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Response Inhibition in AD/HD, CD, Comorbid AD/HD + CD, Anxious, and Control Children: A Meta-analysis of Studies with the Stop Task

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The aim of this study was to investigate whether impaired response inhibition is uniquely related to AD/HD or whether deficits in response inhibition are also evident in other psychopathological disorders. Furthermore, the suggestion was examined that anxiety disorders are associated with abnormally high levels of response inhibition. This paper presents the results of a meta-analysis of eight studies in which response inhibition was assessed with the so-called stop task in five groups of children: children with attention deficit/hyperactivity disorder (AD/HD), children with conduct disorder (CD), children with AD/HD + CD, children with anxiety disorders, and control children. A total of 456 children participated in the 8 studies. All children were in the age range 6–12 years. Consistent and robust evidence was found for a response inhibition deficit in AD/HD. However, response inhibition deficits did not distinguish children with AD/HD from children with CD, nor from children with comorbid AD/HD + CD. Contrary to predictions, anxious children did not demonstrate enhanced levels of response inhibition.

Keywords: Conduct disorder; anxiety disorder, attention deficit hyperactivity disorder, information processing, response inhibition, meta-analysis.

Abbreviations: AD/HD: attention deficit/hyperactivity disorder; BAS: Behavioural Activation System; BIS: Behavioural Inhibition System; CD: conduct disorder; IF: inhibition function; LD: learning disorder; MRT: mean reaction time; NAS: Nonspecific Arousal System; ODD: oppositional defiant disorder; SSRT: stop signal reaction time; ZRFT-slope: slope of the IF plotted as a function of ZRFT (z score of the relative finishing time).

Introduction

Executive functions are those mental control processes that enable self-control and goal-directed behaviour (Barkley, 1996; Denckla, 1994; Lezak, 1983; Pennington & Ozonoff, 1996; Torgesen, 1994). These functions are mediated by the frontal lobes, in particular by the prefrontal cortex and its extended networks (Lezak, 1983; Pennington & Ozonoff, 1996; Torgesen, 1994). A fundamental component of the executive functions is the ability to inhibit inappropriate responding. Barkley (1997) distinguishes three forms of response inhibition: (1) inhibiting prepotent responses, (2) stopping an ongoing response, and (3) inhibiting interference. The ultimate goal of response inhibition is to enhance adaptive functioning (Halperin, McKay, Matier, & Sharma, 1994).

Abnormalities in response inhibition are a central component in the description and explanation of child psychopathological disorders and, in particular, of attention deficit/hyperactivity disorder (AD/HD). Barkley (1994, 1997), for example, argued that AD/HD involves a pervasive deficit in all forms of response inhibition. According to Barkley, this deficit leads to secondary impairments in four executive functions that depend on efficient response inhibition for their execution: working memory, internalisation of speech, self-regulation of affect-motivation-arousal, and reconstitution. As a result,

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children with AD/HD present with a disorder characterised by impaired executive functions, which in turn lead to disturbances in self-control and goal-directed behaviour. These disturbances ultimately result in the behaviours observed in AD/HD.

Quay (1988a, b, 1997) used Gray's (1987) neuropsychological model of brain function to explain the origin of poor response inhibition in AD/HD. In Gray's model, behaviour is explained in terms of the activity of two opposing brain systems: the Behavioural Inhibition System (BIS), which is sensitive to signals of punishment, and the Behavioural Activation System (BAS), which is sensitive to signals of reward. The BIS serves to inhibit behaviour, whereas the BAS controls the initiation of behaviour. Quay proposed that children with AD/HD have a persistently underactive BIS, which results in response inhibition deficits.

A number of other theoretical accounts have emerged in which the inattentive, hyperactive, and impulsive behaviour of children with AD/HD has been suggested to arise from a deficit in response inhibition (Douglas, 1988, 1989; Milich, Hartung, Martin, & Haigler, 1994; Newman & Wallace, 1993; Pennington & Ozonoff, 1996; Wender, 1972). Importantly, Barkley (1994, 1997) has argued that response inhibition deficits are *unique* to AD/HD. This suggestion is also made implicitly in other theoretical accounts of AD/HD (Douglas, 1988, 1989; Pennington & Ozonoff, 1996; Wender, 1972).

The poor performance of AD/HD children on a variety of measures has been taken to support the response inhibition deficit hypothesis for AD/HD. These measures include the Matching Familiar Figures Test (Campbell, Douglas, & Morgenstern, 1971; DuPaul, Anastopoulos, Shelton, Guevremont, & Metevia, 1992; Weyandt & Grant, 1994), the Continuous Performance Task (see for reviews, Barkley, Grodzinsky, & DuPaul, 1992; Corkum & Siegel, 1993; see also Halperin et al., 1994), the Go/No-go Task (Iaboni, Douglas, & Baker, 1995; Milich et al., 1994; Shue & Douglas, 1992), delayed response tasks (Daugherty & Quay, 1991; McClure & Gordon, 1984; Schweitzer & Sulzer-Azaroff, 1995; Solanto, 1990), the Wisconsin Card Sorting Test (see for review, Barkley et al., 1992), and many others.

These measures, however, have been criticised for their poor construct validity and have been considered as too global (Halperin et al., 1994). Performance on these measures may be influenced by many factors other than response inhibition, such as age and IQ (Milich et al., 1994; Schachar & Logan, 1990). The major criticism levelled at these tasks is their failure to clarify the mechanisms underlying impaired response inhibition (Milich et al., 1994; Schachar & Logan, 1990). These criticisms do not apply to the stop task (Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984). This task is purported to measure the ability to interrupt an ongoing response. Several studies have supported the reliability and validity of the stop task as a measure of response inhibition (Kindlon, Mezzacappa, & Earls, 1995; Tannock, Schachar, Carr, Chajczyk, & Logan, 1989).

The stop task is based on a well-established theory of response inhibition, known as the race model (see for review, Logan, 1994; Logan & Cowan, 1984). According to this model, response inhibition depends on a race between, on the one hand, the process underlying response execution, and on the other hand, the inhibitory process. This inhibitory process is triggered by information that tells the subject to discontinue or change a current course of action, such as an error during performance. The process that finishes first determines performance. If the inhibitory process runs to completion first, the response is inhibited. In the opposite case, the ongoing action is completed. In contrast to other measures of response inhibition, the stop task enables one to investigate whether poor response inhibition is due to a deficit in the inhibitory process.

Briefly, the stop task requires fast and accurate execution of a reaction time task, denoted as the primary task. Occasionally, a stop signal is presented, which requires the child to inhibit the response to the primary task. Stop signals are presented at different intervals before the subject's expected response. The shorter the interval, the more difficult it becomes to inhibit the response. Usually, the intervals are chosen such that the shortest interval will yield a probability of inhibition close to 0, whereas the longest interval will produce a probability of inhibition close to 1. A short description of the stop task, the race model, and the main dependent measures in the stop task is provided in the Appendix.

In the last few years, the stop task has been utilised in a series of studies to investigate deficits in response inhibition in children with AD/HD and other psychopathology (Aman, Roberts, & Pennington, in press; Daugherty, Quay, & Ramos, 1993; Jennings, Van der Molen, Pelham, Brock, & Hoza, 1997; Oosterlaan & Sergeant, 1996; Pliszka & Borcherding, 1995; Schachar & Logan, 1990; Schachar & Tannock, 1995; Schachar, Tannock, Marriott, & Logan, 1995). This body of research is reviewed in the current study. The principal aim of this study was to investigate whether data gathered with the stop task supports the response inhibition deficit hypothesis for AD/HD. Furthermore, we investigated whether impaired response inhibition is uniquely related to AD/HD or whether deficits in response inhibition are also evident in other psychopathological disorders. Finally, we examined the suggestion that anxiety disorders are associated with abnormally high levels of response inhibition (Quay, 1988a, b).

Meta-analytic procedures were used to aggregate findings across different studies. One of the advantages of meta-analytic procedures is the greater power to detect group differences (Rosenthal, 1991). This is particularly important given the small groups employed in most studies. Consequently, the failure to detect group differences in these studies might reflect inadequate power rather than the absence of group differences.

The existing literature enabled us to evaluate the following five group comparisons through meta-analysis. For each of these comparisons, hypotheses were derived from current theories of child psychopathology.

AD/HD–controls (*1*). This comparison addresses whether data derived from the stop task lend support for theoretical accounts of AD/HD suggesting that this disorder is characterised by poor response inhibition (Barkley, 1994, 1997; Douglas, 1988, 1989; Newman & Wallace, 1993; Pennington & Ozonoff, 1996; Quay, 1988a, b, 1997; Wender, 1972). Thus, it was predicted

that AD/HD children would demonstrate poor response inhibition compared with controls.

CD–controls (*2*) *and AD/HD–CD* (*3*). As indicated earlier, it has been suggested that deficits in response inhibition are confined to children with AD/HD (Barkley, 1994, 1997; Douglas, 1988, 1989; Pennington & Ozonoff, 1996; Wender, 1972). However, consonant with AD/HD, poor response inhibition has also been described as a core phenomenon in conduct disorder (CD) (Farrington, 1993; Milich et al., 1994; Newman & Wallace, 1993; Quay, 1988a, b, 1993). Thus, response inhibition deficits may not be specific to AD/HD, i.e. impaired response inhibition may not be the *hallmark* of AD/HD.

According to Quay (1988a, b, 1993, 1997), both AD/HD and CD are associated with response inhibition deficits. However, the dysfunction underlying poor response inhibition differs for the two disorders. Quay (1988a,b, 1993) has suggested that CD reflects an overactive BAS that dominates the BIS. According to this view, the excessive BAS activity causes a strong tendency to respond and interferes with the capability for response inhibition. By contrast, poor response inhibition in AD/HD results from an underactive BIS (Quay, 1988a, b, 1997).

Quay referred in particular to children with undersocialised aggressive conduct disorder (American Psychiatric Association, 1987), a group of children currently alluded to as CD–childhood-onset type (American Psychiatric Association, 1994). This type of CD generally emerges prior to age 10 years. Given the age range of children with CD in the present meta-analysis (6–12 years old), Quay's hypothesis concerning CD is particularly relevant.

The existing data derived with the stop task enabled us to investigate both differences between CD and control children and between AD/HD and CD children. The aim was to determine whether response inhibition deficits are unique to children with AD/HD or whether these deficits are also evident in children with CD. The stop task would be particularly useful in clarifying the nature of AD/HD, if it is able to distinguish AD/HD children from children with CD.

AD/HD+CD–AD/HD (*4*). This comparison was designed to investigate whether children with comorbid AD/HD+CD differ from children with AD/HD only. It has been suggested that the combination of AD/HD and CD represents a distinct nosological entity (Biederman, Newcorn, & Sprich, 1991; Schachar & Tannock, 1995). Following Quay's (1988a, b, 1993, 1997) model, one might speculate that the AD/HD+CD group shows greater deficiencies in the capability for response inhibition due to a combination of underactivity in the BIS (resulting in poor response inhibition) and overactivity in the BAS (which may cause an exaggerated tendency to respond). Thus, response inhibition deficits in children with AD/HD+CD might be more pronounced than in children with AD/HD alone.

Anxiety disorder–controls (*5*). In contrast to AD/HD and CD, anxiety disorders have been associated with abnormally high levels of response inhibition. It has been suggested by Quay (1988a, b) that an overactive BIS underlies anxiety disorders. Furthermore, several studies have shown that a strong tendency for response inhibition in children is a powerful predictor of later anxiety disorders (see for review, Biederman, Rosenbaum, Chaloff, & Kagan, 1995). Thus, children with anxiety disorders were predicted to demonstrate enhanced response inhibition in comparison with controls.

Method

Description of the Studies

This review covers studies conducted between 1990 and 1997. Eight independent published and unpublished studies were identified that met the following inclusion criteria: (1) the study contained one or more of the psychopathological groups of interest, and (2) the study included a comparison group of control children. All studies focused on children in the age range 6 to 12 years. A brief description of study attributes is provided in Table 1. In some reports insufficient information was provided to complete the meta-analysis. In these cases, authors were contacted to obtain the missing information[1].

Seven studies contained a group of AD/HD children. Schachar et al. (1995) reported the results for three groups of AD/HD children: a pervasive group, a school-only group, and a home-only group. Only the pervasive group was included in the meta-analysis to avoid the problem of correlated results (Rosenthal, 1991) and to maximise the chance of finding group differences (Luk, Leung, & Yuen, 1991; Schachar & Logan, 1990; Schachar et al., 1995; Van der Meere, Wekking, & Sergeant, 1991).

Children with oppositional defiant disorder (ODD) or CD were included in four of the studies. Five studies included a comorbid AD/HD+CD group. Finally, three studies included children with anxiety disorders. Children with ODD or CD were regarded as a single group and further dealt with under the heading CD. ODD is frequently seen as a milder form of CD, the two disorders are related to the same risk factors and forms of impairment, and ODD to some degree predicts the onset of CD (Achenbach, 1993; American Psychiatric Association, 1991; Lahey, Loeber, Frick, Quay, & Grimm, 1992; Loeber, Green, Kennan, & Lahey, 1995). A total of 456 children participated in the 8 studies.

Dependent Variables

The meta-analysis focused on the following four dependent variables derived from the stop task (a detailed description of these measures is provided in the Appendix):

(1) Mean reaction time (MRT), which measures the latency of the processes involved in response execution. Since this meta-analysis focused on abnormalities in response inhibition, MRT was not the main focus of the present study.
(2) The inhibition function (IF), which reflects the efficiency of the inhibitory mechanism controlling for differences in MRT. Most researchers take the slope of this IF (IF-slope) as an index of the subject's capability for response inhibition. The flatter the IF-slope, the poorer the capability for response inhibition (Logan, 1994).

According to the race model (see for review, Logan, 1994; Logan & Cowan, 1984), the IF is determined by

[1] For two studies data on IF-slope and ZRFT-slope were not included in the meta-analysis. Jennings et al. (1997) did not calculate within-group standard deviations for these two dependent measures. Part of the data from the Schachar and Tannock (1995) study were not available.

Table 1
Studies Included in Meta-analysis: Subject and Task Characteristics

Study	Subjects	Age[a]	Subject selection	Task characteristics
1. Schachar & Logan (1990)[b]	10 Controls 13 AD/HD 9 CD/ODD 14 AD/HD+CD/ODD 13 Emotional disorder[c] 11 LD[d]	10.0 9.3 9.8 9.3 9.9 10.0	AD/HD diagnosis based on parent interview and/or teacher ratings. Other diagnoses based on parent interview alone. Control children were free of any diagnosis. Diagnoses according to DSM III (-R) criteria. Criteria for LD diagnosis were at least average IQ and low reading attainment. All children had IQs $\geqslant$ 80 and were free of medication.	Stop task Primary task: two-choice reaction time task (letters X and O) 25% stop trials, stop signals 500, 400, 300, 200, 100, 0 msec before MRT 432 trials (21.6 min), 2 breaks +96 practice trials
2. Daugherty, Quay, & Ramos (1993)	15 Controls 9 AD/HD 8 CD 11 CD+AD/HD 12 Anxiety-withdrawal	11.0 11.4 11.1 11.7 10.8	Group assignment based on teacher ratings. Psychopathological groups had elevated scores on relevant scale and low scores on other scales. Control children had low scores on all scales.	See Study 1
3. Aman, Roberts, & Pennington (in press)	22 Controls 22 AD/HD[e]	12.1 12.1	Assignment to one of the groups based on parent structured interview *and* parent questionnaires. AD/HD children showed favourable response to stimulants. Diagnoses according to DSM III-R criteria. Children with other psychiatric disorders and reading disability were excluded. All children had IQs $\geqslant$ 80 and were free of medication[f].	Stop task Primary task: see Study 1 1/3 stop signal trials, stop signals 500, 350, 250, 100 msec before MRT 192 trials (9.6 min) +96 practice trials
4. Pliszka & Borcherding (1995)	31 Controls 26 AD/HD (or+ODD) 8 AD/HD+CD 17 AD/HD+overanxious (or+ODD)[d] 18 psychiatric controls[d]	8.8 8.7 8.2 9.4 9.5	AD/HD diagnoses based on parent structured interview *and* teacher ratings. CD diagnoses based on parent structured interview alone. Diagnoses of overanxious disorder based on child structured interview. Absence of psychiatric disorder in controls was assessed with a variety of measures.	Stop task Primary task: two-choice reaction time task (red and green light) 25% stop trials, stop signals 500, 400, 300, 200, 100, 0 msec before MRT 432 trials (21.6 min) +48 practice trials
5. Schachar & Tannock (1995)[g]	16 Controls 22 AD/HD 5 CD 18 AD/HD+CD	9.0 9.2 10.1 8.8	Diagnoses based on parent and/or teacher structured interview. Control children were free of any diagnosis and learning problems. Diagnoses according to DSM III-R criteria. All children had IQs $\geqslant$ 80 and $\leqslant$ 130 and were free of medication.	See Study 6
6. Schachar, Tannock, Marriott, & Logan (1995)[h]	22 Controls 10 Home=only AD/HD[d] 9 School=only AD/HD[d] 14 Pervasive AD/HD	9.2 9.4 9.8 8.7	Diagnoses based on parent *and/or* (depending on AD/HD subtype) teacher structured interview. Control children were free of any diagnosis and LD. AD/HD children had no additional diagnosis of ODD or CD. Diagnoses according to DSM III-R criteria. All children had IQs $\geqslant$ 80 and were free of medication.	Change task Primary task: see Study 1 25% stop signals trials, stop signals 500, 350, 200, 50 msec before MRT 288 trials (14.4 min), 2 breaks +a minimum of 72 practice trials
7. Oosterlaan & Sergeant (1996)	17 Controls 15 AD/HD[i] 18 ODD/CD 20 Anxiety disorder	8.7 9.3 9.3 10.1	Assignment to one of the psychopathological groups based on parent, teacher and child ratings (agreement required between two informants). Control children obtained low scores on all scales of all questionnaires. All children had IQs $\geqslant$ 80 and were free of medication.	Stop task Primary task: spatial compatible two-choice reaction time task (white squares) 25% stop trials, stop signals 500, 350, 200, 50 msec before MRT 256 trials (12.8 min), 1 break +64 practice trials

Table 1 (*cont.*)

Study	Subjects	Age[a]	Subject selection	Task characteristics
8. Jennings, Van der Molen, Pelham, Brock, & Hoza (1997)	26 Controls 40 AD/HD[d, j] 25 AD/HD + ODD/CD[j]	9.8 9.7 9.1	AD/HD diagnosis based on parent structured interview and questionnaires completed by parent and teacher. Diagnoses according to DSM III-R criteria. Control children obtained low scores on parent and teacher questionnaires. All children were free of medication.	Stop task Primary task: simple reaction time task embedded in video game format (stop light changing from red to green) 30% stop trials, stop signals 100 or 200 msec after go stimulus Variable trial length, presentation of task stimuli relative to cardiac cycle and respiratory phase 200 trials (40 min), 1 break + a minimum of 25 practice trials

[a] Mean age in years.
[b] Pathological groups could have additional diagnosis of emotional disorder or LD.
[c] The term emotional disorder encompasses mainly anxiety disorders.
[d] Groups not included in meta-analysis.
[e] AD/HD group obtained high ratings of aggressive and delinquent behaviour on one of the parent questionnaires.
[f] Subjects were tested twice, one week apart. AD/HD children were on methylphenidate in the first session and unmedicated in the second session. The results of the second session were entered into the meta-analysis.
[g] Lower reading attainment in AD/HD and AD/HD + CD groups than in CD and control group. Lower arithmetic attainment in CD and AD/HD + CD groups than in AD/HD and control group.
[h] Five AD/HD children met criteria for LD, two children met criteria for overanxious disorder.
[i] Six children with AD/HD also met criteria for inclusion in ODD/CD group.
[j] Twenty-five of the 40 AD/HD children carried a concurrent ODD diagnosis and three children met criteria for CD. Jennings et al. reported both the results for the full sample and for the subsample of children with comorbid disorders. Three children were dropped from this subsample due to incomplete data. Only data of the subsample were included in the meta-analysis. For this subsample, Jennings et al. did not specify the number of children meeting criteria for ODD and for CD. AD/HD group had lower reading, spelling, and arithmetic attainment than control group. Nine AD/HD children met criteria for LD.

parameters of both the response execution process and the inhibitory process. Using the race model, two measures can be derived to examine whether a deficiency in the inhibitory process underlies a poor IF: stop signal reaction time, and the slope of the IF plotted as a function of ZRFT.

(3) Stop signal reaction time (SSRT), which is an estimate of the latency of the inhibitory process. SSRT is one of the parameters that determines the probability of inhibition given a stop signal. The slower the inhibitory process, the harder it becomes to inhibit the response to the primary task (Logan, 1994).

(4) The slope of the IF plotted as a function of ZRFT (ZRFT-slope). In addition to a slow SSRT, poor response inhibition could reflect two other deficits in the inhibitory process. First, it might indicate that the inhibitory process was triggered less often. Second, poor response inhibition could reflect greater variability in latency of the inhibitory process (Logan, 1994). These two parameters of the inhibitory control process are reflected in the ZRFT-slope.

The following information was obtained for all studies and served as the basis of the meta-analysis: (1) the number of subjects in each group, and for each of the dependent measures, (2) the group mean, and (3) the within-group standard deviation. From these data, effect size estimations and their direction were calculated for all relevant group comparisons within each of the studies. The results of these analyses, in turn, were subjected to meta-analytic procedures (Mullen, 1989; Rosenthal, 1991). All analyses were conducted with the advanced BASIC meta-analysis programme developed by Mullen (1989). Calculations were weighted by sample size. The results were evaluated across studies in terms of their effect size and in terms of their significance levels. Furthermore, diffuse tests were conducted to assess the consistency across studies of both estimated effect sizes and significance levels.

Possible Moderating Variables

Before presenting the results of the meta-analysis, several pertinent study characteristics are described. Each of these study characteristics is evaluated for its potential impact on the findings obtained.

Subject selection. Studies differed sharply in the criteria and the measures that were used to select the various groups (see Table 1). This probably explains part of the heterogeneity in the results across studies.

In five studies diagnoses were made using the third revised edition of the *Diagnostic and statistical manual of mental disorders* (DSM III-R; American Psychiatric Association, 1987). With the exception of the Daugherty et al. (1993) study, pathological groups were selected from clinic referred or otherwise treated children (e.g. placed in special educational facilities).

The presence or absence of psychopathological symptoms was assessed by means of reports from parents, teachers, children, or any combination of these informants. A variety of measures was used across the studies, including standardised rating scales, structured diagnostic interviews, or a combination of both. In some studies, diagnoses were based on the report of a single source. For example, in the Daugherty et al. (1993) study, teachers were the sole informants. In other studies, two or

three informants were used. In the majority of these studies, the reports by the different sources were not necessarily required to converge. Since the degree of consistency between different informants' reports is typically modest (Achenbach, McConaughy, & Howell, 1987), the use of multiple informants results in a greater chance of detecting children with actual disorders.

Other studies used a more restrictive approach to combine the information from different informants. In four studies, children received a diagnosis of AD/HD if both parent and teacher reports indicated its presence. Oosterlaan and Sergeant (1996) used parent and teacher ratings and a self-report anxiety questionnaire to select AD/HD, CD, anxiety disorder and control children. Children were assigned to one of the psychopathological groups only if the reports of two informants converged on this classification.

The distinction between pervasive and situational disorders seems important, at least for AD/HD. Several studies have indicated that children with pervasive AD/HD show greater cognitive deficits than children with situational specific AD/HD symptoms (e.g. Luk et al., 1991; Schachar & Logan, 1990; Schachar et al., 1995; Van der Meere et al., 1991).

Comorbidity. A large body of research has demonstrated that AD/HD shows considerable overlap with a number of other disorders, such as ODD and CD, mood disorders, anxiety disorders, and learning disabilities (Barkley, 1990; Biederman et al., 1991; Semrud-Clikeman et al., 1992). Comorbidity might have influenced children's performance on the stop task.

In all but two of the studies (Oosterlaan & Sergeant, 1996; Pliszka & Borcherding, 1995), AD/HD children were free of comorbid ODD or CD. In the Oosterlaan and Sergeant study, 6 out of the 15 children showed associated "aggressive or delinquent" symptoms. The AD/HD sample studied by Pliszka and Borcherding contained children with AD/HD alone and AD/HD with an additional ODD diagnosis. However, the investigators did not specify the corresponding proportions.

In four studies, children were examined for learning disabilities, although in most of these studies the assessment was limited to reading disabilities (Aman et al., in press; Jennings et al., 1997; Schachar & Logan, 1990; Schachar et al., 1995). In only one study were children with concurrent reading disabilities excluded (Aman et al., in press). The remaining four studies did not report on the possible presence of learning disabilities.

Recent findings highlight the necessity to control for associated learning disabilities. A recent study by Tannock and Marriott (1992) suggested that learning disabilities, and not AD/HD, were associated with poor response inhibition and a slower inhibitory process. Furthermore, Daugherty et al. (1993) have reported a moderate positive correlation between reading achievement and IF-slope.

Conflicting predictions were made regarding the capability for response inhibition in children with anxiety disorders and in children with externalising disorders (AD/HD, CD, and AD/HD+CD). Consequently, the distinction between these two disorders seems important. In four of the eight studies, children with externalising disorders were free of anxiety disorders (Aman et al., in press; Daugherty et al., 1993; Oosterlaan & Sergeant, 1996; Pliszka & Borcherding, 1995). In the Schachar and Logan (1990) study, an additional diagnosis of anxiety disorder was allowed in all psychopathological groups. However, exact figures were not presented. The AD/HD sample of Schachar et al. (1995) included two children with concurrent overanxious disorder. In the remaining two studies, children were not assessed for the possible presence of associated anxiety disorders (Jennings et al., 1997; Schachar & Tannock, 1995).

Gender and age. In five studies, boys served as subjects, whereas in the remaining three studies both sexes were included. Gender, however, does not seem to influence the child's performance on the stop task (Daugherty et al., 1993; Pliszka & Borcherding, 1995). In none of the studies were age differences noted between the groups. This suggests that age is not a confounding variable in any of the studies. The mean age of control children ranged from 8.7 years in the Oosterlaan and Sergeant (1996) study to 12.1 years in the Aman et al. (in press) study.

Intellectual functioning. IQ was assessed in six studies. In five of these studies, children with below-average intellectual functioning (IQ less than 80) were excluded. The remaining two studies did not provide information on the children's IQ (Daugherty et al., 1993; Pliszka & Borcherding, 1995). Since several studies have demonstrated that IQ is not related to children's performance on the stop task (Kindlon et al., 1995; Oosterlaan & Sergeant, 1996; Schachar & Logan, 1990; Schachar et al., 1995), it seems unlikely that group differences in intellectual functioning underlie any of the findings.

Medication. In six studies, children were free of medication or discontinued medication before being tested with the stop task. Daugherty et al. (1993) and Pliszka and Borcherding (1995) did not report on the possible use of medication.

Format of the stop task. In six studies the stop task was used, whereas two studies (Schachar & Tannock, 1995; Schachar et al., 1995) utilised a modification of the stop task, known as the change task (De Jong, Coles, & Logan, 1995; Logan & Burkell, 1986). The stimuli in the change task are identical to those in the stop task. However, the two tasks differ with respect to the demand exerted by the stop signal. In both tasks, the stop signal instructs subjects to inhibit their response to the primary task. In the change task, the stop signal, in addition, requires subjects to immediately re-engage in another response, i.e. to execute the so-called change response. Several studies have indicated that the response execution and inhibitory processes seem to function essentially the same in both the stop task and the change task. However, the change task appears to exert higher cognitive processing demands than the stop task, since it carries the additional demand of re-engaging in another response (De Jong et al., 1995; Logan & Burkell, 1986).

In five studies an identical two-choice reaction time task served as the primary task. In this task, children responded to the letters X and O by pressing one of the two corresponding buttons on a response box. Children responded with a left- and a right-hand finger, usually their index fingers. In those studies in which the change task was used, children responded with two separate fingers of the left hand. Modifications of this primary task were used in two other studies. In the study by Pliszka and Borcherding (1995), children responded to a red and green light. In the Oosterlaan and Sergeant (1996) study, a spatial compatible two-choice reaction time task served as the primary task. Jennings et al. (1977) used a simple reaction time task embedded in a video game format. In this task, a green traffic light signalled children to move a mouse-controlled ice-cream cart to a target area on the computer screen.

With the exception of the study by Jennings et al. (1997), the timing of stimuli was similar for all studies. Trials started with the presentation of a warning signal of 500 msec. Immediately thereafter the primary task stimulus was displayed for 1000 msec. An intertrial interval of 1500 msec was used. In all studies primary task stimuli were presented visually. Jennings et al. investigated the relationship between response inhibition and heartbeat timing. In this study, stimuli were presented relative to the child's cardiac cycle and respiratory phase. Note that the presentation rate of stimuli in information processing tasks has been shown to play a crucial role in the performance of AD/HD children (see Van der Meere, 1996, for review).

Jennings et al. (1997) presented stop signals relative to the onset of the primary task stimulus, whereas in the other studies stop signals were presented relative to the child's MRT (see

Appendix). In all studies, the longest stop signal interval was 500 msec. The shortest stop signal interval varied from 0 msec to 100 msec. In six of the studies, stop signals were presented on 25% of the trials. In the studies by Jennings et al. (1997) and Aman et al. (1995), the percentage of stop trials was somewhat higher: 30% and 33.3%, respectively.

Stop signals were presented in the auditory modality. In the Jennings et al. (1997) study, the stop signal resembled the sound of a car horn. In the other studies, a tone was presented by the computer or through earphones (Oosterlaan & Sergeant, 1996). Where reported, tones were 1 kHz in pitch and 100 msec in duration.

Studies with young adults have demonstrated that the stop task is reasonably robust for differences in the format of the task. First, a number of studies have shown that measures derived from the stop task are not affected by differences in the nature of the primary task. Findings with the stop task do not seem to vary as a function of task difficulty, nor are the results different for tasks that require continuous or discrete responses (see for reviews, Logan, 1994; Logan & Cowan, 1984). Second, the stop task has been found robust for changes in stop signal probability. Typically, the primary task stimulus occurs on every trial and the stop signal occurs on 25% of the trials. Logan (1981) and Logan and Burkell (1986) studied the effect of varying stop signal probability from 10% to 80%. Measures derived from the stop task were not much affected when the stop signal probability varied between 10% and 50%. Third, and finally, Logan (1994; Logan & Cowan, 1984) reviewed studies using auditory and/or visual stop signals and concluded that performance on the stop task is not affected by stop signal modality. In summary, the stop task has been found to be reasonably robust for differences in the nature of the primary task, for differences in stop signal probability, and for differences in stop signal modality. Therefore, it seems unlikely that differences in the format of the stop task underlie any of the findings.

Between-studies differences were noted in the nature and amount of practice before starting the task. Furthermore, studies varied in the number of trials and the number of breaks within the task. Estimates of time-on-task ranged from 9.6 min to 21.6 min (since in the majority of studies the length of the task was not specified, an estimate of time-on-task was obtained by multiplying the number of trials with the trial length). In the Jennings et al. (1997) study, the length of trials varied. In this study, four blocks were administered, each 10 min in duration.

Details on the instructions given to the children varied considerably between studies. Instructions are considered important in the stop task, since the task involves a delicate balance between fast and accurate responding, on the one hand, and inhibition of responses, on the other hand. To enhance the child's performance, Jennings et al. (1997) used feedback, monetary reward, and monetary loss. Where reported, the experimenter remained with the child during the task. However, none of the studies provided information about the role of the experimenter during the task. Most studies failed to report whether the experimenter was aware of the child's group assignment and the purpose of the study. Where reported, subjects were tested individually.

Results

As indicated previously, this meta-analysis concentrates on five group comparisons, namely: (1) AD/HD–controls, (2) CD–controls, (3) AD/HD–CD, (4) AD/HD + CD–AD/HD, and (5) anxiety disorder–controls. Meta-analytic results for each of the dependent measures are presented in Tables 2 through 5. The tables show, for each of the five comparisons: (a) the total number of subjects across studies for the contrasting groups, (b) the number of relevant studies, (c) the weighted mean for both groups, (d) the combined estimated effect size in terms of Cohen's d (1988), and (e) the meta-analytic Z with the respective significance level. In agreement with Cohen's guidelines, effect sizes of .20, .50, and .80 were used as thresholds to define small, medium, and large effects, respectively.

MRT. Meta-analytic results for the latency of responding are presented in Table 2. MRTs of AD/HD and control children were compared in seven studies. The mean effect size ($d = .49$) was close to Cohen's standard of .50 for a medium effect, indicating 32% nonoverlap between the MRT distributions of the two groups. Differences between the other four pairs of groups were small and not significant. Note that the results of all group comparisons were consistent across studies, both in terms of effect sizes and significance levels. This indicates that the results of the studies agree both on the magnitude of the group differences and on the significance of these differences.

Note also that a comparison of weighted mean MRTs of AD/HD and CD children suggests slower MRTs for AD/HD children. The combined effect size and significance level, however, indicated that the two groups did not differ in MRT. This finding could be explained as follows. Across studies, substantial differences were noted in MRTs. For example, MRTs of control children varied from about 350 msec (e.g. Oosterlaan & Sergeant, 1996) to 900 msec (e.g. Schachar & Logan, 1990). These differences reflect variations in the cognitive load of the tasks employed. Since a greater proportion of CD than AD/HD children was tested with a stop task with relatively low cognitive demands (as indexed by the MRT of the control group), CD children showed a substantially shorter weighted mean MRT than AD/HD children.

IF-slope. Table 3 displays the results for IF-slope. Across six studies, AD/HD children had flatter IF-slopes than controls, suggesting impairments in response inhibition. The combined effect size of $d = .94$ exceeds Cohen's threshold for a large effect and corresponds to 53% nonoverlap between the IF-slope distributions of the two groups. Diffuse tests indicated that effect sizes were consistent across studies, whereas heterogeneity was noted for the significance levels. This heterogeneity in significance levels was attributable mainly to the Daugherty et al. (1993) study, in which AD/HD and control children showed highly comparable IF-slopes. In all other studies, AD/HD children showed significantly flatter IF-slopes than controls.

Similar findings were obtained for the comparison between CD and control children. Averaged across three studies, IF-slopes were flatter in CD children than in controls. A medium combined effect size of $d = .56$ was found, which translates into 36% nonoverlap between the IF-slope distributions of the two groups. Results were uniform across studies, both in terms of effect sizes and significance levels.

In three studies AD/HD and CD children were compared. Although AD/HD children had somewhat flatter IF-slopes than children with CD, this difference was small and not significant. Thus, no support was obtained for the hypothesis that AD/HD children would

Table 2
Go Response Mean Reaction Time (*MRT; in Msec*)

	Sample size			Weighted mean			
Group comparison	Group 1	Group 2	Studies	Group 1	Group 2	*d*	Combined *Z*
AD/HD–Controls	121	133	7	772.6	702.5	0.49	3.67*
CD–Controls	40	58	4	612.6	642.0	0.29	1.59
AD/HD–CD	59	40	4	726.1	612.8	0.36	1.73
AD/HD + CD–AD/HD	51	70	4	867.2	847.5	0.16	0.87
Anxious–Controls	45	42	3	623.0	607.7	0.25	1.23

Diffuse comparisons were conducted to assess the homogeneity of effect sizes and significance levels. All group comparisons yielded homogeneous results.
* $p < .001$.

Table 3
Inhibition Function Slope (*IF-slope*)

	Sample size			Weighted mean			
Group comparison	Group 1	Group 2	Studies	Group 1	Group 2	*d*	Combined *Z*
AD/HD–Controls	99	117	6	10.2	14.8	0.94[a]	6.36**[b]
CD–Controls	35	42	3	12.6	16.0	0.56[a]	2.35*[a]
AD/HD–CD	37	35	3	11.5	12.8	0.25[a]	0.73[a]
AD/HD + CD–AD/HD	33	48	3	10.0	8.6	0.17[b]	0.73[b]
Anxious–Controls	45	42	3	14.4	15.9	0.34[a]	1.41[a]

Diffuse comparisons were conducted to assess the homogeneity of effect sizes and significance levels: [a] Homogeneous effect; [b] Heterogeneous effect.
* $p < .05$; ** $p < .001$.

Table 4
Stop Signal Reaction Time (*SSRT; in Msec*)

	Sample size			Weighted mean			
Group comparison	Group 1	Group 2	Studies	Group 1	Group 2	*d*	Combined *Z*
AD/HD–Controls	121	133	7	349.4	246.4	0.64[a]	4.97**[a]
CD–Controls	40	58	4	265.7	248.0	0.51[b]	2.64*[b]
AD/HD–CD	40	59	4	364.7	265.7	0.07[b]	0.37[b]
AD/HD + CD–AD/HD	51	70	4	323.6	361.8	0.13[a]	0.78[a]
Anxious–Controls	45	42	3	231.3	207.6	0.20[a]	0.88[a]

Diffuse comparisons were conducted to assess the homogeneity of effect sizes and significance levels: [a] Homogeneous effect; [b] Heterogeneous effect.
* $p < .01$; ** $p < .0001$.

be more deficient in response inhibition than children with CD. Again, findings were consistent across studies, both in terms of effect sizes and significance levels.

Meta-analysis revealed no differences between IF-slopes of children with comorbid AD/HD + CD and children with AD/HD alone. However, these results need to be interpreted with caution given the inconsistency in both effect sizes and significance levels. In fact, two studies demonstrated no significant differences between both groups (Daugherty et al., 1993; Pliszka & Borcherding, 1995), whereas one study indicated that children with AD/HD alone had flatter IF-slopes than comorbid AD/HD + CD children (Schachar & Logan, 1990).

In three studies children with anxiety disorders were compared with controls. Contrary to the predictions, children with anxiety disorders did not exhibit steeper IF-slopes than controls. Note also that these meta-analytic results were consistent across studies.

SSRT. As indicated earlier, the speed of the inhibitory process is one of the parameters that determines the IF. In other words, poor capability for response inhibition may reflect a relatively slow SSRT (Logan, 1994). The meta-analytic results for SSRT appear in Table 4. Across seven studies, AD/HD children were on average 103 msec slower than control children. A medium combined effect size of $d = .64$ was obtained, which translates into 40% nonoverlap between the two group distributions. Both effect sizes and significance levels were homogeneous across studies.

Similar results were noted for the comparison between CD and control children. Across four studies, SSRT was on average about 18 msec slower in CD children than in controls. The average effect size of $d = .51$ equals Cohen's threshold for a medium effect. This effect size indicates 34% nonoverlap between the SSRT distributions of the two groups. Diffuse tests indicated heterogeneity in terms

Table 5
Inhibition Function Slope Plotted as a Function of ZRFT (ZRFT–slope)

	Sample size			Weighted mean			
Group comparison	Group 1	Group 2	Studies	Group 1	Group 2	*d*	Combined *Z*
AD/HD–Controls	99	117	6	21.0	23.8	0.19	1.31
CD–Controls	35	42	3	19.3	20.7	0.04	0.20
AD/HD–CD	35	37	3	19.3	17.7	0.07	0.04
AD/HD + CD–AD/HD	33	48	3	22.1	20.6	0.12	0.52
Anxious–Controls	42	42	3	21.1	20.7	0.03	0.26

ZRFT = a *z* score which represents the relative finishing time of the inhibitory process and the response execution process in *SD* units (a discussion of this measure is provided in the Appendix). Diffuse comparisons of effect sizes and significance levels indicated homogeneous results for all group comparisons. All *Z* scores nonsignificant.

of effect sizes and significance levels. In fact, three studies supported the direction of the meta-analytic result, and one study revealed faster SSRTs in children with CD (Schachar & Tannock, 1995). However, only Oosterlaan and Sergeant (1996) found that CD children had significantly slower SSRTs than controls.

Although the speed of the inhibitory process was on average almost 100 msec slower in AD/HD children than in CD children, the combined significance level and effect size indicated no differences between the two groups. This finding stems from the sharply conflicting findings obtained in each of the four studies. Two studies revealed substantially faster SSRTs for CD children. In contrast, the other two studies demonstrated that CD children had somewhat slower inhibitory processes. Given these inconsistencies, the meta-analytic findings need to be interpreted with considerable caution.

Comorbid AD/HD + CD children did not differ from children with AD/HD alone. Furthermore, meta-analysis revealed no differences in SSRT between anxious and control children. Note also that these findings were homogeneous both in terms of effect sizes and in terms of significance levels.

ZRFT-slope. Table 5 presents the meta-analytic results for ZRFT-slope. Combined effect sizes and significance levels indicated that there were no differences between the contrasted groups. All groups showed comparable ZRFT-slopes, indicating that group differences in the capability for response inhibition were neither related to the probability of triggering the inhibitory process, nor to differences in variability in latency of the inhibitory process (Logan, 1994). Note also that effect sizes and significance levels were homogeneous.

Summary of Results

Despite important methodological differences between studies, the present meta-analysis revealed fairly consistent results. In this section, we briefly summarise our findings.

First, the major finding was that, compared with controls, both AD/HD and CD children demonstrated flatter inhibition functions, indicating poor response inhibition. In both groups, poor response inhibition was related to a slow inhibitory process, although the findings were inconsistent for CD children. In other words, impairments in response inhibition were not unique to children with AD/HD. Moreover, AD/HD and CD children did not differ in the degree of their impairments. No differences were noted between these two groups in terms of the inhibition function, nor in terms of the inhibitory process. It should be noted, however, that the findings for the speed of the inhibitory process differed sharply between studies.

Second, AD/HD children with and without CD neither differed in their ability to inhibit responses, nor in terms of the inhibitory process. Thus, AD/HD + CD children did not show greater impairments than children with only AD/HD. However, findings were inconsistent between studies for the slope of the inhibition function.

Third, no evidence was found for the prediction of enhanced response inhibition in anxious children. In fact, anxious and control children could not be differentiated on any of the dependent measures.

Finally, the latency of responding on the primary task was slower for AD/HD children than for controls. There were no differences in the speed of responding between the other groups.

Obviously our findings are limited by the restricted number of studies currently available. This holds in particular for the comparisons between CD and control children, between AD/HD and CD children, and between AD/HD + CD and AD/HD children. Some of the meta-analytic results for these group comparisons were heterogeneous. Clearly, these inconsistencies call for more research. Furthermore, we need to mention that our findings for AD/HD were limited by the fact that in two studies some children with this disorder also showed ODD or CD symptomatology. With these limitations in mind, we now discuss the results of the meta-analysis.

Discussion

This paper presents the results of a meta-analysis of studies with the stop task. The aim was to investigate whether data gathered with the stop task supports the response inhibition deficit hypothesis for AD/HD (Barkley, 1994, 1997; Douglas, 1988, 1989; Newman & Wallace, 1993; Pennington & Ozonoff, 1996; Quay, 1988a, b, 1997; Wender, 1972). Furthermore, we investigated whether impaired response inhibition is uniquely related to AD/HD (Barkley, 1994, 1997; Douglas, 1988, 1989; Pennington & Ozonoff, 1996; Wender, 1972) or

whether deficits in response inhibition are also evident in other psychopathological disorders. Finally, we examined the suggestion that anxiety disorders are associated with abnormally high levels of response inhibition (Quay, 1988a, b).

Consistent and robust evidence was found for a response inhibition deficit in AD/HD. Relative to controls, AD/HD children demonstrated flatter inhibition functions, indicating poor response inhibition. Impaired response inhibition was related to a slow inhibitory process. Our findings support recent theoretical notions of AD/HD in which poor response inhibition is suggested to lie at the heart of this disorder (Barkley, 1994, 1997; Douglas, 1988, 1989; Newman & Wallace, 1993; Pennington & Ozonoff, 1996; Quay, 1988a, b, 1997; Wender, 1972).

However, particularly important is that the meta-analysis did not support the notion that response inhibition deficits are unique to AD/HD (Barkley, 1994, 1997; Douglas, 1988, 1989; Pennington & Ozonoff, 1996; Wender, 1972). Similar to AD/HD children, CD children had flatter inhibition functions and slower inhibitory processes compared with controls. Furthermore, response inhibition deficits did not distinguish children with AD/HD from children with CD, nor from children with comorbid AD/HD + CD; the three groups showed similar deficits in terms of their inhibition functions and the latency of the inhibitory process. These collective findings suggest that response inhibition deficits characterise children with behaviour labelled as undercontrolled or externalising (Achenbach, 1991; Achenbach & Edelbrock, 1978). This conclusion confirms the predictions derived from Quay's psychobiological model of child psychopathology (Quay, 1988a, b, 1993, 1997).

The finding that both AD/HD and CD children evidence response inhibition deficits is not surprising. There is considerable overlap in the symptomatology of these two disorders (American Psychiatric Association, 1994; Barkley, 1990; Biederman et al., 1991; Nottelman & Jensen, 1995; Russo & Beidel, 1994). In fact, it is controversial whether AD/HD and CD are distinct clusters of abnormal behaviour (Fergusson, Horwood, & Lloyd, 1991; Hinshaw, 1987; Hinshaw, Lahey, & Hart, 1993). Furthermore, AD/HD has been found to be strongly predictive for the later development of CD (McGee, Williams, & Feehan, 1992; Taylor, Chadwick, Heptinstall, & Dankaerts, 1996). Consequently, it seems plausible that the two disorders share common deficits, as was found in the present research.

Although AD/HD and CD share the impairment in response inhibition, this impairment does not necessarily reflect the same dysfunction. Poor response inhibition in AD/HD children may be part of a general impairment in executive functions, which in turn may be attributable to a frontal lobe dysfunction (Lezak, 1983; Pennington & Ozonoff, 1996; Torgesen, 1994). Recently, such an account of AD/HD has been advanced by Barkley (1994, 1997) and by Pennington and Ozonoff (1996). Indeed, AD/HD children have been found to demonstrate impairments on a variety of tests purported to measure frontal lobe functioning (see for review, Barkley et al., 1992; Pennington & Ozonoff, 1996; see also Grodzinsky & Diamond, 1992; Shue & Douglas, 1992).

A similar explanation for response inhibition deficits in CD children seems less likely. Although there is support for the presence of deficits in executive functioning in CD, Pennington and Ozonoff (1996) pointed out that these findings may be attributed to the presence of comorbid AD/HD. Recent studies that controlled for the presence of AD/HD yield conflicting findings. Some studies have shown that CD is indeed associated with impaired executive functions (Séguin, Pihl, Harden, Tremblay, & Boulerice, 1995; White et al., 1994). Other studies, however, failed to do so (Linz, Hooper, Hynd, Isaac, & Gibson, 1990). The present meta-analysis shows that children with CD, but without AD/HD, exhibit response inhibition deficits. Since response inhibition is regarded as one of the key executive functions, our findings add to the research indicating executive functions deficits in CD.

Besides an explanation in terms of a deficit in executive functioning, poor response inhibition in AD/HD children may also be explained in other ways, for example as reflecting a non-optimal activation state (see, for review, Sergeant & Van der Meere, 1990, 1991; Van der Meere, 1996). In the cognitive energetical model of information processing (Sanders, 1983, 1990), an optimal activation is required to maintain adequate motor processing (Sternberg, 1969a, b). That is, activation is concerned with the readiness to respond (Pribram & McGuiness, 1975; Sanders, 1983, 1990). A non-optimal activation state causes impaired motor processing, which results in difficulties for both the execution and inhibition of responses. Thus, the non-optimal activation hypothesis can account for our finding that AD/HD children had a slower response execution process, as well as for our finding of a response inhibition deficit in these children.

Extensive research supports the hypothesis that AD/HD involves a non-optimal activation state (see, for review, Sergeant & Van der Meere, 1990, 1991; Van der Meere, 1996). Activation is influenced by the presentation rate of stimuli in a task (Sanders, 1983, 1990). A high presentation rate of stimuli causes high levels of activation; a slow event rate causes low levels of activation. A recent study by Van der Meere, Stemerdink, and Gunning (1995) demonstrated that AD/HD children were less able to inhibit inappropriate responding than controls with fast and slow presentation rates of stimuli. However, AD/HD children did not differ from their control peers in a medium stimulus presentation rate.

Particularly compelling for the non-optimal activation state hypothesis is the research with methylphenidate. Stimulants have been found to influence the activation level (Sanders, 1983, 1990; Sergeant & Van der Meere, 1991). Interestingly, research by Tannock and associates with the stop task has shown that methylphenidate ameliorates response inhibition deficits in AD/HD children (Tannock et al., 1989; Tannock, Schachar, & Logan, 1995). Clearly, these findings warrant more research to investigate the relationship between response inhibition and activation state.

Poor response inhibition in AD/HD children may also be understood as a motivational deficit. That is to say, these children do not expend the effort necessary to achieve and maintain optimal performance. In support of this view, it has been found that the performance of AD/HD children relies more strongly on the presence of

contingencies than the performance of control children (Douglas, 1989; Haenlein & Caul, 1987; Newman & Wallace, 1993; Quay, 1988a, b, 1997; Wender, 1972). In several explanatory models of AD/HD, deficits in response inhibition are implicitly linked to motivational factors. Douglas (1989), for example, proposed that AD/HD encompasses a defective capability for inhibition and an unusually strong inclination to seek immediate rewards. As discussed earlier, Quay (1988a, b, 1993, 1997) explained the capability for response inhibition in terms of the activity of the BIS and the BAS. These two brain systems, in turn, are activated by signals of punishment and signals of reward, respectively.

Finally, AD/HD children's lack of response inhibition has been explained as due to delay aversion. On the basis of several studies (Sonuga-Barke, Taylor, & Heptinstall, 1992; Sonuga-Barke, Taylor, Sembi, & Smith, 1992; Sonuga-Barke, Williams, Hall, & Saxton, 1996), Sonuga-Barke (1995) set forth a model that explains the behaviour of AD/HD children in terms of a pervasive aversion of delay. According to this model, disinhibition in children with AD/HD reflects an attempt to reduce delay and does not arise from the inability to inhibit as such. This explanation, however, is unlikely to account for the present findings. In the stop paradigm used in the studies described here, successful inhibition or failing to inhibit did not influence the time that children had to wait before the next trial commenced, since the intertrial interval was fixed. Thus, poor response inhibition could not have served to reduce delay.

An important issue raised by our findings is whether poor response inhibition in AD/HD children represents a stable deficit or a maturational lag in the development. Recently, Barkley (1994, 1997) has suggested that AD/HD is associated with such a delay in the attainment of the capability for response inhibition. Some support for this explanation comes from the Jennings et al. (1997) study (see Table 1). In that study, 10–12-year-old AD/HD boys showed better response inhibition than their 8–9-year-old counterparts. In contrast, no such differences were found for a group of control boys.

Two recent studies (Oosterlaan & Sergeant, 1997; Schachar & Logan, 1990) suggest that the capability for response inhibition and the underlying inhibitory process develop early in childhood. However, both studies were restricted to children aged 8 years and over. At present data for younger children are lacking. Thus, it is currently not possible to estimate the degree of delay in AD/HD children. A proper test of the maturational lag hypothesis would be a longitudinal study of AD/HD children and control children. It seems particularly important to investigate to what extent early emerging impairments in response inhibition predict the onset of externalising behaviour disorders later in childhood.

Meta-analytic findings did not support the prediction that anxiety disorders are characterised by enhanced levels of response inhibition, given their hypothetically overactive BIS (Quay, 1988a, b). We found that anxious and control children differed neither in terms of their inhibition functions, nor in terms of the inhibitory process underlying response inhibition. Our failure to demonstrate strong response inhibition in anxious children might be explained in several ways. First, it is possible that anxious children did not differ from controls because the performance of both groups represents the performance that is maximally feasible. Second, it is possible that anxious children only show enhanced response inhibition in situations that trigger their hypothetically overactive BIS. That is to say, enhanced response inhibition will only be evident when anxious children are presented with signals of impending punishment (Gray, 1987). A third explanation was offered by Newman and Wallace (1993). They argued that increased activity in the BIS might result in heightened activity in the Nonspecific Arousal System (NAS). The NAS mediates the speed and force of responses (Gray, 1987). In this way, the overactive BIS not only leads to increased inhibitory control, but also to faster and more vigorous responding. Increased NAS activity is thought to interfere with the capability to inhibit responses. Finally, the performance of anxious children may have been hampered by worrisome thoughts associated with anxiety (Eysenck & Calvo, 1992). According to this view, worrying (e.g. concern over task performance) pre-empts some of the processing and storage resources of the working memory system. As a result, less processing and storage resources are available and cognitive performance is hampered.

Although the stop task has several advantages over other measures of response inhibition, the paradigm has its limitations. First, since the stop task is purported to measure response inhibition, an important issue is whether children with externalising disorders are also associated with impairments in the ability to inhibit cognitive processes. Second, the stop task is purported to measure one form of response inhibition, i.e. the ability to inhibit an ongoing response. It does not assess other forms of response inhibition (Barkley, 1997) such as inhibiting interference or the ability to inhibit a response over a protracted period of time (Halperin et al., 1994; Masters & Binger, 1978; Sonuga-Barke, 1995). It remains to be seen whether the current findings generalise to these latter definitions of response inhibition.

In summary, this meta-analysis provides consistent and robust support for contemporary accounts of AD/HD in terms of a response inhibition deficit. However, the results do not support the notion that this deficit is uniquely related to AD/HD (Barkley, 1994, 1997; Douglas, 1988, 1989; Pennington & Ozonoff, 1996; Wender, 1972). Poor response inhibition seems to characterise children with externalising disorders. This finding fits well within Quay's model (1988a, b, 1993, 1997), but poses a challenge for models of AD/HD in which deficits in response inhibition are thought to be unique to this disorder (Barkley, 1994, 1997; Douglas, 1988, 1989; Pennington & Ozonoff, 1996; Wender, 1972).

Future studies should be directed to answering whether different processes underlie the similar findings for AD/HD and CD children or whether the similarity in findings points to a common background dysfunction. Furthermore, the task for future research is to uncover the conditions that enhance and challenge the inhibitory control process. This type of research with the stop task will undoubtedly contribute to deepen our knowledge of the mechanisms underlying disinhibition in child psychopathology. Illustrative for this type of research are the studies investigating the effects of methylphenidate on

response inhibition in children with AD/HD (Tannock et al., 1989, 1995). Finally, more attention needs to be devoted to the impact of comorbid disorders, such as learning disabilities.

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Appendix

The Stop Task and the Race Model

The stop task requires fast and accurate execution of a reaction time task, denoted as the primary task. Occasionally, an auditory stop signal is presented, which requires the subject to inhibit the response to the primary task. Stop signals are presented at different stop signal delays. That is, the delay is varied between the onset of the primary task stimulus and the onset of the stop signal. The longer the delay, the more difficult it becomes to inhibit a response. By contrast, the shorter the delay, the easier it becomes to inhibit a response. Usually, delays are chosen such that the longest delay will produce a probability of inhibition close to 0, whereas the shortest delay will yield a probability of inhibition close to 1.

The stop task is based on a well-established theory of inhibition, known as the race model (see for review, Logan, 1994; Logan & Cowan, 1984). According to this model, response inhibition depends on a race between, on the one hand, the response execution process (or go process) that responds to the primary task stimulus (or go signal) and, on the other hand, the inhibitory process (or stop process) that responds to the stop signal. The process that finishes first determines performance.

Thus, if the go process finishes before the stop process, the response is executed. If the stop process finishes before the go process, the response to the primary task stimulus is inhibited.

The faster the go process, the less likely it is that the stop process wins the race with a stop signal presented at a given delay. Consequently, the faster the go process, the lower the probability of inhibition. By contrast, the faster the stop process, the more likely it is that the stop process wins the race, and the higher the probability of inhibition. Many researchers compensate for differences in go signal reaction time between subjects, presenting stop signals relative to mean go signal reaction time. That is, stop signals are presented at different intervals before the subject's expected response, i.e. at different intervals defined as MRT minus delay, where MRT is defined as the mean reaction time calculated across correctly executed responses on go trials. We now discuss the three main dependent variables in the stop task: (1) the inhibition function, (2) the stop signal reaction time, and (3) the ZRFT-slope.

Inhibition Function

The inhibition function (IF) is generated by plotting the probability of inhibition against mean go signal reaction time minus stop signal delay (MRT − delay). This function reflects the efficiency of the inhibitory mechanism controlling for differences in mean go signal reaction time. Most researchers take the slope of this IF (IF-slope) as an index of the subject's capability for response inhibition. This slope is calculated by fitting a regression line to the individual IF. The flatter the slope, the poorer the capability for response inhibition. The steeper the slope, the better the capability for response inhibition.

If groups of subjects are found to differ in their IF, the question arises whether this result reflects differences in the stop process or differences in the go process. Two methods are available to investigate possible differences in the inhibitory process.

According to the race model (see for review, Logan, 1994; Logan & Cowan, 1984), the IF is determined by: (1) the speed of the go process and variability in the speed of the go process, (2) the speed of the stop process and variability in the speed of the stop process, and (3) the probability of triggering the stop process. Thus, a shallow IF could reflect either parameters of the response execution process (i.e. 1) or it could reflect a deficiency in the stop process (i.e. 2 and 3). Using the race model, two measures can be derived to examine whether a deficiency in the stop process underlies a poor IF. The first measure is stop signal reaction time (SSRT). SSRT is an estimate of the latency of the stop process. The second measure is the slope of the IF plotted as a function of ZRFT (ZRFT-slope). ZRFT corrects for all parameters of the response execution process and for SSRT. Differences in the ZRFT-slope reflect differences in the probability of triggering the stop process or differences in the variability of the speed of the stop process. We now describe each of these two measures in detail.

SSRT: The Latency of the Stop Process

The latency of the stop process is not observable, but can be estimated by using the race model. Three methods are available to estimate SSRT (Logan, 1994). We describe the most commonly used method, in which the latency of the stop process is assumed to be constant.

The race model assumes that the stop and the go processes operate independently of one another. Therefore, the distribution of reaction times on go trials can be seen as the distribution of latencies of the go process on stop trials. Since SSRT is assumed to be constant, SSRT can be seen as a point on the time axis of this distribution. At a given stop signal delay, all responses to the right of this point are inhibited because the stop process finishes before the go process. Responses to the left of this point are not inhibited; the go process finishes before the stop process. The proportion of trials that were inhibited is equal to the probability of inhibition, whereas the proportion of trials that were not inhibited is equal to 1 minus the probability of inhibition.

In theory, we integrate the distribution of go signal reaction times from zero to a point in time at which the integral equals the probability of responding given a stop signal (i.e. 1 minus the probability of inhibition). We treat that point as an estimate of the time at which the stop process finished. This time is defined relative to the onset of the go signal (because we use the distribution of go signal reaction times to define it), thus we subtract out stop signal delay to estimate SSRT.

In practice, SSRT is calculated as follows: first, reaction times on go trials are rank ordered on a time axis. Second, we pick the *n*th reaction time, where *n* is defined by the product of the number of reaction times in the distribution and the probability of responding given a stop signal (or 1 minus the probability of inhibition). For example, if there were 100 reaction times in the distribution and the probability of responding given a stop signal was .3, the *n*th reaction time would be the 30th in the rank-ordered distribution. The *n*th reaction time is an estimate of the time at which the stop process runs to completion, relative to the onset of the primary task stimulus. Third, we subtract stop signal delay from the *n*th reaction time and estimate SSRT. For example, if the *n*th reaction time was 545 msec and the stop signal delay was 200 msec, SSRT would be 345 msec. SSRT is calculated for each stop signal delay and then averaged.

ZRFT-slope: The Probability of Triggering the Stop Process and Variability in the Speed of Stop Process

In addition to a slow stop process (SSRT), two other deficiencies in the stop process may underlie poor response inhibition: (1) a low probability of triggering the stop process, and (2) high variability in the latency of the stop process. To investigate whether these two parameters of the stop process explain differences in the IF, a so-called ZRFT transformation is applied to the IF (Logan, 1994). The ZRFT transformation corrects for differences in MRT, for go signal reaction time variability, and for SSRT. Specifically, the probability of inhibition is plotted as a function of a *z* score that represents the relative finishing time of the go process and the stop process in standard deviation units, using the standard deviation of reaction times on the primary task to define these units. ZRFT is obtained with the following formula: ZRFT = (MRT − stop signal delay − SSRT)/standard deviation of reaction times on the primary task. The slope of the IF plotted against ZRFT is known as the ZRFT-slope.

If differences in the IF disappear after correction for ZRFT, this indicates that differences were completely accounted for by differences in MRT, SSRT, and variability in go signal reaction time. That is, differences in the IF were neither related to the probability of triggering the stop process, nor to differences in variability in the latency of the stop process. However, this does not compromise the results for the latency of the stop process (SSRT). Thus, differences in SSRT do not necessarily go together with differences in ZRFT-slope. In contrast, if differences remain after correction for ZRFT, this indicates differences in the probability of triggering the stop process or in SSRT variability. Specifically, a relatively shallow ZRFT-slope indicates that the stop process was triggered less often, or that there was greater variability in latency of the stop process. It is not possible to discriminate between the latter two deficiencies in the stop process.