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Confirmation of an Inhibitory Control Deficit in Attention-Deficit/Hyperactivity Disorder

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The objective of this study was to determine whether deficient inhibitory control distinguishes children with a diagnosis of attention-deficit/hyperactivity (ADHD) disorder, conduct disorder (CD), and comorbid ADHD + CD from normally developing children. Participants were rigorously diagnosed children (age 7 to 12 years) with ADHD ($N = 72$), CD ($N = 13$) or ADHD + CD ($N = 47$) and 33 control children (NC). We studied inhibitory control using the stop-signal paradigm, a laboratory task that assessed the ability to inhibit an ongoing action. The ADHD group had significantly impaired inhibitory control compared to NC, CD, and ADHD + CD children. These results indicate that children with ADHD have deficient inhibition as measured in the stop-signal paradigm and that ADHD occurring in the presence of ADHD + CD may represent a phenocopy of CD rather than a variant of ADHD.

KEY WORDS: Attention deficit hyperactivity disorder; conduct disorder; comorbidity; inhibition.

Attention-deficit/hyperactivity disorder (ADHD) is a common and impairing psychiatric disorder of childhood that affects approximately 3–5% of school age children in the general population (American Psychiatric Association, 1994; Szatmari, Offord, & Boyle, 1989) and 50% of children who are referred for clinic assessment (Offord *et al.*, 1987). ADHD is defined by developmentally inappropriate and impairing levels of inattentiveness, impulsiveness, and hyperactivity commencing in early childhood.

In the early 1970s, Douglas (1972) proposed that attention deficit, rather than excessive activity, was the core abnormality of the disorder. However, deficits in basic or subordinate processes of attention such as memory and encoding have been difficult to identify (e.g., Douglas, 1988; Sergeant & Van der Meere, 1990). Instead, the focus of research and theory has shifted to a deficit in the executive processes that control subordinate cognitive processes, enabling them and directing them, turning them on and off (Barkley, 1997a,b; Logan, 1985; Logan, Schachar, & Tannock, 1997; Meyer & Keiras, 1997). Executive processes are involved in the management of the constant stream of sensory information competing for access to the processes controlling action and in decisions about the appropriateness and timing of action (Denckla, 1996). The effects of deficiencies in executive control processes may be subtle causing adjustment in the parameters of subordinate process; or they may be dramatic, causing cascading effects on subordinate and other executive control process throughout the system.

One particular executive process, inhibition, has been implicated as a potential locus of a core deficit in ADHD (Barkley, 1997a; Pennington & Ozonoff, 1996; Quay, 1997). Inhibition comes into play in situations requiring withholding or sudden interruption of an ongoing action or thought or in the suppression of information that one wishes to ignore. According to this theory, deficient inhibitory control impairs the ability of ADHD children to engage other executive-control strategies to optimize their

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behavior. The direct and cascaded effects of deficient inhibition affects working memory, self-regulation, internal speech, and "reconstitution" (i.e., the ability to reconstruct behavior). A deficit in inhibitory control means that individuals with ADHD act without thinking and therefore miss out on the benefits of these carefully considered control strategies.

Inhibitory control has been studied extensively using the stop-signal paradigm (Logan, 1994), which is a laboratory analog of a situation that requires rapid and accurate execution of a simple motor action and occasional and unpredictable, cessation of this action. The paradigm involves two concurrent tasks, a *go task* and a *stop task*. Typically, the go task involves a choice among stimulus and response alternatives (e.g., discriminating an X from an O). The object of the go task is to respond as quickly and accurately as possible. The stop signal, occurs unpredictably on occasion (typically on 25% of go-task trials), and involves presentation of a signal (typically a tone) that tells participants to completely stop their response to the go task on that trial.

Whether children are able to inhibit on a particular trial depends on the outcome of a race between the go and the stop processes: If the stop task response finishes before the go task response, children will inhibit their response to the go task (Jennings, van der Molen, Brock, & Somsen, 1992; Logan, 1985; Logan, 1994; Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984; Ollman, 1973; Osman, Kornblum, & Meyer, 1986). If the go task response finishes before the stop task response, children will fail to inhibit their response to the go task, responding much as they would if no stop signal had been presented. Thus, inhibitory control depends on the latency of two independent processes; the response to the go signal (go reaction time) and the response to the stop signal (stop-signal reaction time, SSRT). Poor inhibition could result from responding too quickly to the go signal or responding too slowly to the stop signal. The outcome of the race between the go and stop processes depends as well on the interval between the onset of the go signal and the onset of the stop signal (stop-signal delay). Short delay between the go and stop signals increases the probability of inhibiting and long delay increases the probability of responding.

Various methods have been used to calculate SSRT (Logan, 1994). We (Logan, Schachar, & Tannock, 1997) employed a new method of calculating SSRT using a tracking algorithm. The algorithm is designed to find a stop-signal delay that ties the race between the go process and the stop process. The algorithm increases stop-signal delay if children inhibit successfully, and it decreases stop-signal delay if children fail to inhibit. If the increments and decrements are equal in magnitude (we use 50 ms changes), the algorithm converges on a stop-signal delay at which children inhibit 50% of the time. At that point, the go process and the stop process finish at the same time, on average. Go-signal reaction time and stop-signal delay are observable. SSRT is unobservable, but can be estimated simply by subtracting stop-signal delay from mean go-signal reaction time (Logan, 1994). In addition to ease of explanation and simplicity of calculation of SSRT, SSRT can be estimated in less time (10–15 min) than with other methods (30–40 min). Calculated in this way, SSRT is a precise measure of latency of an internally generated, although unobserved, inhibitory control process independent of go-signal reaction time. Compared to individuals with good inhibitory control, those with poor inhibitory control have longer SSRT. The tracking algorithm also takes into account the nature of the strategy that a person adopts to perform the go task (speed and variability). Individuals who respond quickly to the go task have shorter stop-signal delay; individuals with slower go responses or who "wait" for the stop signal have longer stop-signal delays. In either case, the outcome of the race is biased in the same way and SSRT can be calculated if, on average, individuals inhibit on 50% of trials.

We were the first to demonstrate that ADHD children are deficient in their ability to inhibit responses in the stop-signal paradigm (Schachar & Logan, 1990). This basic finding has been replicated in several studies. In a meta-analysis of published studies, Oosterlaan, Logan, and Sergeant (1998) observed that a total of 121 ADHD children had an average SSRT of 349 ms, whereas 133 normal controls had an average SSRT of 246 ms ($p < .05$). All of these studies predate DSM-IV and none employed the tracking algorithm to calculate SSRT. Therefore, the first objective of the current study is to replicate the deficit in ADHD using individuals diagnosed according to DSM-IV diagnostic criteria and using the tracking method for estimating SSRT.

Our previous research indicated that an inhibitory control deficit as measured by the stop-signal paradigm was specific to ADHD: Children with learning disability, anxiety, or conduct disorder not associated with ADHD have SSRTs similar to those of normally developing control children (Schachar & Logan, 1990; Schachar, Tannock, Marriott, & Logan, 1995). By comparison, the review of Oosterlaan *et al.* (1998) reported that children with a diagnosis of conduct disorder (CD) showed a small but significant deficit in SSRT compared with normally developing children (266 ms vs. 248 ms, $p < 0.05$). The same meta-analysis found a difference between CD and ADHD that was much larger in absolute terms (266 ms vs. 365 ms). However, the difference between ADHD and

CD groups was not significant because of the greater variability among individuals diagnosed with ADHD. Few children with CD not associated with ADHD have been included in these studies (total of 40–59). Consequently, the second objective of the current study is to reexamine inhibitory control in children with a diagnosis of CD. Finally, the current study will also compare the performance of those children with ADHD and those with concurrent ADHD and CD (ADHD + CD).

METHOD

Participants

There were 132 participants (111 boys, 21 girls) ranging in age from 7 to 12 years ($M = 9.1$, $SD = 1.5$) who were referred for assessment of disruptive behavior to the outpatient department of psychiatry of an urban pediatrics hospital. Thirty-three normal control children (20 boys, 13 girls) ranging in age from 7 to 12 years ($M = 9.3$, $SD = 1.5$) were recruited through advertisements. The parents of all participants gave written consent and all children gave verbal assent. The study was approved by the institutional review board.

Children were excluded from the study if they had a history or evidence of a neurological disorder (e.g., epilepsy), a chronic or serious medical problem, psychosis, or a clinically significant mood or anxiety disorder. They were also excluded if they had verbal and performance IQ of less than 80. Any participant receiving stimulant medication for ADHD had their medication withdrawn for 48 hours preceding testing. The latter exclusion ensured that there were no drug effects on cognitive performance (Tannock, Schachar, Carr, Chajczyk, & Logan, 1989).

Diagnostic Measures

The participants were assigned a diagnosis on the basis of the results of a semistructured interview with parents (Parent Interview for Child Symptoms, PICS; Schachar & Ickowicz, 1994; unpublished manuscript) and with each child's classroom teacher conducted by telephone (Teacher Telephone Interview, TTI; Schachar & Tannock, 1990; unpublished manuscript). Each interview was administered by an experienced clinician trained to a high level of agreement (K of .80) with criterion interviews. The PICS interview covers DSM-IV symptoms of ADHD, CD, oppositional defiant disorder (ODD), as well as anxiety, mood, and other single symptom and internalizing disorders. The TTI covers ADHD, CD, and ODD and screens for other disorders.

Diagnostic Criteria

To be classified as ADHD, children had to meet DSM-IV criteria for ADHD (age of onset, severity, and type of symptom) defined as at least six of nine inattentive or hyperactive–impulsive symptoms, or both. In order to ensure pervasive impairment, we required that children meet criteria for ADHD in the parent or teacher interview but also exhibit a minimum of four inattentive *or* four hyperactive–impulsive symptoms according to the other informant. ADHD was diagnosed in 17 additional cases that had very high ADHD symptom scores (more than 15 of 18 possible inattentive and hyperactive–impulsive symptoms) in one interview and at least three inattentive or three hyperactive–impulsive symptoms on the other interview.

CD was diagnosed if two or more DSM-IV criteria for CD were reported by parents or teacher ($N = 29$), or if one CD symptom was reported in addition to four ODD symptoms ($N = 40$). CD criteria were adjusted because of the young age of our sample (see DSM-IV).

Normal controls were assessed in the same way, and to be included, they had to be free of all psychiatric disorder. Based on these criteria, children were assigned to one of four groups; normal control, ADHD, CD, and ADHD + CD.

Reliability of the PICS interview was assessed using audiotapes of 25 interviews by a second, trained rater who was blind to diagnosis. Agreement on the presence of diagnosis was high to moderate (ADHD $K = .84$; ODD $K = .84$; CD $K = .50$).

Reading proficiency was assessed with the Word Attack subtest from the Woodcock Reading Mastery Tests–Revised (WRMT; Woodcock, 1987).

Apparatus and Stimuli

The stimuli for the stop-signal paradigm were presented on a desktop computer equipped with headphones through which auditory signals were presented. The stimuli for the go task were the uppercase letters X and O (1.25 inches in height) presented in the center of the screen for 1000 ms. Each trial was preceded by a 500-ms fixation point, presented in the center of the screen and then extinguished. The screen remained blank for 1000 ms. Consequently, each trial included a period of 2.5 s in which the child could respond to the primary task in accordance with the task's demands. The stop signal was a 500-ms, 1000-Hz tone generated by the computer and delivered through headphones at a comfortable listening volume. Stop signals occurred unpredictably on 25% of go-task

trials, and involved presentation of a tone that instructed children to completely stop their response to the go task on that trial. Responses were recorded with a hand-held response box with buttons labeled with either an "X" or an "O."

The stop-signal delay (the interval between the presentation of the go signal and the stop signal) was altered dynamically after every stop-signal trial, depending on the subject's performance. If an individual inhibited their response on a particular trial, the stop-signal delay was reset so that it appeared 50 ms later on the subsequent stop trial. If the individual responded on a particular stop-signal trial, the stop delay was shortened by 50 ms.

Procedure

Children were tested individually in a quiet room in the presence of an examiner who read a uniform set of instructions. The task was presented in 10 blocks, the first two of which were practice. Each block consisted of 32 trials; 24 go-signal trials without stop signals and 8 trials that included a stop signal. The X and O comprising the go-signals occurred equally often in each block. Stop signals were presented in 25% of trials and occurred equally often with each of the two go-signal letters. In the first of 10 blocks, children were presented with the go and the stop signals but were instructed to ignore the stop tones and practice responding quickly and accurately to the go signal by pressing the appropriate response button identified by X and O labels. Children were instructed to keep separate fingers of their left hand on the X and O buttons throughout the experiment. In the second practice block, children were informed about the stop signal and instructed to stop their response to the go signal when they heard the stop tone. Children were encouraged to continue responding to the go signal as quickly and as accurately as possible if no stop signal were presented. They were told that stop signals occurred in such a way that sometimes it would be difficult to stop and sometimes not. Stop-signal delay was set initially at 250 ms and then reset to 250 ms at the beginning of the eight experimental blocks. After the third and sixth experimental block, go reaction time was presented on the screen and children were reminded about the importance of maintaining the speed and accuracy of their responses to the go signal. Go reaction time, standard deviation of go reaction time, and SSRT were calculated for each of the eight experimental blocks and for the entire task.

Analysis

To detect inter-group differences in symptom severity and in clinical characteristics, we compared diagnostic groups using one-way analysis of variance (ANOVA) for continuous variables with post hoc tests (Tukey–HSD test) and χ^2 for categorical variables with a significance level of .05.

Accuracy, probability of inhibition, and SSRT in each test block was examined to determine whether the individual had generally complied with the requirements of the task. Unacceptable performance in any block was characterized by; inhibiting on all or none of the stop-signal trials, fewer than 66% correct responses to the go task, or an SSRT that was less than 50 ms. Participants who failed the first two criteria were excluded from further analyses because such performance would yield questionable estimates of their SSRT (Band, 1997). Participants who failed the last criterion, however, were not excluded because their SSRT could be estimated on the basis of their valid performance on the first three blocks using regression analysis (Norman & Streiner, 1993; see below for details). Planned comparisons of ADHD and NC and of ADHD and CD were conducted to test hypotheses about the specificity of the observed inhibition deficit (Keppel, 1982). In addition, we conducted analyses of covariance to assess the effect of reading ability, age, IQ, and gender on the relationship of ADHD and inhibitory control.

RESULTS

Eleven children (5% of those tested) were excluded because their overall go-task accuracy on the stop-signal paradigm was less than 66%. These excluded children were distributed equally across the four diagnostic groups. A further four children were excluded because they were extreme multivariate outliers (greater than 3 *SD* above mean) on multiple performance measures (SSRT, go reaction time, accuracy) (Tabachnick & Fidel, 1998).

Of the remaining 165 participants, 21 had at least one block in which SSRT was less than 50 ms. SSRT of this magnitude were never observed even among normal adults in a study of the development of inhibitory control (Williams, Ponesse, Schachar, Logan, & Tannock, 1999). These 21 children were more likely to have CD (31%) than the remaining participants (e.g., ADHD 9%) but did not differ in gender, IQ, or reading performance.

No abnormally fast SSRTs were observed in any of the first three experimental blocks. Consequently, we estimated SSRT by using performance on blocks 1, 2, and 3 in a regression equation (Norman & Streiner, 1993). The regression equation was calculated for those participants who had valid data for all blocks. The SSRTs for blocks 1 through 3 were fit into a model to predict overall SSRT. A strong regression model that accounted for 76% of the

Table I. Comparison of Diagnostic Groups on Demographic and Behavioral Characteristics

Attribute	NC ($n=33$)	ADHD ($n=72$)	CD ($n=13$)	ADHD + CD ($n=47$)	$F(3,161)$	$p<$	Contrasts[a]
Age (mean years)	9.3 (1.5)	9.0 (1.4)	9.5 (1.4)	9.2 (1.5)	0.73	.54	
IQ	110.8 (15.0)	96.0 (12.4)	109.6 (11.6)	103.1 (14.7)	10.4	.001	ADHD < NC, CD, ADHD + CD
WRMT[b]	94.5 (14.0)	86.2 (16.0)	94.5 (22.7)	93.0 (14.3)	3.07	.03	
Teacher-rated symptoms							
ADHD	1.6 (2.2)	9.7 (2.9)	3.8 (3.5)	10.2 (3.2)	77.5	.001	NC, CD < ADHD, ADHD + CD
CD	0.0 (0.0)	0.0 (0.0)	1.9 (3.0)	1.3 (1.5)	20.6	.001	NC, ADHD < CD, ADHD + CD
ODD	0.2 (0.9)	0.8 (1.4)	2.6 (3.0)	2.9 (2.8)	16.6	.001	NC, ADHD < CD, ADHD + CD
Parent-rated symptoms							
ADHD	2.8 (3.4)	10.2 (3.5)	8.5 (4.9)	11.7 (3.6)	42.7	.001	CD < ADHD + CD; NC < ADHD, CD, ADHD + CD
CD	0.0 (0.2)	0.0 (0.2)	1.8 (1.6)	2.0 (1.6)	49.6	.001	NC, ADHD < CD, ADHD + CD
ODD	0.9 (1.2)	2.3 (2.2)	5.2 (2.8)	4.9 (2.2)	30.9	.001	NC < ADHD < CD, ADHD + CD
Sex ratio (m/f)	3:2	4:1	6:1	9:1	$\chi^2(3)=10.3$	.02	

[a] Tukey's HSD, $p < .05$.
[b] WRMT = Woodcock Reading Mastery Test (Word Attack).

Table II. Comparison of Diagnostic Groups on Stop-Signal Paradigm Performance

Characteristic	NC ($n=33$)	ADHD ($n=72$)	CD ($n=13$)	ADHD + CD ($n=47$)	$F(3,161)$	$p<$	Planned comparisons
SSRT (ms)	264.3 (75.7)	331.8 (149.3)	293.8 (88.3)	270.4 (96.9)	3.71	.01	ADHD < NC, ADHD + CD
Go RT (ms)	579.3 (107.6)	663.8 (132.7)	643.0 (101.0)	647.5 (137.0)	3.39	.02	ADHD < NC
Go RT variability	215.1 (59.9)	247.3 (84.5)	256.7 (76.8)	228.9 (76.9)	1.75	.16	
Probability of inhibition	51.1 (2.2)	51.3 (4.1)	52.7 (4.0)	52.2 (4.5)	1.08	.36	

variance was obtained [$F(3,144)=123.6$, $p < .00001$]. Estimated SSRT scores for those 21 children were calculated by substituting their scores for blocks 1, 2, and 3 into the following derived equation:

$$\begin{aligned} SSRT_{predicted} = {} & 22.659289 + SSRT_{block1} * .205970 \\ & + SSRT_{block2} * .347271 \\ & + SSRT_{block3} * .356355 \end{aligned}$$

Seventy-two participants met criteria for ADHD, 13 for CD, 47 for ADHD + CD, and 33 for NC. No difference among diagnostic groups were identified in age or rate of reading ability (Table I). Children in the ADHD, CD, and ADHD + CD groups were more likely to be males than those in the NC group. IQ differed among the groups. The association between IQ and SSRT, the primary measure of inhibitory control, was significant but weak ($r = .17$, $p < .05$).

The four diagnostic groups differed in severity of ADHD, ODD, and CD symptoms as expected (see Table I). Post-hoc comparisons found that the ADHD and ADHD + CD groups did not differ in severity of ADHD symptom severity (as reported by parents or teachers). However, the two ADHD groups (ADHD, ADHD + CD) had significantly higher ADHD symptom counts than did the two non-ADHD groups (NC, CD). The CD and ADHD + CD groups did not differ in severity of CD and ODD as reported by parents or teachers, but the two CD groups (ADHD + CD, CD) had significantly higher CD and ODD symptom counts than the two non-CD groups (ADHD, NC).

Table II shows stop-signal paradigm performance for children in each of the four diagnostic groups. The probability of inhibition was very close to 50%, indicating that the tracking algorithm was working well. No difference in probability of inhibition was observed among the four groups. Moreover, the four diagnostic groups did not differ in variability of go reaction time.

Groups differed in SSRT (Table II). Planned comparison indicated that the ADHD group had slower SSRT than the NC group [$F(1,161)=7.36$, $p < .01$], and the ADHD + CD [$F(1,161)=7.67$, $p < .01$] group. The differences between ADHD and CD groups [$F(1,161)=1.14$, n.s.] and between the CD and NC groups [$F(1,161)=.58$, n.s.] were not significant.

Groups also differed in go RT (Table II). Planned comparison indicated that the ADHD group had slower go RT than the NC group [$F(1,161)=9.9$, $p < .005$].

The ADHD group did not differ from the ADHD + CD [$F(1,161) = .5$, n.s.] or the CD groups [$F(1,161) = .3$, n.s.] and the CD and NC groups did not differ [$F(1,161) = 2.3$, n.s.].

Analysis of covariance was conducted to assess the effect of potential confounds (age, IQ, gender, reading disability). In these analyses, IQ, age, gender, and reading scores were not significant covariates of inhibitory control. These results were unchanged when analyses were rerun omitting those individuals for whom SSRT was estimated.

DISCUSSION

The first objective of this study was to confirm the presence of an inhibition deficit in children with a diagnosis of ADHD as measured by the new tracking version of the stop-signal paradigm. In previous research, various versions of the stop-signal paradigm have been used. These versions varied in task length, method of setting of delay, proportion of stop-signal trials, and interstimulus interval. Moreover, previous studies varied in sampling frame and diagnostic procedures. Despite these methodologic differences, studies have been remarkably consistent in observing significantly longer SSRT—evidence of deficient inhibition—in ADHD than in normal control children (Oosterlaan, Logan, & Sergeant, 1998). In the current study, as in previous research, ADHD children were approximately 70 ms slower than normal children to stop an ongoing action, an effect of about one-half standard deviation in magnitude.

The deficit in inhibitory control observed in ADHD was not attributable to differences in age, IQ, or gender. Slowing of all responses did not appear to account for the inhibitory control deficit. ADHD, CD, and ADHD + CD children had slower go reaction times than normal control children, but only the ADHD group was slower to stop. The magnitude of the deficit in stopping was as great as that in going: The difference between normal controls and ADHD children was 84 ms for go-signal reaction time and 68 ms for SSRT, even though go reaction times were twice as long as SSRT. Furthermore, differences in reading ability by itself did not account for the observed differences. Other research has shown that children with ADHD and comorbid reading disability may have the most pronounced deficits in inhibition (Purvis & Tannock, in press). The association of inhibition and RD may reflect the important role of inhibitory mechanisms in language comprehension (Carr & Dagenbach, 1990) as well as in regulation of behavior.

The tracking version of the stop-signal paradigm worked well for the majority of participants. One gauge of how well the staircase tracking version worked is the probability of stopping given a stop signal. The objective of the staircase method is to locate the delay at which the race between the go response and the stop response processes are tied on average. The closer to probability of .5 for inhibiting given a stop signal, the closer the two processes are to being tied. Over all participants, we observed a mean probability of inhibition of 51.7%. In addition, the mean probability of inhibition did not vary across groups. The primary advantages of the tracking method are the simplicity of calculating SSRT and the brevity of the task (15 min compared with 40 min for earlier versions).

However, the new version of the task did not work well for everyone. In a small group of children, accuracy of performance on the go task was so low that one could not assume that the children was engaged in the task. SSRT cannot be estimated accurately under these circumstances (Band, 1997). For other children, SSRTs were unrealistically fast. In one study in the general population (Williams *et al.*, 1999), we found no adults or children who achieved SSRT of 50 ms or less. In the current study, the majority of these unrealistically fast SSRT arose after the third block of trials. It was at that point in the procedure that we paused the task briefly to remind children of the requirements of the task, namely that they were to respond as quickly and as accurately as possible without making errors on the go task. This reminder resulted in a speeding of reaction time to go task stimuli in subsequent blocks. The tracking algorithm responds slowly to sudden fluctuations in go reaction time because delay shifts only following stop-signal trials. Stop signals occur on 25% of trials and delay shifts only by 50 ms in each step. Consequently, sudden shifts in go reaction time may result in inaccurate SSRT estimates. Optimally, children should be reminded to respond quickly and accurately following each block of trials. Alternately, a regression approach can be used to estimate SSRT from blocks of trials in which SSRT is not unrealistically fast.

The second objective of the current study was to examine the specificity of inhibitory control deficit in ADHD. Two previous studies had indicated that an inhibitory control deficit as measured by the stop-signal paradigm was specific to ADHD (Schachar & Logan, 1990; Schachar *et al.*, 1995). Children with CD, anxiety disorder, or learning disability did not differ from normal controls. The comparison with CD is particularly compelling because CD is another externalizing disorder. However, few children with CD not associated with ADHD have been included in previous studies.

The current study identified only 13 children who had significant CD symptoms and minimal ADHD symptoms. Our results supported the conclusion that CD children do

not differ in inhibitory control from normally developing children. Although they did not show a deficit relative to ADHD children, the performance of the CD group was more like that of the NC and the ADHD + CD groups. The validity of this finding has to be seen in the light of the small number with "pure" CD available for study. It seems that most children who meet diagnostic criteria for CD also meet criteria for ADHD. Available research is rather consistent (cf. Oosterlaan, Logan, & Sergeant, 1998). This research indicates that deficient inhibition either is specific to ADHD among the disruptive behavior disorders or at least ADHD is associated with a more severe deficit than CD.

The third purpose of the present study was to determine whether those with combined ADHD and CD exhibit the same deficit as those with ADHD alone. There are four possible hypotheses to account for true comorbidity of ADHD and CD (Caron & Rutter, 1991). Briefly, these hypotheses are that (1) ADHD + CD is a hybrid of pure ADHD and pure CD (i.e., has the unique characteristics of both pure disorders) because the presence of risk factors for one disorder increases the probability of the risk factors for the second disorder; (2) ADHD, CD, and ADHD + CD reflect a single underlying disorder with similar risk factors (e.g., developmental, cognitive, and psychosocial) differing only in surface manifestations, such as developmental course or severity; (3) the comorbid condition is a third, distinct condition that differs from both pure disorders; and (4) one condition can produce a symptomatic phenocopy of the other disorder but without the second disorder's underlying deficits.

Our two previous studies had reached conflicting conclusions concerning the comorbidity of ADHD and CD. In the first study (Schachar & Logan, 1990), we observed a deficit in ADHD but not in ADHD + CD. We concluded that ADHD + CD might represent a phenocopy of true ADHD and would be more accurately conceived as a variant of CD. This conclusion supports hypothesis 4. In a subsequent study (Schachar & Tannock, 1995), the ADHD + CD group performed as poorly as the ADHD group on the stop-signal paradigm and shared the characteristic of high exposure to psychosocial adversity that was found in the CD group. We concluded that ADHD + CD represented a hybrid of ADHD and CD characteristics supporting hypothesis 1. The results of Oosterlaans *et al.*'s meta-analysis (1998) supported the hybrid hypothesis: Although ADHD + CD children had faster SSRTs (324 ms) than those with ADHD (362 ms) the difference was not significant. In the current study, we observed that the performance of the ADHD + CD individuals was comparable to that of CD and normal controls on the stop signal paradigm and was significantly faster than individuals with "pure" ADHD. This result is in agreement with our first study and supports the phenocopy hypothesis 4.

In addition, the phenocopy hypothesis is supported by available family–genetic studies. Faraone *et al.* (1991) compared rate and type of disorder among the relatives of probands with ADHD or ADHD + CD. They found that ADHD was common among the relatives of both groups, that CD was evident only among relatives of ADHD + CD probands and that ADHD and CD cosegregated among relatives of ADHD + CD probands. They concluded that ADHD + CD was distinct from ADHD. However, they had insufficient CD participants to determine whether ADHD + CD was a variant of CD or a distinct entity. Studies of neuropsychological deficit in these groups have concluded that ADHD + CD may represent a unique entity (Schachar & Tannock, 1995, for review).

A phenocopy of ADHD could arise if disruptive children do not attend to tasks or sit still as a result of noncompliance rather than as a result of a cognitive deficit. Alternatively, there may be a halo effect operating, whereby disruptive children are described as hyperactive, impulsive and inattentive by their teachers and parents even though their actual behavior as directly observed might not show them to be (Abikoff, Courtney, & Koplewicz, 1991; Schachar, Sandberg, & Rutter, 1986).

Results to date indicate that the combined ADHD + CD phenotype may be a heterogeneous mixture of (1) ADHD children who, as a consequence of their poor self-regulation, have developed secondary CD (i.e., a true hybrid) and (2) CD children who exhibit or are thought to exhibit ADHD-like behaviors as a reflection of their CD. The uniqueness of the combined group in any particular study will depend on the proportion of individuals in each of these subgroups of ADHD + CD children. If this hypothesis is correct, ADHD + CD children with deficient inhibition should be similar to ADHD children whereas ADHD + CD children without deficient inhibition should be similar to CD.

The possibility that ADHD + CD may be a phenocopy of ADHD is intriguing and raises important questions for research into the etiology of ADHD. If phenocopies of ADHD occur, it is possible that some children with disruptive behavior problems may be incorrectly diagnosed as ADHD in research samples. Laboratory tests do not have an established role in clinical practice. However, inclusion of more "objective" nonbehavioral, diagnostic markers such as laboratory measures of cognitive deficits could be helpful in research. They could be used to improve diagnosis of apparently unaffected individuals or to define more homogeneous subtypes of affected individuals for genetic analysis (Tsuang, Faraone, & Lyons, 1993). Moreover, cognitive measures may improve our understanding

of the underlying deficits in various disorders. They point to functional impairments and specific pathology of particular brain regions or neural networks.

In summary, this study confirms the presence of an inhibitory control deficit among ADHD children diagnosed according to DSM-IV criteria as measured by a new tracking version of the stop-signal paradigm. The deficit was not evident among children with comorbid ADHD + CD, suggesting that the latter might be a variant of CD rather than of ADHD. The findings have implications for research that depends on accurate diagnosis.

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