

Response Execution and Inhibition in Children with AD/HD and Other Disruptive Disorders: The Role of Behavioural Activation

Anouk Scheres, Jaap Oosterlaan, and Joseph A. Sergeant

Free University of Amsterdam, The Netherlands

This study was aimed at (a) replicating findings of slow and variable response execution and slow response inhibition in Attention Deficit/Hyperactivity Disorder (AD/HD), (b) investigating whether these deficits are specifically related to AD/HD or may also be observed in Oppositional Defiant Disorder (ODD), and children comorbid for AD/HD+ODD, and (c) examining the role of activation level in task performance of children with AD/HD. To meet these aims, the stop paradigm was administered at three levels of activation, using a slow, medium, and fast presentation rate of stimuli, to 4 groups of children: 24 AD/HD children, 21 children with ODD, 27 children with comorbid AD/HD+ODD, and 41 normal controls. As hypothesized, children with AD/HD exhibited a slow response execution process with considerable variability in the speed of responding compared to normal controls. Slow response execution was also observed in the comorbid AD/HD+ODD group but not in the pure ODD group. Larger variability in the speed of responding was common to all disruptive groups compared with controls. In contrast to our hypothesis, no group differences emerged for inhibitory functioning. Finally, the slow event rate condition caused a further deterioration in the speed of the response execution process in both the AD/HD group and ODD group.

Keywords: ADD, ADHD, inhibition, activation, event rate, ODD/CD.

Abbreviations: AD/HD: Attention Deficit/Hyperactivity Disorder; CBCL: Child Behavior Checklist; CD: conduct disorder; DBD: Disruptive Behavior Disorder Rating Scale; IOWA CTRS: IOWA Conners Teacher Rating Scale; ISI: interstimulus interval; MRT: mean reaction time; NC: normal control; ODD: Oppositional Defiant Disorder; RT: reaction time; SSRT: stop signal reaction time; TRF: Teacher Rating Form.

Introduction

In the *Diagnostic and statistical manual of mental disorders* (DSM-IV, American Psychiatric Association, 1994), Attention Deficit/Hyperactivity Disorder (AD/HD) is characterized by the following symptom domains: inattention, impulsivity, and hyperactivity.

Some researchers have proposed poor response inhibition to be *the* central impairment in AD/HD. Inhibitory control is one of the executive functions, mediated by the prefrontal cortex. In one authoritative theoretical model, Barkley (1997) proposed that a deficit in behavioural inhibition (as the primary executive, self-regulatory act, necessary for performing other executive functions) is the core deficit of AD/HD. Quay (1988, 1997) suggested that AD/HD children have an underactive Behavioural Inhibition System (BIS), which results in deficient response inhibition.

In contrast to the inhibition hypothesis, other researchers have proposed that AD/HD is a manifestation of an underlying self-regulatory defect, defined as a failure to allocate adequate effort to meet task demands

(Douglas, 1999). A comparable position explains deficient task performance in AD/HD in terms of a nonoptimal energetic state, in particular activation (Sergeant, Oosterlaan, & Van der Meere, 1999; Van der Meere, 1996). Sanders (1983, 1998) defines activation as a behavioural “tonic readiness to respond”. According to the cognitive-energetic model, an optimal activation state is a prerequisite for readiness for motor action (Sanders, 1983, 1998). Behavioural activation influences motor adjustment, and the primary task variables influencing behavioural activation are drugs, loud noise, sleep deprivation, and ISI (interstimulus interval) (Frowein, 1981). The activation level increases (becomes higher) with an increase in the presentation rate of stimuli. In contrast, as ISI becomes longer, behavioural activation declines (Sanders, 1998). In several studies it has been shown that performance of AD/HD children is impaired when a long ISI or preparatory interval is used (Zahn, Kruesi, & Rapoport, 1991), or long delays between cue and target (Swanson et al., 1991; Tomporowski, Tinsley, & Hager, 1994), long prereponse delays (Sonuga-Barke & Taylor, 1992), and slow event rates (Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989; Conte, Kinsbourne, Swanson, Zirk, & Samuels, 1986; Van der Meere, Shalev, Börger, & Gross-Tsur, 1995; Van der Meere, Stemerding, & Gunning, 1995; Van der Meere, Vreeling, & Sergeant, 1992). The typical slow and variable response style of

Requests for reprints to: Anouk Scheres, Department of Clinical Neuropsychology, Free University of Amsterdam, De Boelelaan 1109, 1081 HV Amsterdam, The Netherlands (E-mail: apj.scheres@psy.vu.nl).

children with AD/HD generally improves, when the presentation rate of stimuli is fast. However, in two studies, it was shown that both slow and fast event rate conditions induced poor performance in children with AD/HD, compared to a medium event rate (Chee et al., 1989; Van der Meere, Stemerink, et al., 1995). In Chee's study, the group differences in the fast event rate condition disappeared after correction for the time-on-task effect. In the study by Van der Meere et al. only the impulsivity measure was shown to be dependent on event rate but not the response execution measures. In a recent review, it was concluded that the performance of children with AD/HD is strongly dependent on the behavioural activation state of the child (Sergeant et al., 1999). Although it is not yet clear whether children with AD/HD suffer from a suboptimal activation state (showing poor performance under slow event rate conditions), or from an inability to adjust their activation state (to high and low events rates), AD/HD performance seems to be dependent on behavioural activation.

In several studies, the stop paradigm (Logan & Cowan, 1984) has been used to contrast AD/HD children with normal controls and with conduct disordered (CD) children (see for review Oosterlaan, Logan, & Sergeant, 1998; Tannock, 1998). The stop paradigm is a task that enables one to measure the speed of both the response execution and response inhibition processes. In previous studies it has been demonstrated that children with AD/HD have slower and more variable response execution processes as well as slower inhibitory processes compared to normal controls (Oosterlaan & Sergeant, 1998a, b; Pliszka, Borchering, Spratley, Leon, & Irick, 1997; Schachar, Tannock, Marriott, & Logan, 1995; see for review Oosterlaan et al., 1998; Sergeant et al., 1999). In some studies, slow and variable response execution has been reported to be related not only to children with AD/HD, but also to children with aggressive symptoms (Oosterlaan & Sergeant, 1996, 1998a, b). However, in another study, slow response execution processing was only observed in children with AD/HD and children with AD/HD+CD, but not in children with only CD (Schachar & Tannock, 1995). Poor inhibitory performance has in some studies been shown to be specifically related to AD/HD: the inhibition deficit was not observed in children with CD (Schachar & Logan, 1990; Schachar & Tannock, 1995; Schachar, Tannock, & Logan, 1993). However, a recent meta-analysis demonstrated deficient inhibitory control in both AD/HD and Oppositional Defiant Disorder (ODD)/CD (Oosterlaan et al., 1998). Thus, at present it remains unclear whether deficiencies in response execution and inhibition are specifically related to AD/HD or also evident in children with ODD/CD and comorbid AD/HD+ODD/CD.

The aim of the current study was threefold. The first aim was to replicate findings of slow and variable response execution, and deficient response inhibition in AD/HD. Second, it was examined whether poor response execution and inhibition is uniquely related to AD/HD, or whether these deficiencies are also observed in children with ODD/CD and with comorbid AD/HD+ODD/CD. Third, the present study directed attention to the effects of behavioural activation level on response execution ("go") and the inhibition ("stop") process. We hypothesized that (a) children with AD/HD would show slow and variable response execution processes, as well as slow response inhibition processes, (b) poor response execution and inhibition would not only be related to

AD/HD but might also be observed in children with ODD/CD and possibly in children comorbid for AD/HD+ODD/CD, and (c) children with AD/HD would perform worse than normal control children, especially when the behavioural activation level is low. Since task performance in children with AD/HD, in general, normalizes when the event rate is fast and decreases in slow event rate conditions, it was hypothesized that poor response execution and inhibition in children with AD/HD are dependent on behavioural activation level. That is, poor executive and inhibitory control may be manifestations of a nonoptimal activation level, rather than being the core deficit of AD/HD. Van der Meere, Stemerink et al., (1995) showed that in children with AD/HD the ability to inhibit a response was dependent on the event rate of stimuli. An inhibition-deficit explanation of the information-processing performance of AD/HD needs to show that disinhibitory responding occurs *independently* of task event rate (Sergeant et al., 1999).

In order to test these hypotheses, three psychopathological groups were compared with healthy controls: a pure AD/HD group without comorbid ODD/CD symptoms, a pure ODD/CD group without comorbid AD/HD symptoms, and a comorbid AD/HD+ODD/CD group. ODD/CD symptoms are frequently comorbid with AD/HD (e.g., Angold, Costello, & Erkanli, 1999). The relevance of including a comorbid group is evident from a study by Schachar and Tannock (1995). They studied the basis of the comorbidity of AD/HD and CD by comparing the performance of children with AD/HD, CD, and AD/HD+comorbid CD on several factors, such as cognitive performance. Several hypotheses on the nature of the comorbidity of AD/HD with CD exist, such as AD/HD+CD being a hybrid of pure AD/HD and pure CD; another hypothesis is that the comorbid condition is a distinct condition that is different from the two pure disorders of AD/HD and CD. If the comorbid group shows a pattern of performance on the stop paradigm with the characteristics of both pure groups, the hybrid theory on comorbidity is supported. A unique pattern of performance in the comorbid group, however, would support the idea of AD/HD+ODD/CD being a distinct disorder.

The stop paradigm was administered to these four groups at three event rates in order to investigate the relationship between the go and the stop processes, on the one hand, and the behavioural activation level, on the other hand. This may enable us to determine how far deficiencies in response execution and inhibition, as measured in the stop paradigm, are in fact manifestations of a nonoptimal behavioural activation state.

Method

Subjects and Selection Criteria

One hundred and fifteen children in the age range of 7 to 12 years participated in this study. The participants were assigned to one of the four groups, i.e., the normal control group (NC), the AD/HD group, the ODD/CD group, or the comorbid (AD/HD and ODD/CD) group. The three psychopathological groups were selected from 14 special education services, which specialize in the education of children with extreme behavioural problems. Of all Dutch children in the age range of 6 to 12 years, 2.2% attend these special educational services (Central Office for Statistics, personal communication). The normal control children were selected from six regular schools. Schools were located throughout the country.

In order to select participants, a two-stage procedure was used. In the first stage, 1504 households (876 parents of children who were placed in special schools and 628 parents of children in regular schools) received information on the study, an informed consent form, and two child behavior questionnaires. If parents were willing to participate, they signed the informed consent form and completed the questionnaires. Questionnaires were the Disruptive Behavior Disorder Rating Scale (DBD; Oosterlaan, Scheres, Antrop, Roeyers, & Sergeant, 2000; Pelham, Gnagy, Greenslade, & Milich, 1992) and the Child Behavior Checklist (CBCL; Achenbach, 1991a; Verhulst, Van der Ende, & Koot, 1996a). The DBD consists of: (a) two subscales composed of the DSM-IV items for AD/HD, i.e., an Inattention subscale and an Hyperactivity/Impulsivity subscale, (b) a scale composed of the DSM-IV items for ODD, and (c) a scale composed of the DSM-IV items for CD. Items were rated on a scale ranging from 0 to 3. The DBD was used to select participants for the study. The major advantage of this rating scale is that it includes statements listed as behavioural descriptors of the disorders AD/HD, ODD, and CD in the DSM-IV. Parents of 576 children completed the questionnaires (response rate 38.3%). There were 337 children who met the inclusion criteria for one or more of the four groups (see below), and these children entered the second stage.

At stage two, teachers completed the DBD, the Teacher Rating Form (TRF; Achenbach, 1991b; Verhulst, Van der Ende, & Koot, 1996b), and the IOWA Conners Teacher Rating Scale (IOWA CTRS; Oosterlaan, Prins, & Sergeant, 1992; Pelham, Milich, Murphy, & Murphy, 1989). Three hundred and two sets of completed questionnaires were received (response rate 89.6%).

For a child to be included in one of the three psychopathological groups, both parent *and* teacher ratings had to meet inclusion criteria for that particular group. In this way the criterion of pervasiveness of the disorder was met. The inclusion criteria that were used are based on the DSM-IV symptoms for AD/HD, ODD, and CD. Inclusion criteria for the AD/HD group were: a rating of 12 or more on the Inattention subscale and/or on the Hyperactivity/Impulsivity subscale of both the parent and the teacher DBD. AD/HD inattentive subtype was defined as: (a) a rating of 12 or more on the Inattention subscale of both the parent and the teacher DBD, and (b) a rating lower than 12 on the Hyperactivity/Impulsivity subscale by at least one informant. AD/HD hyperactive/impulsive subtype was defined as: (a) a rating of 12 or more on the Hyperactivity/Impulsivity subscale of both the parent and the teacher DBD, and (b) a rating lower than 12 on the Inattention subscale by at least one informant. AD/HD combined subtype was defined as: (a) a rating of 12 or more on the Hyperactivity/Impulsivity subscale of both the parent and the teacher DBD, and (b) a rating of 12 or more on the Inattention subscale on both the parent and the teacher DBD. To be included in the ODD/CD group, the following criteria had to be met: (a) a rating of at least 8 on the ODD scale or a rating of at least 6 on the CD scale of the parent DBD, and (b) a score of at least 8 on the ODD scale or a score of at least 6 on the CD scale of the teacher DBD. To be assigned to the comorbid group, the criteria of both the AD/HD group and the ODD/CD group had to be met. In order to exclude children with psychotic symptoms, an additional criterion for all three psychopathological groups was that the child was rated at or below the 75th percentile on the Thought Problem scale of the CBCL and the TRF.

To be assigned to the normal control group both parents and teachers were required to rate the child (a) below the critical values of all the scales of the DBD, (b) at or below the 75th percentile on all the scales of the CBCL and the TRF, and (c) below the suggested cutoff scores on the Inattention/Overactivity scale and the Oppositional/Defiant scale of the IOWA CTRS (Pelham et al., 1989).

There were 154 children who met criteria for membership of one of the four groups. However, 39 children did not participate in the experiment for various reasons. The most important

reason for exclusion at this stage was use of medication that could not be discontinued (pipamperon or clonidine) ($N = 20$). Other children dropped out because of moving house, finishing school, or parents who withdrew their consent. The remaining 115 children participated in the experiment. Five AD/HD children, five comorbid children, and one ODD/CD child used methylphenidate (Ritalin®), but discontinued the use of this medication at a minimum of 18 hours prior to the experiment.

All children who were assigned to the ODD/CD group appeared to be ODD children and will therefore be referred to as the ODD group. Two children were excluded prior to data analyses: one because of an extreme low IQ (48), and the other because of a diagnosis of Asperger syndrome. The groups consisted of 24 AD/HD children, 21 ODD children, 27 comorbid children, and 41 normal control children. The AD/HD group consisted of 9 pervasively inattentive children, 6 pervasively hyperactive/impulsive children, 7 pervasively combined type children, and 2 children who were defined as inattentive by one rater and hyperactive/impulsive by the other rater. A Student Newman-Keuls procedure (overall α set at .05) showed that the groups did not differ with respect to age. The normal control group had fewer male subjects and a higher mean IQ than to the other groups (see Table 1). Correlations showed, however, that the dependent variables of the stop paradigm were significantly correlated with age only, and not with IQ or gender (data available from the first author). Each of the three psychopathological groups could be distinguished from one another and from the normal control group on the DBD scales that were used as the criterion measures. In addition, the selected groups differed from one another on a number of other scales. As would be predicted, the AD/HD group and the comorbid group showed the highest scores on the Attention scale of the CBCL and the TRF, and on the Inattention/Overactivity scale of the IOWA CTRS. The ODD group and the comorbid AD/HD+ODD group, as would be expected, showed the highest scores on the Aggression and Delinquency scales of the CBCL and the TRF, and on the Oppositional/Defiant scale of the IOWA CTRS (see Table 1). This supports the behavioural distinctiveness of the four groups.

Stop Paradigm

The stop paradigm involves two types of trials: go trials and stop trials. Go trials were aeroplanes, presented for a period of 300 ms at the midpoint of the computer screen. Immediately before the go stimulus onset, a fixation point (200 ms in duration) appeared on the screen. If the aeroplane pointed to the right, subjects were required to press the right response button. If the plane pointed to the left, subjects were instructed to press the left button. Stop trials consisted of a go trial and a stop signal (a 1000 Hz tone, 50 ms in duration), presented through earphones. The stop signal was usually presented shortly after the aeroplane, but could also be presented concurrently with or shortly before the aeroplane, dependent on the child's performance (see below). Children were instructed not to press either of the two buttons when the plane was followed by the tone. Seventy-five per cent of the trials were go trials, and 25% were stop trials. The stop paradigm allows measurement of both response execution (go trials) and response inhibition (stop trials).

Trials were presented in blocks of 32. Within a block the plane pointed equally often to the right or to the left. Stop signals were balanced for right and left go trials. Stop trials were presented randomly within each block with the restriction that two stop trials were presented in succession only once in each block.

The task commenced with four practice blocks, to make sure that the children were familiar with the paradigm. In the first two practice blocks only go trials were presented. During practice of the go task, children were encouraged with standardized instructions to respond as quickly as possible without making too many errors. In the last two practice blocks, 25% of

Table 1
Means, Standard Deviations, and Pairwise Group Comparisons for IQ, Age, and Rating Scale Scores

Measure	Group								Pairwise group comparisons ^b
	AD/HD (A) N = 24 (18) ^a		OOD (O) N = 21 (19) ^a		Comorbid (C) N = 27 (25) ^a		NC (N) N = 41 (24) ^a		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
IQ	92.2	15.1	86.9	14.7	82.7	12.6	105.0	25.0	N > A, O, C
Age	10.1	1.5	10.7	1.3	10.9	1.5	10.2	1.6	n.s.
CBCL									
Attention	70.6	6.6	64.7	8.4	72.3	7.8	51.0	1.9	A, C > O > N
Aggressive ^c	65.3	7.6	70.8	8.6	74.6	9.4	50.2	0.8	O, C > A > N
Delinquent ^d	59.5	7.2	65.1	8.4	67.8	7.8	50.4	1.2	O, C > A > N
TRF									
Attention	62.7 ^e	4.9	56.9	4.5	63.0	4.8	50.3	1.0	A, C > O > N
Aggressive ^c	62.8 ^e	6.8	68.0	7.4	74.7	11.0	50.3	0.9	C > O, A > N
Delinquent ^d	56.6 ^e	6.6	63.2	6.1	67.4	10.7	50.7	1.8	C > O, A > N
DBD parents									
Inattention	14.7	5.1	12.0	5.1	14.9	4.6	1.6	2.3	A, O, C > N
H/I ^f	15.0	5.4	12.0	3.9	15.1	4.5	1.5	1.8	A, C > O > N
ODD	8.1	3.9	13.3	3.8	12.7	3.7	1.5	1.9	O, C > A > N
CD	2.0	2.2	3.6	3.7	3.9	2.7	0.2	0.5	O, C > A > N
DBD teacher									
Inattention	14.8	3.8	7.3	3.1	14.8	3.9	0.6	1.2	A, C > O > N
H/I ^f	12.6	5.3	7.3	4.3	13.9	4.6	0.5	1.4	A, C > O > N
ODD	6.8	5.3	12.6	4.3	14.0	4.4	0.1	0.4	O, C > A > N
CD	2.0	2.8	4.5	3.5	5.5	4.8	0.0	0.0	O, C > A > N
IOWA CTRS									
I/O ^g	8.0	2.3	5.3	2.3	8.7	2.6	0.6	1.2	A, C > O > N
O/D ^h	4.4	3.1	7.3	2.0	8.1	3.4	0.1	0.3	O, C > A > N

^a Number of males.

^b Student Newman-Keuls (α set at .05).

^c Aggressive Behavior Scale.

^d Delinquent Behavior Scale.

^e N = 23.

^f Hyperactivity/Impulsivity scale.

^g Inattention/Overactivity scale.

^h Oppositional/Defiant scale.

the trials were stop trials. During practice of the stop task, children were instructed to work as quickly as possible and not to try to respond when they heard the stop signal.

After practice, participants were administered 6 experimental blocks of 32 trials. Event rate was manipulated to examine the effect of behavioural activation state on response execution and inhibition. The stop paradigm was administered using three different event rates. The event rate is the total time of the duration of the fixation point, of the go stimulus, and the interval prior to the fixation point. The duration of the fixation point (200 ms) and the duration of the go stimulus (300 ms) were held constant across all conditions. The ISI varied. In the fast condition it was 1.5 s, in the medium condition it was 3.5 s, and in the slow condition it was 7.5 s. The total duration of the event rates was thus 2, 4, and 8 s. For each event rate condition, 2 blocks of 32 trials were administered. Before each condition, subjects were administered a practice block of 16 trials in order to become accustomed to that particular event rate. The order of event rate conditions was balanced for each of the four groups, using a Latin square design.

Dependent Variables and the Race Model

The main dependent variables reflecting the response execution process are mean reaction time on go trials (MRT), standard deviation of the reaction times on go trials (SD), and the percentage correct responses on go trials. MRT and SD

were calculated across correct responses on go trials. The dependent variable that reflects the latency of the inhibitory process is the stop signal reaction time (SSRT). MRT is a variable that can be observed. SSRT cannot be observed, because the response to a stop signal is a covert one. Therefore, SSRT has to be estimated. This can be done using the race model (Logan & Cowan, 1984). This model assumes that the go process and the stop process are independent. The go stimulus triggers the go process and the stop signal initiates the stop process. The process that finishes first wins the race. If the go process wins the race, the response is executed. If the stop process finishes first, the response is inhibited. The outcome of the race depends on the speed and the variability of the go process, the delay between go stimulus and stop signal, and the speed and the variability of the stop process. In the present study, a tracking algorithm was used to vary dynamically the delay between go stimulus and stop signal, contingent on the subject's performance (Osman, Kornblum, & Meyer, 1986, 1990; Logan, Schachar, & Tannock, 1997). The initial delay between go stimulus and stop signal was 250 ms. If the subject inhibited his/her response, the delay on the next stop trial decreased by 50 ms. If the subject failed to inhibit his/her response, the delay on the next stop trial increased by 50 ms. By using this tracking algorithm, it was established that all subjects inhibited on average 50% of the stop trials. Therefore, on average the go process and the stop process finish at the same time. Thus, the finishing time of the go process can be used to estimate the SSRT. SSRT can be calculated by subtracting the mean delay from the mean go reaction time.

WISC-R

In addition to the stop paradigm, two subtests of the Revised Wechsler Intelligence Scale for Children (WISC-R) were administered to assess intelligence. These subtests were Vocabulary and Block Design. The estimation of the IQ as obtained by these subtests correlates $r = .90$ with the Full Scale IQ (Groth-Marnat, 1997).

Procedure

When subjects entered the experimental room, they were first informed of the purpose of the experiment and of the duration and the nature of the tasks that they were going to perform. Two subtests of the WISC-R were administered in the same order (Vocabulary–Block Design) for all subjects. Following this, children performed the stop paradigm.

During the practice blocks, children received feedback on the speed and response accuracy of their performance, in order to reach an optimal task performance. During practice of the go task, standard instructions were given in which children were directed to respond as quickly and as accurately as possible when they detected an aeroplane. When the stop task was introduced, children were directed to keep on working as quickly as possible, and to inhibit their response when they heard the stop signal. During the experimental blocks, children were encouraged to work as quickly as possible as soon as they slowed down. All children were instructed to press the response buttons by using their index fingers, to keep their fingers on the buttons, and to keep their eyes focused on the screen during the task. The experimenter remained with the child during the task. A second computer screen was placed next to the monitor that the child was looking at, which showed for each trial the reaction time, the type of trial (go or stop), the accuracy of the response, and the number of *SDs* that a reaction time deviated from the MRT as measured in the previous block of trials. The experimenter could follow the performance of the child exactly and give instructions when necessary. The order of the event rate conditions was 2-4-8, 4-8-2, or 8-2-4. Order was balanced over the groups using a Latin square design. After the second condition, a short break was scheduled.

Statistical Analyses

The dependent measures (MRT, *SD*, percentage correct responses on go trials, and SSRT) were analyzed using ANOVAs with group as the between-subjects factor (four levels) and event rate as a repeated measure within-subjects factor (three levels). To model the form of the main effects of event rate, trend analyses were performed. Since the trends were expected to be linear, only linear trends will be reported. To interpret the main effects of group, and group by event rate interactions, each psychopathological group was contrasted against the normal control group, and post hoc Tukey procedures were used. Overall α was set at .05.

In addition to this categorical approach, we used the rating scale data as dimensions and applied a multiple regression analysis to predict the dependent variables using composite measures of AD/HD as well as of ODD/CD. The composite measures were comprised of scale scores on the DBD.

Results

ANOVA

Response execution. The results for measures of response execution are depicted in Table 2 and in Fig. 1 (MRT and variability of reaction times). It was found that the task manipulation was successful: A main effect of event rate was observed for MRT, $F(2, 218) = 46.4$, $p < .001$, for variability of reaction times, $F(2, 218) = 7.0$, $p = .001$, and for response accuracy, $F(2, 218) = 20.4$, $p < .001$ for the percentage correct responses on go trials. Slower event rates yielded slower and more variable

reaction times, and fewer errors: linear trends; $F(1, 109) = 72.8$, $p < .001$ for MRT, $F(1, 109) = 7.8$, $p < .05$ for variability of reaction times, and $F(1, 109) = 22.2$, $p < .001$ for percentage correct responses on go trials.

A main effect of group was found for MRT, $F(3, 109) = 3.2$, $p = .03$, and variability of reaction times, $F(3, 109) = 6.3$, $p = .001$. As hypothesized, contrast tests revealed that the AD/HD group had slower, $F(1, 109) = 5.6$, $p = .02$, and more variable reaction times, $F(1, 109) = 13.4$, $p = .000$, than healthy controls. Similarly, the comorbid group showed slower, $F(1, 109) = 7.7$, $p = .006$, and more variable reaction times, $F(1, 109) = 9.7$, $p = .002$, than normal controls. Children with ODD exhibited greater variability of reaction times compared to normal control children, $F(1, 109) = 9.2$, $p = .003$. No significant group differences were detected for response accuracy, $F(3, 109) = 0.89$, n.s.

Group by event rate interactions were found for the speed, $F(6, 218) = 2.2$, $p = .046$, and the variability, $F(6, 218) = 2.5$, $p = .02$, of the response execution process. As would be predicted by the activation hypothesis, contrast tests indicated that the interaction effect for MRT was due to the AD/HD–normal control comparison, $F(2, 218) = 5.2$, $p = .006$; linear trend, $F(1, 109) = 7.6$, $p < .05$. A post hoc Tukey procedure revealed that the difference for MRT between the AD/HD and the normal control group was the most pronounced in the slowest event rate condition, $F(1, 63) = 4.2$, $p = .04$ for the fast condition, $F(1, 63) = 4.8$, $p = .03$ for the medium condition, $F(1, 63) = 9.9$, $p = .003$ for the slow condition.

The interaction effect for the variability of reaction times was due to the AD/HD–normal control comparison, $F(2, 218) = 3.4$, $p = .036$; linear trend, $F(1, 109) = 5.6$, $p < .05$, and due to the ODD–normal control comparison, $F(2, 218) = 4.0$, $p = .019$; linear trend, $F(1, 109) = 6.6$, $p = .01$. As would be predicted by the activation hypothesis, a post hoc contrast procedure revealed that the difference in the variability of reaction times between AD/HD and normal controls increased, when the event rate became slower, $F(1, 63) = 8.6$, $p = .005$ fast event rate; $F(1, 63) = 10.4$, $p = .002$ medium event rate; $F(1, 63) = 16.7$, $p < .001$ slow event rate. The ODD group had a larger RT variance than normal controls especially in the slowest event rate condition, $F(1, 60) = 5.9$, $p = .02$ fast event rate; $F(1, 60) = 6.2$, $p = .02$ medium event rate; $F(1, 60) = 13.8$, $p < .001$ slow event rate.

Response inhibition. The tracking algorithm was successful: The mean percentage of inhibition across groups and event rate conditions was 50.4. The percentage of inhibition that maximally deviated from 50% was observed in the ODD group in the fast event rate condition (53.9%; see Table 2). For the percentage of inhibition, no main effect of event rate was detected, $F(2, 218) = 0.03$, n.s. No group differences were noted for percentage of inhibited responses, $F(3, 109) = 1.3$, n.s. This result was expected, since the tracking algorithm was used, which ensures that each child in each condition produces approximately 50% inhibition on the stop trials.

In order to estimate SSRT using the subtraction method suggested by Logan et al. (1997), the percentage of inhibition has to be 50% for each individual. If the percentage of inhibited responses deviates from 50%, it cannot be assumed that the go process and the stop process finish, on average, at the same time. Since the percentage of inhibition differed from 50% in some

Table 2
Group Means and Standard Deviations for Stop Paradigm Measures in the Three Event Rate Conditions

Measure	Group							
	AD/HD		ODD		Comorbid		NC	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
MRT								
Event rate 2	427.1	72.6	411.9	79.4	440.6	100.2	392.4	61.6
Event rate 4	444.8	84.4	436.1	83.8	464.6	113.0	400.6	75.4
Event rate 8	495.2	114.6	466.0	95.5	481.7	118.7	419.7	78.4
Variability of RT								
Event rate 2	103.8	34.1	99.4	28.6	100.1	29.8	84.2	20.1
Event rate 4	101.6	34.9	97.4	37.4	105.0	41.4	77.9	24.3
Event rate 8	118.2	39.9	116.2	40.3	104.1	36.3	80.8	32.8
Percentage correct on go trials								
Event rate 2	89.1	7.1	88.4	10.7	91.1	9.2	91.1	9.0
Event rate 4	93.8	6.2	93.3	9.0	92.6	9.5	94.7	5.7
Event rate 8	93.9	5.2	91.1	8.9	94.5	5.1	95.2	4.4
Percentage inhibition								
Event rate 2	49.5	10.6	53.9	7.8	48.1	9.5	49.4	8.5
Event rate 4	50.5	7.1	52.1	8.2	47.9	7.8	51.2	7.8
Event rate 8	51.0	6.3	49.1	11.7	51.6	9.1	50.3	7.6
SSRT (subtraction)^a								
Event rate 2	265.1	94.0	231.5	121.4	228.5	103.7	227.8	88.3
Event rate 4	233.3	128.4	235.8	118.5	209.8	119.9	215.6	86.6
Event rate 8	300.3	135.7	296.1	130.3	248.6	135.2	238.1	92.9
SSRT (integration)^a								
Event rate 2	258.5	113.9	215.5	124.0	230.0	113.1	226.1	97.4
Event rate 4	218.6	139.9	222.9	126.7	206.5	123.2	209.3	91.3
Event rate 8	275.0	131.4	283.7	151.0	234.2	145.0	232.4	96.9

^a See text.

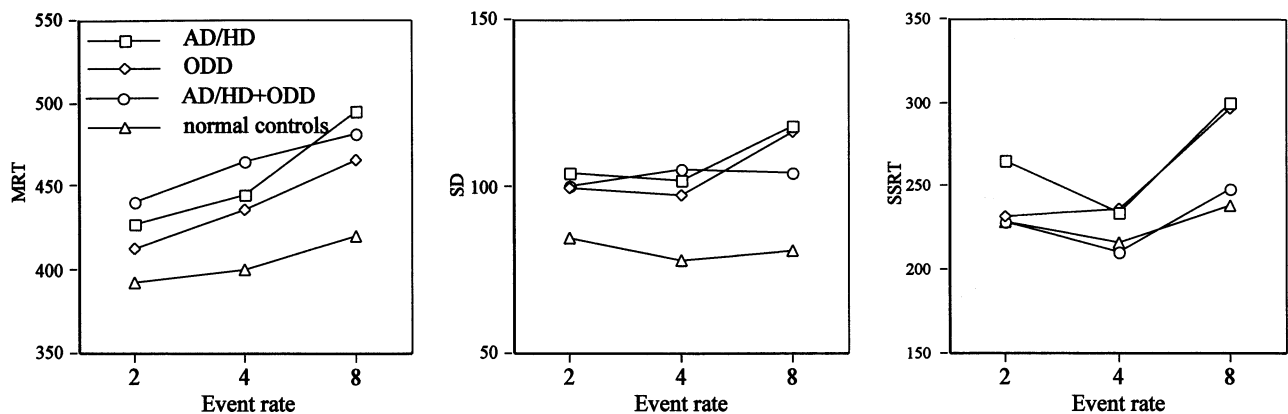


Figure 1. The stop paradigm variables as a function of the event rate condition for AD/HD, for ODD, for comorbid AD/HD+ODD, and for normal controls. Mean reaction time (MRT) is depicted in the left panel, standard deviation of reaction times (SD) is depicted in the middle panel, and stop signal reaction time (SSRT) is shown in the right panel.

children, the SSRT was also calculated using the so-called integration method (Logan, 1994). Using this method, the percentage of inhibition does not need to be equal for all subjects. SSRT is calculated for each individual's percentage of inhibition (see Logan, 1994).

A main effect of event rate was found for SSRT as calculated by the subtraction method, $F(2, 218) = 12.8$, $p < .001$ (see Fig. 1). Across groups, the latency of the stop process increased as the event rate became slower: linear trend, $F(1, 109) = 11.9$, $p = .001$. Contrary to predictions, no significant group differences were found for SSRT, $F(3, 109) = 1.1$, n.s., nor was there an interaction observed between groups and event rate,

$F(6, 218) = 1.1$, n.s. This means that all the groups showed comparable latencies for their stop process in all event rate conditions (see Fig. 1). A contrast test was used to compare the AD/HD group with the normal controls. The difference for SSRT between AD/HD and healthy controls did not reach significance, $F(1, 109) = 2.6$, $p = .11$; effect size $d = 0.39$.

The same analyses were applied to SSRT as calculated by the integration method. This analysis yielded similar results: a main effect of event rate, $F(2, 218) = 8.4$, $p < .001$, no group differences, $F(3, 109) = 0.5$, n.s.; effect size $d = 0.26$, and no group by event rate interaction, $F(6, 218) = 1.2$, n.s.

Table 3

Multiple Regression Analyses with the Predictors Age Entered at Step 1, AD/HD Entered at Step 2, and ODD/CD Entered at Step 3 (Model 1), and ODD/CD Entered at Step 2, and AD/HD Entered at Step 3 (Model 2)

Predictor	Dependent measures											
	MRT			SD			SSRT			% correct		
	β	R ²	ΔR^2	β	R ²	ΔR^2	β	R ²	ΔR^2	β	R ²	ΔR^2
Fast event rate												
Step 1, age	-.29	.08	.08*	-.25	.06	.06*	-.34	.12	.12**	.34	.12	.12**
Step 2, AD/HD	-.31	.18	.10*	.34	.17	.11**	.09	.13	.01	-.05	.12	.00
Step 3, ODD/CD	.16	.19	.01	.22	.19	.02	-.15	.14	.01	-.06	.12	.00
Step 2, ODD/CD	.31	.17	.09*	.35	.18	.12**	.00	.12	.00	-.06	.12	.00
Step 3, AD/HD	.19	.19	.02	.18	.19	.01	.20	.14	.02	.00	.12	.00
Medium event rate												
Step 1, age	-.27	.08	.08*	-.28	.08	.08*	-.22	.05	.05	.14	.02	.02
Step 2, AD/HD	.36	.20	.13**	.40	.24	.16**	.04	.05	.00	-.09	.03	.01
Step 3, ODD/CD	.22	.22	.02	.23	.26	.02	.05	.05	.00	-.03	.03	.00
Step 2, ODD/CD	.37	.21	.13**	.41	.24	.16**	.05	.05	.00	-.08	.02	.01
Step 3, AD/HD	.19	.22	.02	.23	.26	.02	.00	.05	.00	-.06	.03	.00
Slow event rate												
Step 1, age	-.36	.13	.13**	-.35	.12	.12**	-.19	.04	.04	.12	.01	.01
Step 2, AD/HD	.37	.26	.13**	.43	.31	.19**	.13	.05	.02	-.12	.03	.01
Step 3, ODD/CD	.10	.27	.00	.15	.31	.01	-.02	.05	.00	-.14	.04	.01
Step 2, ODD/CD	.33	.23	.10**	.40	.27	.15**	.09	.05	.01	-.15	.04	.02
Step 3, AD/HD	.29	.27	.04	.33	.31	.05*	.14	.05	.01	-.02	.04	.00

* $p < .05$; ** $p < .001$.

Multiple Regression Analyses

In this section, AD/HD and ODD/CD symptoms are considered from a dimensional rather than a categorical approach (Nigg, Hinshaw, Carte, & Treuting, 1998). It was expected that regression models would provide converging evidence with the previously described results using ANOVA.

Two stepwise univariate regression models were run for each event rate condition to investigate the relative contribution of the predictors (see below) to the proportion explained variance of the dependent variables. The predictors entered in the first model were age (step 1), a composite measure of the DBD scales measuring AD/HD (step 2), and a composite measure of the DBD ODD and CD scales (step 3). To control for a possible confounding effect of age on the predictors AD/HD and ODD/CD, age was entered at step 1. Since AD/HD and ODD/CD symptoms are highly correlated ($r = .76$), the ODD/CD predictor cannot account for much variance in the dependent variables because it is entered at the last step. Therefore, in the second model, age was entered at step 1 again, ODD/CD symptoms were entered at step 2, and AD/HD symptoms were entered at step 3. The composite AD/HD score was created by calculating the mean of the parent DBD Inattention and Hyperactivity/Impulsivity scales, and the teacher DBD Inattention and Hyperactivity/Impulsivity scales. The composite ODD/CD score was created by calculating the mean of the parent DBD ODD and CD scales and the teacher DBD ODD and CD scales.

The regression analyses showed that the power of AD/HD as a predictor of response execution measures increased with the event rate. For MRT, the proportion of variance explained by AD/HD symptoms (step 2) was 10% in the fast event rate condition ($p = .002$), 13% in the medium event rate condition ($p < .001$), and 13% in the slow event rate condition ($p < .001$). For variability of reaction times, AD/HD symptoms explained 11%

variance in the fast condition ($p < .001$), 16% in the medium condition ($p < .001$), and 19% in the slow condition ($p < .001$). However, no relevant proportion of the variance in the percentage correct responses on go trials could be accounted for by AD/HD or ODD/CD. Contrary to the predictions, no relevant proportion of the variance of SSRT could be accounted for by AD/HD or ODD/CD symptoms in the three event rate conditions (see Table 3).

Thus, the slower the event rate, the larger the proportion of variance in MRT and SD that is accounted for by AD/HD symptoms. This finding is in agreement with the results of the ANOVA: the AD/HD group showed slower and more variable reaction times compared to normal controls, especially in the slow event rate condition.

When the order of entry of the predictors AD/HD and ODD/CD in the model was reversed, ODD/CD symptoms accounted for about the same proportion of variance in MRT and SD as did AD/HD symptoms in the procedure described above (see Table 3). AD/HD and ODD/CD symptoms are highly correlated, and therefore most of the variance that could have been accounted for by ODD/CD had already been accounted for by AD/HD. Similarly, when ODD/CD symptoms are entered first, AD/HD symptoms cannot account for additional variance, thus indicating that both AD/HD and ODD/CD have comparable predictive power for MRT and variability of reaction times in the stop paradigm.

Discussion

This study aimed at replicating previous findings of slow and variable response execution processes as well as slow response inhibition processes in children with AD/HD. Furthermore, the specificity of deficient response execution and inhibition was examined: the question was asked whether slow and variable response

execution and slow inhibition was uniquely related to children with AD/HD, or also evident in children with ODD and children with comorbid AD/HD+ODD. Finally, this study explored the possibility that a non-optimal behavioural activation level is the explanatory factor for deficits in both the go and the stop processes in AD/HD. The main findings were that (a) AD/HD and comorbid AD/HD+ODD is associated with slow response execution, and AD/HD, ODD, and AD/HD+ODD is associated with variable response execution, (b) neither AD/HD nor ODD, nor comorbid AD/HD+ODD, is associated with slow inhibitory processing as measured here, and (c) in both AD/HD and ODD the deficiency in response execution increases in the condition with a slow event rate, compared to medium and fast event rate conditions. Deficient response execution of the comorbid AD/HD+ODD group, however, does not further deteriorate in the slow event rate condition.

It was shown that the event rate manipulation was effective, i.e. all subjects showed an increase on mean reaction time, percentage correct responses to go trials, and SSRT, with an increase in event rate. The fact that event rate influenced SSRT means that the speed of the inhibition process in children is *not* constant, but is dependent on a primary task characteristic such as event rate. Although research in adults has shown that SSRT is about 200 ms, independent of the difficulty of the go task, the current study showed that in children aged 6–12 years, SSRT varies with the event rate condition.

As hypothesized, we replicated the findings of slow response execution processes and high variability in reaction times in AD/HD (e.g., Oosterlaan & Sergeant, 1998a, b; Pliszka et al., 1997; Schachar et al., 1995; see for review Oosterlaan et al., 1998; Sergeant et al., 1999).

Second, we found that slow response execution was not specifically related to AD/HD: it was also observed in the comorbid AD/HD+ODD group. However, the ODD group was not significantly slower than the normal control group. This may suggest that the slowness in responding in the comorbid group is due to the symptoms of AD/HD. This result is in agreement with a finding reported by Schachar and Tannock (1995), who observed slow responding in AD/HD and AD/HD+CD but not in pure CD. It can be interpreted as support for the distinctiveness of AD/HD and ODD. In addition, Oosterlaan and Sergeant (1996) found slow reaction times in AD/HD but not in an aggressive group, which supports the distinctiveness of these two groups. However, in two other studies, slow reaction times were found in AD/HD as well as in an aggressive group (Oosterlaan & Sergeant, 1998a, b). Variability in reaction times was observed in all the disruptive groups. This shows that variable response execution is not specific to AD/HD, but also evident in ODD and comorbid AD/HD+ODD. This finding would support the hypothesis that AD/HD, ODD, and comorbid AD/HD+ODD reflect a single underlying disorder (Schachar & Tannock, 1995). The finding of high variability in reaction times in all disruptive groups is in agreement with findings of previous studies that demonstrated a high variability in response times in an AD/HD group as well as in an aggressive group (Oosterlaan & Sergeant, 1996, 1998a, b).

In the current study, previous findings of slow response inhibition in AD/HD were not replicated (e.g., Oosterlaan & Sergeant, 1998a, b; Pliszka et al., 1997;

Schachar et al., 1995). Although the AD/HD group showed slower SSRTs than normal controls, this difference was not significant. The inhibition deficit in AD/HD children found in previous studies seems to be a robust effect with a medium effect size ($d = 0.64$) (Oosterlaan et al., 1998), thus the present finding requires explanation.

A factor that is possibly responsible for this failure to replicate is the type of stop paradigm used here. In the current study, a stop paradigm with a tracking algorithm was used, which dynamically varied the delay between go and stop signal, contingent to the child's inhibitory performance. This results in an inhibition rate of .5 in all children. In previous studies that reported group differences on the speed of the inhibitory process, a version of the stop paradigm was used with a number of fixed delays (usually four) between the stop signal and the expected response. This results in four different inhibition rates and these rates can be different for subjects. In this study the stop paradigm with tracking algorithm was employed, since it has been demonstrated that this procedure has several methodological and practical advantages compared to the fixed delay procedure (Band, 1997). First, in contrast to the fixed delay paradigm, the tracking procedure does not depend on the assumption that the inhibition process has a constant latency. Second, it has been demonstrated that SSRT is most reliably estimated around a central delay where the inhibition rate is .5. Third, the tracking algorithm corrects for the tendency to wait for the stop signal. Fourth, it has the advantage that SSRT can be calculated reliably using fewer stop trials than in the fixed delay version of the paradigm. This is a crucial point, especially in a study with a repeated measures design.

However, it might be possible that the stop paradigm with the tracking algorithm in one way or another does not measure the same SSRT as the paradigm with fixed delays. It is noted here that the SSRT as obtained by the current task and procedure is in fact more reliable than the SSRT as measured in previous stop paradigm research. In a pilot study that was conducted to compare the two versions of the paradigm, the SSRTs obtained by the tracking paradigm and the fixed delay paradigm showed a robust correlation ($r = .80$, $p = .02$) (data available from the first author). This argues for convergence between the two paradigms in measuring the same inhibitory process.

A second possible explanation for our failure to replicate poor response inhibition in AD/HD is that there was not enough power to detect group differences. In a meta-analysis, Oosterlaan et al. (1998) reported a medium effect size of SSRT differences between AD/HD and normal controls ($d = 0.64$). To detect this effect with a power of .80, 22 subjects were required for each group. This requirement was met here and thus the groups were sufficiently large to measure a difference between AD/HD and normal controls for SSRT. Furthermore, the more stop trials that are administered, the more reliable the estimated SSRT. In this study across the three event rate conditions 48 stop trials were used to study group differences, which exceeds the number of 40 stop trials suggested by Band (1997).

An alternative argument to explain our findings could be that the pathological groups were not severely impaired. This argument, however, seems unlikely for a number of reasons. First, the groups were clearly different on the relevant clinical scales. Second, the inclusion criterion of pervasiveness was applied to all pathological

groups. Third, samples were drawn from children who attended special school services for children with extreme behavioural problems (2.2% of Dutch children in the age range 6 to 12 years attend these school services). Fourth, 20 children were excluded from participating in the experiment because of medication use—pipamperon (Dipiperon®) or clonidine (Dixarit®)—and 11 children who participated used methylphenidate (Ritalin®). Fifth and finally, the disruptive groups differed from the normal controls on the go process. Thus, it seems unlikely that the pathological groups were not significantly impaired.

This is not the first study that has failed to detect an inhibition problem in children with AD/HD. Jennings, Van der Molen, Pelham, Debski, and Hoza (1997) did not find a group difference for SSRT when they compared an AD/HD group with normal controls. It was only when they compared the young AD/HD children with comorbid ODD or CD with the normal control group that a difference emerged. Daugherty, Quay, and Ramos (1993) did not detect group differences for SSRT. Schachar et al. (1995) did not find a group difference for SSRT when they compared their AD/HD group with the normal control group. It was only when they compared a subsample of pervasive AD/HD children with the normal control group that a group difference emerged. However, in a meta-analysis which was conducted on several stop paradigm studies, including the two that did not find group differences, it was shown that the effect size of SSRT differences between AD/HD children and normal controls was a medium one; $d = 0.64$ (Oosterlaan et al., 1998). Therefore, we cannot conclude from only the current study that the previous findings of SSRT differences between AD/HD children and normal controls is not robust.

Since the AD/HD group did not show slow response inhibition, the specificity issue of deficient response inhibition cannot be discussed here. However, since the ODD group and the comorbid AD/HD+ODD group did not show slower SSRTs compared to normal controls, the present finding suggests that children with disruptive behavior disorders do not have an inhibition problem as measured in this study. This finding would support the hypothesis that AD/HD, ODD, and comorbid AD/HD+ODD reflect one underlying disorder (Schachar & Tannock, 1995).

The third aim of the current study was to investigate whether children with AD/HD show poor task performance in particular with a low behavioural activation level. First, the relationship between behavioural activation level and response execution is discussed, followed by a discussion of the inhibition findings for the different activation levels.

AD/HD children showed disproportionate slower reaction times and higher variability in reaction times than normal control children in the slow event rate (low activation) condition. This supports the activation hypothesis (Sergeant et al., 1999). When a low activation level is induced, children with AD/HD have more problems than normal control children to maintain the performance they showed with a high or a medium event rate. These results support the hypothesis of a suboptimal activation state in children with AD/HD rather than the hypothesis of the inability to adjust the activation state to both slow and fast event rate conditions. As reviewed in the Introduction, most previous studies showed that response execution processes in AD/HD children are

impaired especially in slow event rate conditions. These results have been variously interpreted, dependent on the nature of the task and the theoretical framework that was used: as a deficit in sustained attention (Chee et al., 1989; Conte et al., 1986; Zahn, Kruesi, & Rapoport, 1991), as a reduced arousal level underlying sensitivity to situational context in hyperactivity (Conte et al., 1986; Zentall & Zentall, 1983), as support for the activation hypothesis (Van der Meere, Shalev, et al., 1995), as an unusual sensitivity to delay (Sonuga-Barke & Taylor, 1992), as sensitivity to temporal structure of the task, and increased difficulty in motor preparation (Van der Meere et al., 1992), and as a state regulation deficiency (Van der Meere, Stemerding, et al., 1995; see also Douglas, 1999). The current findings could be interpreted in terms of under-arousal, under-activation, poor motor preparation, sensitivity to delay, or poor self-regulation.

The effects of activation level on parameters of the response execution process in AD/HD were also observed in children with ODD, suggesting that activation plays a major role in task performance not only of children with AD/HD, but also of those with ODD. The comorbid group, however, showed a deficit in response execution that was constant over the three event rate conditions. This unique pattern of responding of the comorbid group would support the hypothesis that the comorbid condition is a distinct condition which is different from both pure disorders (Schachar & Tannock, 1995). In a previous study (Chee et al., 1989), the effects of different event rates were studied comparing AD/HD with CD, comorbid AD/HD+CD, and normal controls. A continuous performance test was used, and the presentation rate uniquely affected children with AD/HD. Our finding of the comorbid group is in agreement with the findings of Chee et al. However, our findings for the ODD group contrast with the results obtained by Chee et al. Two explanations for this discrepancy in findings may be offered: (a) a different paradigm was studied by Chee et al., and (b) the groups were different (CD in Chee et al.'s study versus ODD in our study). In another study, Zahn et al. (1991) compared a group of boys with disruptive behaviour disorders with normal control boys. In contrast to our findings, it was shown that the boys with disruptive behaviour disorders (who met criteria for AD/HD and ODD/CD) had disproportionately slow reaction times compared to normal controls on trials with longer preparatory intervals, suggesting that children with comorbid AD/HD+ODD/CD are sensitive to slow event rates. The results of these studies are not in agreement as to whether sensitivity to activation level is specifically related to AD/HD.

Although the difference between the AD/HD and the normal control group for SSRT was most pronounced in the slow event rate condition, the group by event rate interaction did not reach statistical significance. Since this is the first study in AD/HD in which the stop paradigm was studied under different event rate conditions, full comparison with previous results is not possible. However, Van der Meere, Stemerding et al. (1995) used a go/no-go task in different event rate conditions to compare an AD/HD group with normal control children on inhibitory control. Van der Meere et al. showed that children with AD/HD inhibited less than normal controls in both the fast and the slow condition. These findings were interpreted in terms of activation: children with AD/HD are easily underactivated (in the slow event rate condition) and easily overactivated (in the fast event rate

condition). The dependence of response inhibition on event rate in the AD/HD group as shown by Van der Meere et al. was not found in the current study. An explanation for the discrepant findings could be the difference in tasks used between that and the current study.

Taken together, the results of the current study show that children with AD/HD and comorbid AD/HD+ODD show slow execution processes. Slow response execution seems to be due to symptoms of AD/HD. The high variability in reaction times is not specific to a single group, since it is observed in AD/HD, ODD, and AD/HD+ODD. Furthermore, this study failed to replicate previous findings of slow response inhibition in children with AD/HD and/or ODD symptoms. Future research should show whether the nature of the stop paradigm has been crucial for this failure to replicate poor response inhibition in AD/HD. Response execution in children with AD/HD and in children with ODD is sensitive to event rate. This finding can be interpreted as support for the activation hypothesis in AD/HD (Sergeant et al., 1999). However, response inhibition was not dependent on event rate. On the one hand, it could be possible that only response execution processes and not response inhibition processes are sensitive to the level of activation in AD/HD. On the other hand, if the primary problem in children with AD/HD were a nonoptimal activation state, then it would be expected that all dependent measures of a task are dependent on the activation state. Therefore, from this study it cannot be concluded that a suboptimal activation state is the core problem in children with AD/HD. However, behavioural activation state does seem to play an important role in response execution in AD/HD. In the current study, we focused on the role of activation in response execution and response inhibition. It was not the intention of this study to explain all the behavioural symptoms in children with AD/HD in terms of only suboptimal activation. From the results of this study, it may be suggested that a low behavioural activation state may be an underlying problem for deficient task performance which is not specifically related to AD/HD, but may also play a role in deficient task performance in ODD but, interestingly, not in children comorbid for AD/HD+ODD. However, alternative explanations for the event rate findings, such as delay aversion (Sonuga-Barke & Taylor, 1992), or a self-regulation deficit (Douglas, 1999), are available. Concerning the distinctiveness of the disorders, the findings of the speed of the response execution process suggest that AD/HD and ODD are distinctive disorders. The finding of the effect of activation level on response execution suggests that comorbid AD/HD+ODD is a distinct condition. However, on other variables (variability in responding and speed of inhibiting) the disorders show a similar pattern of performance. It remains for future studies to attempt to demonstrate the neuropsychological distinctiveness of AD/HD, ODD/CD and the comorbid condition AD/HD+ODD in order to revise current models of these disorders (Barkley, 1997; Douglas, 1999; Sergeant, Oosterlaan & Van der Meere, 1999; Sonuga-Barke & Taylor, 1992).

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