was also present. This is an important finding as most studies investigating working memory in ADHD did not control for the presence of RD. Consequently, working memory problems could have been mistakenly attributed to ADHD due to a high number of children with comorbid RD. It has been reported by others that when learning disabilities (LD) are controlled for, EF deficits are more prevalent in children with LD or comorbid LD+ADHD than in children with only ADHD (see Lazar & Frank, 1998). However, it must be noted that in the latter study the LD sample was not specific to reading problems alone.

This finding of EF problems in RD, together with other studies reporting EF problems in other disorders (see Sergeant et al., 2002), questions Barkley's EF theory which states that these deficits are specific for ADHD (Barkley, 1997). In addition, the absence of evidence for EF deficits in ADHD when a baseline measure of functioning is included, as has also been reported by others (e.g., Andreou et al., 2007; Rommelse et al., 2007), is in line with the current opinion of several researchers that EF deficits are not the central cause of ADHD for all or most cases (Halperin & Schulz, 2006; Nigg et al., 2005; Willcutt, Doyle, et al., 2005). Researchers however acknowledge the fact that problems with EF are characteristic for a considerable portion of individuals with ADHD but plead for a multiple deficits/pathways view of ADHD with EF deficits being only one of several possible causes (Nigg et al., 2005; Pennington, 2005; Sonuga-Barke, 2005).

Motor speed and within-subject variability

In contrast to the frequent observation that children with ADHD are slower in performing motor responses (Barkley, 2005; Purvis & Tannock, 2000), we did not find differences in RT between children with or without ADHD in the simple RT task (Go/no-go), nor in the two-choice RT task (n-back). One possible explanation lies in the inter-trial interval of both RT tasks (2 seconds in the Go/no-go tasks and maximum 4 seconds in the n-back tasks). It has frequently been found that children with ADHD are only impaired in their speed of responding when a slow event rate is used and not when a fast to moderate event rate is used (Sergeant, 2000). This observation can be explained by the state regulation hypothesis, which states that children with ADHD get under-activated under slow event rate conditions and are not able to allocate enough effort to adjust this under-activation (van der Meere, 2005).

The children with ADHD in our study were however more variable in their RTs than children without ADHD. This is currently the most consistent finding concerning ADHD associated deficits and has therefore been put forward as a candidate endophenotype of ADHD (Bidwell et al., 2007; Castellanos et al., 2005; Kuntsi et al., 2005; Lijffijt et al., 2005). However, in the current study higher within-subject variability was also associated with RD (when the n-back and RAN tasks had to be performed), a finding also reported in some previous studies (e.g., Burgio-Murphy et al., 2007). Marked within-subject variability has also been reported in other clinical disorders, like schizophrenia, depression and borderline personality (Kaiser et al., 2008). These findings seem to question the uniqueness of greater within-subject variability to ADHD. However, it is possible that different factors underlie this higher variability in ADHD and RD. Greater SD-RT can be the result of a general process (greater deviation at both sides of the mean RT) or of a more specific process (greater deviation only in the slow or the fast end of the RT distribution). Children with ADHD have been found to show more abnormally slow responses instead of general inconsistency, which can be interpreted as the occurrence of attentional lapses during information processing (Hervey et al., 2006; Leth-Steensen, Elbaz, & Douglas, 2000) or an inability to efficiently regulate one's activation state to meet the demands of a task (Douglas, 1999; Sergeant, 2000; van der Meere, 2005). As state regulation problems have not been associated with RD, other factors may underlie the higher variability in RD. Further research is needed to address this issue.

Nature of the comorbidity

The nature of the comorbidity was explored by comparing the profile of deficits of the comorbid group with that of the single disorder groups. We evaluated both the kind of deficits they exhibited (qualitative comparison) as well as the level of impairment they displayed (quantitative comparison of the means). Most studies concerning the nature of the comorbidity of ADHD and RD support the view that ADHD and RD represent separate disorders and that the comorbid group exhibits the deficits of both single disorders (e.g., Dykman & Ackerman, 1991; Klorman et al., 1999; Nigg et al., 1998; Purvis & Tannock, 1997, 2000; Seidman, Biederman, Monuteaux, Doyle, & Faraone, 2001; Willcutt, Pennington,

et al., 2005). Our study also found that the comorbid group showed all the deficits of the pure groups and did not exhibit any deficits that were not present in one of the pure groups, as has been found in some other studies (e.g., Bental & Tirosh, 2007; Rucklidge & Tannock, 2002). This finding, together with the absence of ADHD x RD interactions, argues against the hypothesis that the comorbid condition is a separate cognitive subtype.

With respect to the level of impairment, we found that the comorbid group did not differ from the single disorder group when only one of the pure groups was impaired on the measure (e.g. phonological and RAN tasks, memory difference score). Two of the measures were associated with impairments in both disorders. Children with ADHD and children with RD showed greater SD-RT performing the n-back task, and the level of impairment in the comorbid group was similar to that of the single disorder groups. Greater SD-RT is therefore a potential shared cognitive risk factor in RD and ADHD. Both children with ADHD and children with RD also showed a general tendency to make more errors in the Go/no-go tasks. On this measure the comorbid group was most severely, almost double, impaired; on all Go/no-go tasks, independent of modality and inhibition condition, the mean percentage of commission errors was nearly equal to the sum of the percentages of the pure groups. This interesting finding, which has been discussed above, underlines the importance of the inclusion of the comorbid group when investigating cognitive deficits in ADHD and RD.

All taken together, our findings fit well within the view that ADHD and RD have some cognitive risk factors that are shared, but also have some independent underlying factors (Banaschewski et al., 2005; Shanahan et al., 2006). Our results suggest that greater withinsubject variability could be a shared underlying deficit, while the higher error rate could be the result of different underlying mechanisms in both disorders. Obviously, more research is needed to investigate these possibilities. To date, research has not succeeded in finding a specific core deficit in ADHD (Andreou et al., 2007) and several researchers are starting to claim that a single deficit view may not be sufficient to understand the complex nature of ADHD (e.g., Nigg et al., 2005; Pennington, 2005; Sonuga-Barke, 2005). Our results suggest that this may well be the case for RD too. The proposed core deficit in phonological processing (Snowling, Bishop, & Stothard, 2000) can not explain why children with RD exhibit greater within-subject variability in linguistic and non-linguistic tasks, have problems memorizing purely visual material, and make more errors in non-phonological speeded RT tasks. It is therefore recommended to adopt a multiple deficit model to guide future research into the aetiology of RD too, as has been suggested by other researchers (e.g., Pennington, 2006).

Implications

The diagnosis of ADHD is still a controversial matter (Halperin & Schulz, 2006). Therefore, it is important to search for endophenotypes to identify valid cognitive / laboratory measures that can more reliably diagnose ADHD than the currently used behavioural measures (Nigg, Blaskey, Huang-Pollock, & Rappley, 2002; Purvis & Tannock, 1997). The results of the current study suggest that more research is needed into other domains than the currently most studied executive functions (inhibition and working memory) as these are possibly not uniquely associated with ADHD and therefore are not good candidates for diagnostic purposes.

As the comorbid disorder is associated with the cognitive deficits of both single disorders, treatment should be directed to both types of problems (Purvis & Tannock, 2000). It is therefore very important that during the diagnostic process the presence of symptoms of the other disorder is also evaluated in order to implement the correct treatment program. Since researchers are beginning to pay attention to comorbidities in the investigation of the cognitive deficits of ADHD, evidence is gathering that certain deficits are not as uniquely associated with ADHD as previously thought. Theories of ADHD and also of RD should be adjusted in order to be able to account for these new findings.

Limitations

The current study had some limitations that need to be mentioned. In view of the relative small sample sizes in all groups, the reported results will need to be replicated by future studies with larger samples. Due to these small sample sizes, it was not possible to distinguish between ADHD subtypes. Future research should investigate whether the reported effects apply for each of the ADHD subtypes as it has been suggested that they may differ in the cognitive profile they exhibit (Nigg et al., 2002). It should also be mentioned that we only studied response inhibition and working memory, and therefore we do not know whether our findings also apply to other EF domains. Future research should use control/baseline measures to examine whether impaired performance on other EF tasks also relates to a general inaccurate response style, or represents a true EF deficit. Lastly, the domains of interest of our study were examined only by means of behavioural measures and these can not give us information about underlying/covert processes (e.g., efficiency of the error monitoring mechanism). The latter processes could be more sensitive to differentiate ADHD from RD and controls as differences in brain processes have been found in the absence of differences in overt measures (Banaschewski & Brandeis, 2007). Further research with ERP measures should be conducted to study the nature of deficits found in both disorders (e.g. the higher error rate in simple RT tasks).

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References

- Achenbach, T.M. (1991). Manual for the Child Behaviour Checklist: 4-18 and 1991 profile.Burlington, VT: University of Vermont.
- Alderson, R.M., Rapport, M.D., & Kofler, M.J. (2007). Attention-deficit/hyperactivity disorder and behavioral inhibition: A meta-analytic review of the stop-signal paradigm. *Journal of Abnormal Child Psychology*, 35, 745-758.
- Andreou, P., Neale, B.M., Chen, W., Christiansen, H., Gabriels, I., Heise, A., Meidad, S.,
 Muller, U.C., Uebel, H., Banaschewski, T., Manor, I., Oades, R., Roeyers, H.,
 Rothenberger, A., Sham, P., Steinhausen, H., Asherson, P., & Kuntsi, J. (2007).
 Reaction time performance in ADHD: Improvement under fast-incentive condition
 and familial effects. *Psychological Medicine*, *37*, 1703-1715.
- August, G.J., & Garfinkel, B.D. (1990). Comorbidity of ADHD and reading disability among clinic-referred children. *Journal of Abnormal Child Psychology*, *18*, 29-45.
- Baddeley, A. (1992). Working memory. Science, 255, 556-559.
- Banaschewski, T., & Brandeis, D. (2007). Annotation: What electrical brain activity tells us about brain function that other techniques cannot tell us a child psychiatric perspective. *Journal of Child Psychology and Psychiatry*, *48*, 415-435.
- Banaschewski, T., Hollis, C., Oosterlaan, J., Roeyers, H., Rubia, K., Willcutt, E., & Taylor, E. (2005). Towards an understanding of unique and shared pathways in the psychopathophysiology of AD/HD. *Developmental Science*, *8*, 132-140.
- Barkley, R.A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*, 65-94.
- Barkley, R.A. (2005). Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment. New York: Guilford.

- Bental, B., & Tirosh, E. (2007). The relationship between attention, executive functions and reading domain abilities in attention deficit hyperactivity disorder and reading disorder: A comparative study. *Journal of Child Psychology and Psychiatry*, 48, 455-463.
- Berument, S.K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *British Journal of Psychiatry*, *175*, 444-451.
- Bidwell, L.C., Willcutt, E.G., DeFries, J.C., & Pennington, B.F. (2007). Testing for neuropsychological endophenotypes in siblings discordant for attentiondeficit/hyperactivity disorder. *Biological Psychiatry*, 62, 991-998.
- Bruin, K.J., & Wijers, A.A. (2002). Inhibition, response mode, and stimulus probability: A comparative event-related potential study. *Clinical Neurophysiology*, 113, 1172-1182.
- Brus, B.T., & Voeten, M.J.M. (1973). *Een-Minuut-Test* [One-Minute-Test]. Nijmegen, The Netherlands: Berkhout.
- Burgio-Murphy, A., Klorman, R., Shaywitz, S.E., Fletcher, J.M., Marchione, K.E., Holahan,
 J., Stuebing, K.K., Thatcher, J.E., & Shaywitz, B.A. (2007). Error-related eventrelated potentials in children with attention-deficit/hyperactivity disorder, oppositional
 defiant disorder, reading disorder, and math disorder. *Biological Psychology*, 75, 7586.
- Castellanos, F.X., Sonuga-Barke, E.J.S., Milham, M.P., & Tannock, R. (2006). Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences*, 10, 117-123.
- Castellanos, F.X., Sonuga-Barke, E.J.S., Scheres, A., Di Martino, A., Hyde, C., & Walters, J.R. (2005). Varieties of attention-deficit/hyperactivity disorder-related intraindividual variability. *Biological Psychiatry*, 57, 1416-1423.

- Castellanos, F.X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: The search for endophenotypes. *Nature Reviews Neuroscience, 3*, 617-628.
- Denckla, M.B., & Rudel, R.G. (1976). Rapid Automatized Naming (RAN) Dyslexia differentiated from other learning-disabilities. *Neuropsychologia*, *14*, 471-479.
- Depessemier, P., & Andries, C. (2009). Test voor Gevorderd Lezen en Schrijven [Test for Advanced Reading and Writing]. Antwerpen, Belgium: Garant.
- Dimoska, A., & Johnstone, S.J. (2008). Effects of varying stop-signal probability on ERPs in the stop-signal task: Do they reflect variations in inhibitory processing or simply novelty effects? *Biological Psychology*, 77, 324-336.
- Douglas, V.I. (1999). Cognitive control processes in attention-deficit/hyperactivity disorder.
 In H.C. Quay, & A.E. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 105-138). New York: Kluwer Academic.
- Dykman, R.A., & Ackerman, P.T. (1991). Attention deficit disorder and specific reading disability: Separate but often overlapping disorders. *Journal of Learning Disabilities*, 24, 96-103.
- Faraone, S.V., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The worldwide prevalence of ADHD: Is it an American condition? *World Psychiatry*, *2*, 104-113.
- Farmer, M.E., & Klein, R.M. (1995). The evidence for a temporal processing deficit linked to dyslexia: A review. *Psychonomic Bulletin & Review*, 2, 460-493.
- Grégoire, J. (2000). *L'évaluation clinique de l'intelligence de l'enfant* [The clinical evaluation of the child's intelligence]. Liège, Belgium: Mardaga.
- Halperin, J.M., & Schulz, K.P. (2006). Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. *Psychological Bulletin*, *132*, 560-581.

- Hervey, A.S., Epstein, J.N., Curry, J.F., Tonev, S., Arnold, L.E., Conners, C.K., Hinshaw, S.P., Swanson, J.M., & Hechtman, L. (2006). Reaction time distribution analysis of neuropsychological performance in an ADHD sample. *Child Neuropsychology*, 12, 125-140.
- Hinshaw, S.P. (1992). Externalizing behaviour problems and academic underachievement in childhood and adolescence: Causal relationships and underlying mechanisms. *Psychological Bulletin*, 111, 127-155.
- Hollingshead, A.B., & Redlich, F.C. (1958). Social Class and Mental Illness. New York: John Wiley & Sons.
- Jonkman, L.M., van Melis, J.J.M., Kemner, C., & Markus, C.R. (2007). Methylphenidate improves deficient error evaluation in children with ADHD: An event-related brain potential study. *Biological Psychology*, *76*, 217-229.
- Kaiser, S., Roth, A., Rentrop, M., Friederich, H., Bender, S., & Weisbrod, M. (2008). Intraindividual reaction time variability in schizophrenia, depression and borderline personality disorder. *Brain and Cognition*, 66, 73-82.
- Kaufman, A.S., Kaufman, J.C., Balgopal, R., & McLean, J.E. (1996). Comparison of three
 WISC-III short forms: Weighing psychometric, clinical, and practical factors. *Journal* of Clinical Child Psychology, 25, 97-105.
- Kibby, M.Y., Marks, W., Morgan, S., & Long, C.J. (2004). Specific impairment in developmental reading disabilities: A working memory approach. *Journal of Learning Disabilities*, 37, 349-363.
- Klein, C., Wendling, K., Huettner, P., Ruder, H., & Peper, M. (2006). Intra-subject variability in attention-deficit hyperactivity disorder. *Biological Psychiatry*, *60*, 1088-1097.
- Klorman, R., Hazel-Fernandez, L.A., Shaywitz, S.E., Fletcher, J.M., Marchione, K.E., Holahan, J.M., Stuebing, K.K., & Shaywitz, B.A. (1999). Executive functioning

deficits in ADHD are independent of oppositional defiant or reading disorder. *Journal* of the American Academy of Child and Adolescent Psychiatry, 38, 1148-1155.

- Kort, W., Schittekatte, M., van den Bos, K.P., Vermeir, G., lutje Spelberg, H.C., Verhaeghe,
 P., & van der Wild, S. (2005). *Dyslexie Screening Test (DST-NL). Handleiding*[Manual for the Dyslexia Screening Test]. London: Harcourt Test Publishers.
- Kuntsi, J., Andreou, P., Ma, J., Börger, N.A., & van der Meere, J.J. (2005). Testing assumptions for endophenotype studies in ADHD: Reliability and validity of tasks in a general population sample. *BMC Psychiatry*, *5*, 40.
- Lazar, J.W., & Frank, Y. (1998). Frontal systems dysfunction in children with attentiondeficit/hyperactivity disorder and learning disabilities. *The Journal of Neuropsychiatry* and Clinical Neurosciences, 10, 160-167.
- Leth-Steensen, C., Elbaz, Z.K., & Douglas, V.I. (2000). Mean response times, variability, and skew in the responding of ADHD children: A response time distributional approach. *Acta Psychologica*, *104*, 167-190.
- Lijffijt, M., Kenemans, J.L., Verbaten, M.N., & van Engeland, H. (2005). A meta-analytic review of stopping performance in attention-deficit/hyperactivity disorder: Deficient inhibitory motor control? *Journal of Abnormal Psychology*, *114*, 216-222.
- Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A meta-analysis of working memory impairments in children with attention-deficit/hyperactivity disorder.
 Journal of the American Academy of Child and Adolescent Psychiatry, 44, 377-384.
- Miyake, A., Friedman, N.P., Emerson, M.J., Witzki, A.H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex frontal lobe tasks: A latent variable analysis. *Cognitive Psychology*, *41*, 49-100.
- Neuhaus, G.F., Carlson, C.D., Jeng, W.M.M., Post, Y., & Swank, P.R. (2001). The reliability and validity of rapid automatized naming scoring software ratings for the

determination of pause and articulation component durations. *Educational and Psychological Measurement, 61,* 490-504.

- Nigg, J.T., Blaskey, L.G., Huang-Pollock, C.L., & Rappley, M.D. (2002). Neuropsychological executive functions and DSM-IV ADHD subtypes. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 59-66.
- Nigg, J.T., Hinshaw, S.P., Carte, E.T., & Treuting, J.J. (1998). Neuropsychological correlates of childhood attention-deficit/hyperactivity disorder: Explainable by comorbid disruptive behavior or reading problems? *Journal of Abnormal Psychology*, 107, 468-480.
- Nigg, J.T., Willcutt, E.G., Doyle, A.E., & Sonuga-Barke, E.J.S. (2005). Causal heterogeneity in attention-deficit/hyperactivity disorder: Do we need neuropsychologically impaired subtypes? *Biological Psychiatry*, *57*, 1224-1230.
- Oosterlaan, J., Logan, G.D., & Sergeant, J.A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD+CD, anxious, and control children: A meta-analysis of studies with the stop task. *Journal of Child Psychology and Psychiatry*, *39*, 411-425.
- Parmenter, B.A., Shucard, J.L., Benedict, R.H.B., & Shucard, D.W. (2006). Working memory deficits in multiple sclerosis: Comparison between the n-back task and the paced auditory serial addition test. *Journal of the International Neuropsychological Society*, *12*, 677-687.
- Pelham, W., Gnagy, E.M., Greenslade, K.E., & Milich, R. (1992). Teacher ratings of DSM-III-R symptoms for the disruptive behaviour disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 210-218.
- Pennington, B.F. (2005). Towards a new neuropsychological model of ADHD: Subtypes and multiple deficits. *Biological Psychiatry*, *57*, 1221-1223.

- Pennington, B.F. (2006). From single to multiple deficit models of developmental disorders. *Cognition, 101,* 385-413.
- Pennington, B.F., Groisser, D., & Welsh, M.C. (1993). Contrasting cognitive deficits in attention deficit hyperactivity disorder versus reading disability. *Developmental Psychology*, 29, 511-523.
- Pennington, B.F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *37*, 51-87.
- Pontius, A.A. (1973). Dysfunction patterns analogous to frontal lobe system and caudate nucleus syndromes in some groups of minimal brain dysfunction. *Journal of the American Medical Women's Association, 26,* 285-292.
- Purvis, K.L., & Tannock, R. (1997). Language abilities in children with ADHD, reading disabilities, and normal controls. *Journal of Abnormal Child Psychology*, 25, 133-144.
- Purvis, K.L., & Tannock, R. (2000). Phonological processing, not inhibitory control, differentiates ADHD and reading disability. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 485-494.
- Ramautar, J., Kok, A., & Ridderinkhof, K.R. (2004). Effects of stop-signal probability in the stop-signal paradigm: The N2/P3 complex further validated. *Brain and Cognition*, 56, 234-252.
- Rashid, F.L., Morris, M.K., & Morris, R. (2001). Naming and verbal memory skills in adults with attention deficit hyperactivity disorder and reading disability. *Journal of Clinical Psychology*, 57, 829-838.
- Roberts, R.J., & Pennington, B.F. (1996). An interactive framework for examining prefrontal cognitive processes. *Developmental Neuropsychology*, *12*, 105-126.

Rommelse, N.N.J., Altink, M.E., de Sonneville, L.M.J., Buschgens, C.J.M., Buitelaar, J., Oosterlaan, J., & Sergeant, J.A. (2007). Are motor inhibition and cognitive flexibility dead ends in ADHD? *Journal of Abnormal Child Psychology*, *35*, 957-967.

- Rommelse, N.N.J., Altink, M.E., Oosterlaan, J., Beem, L., Buschgens, C.J.M., Buitelaar, J., & Sergeant, J.A. (2008). Speed, variability, and timing of motor output in ADHD: Which measures are useful for endophenotypic research? *Behavior Genetics*, 38, 121-132..
- Rucklidge, J.J., & Tannock, R. (2002). Neuropsychological profiles of adolescents with ADHD: Effects of reading difficulties and gender. *Journal of Child Psychology and Psychiatry*, *43*, 988-1003.
- Savage, R., Lavers, N., & Pillay, V. (2007). Working memory and reading difficulties: What we know and what we don't know about the relationship. *Educational Psychology Review*, 19, 185-221.
- Seidman, L.J., Biederman, J., Monuteaux, M.C., Doyle, A.E., & Faraone, S.V. (2001). Learning disabilities and executive dysfunction in boys with ADHD. *Neuropsychology*, 15, 544-556.
- Semrud-Clikeman, M., Guy, K., Griffin, J.D., & Hynd, G.W. (2000). Rapid naming deficits in children and adolescents with reading disabilities and attention deficit hyperactivity disorder. *Brain and Language*, 74, 70-83.
- Sergeant, J. (2000). The cognitive-energetic model: An empirical approach to attentiondeficit/hyperactivity disorder. *Neuroscience and Biobehavioral Reviews*, 24, 7-12.
- Sergeant, J.A., Geurts, H., & Oosterlaan, J. (2002). How specific is a deficit of executive functioning for attention-deficit/hyperactivity disorder? *Behavioural Brain Research*, 130, 3-28.
- Shaffer, D., Fisher, P., Lucas, C., Dulcan, M., & Schwab-Stone, M.E. (2000). NIMH Diagnostic Interview for Children Version IV (NIMH DISC-IV): Description,

differences from previous versions, and reliability of some common diagnosis. *Journal* of the American Academy of Child and Adolescent Psychiatry, 39, 28-38.

- Shanahan, M.A., Pennington, B.F., Yerys, B.E., Scott, A., Boada, R., Willcutt, E.G., Olson,
 R.K., & DeFries, J.C. (2006). Processing speed deficits in attention
 deficit/hyperactivity disorder and reading disability. *Journal of Abnormal Child Psychology*, *34*, 585-602.
- Simmonds, D.J., Pekar, J.J., & Mostofsky, S.H. (2008). Meta-analysis of go/no-go tasks demonstrating that fMRI activation associated with response inhibition is task-dependent. *Neuropsychologia*, *46*, 224-232.
- Snowling, M., Bishop, D.V.M., & Stothard, S.E. (2000). Is preschool language impairment a risk factor for dyslexia in adolescence? *The Journal of Child Psychology and Psychiatry and Allied Disciplines, 41,* 587-600.
- Sonuga-Barke, E.J.S. (2005). Causal models of attention-deficit/hyperactivity disorder: From common simple deficits to multiple developmental pathways. *Biological Psychiatry*, 57, 1231-1238.
- Stein, J. (2001). The sensory basis of reading problems. *Developmental Neuropsychology*, 20, 509-534.
- Swanson, H.L., Ashbaker, M.H., & Lee C. (1996). Learning-disabled readers' working memory as a function of processing demands. *Journal of Experimental Child Psychology*, 61, 242-275.
- Swanson, H.L., Mink, J., & Bocian, K.M. (1999). Cognitive processing deficits in poor readers with symptoms of reading disabilities and ADHD: More alike than different? *Journal of Educational Psychology*, 91, 321-333.
- Tabachnick, B.G., & Fidell, L.S. (2007). Using multivariate statistics (5th ed.). Boston: Allyn and Bacon.

- Tannock, R., Martinussen, R., & Frijters, J. (2000). Naming speed performance and stimulant effects indicate effortful, semantic processing deficits in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology*, 28, 237-252.
- Toplak, M.E., Dockstader, C., & Tannock, R. (2006). Temporal information processing in ADHD: Findings to date and new methods. *Journal of Neuroscience Methods*, 151, 15-29.
- van den Bos, K.P., & lutje Spelberg, H.C. (2007). *Continu Benoemen en Woorden Lezen* [Continuous Naming and Word Reading].Amsterdam, The Netherlands: Boom Testuitgevers.
- van den Bos, K.P., lutje Spelberg, H.C., Scheepsma, A.J.M., & de Vries, J.R. (1994). *Handleiding de Klepel* [Manual for the Klepel]. Nijmegen, The Netherlands: Berkhout.
- van der Meere, J.J. (2005). State regulation and attention deficit hyperactivity disorder. In D. Gozal, & D.L. Molfese (Eds), *Attention deficit hyperactivity disorder: From genes to patients* (pp. 162–213). Totowa, NJ: Humana Press Inc.
- Wechsler, D. (1991). *Wechsler Intelligence Scale for Children* (3rd ed.). San Antonio, TX: The psychological Corporation.
- Weyandt, L.L. (2005). Executive function in children, adolescents, and adults with attention deficit hyperactivity disorder: Introduction to the special issue. *Developmental Neuropsychology*, 27, 1-10.
- Wiersema, J.R., van der Meere, J.J., & Roeyers, H. (2005). ERP correlates of impaired error monitoring in children with ADHD. *Journal of Neural Transmission*, 112, 1417-1430.
- Willcutt, E.G., Doyle, A.E., Nigg, J.T., Faraone, S.V., & Pennington, B.F. (2005). Validity of the executive function theory of ADHD: A meta-analytic review. *Biological Psychiatry*, 57, 1336-1346.

- Willcutt, E.G., Pennington, B.F., Boada, R., Ogline, J.S., Tunick, R.A., Chhabildas, N.A., & Olson, R.K. (2001). A comparison of the cognitive deficits in reading disability and attention-deficit/hyperactivity disorder. *Journal of Abnormal Psychology*, *110*, 157-172.
- Willcutt, E.G., Pennington, B.F., Olson, R.K., Chhabildas, N., & Hulslander, J. (2005).
 Neuropsychological analyses of comorbidity between reading disability and attention deficit hyperactivity disorder: In search of the common deficit. *Developmental Neuropsychology*, 27, 35-78.
- Williams, B.R., Strauss, E.H., Hultsch, D.F., Hunter, M.A., & Tannock, R. (2007). Reaction time performance in adolescents with attention-deficit/hyperactivity disorder:
 Evidence of inconsistency in the fast and slow portions of the RT distribution. *Journal of Clinical and Experimental Neuropsychology*, 29, 277-289.
- Wright, B.A., Bowen, R.W., & Zecker, S.G. (2000). Nonlinguistic perceptual deficits associated with reading and language disorders. *Current Opinion in Neurobiology*, 10, 482-486.
- Wu, K.K., Anderson, V., & Castiello, U. (2002). Neuropsychological evaluation of deficits in executive functioning for ADHD children with or without learning disabilities. *Developmental Neuropsychology*, 22, 501-531.

Table 1

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Means of the Four Grou	ps on Descriptive	and Diagnostic	Measures
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	<u> </u>	1 D I I D			
	Control	ADHD	RD	ADHD+RD	
	(<i>N</i> =19)	(<i>N</i> =19)	(<i>N</i> =17)	(<i>N</i> =21)	
Measure	M (SD)	M (SD)	M (SD)	M (SD)	F
Age	120.5 (17.7)	127.2 (18.9)	127.1 (14.4)	122.7 (15.6)	0.72
SES	4.4 (0.7)	4.2 (0.7)	4.0 (0.8)	3.8(1.1)	1.73
Sex (M:F)	11:8	16:3	9:8	17:4	a
FSIQ	108.7 (12.9)	106.8 (15.9)	107.9(10.1)	105.3 (12.2)	0.25
ADHD symptoms ^b	$1.7_{a}(1.4)$	13.1 _b (4.6)	$4.0_{a}(3.0)$	11.8 _b (4.9)	41.31***
ODD symptoms ^b	$0.9_{a}(1.2)$	6.9 _b (2.9)	$1.4_{a}(1.6)$	$5.4_{b}(3.2)$	28.50***
CD symptoms ^b	0.1 (0.2)	1.8 (1.5)	0.2 (0.3)	1.0 (1.1)	
Reading score ^c	$11.4_{a}(2.5)$	9.3 _b (2.1)	4.3 _c (1.9)	$4.3_{\rm c}(1.5)$	61.06***

Note. ADHD = attention-deficit/hyperactivity disorder. RD = reading disorder. SES = socioeconomic status. FSIQ = full-scale intelligence quotient. ODD = oppositional defiant disorder. CD = conduct disorder. Means with different subscripts are significantly different by Bonferroni post hoc tests. ^a $\chi^2(3) = 6.67$.

^b Mean of teacher and parent Disruptive Behavior Disorder Rating Scale (DBDRS) raw score.

^c Mean of Klepel and One-Minute-Test standard score.

*** *p* <.001

Table 2

Unad	iusted Means	of the Four	Groups on	Linguistic and	Neuropsycho	logical Measures

	Control	ADHD	RD	ADHD+RD		
	(<i>N</i> =19)	(<i>N</i> =19)	(<i>N</i> =17)	(<i>N</i> =21)	Main effects F	
Measure	M (SD)	M (SD)	M (SD)	M (SD)	ADHD	RD
Phonology						
% correct	83.9 _a (8.2)	77.9 _a (12.9)	61.1 _b (18.9)	57.3 _b (15.4)	2.25	43.70***
Mean tempo (s)	173.5 _a (68.1)	218.2 _{ab} (130.0)	279.6 _b (134.9)	309.7 _b (119.9)	1.96	13.74***
RAN times (s)						
Total pause time	$17.1_{a}(6.7)$	18.5 _{ab} (8.8)	24.4 _{bc} (8.4)	$26.4_{\rm c}(8.4)$	0.44	18.80***
SD item pause times	$1.2_{a}(0.5)$	$1.0_{a}(0.3)$	1.7 _b (0.8)	$1.9_{b}(0.6)$	0.00	29.62***
Response Inhibition						
Low inhibition (50-50)						
% commission errors	$7.6_{a}(6.3)$	14.9 _b (8.9)	$11.6_{ab}(6.2)$	24.8 _c (15.5)	16.31***	11.10**
RT (ms)	521.8 (86.6)	562.7 (90.3)	541.4 (79.7)	518.0 (85.7)	0.19	0.40
SD-RT (ms)	147.4 (44.4)	183.3 (42.9)	166.9 (41.4)	174.2 (40.1)	4.89*	0.29

	Control	ADHD	RD	ADHD+RD		
	(<i>N</i> =19)	(<i>N</i> =19)	(<i>N</i> =17)	(<i>N</i> =21)	Main effects F	
Measure	M (SD)	M (SD)	M (SD)	M (SD)	ADHD	RD
High inhibition (80-20)						
% commission errors	17.9 _a (10.9)	30.9 _b (15.6)	26.6 _b (8.9)	45.9 _c (17.8)	20.43***	18.29***
RT (ms)	518.7 (98.9)	521.9 (101.8)	504.0 (85.7)	475.9 (79.4)	0.35	2.06
SD-RT (ms)	151.3 (51.2)	182.6 (57.3)	174.2 (55.4)	167.5 (44.0)	1.06	0.10
Inhibition difference score	10.3 (7.3)	16.0 (9.8)	15.0 (6.7)	20.5 (8.3)	0.33	0.04
Working Memory						
% errors 0-back	3.6 _a (2.1)	7.1 _{ab} (5.1)	$4.4_{a}(3.5)$	9.0 _b (4.8)	13.59***	1.22
% errors 1-back	$4.5_{a}(1.5)$	11.6 _b (1.4)	10.0 _{ab} (1.5)	14.1 _b (1.4)	15.79***	14.64***
Memory difference score	$0.9_{a}(1.8)$	4.5 _a (6.6)	5.6 _a (4.4)	5.1 _a (7.3)	0.04	5.80*
RT (ms)	850.7 (127.2)	863.7 (159.2)	875.0(127.2)	881.3 (126.4)	0.09	0.44
SD-RT (ms)	240.2 _a (53.2)	270.7 _{ab} (48.4)	266.4 _{ab} (62.2)	292.9 _b (27.2)	6.36*	4.58*

Note. ADHD = attention-deficit/hyperactivity disorder. RD = reading disorder. RAN = Rapid Automatized Naming. SD = standard deviation. RT = reaction time. SD-RT = standard deviation of RTs. Means with different subscripts are significantly different by Bonferroni post hoc tests.

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



Figure 1. Meaningless symbols used in one condition of the Go/no-go task

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Figure 2. Meaningless symbols used in one condition of the n-back task.