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Deficient Inhibitory Control in Attention Deficit Hyperactivity Disorder

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The purpose of this study was to examine two executive control processes — response inhibition and re-engagement of responses after inhibition in children with attention deficit hyperactivity disorder (ADHD). Thirty-three children with ADHD and 22 normal control children of similar age (7 to 11 years) and mean IQ (107) were tested with the change paradigm. ADHD subgroups were defined by the context in which the ADHD symptoms predominated (in the home only; at school only; and in both, i.e., pervasive ADHD). Children with marked oppositional defiant or conduct disorder were excluded. Children with ADHD exhibited deficits in inhibitory control and in response re-engagement. Deficits were greatest in pervasive ADHD and, to a lesser extent, in those with ADHD limited to the school context. ADHD limited to the home context showed the least deficit. These results replicate an earlier study that found deficient inhibitory control in pervasive ADHD and demonstrate that the deficit in ADHD involves a second aspect of executive control.

Attention deficit hyperactivity disorder (ADHD) as defined in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed. rev.) (DSM-III-R; American Psychiatric Association, 1987), the most common psychiatric disorder of childhood, affects 5% to 10% of school-aged children (e.g., Offord, Boyle,

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Fleming, Blum, & Grant, 1989). The impulsiveness, inattentiveness, and overactivity that characterize the syndrome have been attributed to impairment of the higher-level cognitive functions referred to as *self-regulation* or *executive control* (Douglas, 1983, 1988; Logan & Cowan, 1984; Schachar & Logan, 1990a; Shue & Douglas, 1992). Executive control involves multiple processes including the capacity to initiate, inhibit, and alter actions according to circumstances (Lezak, 1983; Pribram, 1973). This study aims to replicate a previous finding of deficient inhibitory control in subjects with ADHD (Schachar & Logan, 1990a) and to investigate the possibility of a deficit in a second aspect of executive control, altering an action after a response has been inhibited.

In our previous research (Schachar & Logan, 1990a), we were concerned with one aspect of executive control, the ability of children to inhibit a planned action. This is an important executive control option in circumstances requiring efficient reactions to changes in the environment or errors in performance (e.g., checking a swing at a bad pitch in baseball; stopping oneself instead of chasing a ball into traffic). Compared with actions that are well controlled, those that are poorly controlled are likely to run on to completion. The behavioral manifestations of ADHD, in part, may be attributed to such deficient inhibitory control (Douglas, 1983, 1989; Schachar, Tannock, & Logan, 1993).

We studied inhibitory control in ADHD using the stop-signal paradigm — a well-established and theoretically derived method for studying the central control of the processes involved in the inhibition of a planned action or thought (Logan, 1981, 1985; 1994; Logan & Cowan, 1984). The stop-signal paradigm is a laboratory analogue of a real-life situation that requires rapid and accurate execution of an action and, on occasion, cessation of this action. In the stop-signal paradigm, subjects are engaged in a primary task (a forced-choice reaction-time task) and, occasionally and unpredictably, they are presented with a stop signal (a tone) that instructs them to withhold the motor response to the primary task. The main datum collected is whether or not subjects withhold their responses to trials in which the stop signal occurred.

Using the stop-signal paradigm, we found that children with a diagnosis of attention deficit disorder with hyperactivity (ADDH) as defined in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.) (DSM-III; American Psychiatric Association, 1980) had flatter inhibition functions than normal control children, indicating deficient inhibitory control (Schachar & Logan, 1990a). Deficits were greater in children with pervasive ADDH (i.e., they met criteria for a diagnosis of ADDH both at home and at school) compared with those with situational ADDH (i.e., they met criteria for a diagnosis of ADDH at home or at school but not in both situations). However, the numbers of pervasive and situational ADDH subjects were too few to permit firm conclusions.

The primary objective of the current experiment was to replicate our previous observation of deficient inhibitory control (in subjects with ADHD) and to examine a second aspect of executive control. Cognitive flexibility (the ability to switch rapidly and appropriately from one thought or action to another, Grattan & Eslinger, 1990) involves two sets of executive control processes: namely, the ability to inhibit an ongoing action or response (response inhibition) and the ability to execute an alternative response after inhibition of the current action (response re-engagement) (Logan & Burkell, 1986; Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984). Consequently, the second objective of the current study was a more direct examination of the ability to shift to or re-engage an alternative response after inhibition of an ongoing response. The question of deficient re-engagement of alternative responses has been investigated previously, usually with the Wisconsin Card Sorting Task (WCST; Chelune, Ferguson, Koon, & Dickey, 1986; Dyme, Sahakian, Golinko, & Rabe, 1982; Gorenstein, Mammato, & Sandy, 1989). Typically, children with a diagnosis of ADHD make more perseverative errors in the face of changing task demands. However, performance on the WCST may be a function of a range of factors in addition to the ability to switch to an alternative response, including task comprehension, speed of response, and the ability to stop an ongoing response (Schachar *et al.*, 1993). In this experiment, we tested subjects with ADHD using the change paradigm (Logan & Burkell, 1986), a measure that distinguishes between speed of response, ability to inhibit, and ability to switch to an alternative response.

The change paradigm presents the subject with stimuli that are identical to those of the stop-signal paradigm (Logan & Cowan, 1984). The change paradigm is identical to the stop-signal paradigm in the response inhibition required when stop signals are presented, but has the additional requirement of an immediate, separate, and overt response to the stop signal. This response to the stop signal constitutes the secondary task. The change paradigm permits distinction among the various processes involved in response re-engagement, as will be described below. We predicted that ADHD would be associated with deficits both in the inhibition of ongoing responses and in the re-engagement of a secondary response. A general deficit in executive control would be implicated if ADHD were associated both with deficient inhibition and re-engagement processes.

The second objective of this experiment was the replication and extension of our previous research about the distinction between pervasive and situational ADHD (Schachar & Logan, 1990a). Epidemiological studies have indicated that approximately 16% of children with ADHD meet the criteria for ADHD both at home and at school and are considered pervasively ADHD. Subjects with situational ADHD comprise the 73%

of children with ADHD who meet the criteria at school only and the 11% who meet the criteria at home only (Szatmari, Offord, & Boyle, 1989). Children with both situational and pervasive ADHD qualify for a diagnosis according to the DSM-III and DSM-III-R. However, previous research has found evidence that supports the validity of the distinction between pervasive ADHD and situational ADHD. Subjects with pervasive ADHD perform worse than subjects with situational ADHD on a range of neuropsychological and psychoeducational tests (Boudreault *et al.*, 1988; Sandberg, Rutter, & Taylor, 1978; Schachar, Rutter, & Smith, 1981; Taylor, Schachar, Thorley, & Wieselberg, 1986; Taylor, Sandberg, Thorley, & Giles, 1991), as well as exhibiting greater psychosocial adversity, more severe developmental delay, and worse prognosis (see Schachar, 1991, for a review).

Recent evidence indicates that an additional distinction may be necessary between situational ADHD defined in the home context only and situational ADHD defined in the school context only. The combination of home-only and school-only situational ADHD may result in a group of children with situational ADHD that has the characteristics of a mild form of pervasive ADHD and could obscure potentially important distinctions among these subtypes (Costello, Loeber, & Strouthamer-Loeber, 1991). There were insufficient numbers of children with home- and school-only ADHD in the study of Schachar and Logan (1990a) to permit an examination of inhibitory control in school-only, home-only, and pervasive ADHD. Accordingly, to achieve the second objective of our experiment, we used the change paradigm to compare the response inhibition and reengagement of context-dependent ADHD subtypes.

METHOD

Subjects

The subjects in this experiment were 33 boys (age range 7 to 11 years) referred for assessment of disruptive behavior to the outpatient Departments of Psychiatry or Pediatrics at The Hospital for Sick Children, Toronto, Canada. Twenty-two children seen in the general pediatrics department who had uncomplicated medical problems and were free of psychiatric or learning problems served as normal controls. Informed consent was obtained from the parents of all subjects and assent from all subjects themselves. Children were excluded from the study if they showed evidence of a neurological disorder such as epilepsy or a chronic and serious medical problem (asthma), had a history or evidence of psychosis, or

had an estimated full-scale IQ of less than 80. Children with an additional or exclusive DSM-III-R diagnosis of conduct disorder (CD) or severe oppositional defiant disorder (ODD) were excluded from the current sample because these diagnoses were not associated with deficient inhibitory control (Schachar & Logan, 1990a) or attentional capacity (Schachar & Logan, 1990b). Although four children were taking methylphenidate on a regular basis before the study, all children were free of any medication for a minimum of 48 hours preceding testing.

Diagnostic Assessment of the Child. The subjects were assigned a diagnosis on the basis of the results of a diagnostic interview with the parent(s) (Parent Interview for Child Symptoms, PICS; Schachar & Wachsmuth, 1989, unpublished data) and an interview of each child's teacher conducted by telephone (Teacher Telephone Interview, TTI; Schachar & Tannock, 1990, unpublished data).

The interrater reliability of these interviews was assessed by a child psychiatrist who rated videotaped (PICS) and audiotaped (TTI) interviews. Agreement among symptom scores derived from these interviews with scores for related symptom dimensions derived from the Ontario Child Health Study (OCHS) scales (Boyle *et al.*, 1987) was used to assess the convergent validity of the PICS and the TTI.

Psychoeducational Screening Assessment. The psychoeducational screening consisted of the reading and arithmetic subtests of the Wide Range Achievement Test — Revised (WRAT-R; Jastak & Wilkinson, 1984) and the Vocabulary and Block Design subtests from the Wechsler Intelligence Scale for Children — Revised (Wechsler, 1974).

Child Diagnosis

Diagnoses were assigned according to DSM-III-R criteria. Three subtypes of ADHD were diagnosed: home-only ADHD, school-only ADHD, and pervasive ADHD. Pervasive ADHD was diagnosed if eight or more ADHD symptoms were reported on both the PICS and TTI. Home-only ADHD was diagnosed if eight or more DSM-III-R ADHD symptoms were reported on the PICS but fewer than eight symptoms were reported by the teacher. School-only ADHD was diagnosed if eight or more ADHD symptoms were reported on the TTI but fewer than eight symptoms were reported by parents.

A diagnosis of emotional disorder (ED) was assigned to children meeting the criteria on the PICS interview for one of the following disorders: overanxious, minor depressive, phobic, obsessive–compulsive, or separation anxiety disorder.

Several steps were taken to confirm the validity of the distinction among the ADHD subtypes. First, we compared ADHD subtypes on symptom-severity scores for ADHD, ODD, CD, and ED, which we calculated by summing the number of symptoms in each setting on the PICS and TTI.

Second, we compared ADHD subtypes on an alternative measure of disorder, the OCHS scale (Boyle *et al.*, 1987). These comparisons were necessary to determine whether context-dependent ADHD was confounded by the severity of the ADHD. For example, the pervasive ADHD group could have exhibited more ADHD symptoms at school than the school-only ADHD subjects or more ADHD symptoms at home than the home-only ADHD subjects. Moreover, the validity of the contrast between school-only and home-only ADHD could have been weakened if one group had many more symptoms in the defining situation than the other group. For example, school-only ADHD subjects might have had more ADHD symptoms at school than home-only subjects had at home. In addition, we checked the distribution of ADHD symptom counts for school-only and home-only ADHD groups to determine whether the validity of our contrasts among groups was undermined by a large number of subjects who just failed to meet criteria for pervasive ADHD (e.g., seven rather than eight symptoms in the setting in which they failed to reach the threshold for diagnosis).

Stop-Signal Paradigm

According to the model of Logan and colleagues (Logan & Cowan, 1984; Logan, *et al.*, 1984), the probability of inhibiting a response on a stop-signal trial depends on the outcome of a race between two sets of processes — the go or primary-task processes and the stopping or inhibition processes. If the primary-task processes win the race, the response will occur. If the inhibition or stopping process wins the race, the response will not occur. The interval between the presentation of the primary-task stimulus and the presentation of the stop signal biases the outcome of the race: If the stop signal occurs early enough, subjects never respond; if the stop signal occurs late enough, subjects always respond. In addition, the model predicts that, independent of the speed of the stopping process, more responses will be inhibited if primary-task reaction times are slow rather than fast. An important feature of the change paradigm is that it permits control for the effect on inhibition of variation in primary-task reaction times among individual subjects or groups by defining and presenting the stop-signal interval relative to each subject's mean primary-task reaction time (see Appendix).

The speed and variability of the primary-task processes can be measured directly from performance on trials in which no stop signal is presented (no-signal trials). The stopping process cannot be observed directly. However, the efficiency of the stopping process can be inferred from the subjects' ability to inhibit on stop-signal trials (Logan & Cowan, 1984; Logan *et al.*, 1984). The slope of the inhibition function, which relates the probability of inhibiting a response to stop-signal interval, reflects the efficiency of inhibition. Inhibition functions will be flatter with slower or less efficient inhibitory control. The latency of the internal response to the stop signal or stop-signal reaction time (SSRT) can be calculated from the observed distribution of primary-task reaction times (on no-signal trials) and the probability of inhibition (see Appendix in Tannock, Schachar, & Logan, 1995). Faster SSRT indicates more efficient inhibitory control.

In addition to SSRT, the slope of inhibition functions is influenced by the variability in primary-task reaction time, variability in SSRT, and probability that the stopping process will be triggered. More responses will be withheld at any given SSRT with less variable primary-task latencies. Inhibition functions will be flatter when the SSRT is more variable or if the stopping process is not triggered on a proportion of trials. It is important to distinguish among these factors because the variability in SSRT and the probability of triggering the stopping response reflect the efficiency of inhibitory control, whereas primary-task variability does not.

Logan and Cowan (1984) described a method for distinguishing among the effects of these factors (known as *ZRFT*; see Appendix in Tannock *et al.*, in press). If the inhibition functions of different subjects, groups, or conditions are not equivalent when the probability of inhibition is plotted as a function of ZRFT at each interval, we conclude that the shallower functions represent deficiencies in inhibitory control in addition to longer SSRT; either the inhibitory process is more variable, or it is triggered less often.

Apparatus and Stimuli

The primary-task stimuli were the uppercase letters X and O presented on an Apple IIe computer connected to a specialized Cognitive Testing Station (Digitry Company, Inc., Maine), which allowed direct and precise control of the stimulus presentation, as well as the collection of response times with millisecond accuracy. Each letter, presented one at a time in the center of the screen, was 4 mm wide and 6 mm high and, when viewed at a distance of 1.0 m, subtended 0.23° × 0.34° of visual angle. The stop signal was a 1-kHz tone (beep), 100 ms in duration, generated and presented by the computer. Half of the stop-change signals occurred with an X and half with an O. This

signal was presented after the primary-task stimulus but before the subject's expected primary-task response. On a given stop-signal trial, the beep was presented either 500, 350, 200, or 50 ms before the expected primary-task response time. The details of the stop-signal intervals and the manner in which they were generated are presented in the Appendix.

Each trial began with a fixation point illuminated for 500 ms. It was followed by the letter for that trial, displayed for 1 s and then extinguished. The screen remained blank for an interval of 1.5 s. Thus, each trial included a period of 2.5 s in which the subject could respond to the primary task or to the stop signal in accordance with the task's demands.

The test trials were presented in six blocks of 48 trials (total of 288 test trials), with a short break between the third and fourth blocks. The two letter stimuli and each stop-signal interval occurred equally often in each block. The combination of letter and interval was counterbalanced approximately within blocks and completely across adjacent blocks. Stop signals were presented in 25% of the trials (72 trials), occurring equally often at each of four stop-signal intervals, so that a total of 18 stop signals occurred at each stop-signal interval. The sequence of primary-task stimuli, stop signal, and stop-signal intervals was pseudorandomized with the following constraints: No more than three stop signals could occur in succession (and each stop-signal interval should be equally likely to follow immediately each other interval, but the same stop-signal interval should not occur on successive trials); and the positions of specific stop-signal intervals within given blocks should be evenly distributed, so that particular intervals would not be concentrated in particular portions of blocks. The six-block section of test trials lasted approximately 30 min.

Procedure

Subjects were tested individually in a quiet room in the presence of an examiner. Preceding the test trials, subjects were given three types of practice blocks, each of which could be performed a maximum of three times. Repetitions of specific practice blocks were carried out at the experimenter's discretion; the goal was the subject's full understanding of the demands of the task to that point.

The first block consisted of 24 presentations of either X or O, and subjects were simply required to press the appropriate response button (either one of the two left-most buttons on a response box, 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. They were told to respond as quickly and accurately as possible.

In the second type of practice block (24 trials), subjects were introduced to the stop signal and were told that upon hearing a beep they should try to withhold their responses. They were instructed that, if a beep was not heard, they should still press the button indicated by the letter on the screen. They were also told not to wait for the stop signal because it would not occur very often. It was explained that the stop signals would occur in such a way that sometimes subjects would be able to stop their responses and sometimes not, and that they should simply attempt to stop their responses on as many stop-signal trials as possible.

In the third type of practice (24 trials), subjects were told that, in addition to withholding their X or O response when they heard a beep, they should press the right-most button on the response box, which was called the *beep button.* They were told to respond as quickly as possible whenever the tone occurred, whether or not they were able to inhibit their response to the letter. The second and third types of practice block were identical in format to the test trial blocks.

Dependent Measures

Response Inhibition. Measures and procedures for data analysis were derived from the horse-race model of inhibitory control (see Appendix; Logan & Cowan, 1984; Logan *et al.*, 1984). The change paradigm yields the same measures of inhibitory control as the stop-signal paradigm: (1) the slope of the inhibition function (p-inhibit slope) generated from the probability of inhibition plotted against the stop-signal interval, which provides an index of inhibitory control; (2) the mean SSRT, which is a measure of the latency of the internal response to the stop signal; and (3) the ZRFT slope, which determines the effect on inhibition functions of variability in the latency of the inhibitory control process (SSRT variability) and the probability with the inhibitory control process is triggered while it controls for the variability in primary-task latency (see Appendix in Tannock *et al.*, in press, for details of all measures).

Response Re-Engagement of Secondary Task. Secondary response reengagement was measured directly as the mean latency and variability of the response to the change task, given the successful inhibition of the primary-task response [change-RT (RT = response time) and change-SD (SD = standard deviation), respectively].

Primary-Task Response. In the change paradigm, the processes involved in execution of the primary responses were measured directly; they included the mean latency and variability of the response to the primary task (primary-RT and primary-SD).

Statistical Analysis

Because the study was designed to replicate and extend our previous research, we were interested in three specific hypotheses about the difference among ADHD context-specific subgroups and normal controls. Specifically, we were interested in comparing (1) normal controls and all ADHD subjects combined into a single group (pervasive, home-only, and school-only); (2) normal controls and subjects with pervasive ADHD; and (3) subjects with home-only ADHD and school-only ADHD. The first comparison addressed the presence of deficits among all ADHD subjects and arose from the observation in our previous study that ADHD subjects had deficient inhibitory control compared with that of normal children. This is the comparison typical of most studies of subjects with ADHD in which normal control subjects are compared with ADHD groups consisting of various context-dependent subtypes of ADHD. The second contrast addressed the presence of a deficit in subjects with pervasive ADHD. The third contrast examined differences in the two subtypes of situational ADHD — school-only and home-only ADHD.

Accordingly, three focused F tests (contrasts or planned comparisons with numerator $df = 1$) were applied to each dependent measure, rather than an overall analysis of variance and an omnibus F test with numerator degrees of freedom greater than 1, which would need to be followed by multiple *post hoc* comparisons (e.g., six would be required in the present study). Each contrast was tested against the error term from the omnibus F.

Contrast analysis affords much greater statistical power and clearer substantive interpretation of research results than nonfocused omnibus tests (Keppel, 1982; Rosenthal & Rosnow, 1985). Homogeneity of variance for each comparison was tested with the Bartlett Box F test and separate variance estimates were used for contrasts with nonhomogeneous variance. Pearson correlations were used to examine the association of IQ, and execution, inhibition, and re-engagement of action.

The symptom severity and clinical characteristics of ADHD subgroups were compared in a one-way ANOVA. Newman-Keuls (Winer, 1971) were conducted to locate differences among subgroups because we had no *a priori* hypotheses about symptom severity and clinical characteristics.

RESULTS

Reliability and Validity of Diagnostic Method

In 20 PICS drawn from a previous sample (Schachar & Logan, 1990a) and in 15 assessments from the current sample, interrater agreement for diagnosis of ADHD, ODD or CD, and ED was 100%. Some

disagreement on ratings of individual symptoms was noted but did not affect diagnostic agreement. On the TTI, substantial agreement was obtained between two raters for diagnoses of ADHD (k = .76), ODD (k = .76), and CD (k = .83).

Convergent validity of the PICS and TTI was moderate to high. For example, ADHD symptom counts derived from the PICS and the OCHS scale completed by the parent (based on the number of symptoms that were rated as "often or very true") were highly correlated (.65, $p < .001$), and the same was true for counts of ODD (.68, $p < .001$), CD (.44, $p < .001$), and ED (.49, $p < .001$) symptoms. Agreement between the TTI and teacher OCHS scale symptom dimensions was even greater: ADHD, .74 ($p < .001$), ODD, .66 ($p < .001$), and CD, .55 ($p < .001$).

Groups did not differ in mean age (9.2 years) [$F(3, 51) < 1$, n.s.], mean IQ (107) [$F(3, 51) < 1$, n.s.] or, mean WRAT-R reading or arithmetic scores (Table I).

As shown in Table II, ADHD subgroups differed in the number of ADHD, ODD, CD, and ED symptoms reported by their parents and teachers. However, there was no evidence that severity of the ADHD confounded the ADHD subtype. The pervasive ADHD subgroup did not exhibit a greater number of ADHD symptoms than their situational ADHD counterparts within a specific context.

Moreover, inspection of mean symptom counts suggests that subjects with school-only and home-only ADHD did not differ in the severity of their symptoms either in the setting that generated their diagnosis or in the other setting.

Although subjects with situational ADHD (home-only or school-only) did not reach the diagnostic threshold for ADHD in one of the two settings (e.g., school setting for the home-only ADHD group), they had more ADHD symptoms than normal control children across both the situation that generated their diagnosis and the situation in which they failed to meet the diagnosis. However, there were no subjects who just failed to meet the criteria for pervasive ADHD (i.e., by scoring more than eight ADHD symptoms on either the PICS or TTI and only seven on the other interview); only three subjects obtained ADHD scores of eight or more on one measure and six on the other.

There was a nonsignificant trend for the parents to report more symptoms of ED for subjects with home-only ADHD than for either subjects with school-only or pervasive ADHD. Only two children met the diagnostic criteria for ED: one subject with pervasive and one with home-only ADHD. In both cases, the diagnosis was overanxious disorder.

Table I. Mean Age, IQ, Reading, and Arithmetic Scores for Subjects in Each ADHD Subgroup and Normal Controls

	Mean scores (standard deviations)[a]							
Measure	Normal controls (22)		Home-only ADHD (10)		School-only ADHD (9)		Pervasive ADHD (14)	
Age	9.2	(1.5)	9.4	(1.5)	9.8	(0.8)	8.7	(1.1)
IQ	111.8	(12.2)	105.8	(16.4)	105.1	(12.8)	103.1	(11.9)
WRAT-R								
Reading	100.9	(16.2)	95.5	(13.3)	97.6	(18.5)	92.5	(17.1)
Arithmetic	99.3	(12.6)	97.6	(9.1)	89.0	(8.8)	96.2	(7.0)

[a] There were no significant intergroup differences for any variable. ADHD = attention deficit hyperactivity disorder.
[b] WRAT-R = Wide-Range Achievement Test — Revised.

Table II. Mean Number of Symptoms on Parent and Teacher Interviews and on the OCHS Scale for Subjects in Each ADHD Subgroup and Normal Controls[a]

	Mean scores (standard deviations)				
Informant scale	Normal controls (22)	Home-only ADHD (10)	School-only ADHD (9)	Pervasive ADHD (14)	Significant contrast (df = 51)
Parent Interview					
ADHD	1.5 (1.7)	10.2 (2.0)	4.1 (2.6)	10.4 (2.4)	PER, HO, SO > NC; PER, HO > SO
ODD	0.5 (0.9)	3.0 (2.6)	1.2 (1.0)	2.1 (1.5)	PER, HO > NC; HO > SO
CD	0.1 (0.3)	0.3 (0.5)	0.0 (0.0)	0.7 (0.8)	PER > NC : PER > SO
ED	0.6 (0.7)	1.9 (1.9)	0.7 (0.7)	1.0 (1.6)	
OCHS scales					
ADHD	0.5 (1.0)	6.3 (4.4)	3.3 (1.1)	6.8 (2.8)	PER, HO, SO > NC; PER, HO > SO
ODD	0.3 (0.7)	2.2 (2.8)	0.8 (0.8)	2.1 (2.1)	PER, HO > NC
CD	0.1 (0.2)	0.3 (0.7)	0.0 (0.0)	0.6 (0.8)	PER > NC; PER > SO
ED	0.7 (1.0)	3.0 (1.4)	1.4 (0.7)	3.4 (2.3)	PER, HO > NC; PER > SO
Teacher Interview					
ADHD	0.9 (1.3)	4.1 (2.3)	10.3 (1.9)	11.3 (1.7)	PER, HO, SO > NC; PER, SO > HO
ODD	0.4 (1.3)	1.6 (1.7)	2.8 (2.4)	2.8 (2.4)	PER, SO > NC
CD	0.0 (0.0)	0.5 (0.5)	0.3 (0.7)	0.6 (0.9)	PER > NC
OCHS scales					
ADHD	0.5 (1.4)	3.3 (3.8)	5.1 (2.1)	6.6 (4.8)	PER, SO, HO > NC; PER > HO
ODD	0.1 (0.2)	1.3 (1.9)	1.3 (1.7)	1.2 (1.5)	PER, SO > NC
CD	0.0 (0.0)	0.6 (1.6)	0.0 (0.0)	0.2 (0.6)	
ED	0.6 (1.7)	1.3 (1.6)	1.4 (1.2)	1.7 (1.3)	

[a] ADHD = attention deficit hyperactivity disorder symptoms, ODD = oppositional defiant disorder symptoms, CD = conduct disorder symptoms, ED = emotional disorder symptoms, PER = pervasive ADHD, HO = home-only ADHD, SO = school-only ADHD, NC = normal controls. The number of subjects is enclosed in parentheses.

Change-Paradigm Performance

The scores of measures of response inhibition, re-engagement, and primary-task response for each group are presented in Table III. The probability of inhibition at each stop-signal interval for each diagnostic group is presented in Fig. 1.

Response Inhibition. When all three ADHD subgroups were considered as a single group, they were not significantly different from normal subjects in inhibitory control (mean SSRT, inhibition slopes, ZRFT). However, differences in inhibitory control among subgroups were noted. Compared with normal controls, the pervasive subjects had significantly flatter inhibition slopes (Fig. 1a) [$t(51) = 2.0, p < .05$] and were on average, 120 ms slower in their mean SSRT [$t(51) = 2.0, p < .05$]. The fact that differences in inhibition slopes were eliminated when the probability of inhibition at each stop-signal interval was plotted against ZRFT [$t(51) < .1$, n.s.] (Fig. 1b) indicates that the inhibition processes of pervasively ADHD subjects were slower but not any more variable or any less likely to be triggered. The p-inhibition slopes and SSRT of school-only subjects fell midway between the scores for normal controls and pervasive ADHD subjects but did not differ significantly from those of the home-only group.

Response Re-engagement of Secondary Task. Subjects with ADHD, considered as a single group, demonstrated deficient response re-engagement. ADHD subjects had longer mean change-task RT [$t(51) = 2.9, p < .01$], and more variable change-task RT [$t(51) = 4.0, p < .01$]. However, compared with normal controls, the subjects with pervasive ADHD showed the worst change-task performance of all ADHD subgroups. The subjects with pervasive ADHD were slower [$t(51) = 3.4, p < .01$] and more variable [$t(51) = 3.8, p < .001$] in their change-task RT compared with those of normal controls. Compared with normal controls, the pervasive ADHD subjects were 140 ms slower. No significant differences in change-task performance were noted in the latency of change-task responses between subjects with school-only and home-only ADHD, but the responses of the school-only subgroup were significantly more variable than those of the home-only subgroup [$t(51) = 2.1, p < .05$].

Primary-Task Response. Compared with normal controls, the combined ADHD subjects had more variable primary-task RT [$t(51) = 2.3, p < .05$]. No difference was observed between normal and the combined ADHD subjects in speed of primary-task responses. However, the pervasive ADHD group were, on average, 100 ms slower [$t(51) = 2.3, p < .05$], and more variable [$t(51) = 3.1, p < .01$] than normal controls in the execution of primary-task responses. Home-only and school-only groups did not differ in speed or in variability of their primary-task responses.

Table III. Cognitive Performance: Response Inhibition, Re-Engagement, and Primary-Task Response Measures for Subjects in Each ADHD Subgroup and Normal Controls[a]

Cognitive measures	Mean scores (standard deviations)								Planned contrast[b] *F* value		
	Normal controls (22)		Home-only ADHD (10)		School-only ADHD (9)		Pervasive ADHD (14)		1	2	3
Inhibition											
P-inhibit slope	13.1	(4.0)	12.2	(4.8)	10.2	(4.2)	9.7	(6.5)	1.7	2.1[c]	.9
SSRT	354.9	(94.1)	383.4	(152.6)	425.2	(189.0)	472.3	(258.6)	.5	2.0[c]	1.5
ZRFT slope	26.1	(9.1)	27.4	(9.6)	22.2	(9.3)	24.1	(12.8)	.6	.6	1.1
Re-engagement											
Change-RT	571.6	(124.5)	616.5	(112.9)	682.7	(113.9)	712.6	(144.5)	3.3[d]	2.8[d]	1.1
Change-SD	147.8	(58.7)	172.3	(54.0)	245.1	(85.6)	225.4	(103.7)	3.1[d]	3.0[d]	2.1[c]
Primary-task											
Primary-RT	718.8	(137.1)	777.5	(120.9)	747.6	(151.6)	840.7	(202.1)	1.6	2.3[c]	.4
Primary-SD	199.0	(57.5)	229.9	(57.7)	233.6	(87.4)	280.8	(102.0)	2.3[c]	3.1[d]	.1

[a] ADHD = attention deficit hyperactivity disorder; SSRT = stop-signal reaction time; RT = response time; SD = standard deviation. For explanation of ZRFT, see text.

[b] Planned contrasts: 1 = normal controls versus all ADHD subjects combined into a single group; 2 = normal control versus pervasive ADHD; 3 = home-only ADHD versus school-only ADHD.

[c] $p < .05$.

[d] $p < .01$.

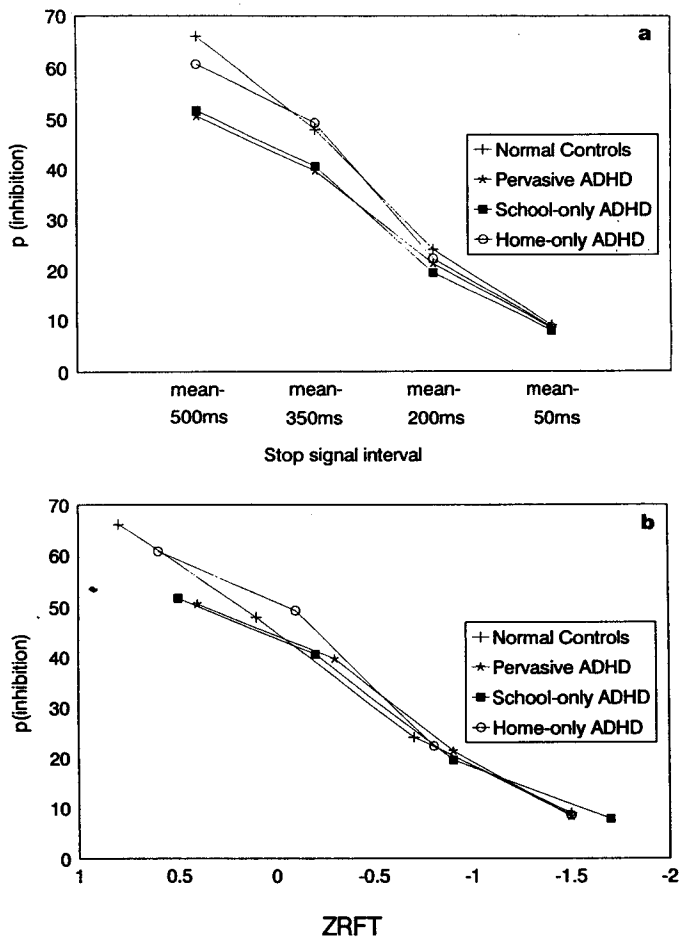

Fig. 1. (A) Probability of inhibition at each stop-signal interval for subjects in each ADHD subgroup and normal controls. (B) Probability of inhibition by ZRFT for subjects in each ADHD subgroup and normal controls. ADHD = attention deficit hyperactivity disorder. For explanation of ZRFT, see main text.

Association with IQ. Across all subjects, IQ was significantly correlated with median primary-task RT (-.31, $p < .01$) but not with p-inhibition slope (-.09), ZRFT slope (-.15), mean SSRT (.03), or mean secondary-task RT (-.02). Primary-task reaction time was correlated with change-RT (.32, $p < .01$), but SSRT was not correlated with either primary-task reaction time (.17) or with change-RT (.24).

DISCUSSION

In this experiment, we used the change paradigm to examine directly the efficiency of the internally generated acts of control involved in the inhibition and re-engagement of action. Inhibitory control and the ability to shift rapidly to the execution of an alternative response are executive control functions of the cognitive system. Executive control functions determine how various mental processes (e.g., encoding, recognition, retrieval) with work together in the performance of a task. Children require executive control to choose, construct, execute, and maintain optimal strategies for performing a task, as well as to inhibit and alter strategies that become inappropriate. The change paradigm assesses two of these functions — inhibition and re-engagement — and is analogous to a wide range of situations in which a discrete course of action must be stopped and altered in response to an error or to changing circumstances. Consequently, inferences about the control required in the change paradigm may be generalized to a range of similar circumstances. Moreover, the control involved in these circumstances may differ only in degree from more subtle acts of control in which action is adjusted but not stopped (see Logan, in press).

The central finding of the change paradigm used in this study is that children with pervasive ADHD have a deficit in inhibitory control. The results of the current study replicate and extend the findings of our earlier research (Schachar & Logan, 1990a): We observed that deficient inhibitory control in children with ADHD was most evident in those children with pervasive symptoms. In the current experiment, we replicated our earlier finding of deficient inhibitory control in a larger sample of subjects with pervasive ADHD than in the previous study 14 vs. 5). In addition to demonstrating deficient inhibitory control in subjects with ADHD, these results indicate that pervasive ADHD is associated with a deficit in a second executive control process — the engagement of an alternate action after inhibition of an ongoing action.

It is more difficult to determine whether inhibitory control differs in kind or in degree among subjects with pervasive, school-only, or home-only ADHD. In the previous study (Schachar & Logan, 1990a), there were too few subjects with home-only and school-only ADHD for adequate comparison. In the current study, the deficit in inhibitory control was greatest in subjects with pervasive ADHD. However, the school-only subgroup was rather similar to the pervasive group. The home-only ADHD subgroup showed no deficit in inhibitory control but the numbers were too small to reach definite conclusions. Subjects with home-only ADHD comprised one in eight of the subjects with ADHD in our clinic

sample, approximately the same proportion as are found in the general childhood population (Szatmari *et al.*, 1989). The distinction between these context-dependent subgroups is interesting and merits further study. For example, will this distinction prove to be stable over time? The validity of these subgroup distinctions depends upon the reliability of the diagnostic methods. We are quite confident in the detailed assessment procedure we used in this study; they were reliable and able to predict scores on an alternative measure of disorder. However, larger samples will be required to reach definitive conclusions.

These results raise two important questions about the specificity of the inhibitory control deficit in children with pervasive ADHD and about the specificity of the psychological processes involved. The results of the previous study (Schachar & Logan, 1990a) indicated that deficient inhibitory control most clearly distinguishes children with ADHD from normally developing children. Children with academic underachievement, ED, or CD in that study (Schachar & Logan, 1990a) may have shown some deficit, but they were not distinguishable from normal children. In addition, deficient inhibitory control was most marked in children with ADHD not associated with CD, rather than in children with combined ADHD and CD. We are currently attempting to replicate this latter finding using the change paradigm. The current sample included only those subjects with ADHD without concurrent CD or severe ODD to replicate the finding of the previous study. The specificity of executive control deficits to ADHD merits further research.

These results also raise questions about the nature of the cognitive deficit underlying ADHD. Is it a generalized deficit in the speed of processing and response, a generalized deficit in executive control, or a specific deficit in inhibitory control? The fact that pervasive ADHD was associated with slower go, stop, and switch processes suggests a generalized deficit in speed of response. Numerous studies have found slower reaction times in subjects with ADHD (e.g., Sergeant & Scholten, 1985a, 1985b).

However, several findings argue against this conclusion and in favor of a specific-deficit hypothesis. First, the association between primary-task reaction time and latency of the inhibitory control process (SSRT) was not significant, suggesting independence of these two processes. Second, we have previously and directly investigated the hypothesis that a generalized deficit in attentional capacity might impair the ability to detect and respond to the stop signal in the stop-signal paradigm (Schachar and Logan, 1990b). To do this, we used a dual-task version of the stop-signal paradigm. The dual-task paradigm used the same stimuli as the stop-signal paradigm, only the response requirements differed. In the dual-task paradigm, subjects responded to both primary and stop signals (secondary task). The amount

of dual-task interference provided a measure of residual attentional capacity. We found no evidence of deficient attentional capacity in subjects with ADHD. Third, in the current study, we have found only a modest correlation between IQ and measures of inhibitory control, but a much stronger association of IQ with primary-task latency. Fourth, in an acute trial of methylphenidate in a separate sample, we found a different dose-response relationship for measures of inhibition than was found for measures of primary-task and secondary-task latencies (Tannock *et al.*, 1995). Finally, the change paradigm (as well as the stop-signal paradigm) is designed to control for differences in primary-task reaction time. Stop-change tones are presented in relationship to each subject's mean primary-task reaction time. Moreover, the ZRFT correction allows us to determine whether differences in primary-task reaction-time variability account for differences in inhibition. According to the results of this experiment, primary-task variability does not account for observed differences in inhibitory control.

Are the deficits in inhibitory control and response re-engagement related or independent? The fact that both processes are deficient in children with pervasive ADHD suggests a generalized deficit in executive control, as does a previous finding that pervasive ADHD is associated with a deficit in sustained attention (Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989).

However, there is evidence supporting the proposition that multiple independence deficits exist in subjects with ADHD. In the current experiment, independence of the processes involved in inhibition and re-engagement was suggested by the nonsignificant correlation of SSRT and change-RT. Also, a previous trial of methylphenidate conducted by Tannock *et al.* (1995) demonstrated different dose–response relationships for the processes of inhibition and response alteration. Finally, previous research (De Jong, Coles, Logan, & Gratton, 1990; Jennings, van der Molen, Brock, & Somsen, 1992; Logan & Burkell, 1986; Logan & Cowan, 1984) on adults with the stop-signal paradigm indicates that the response inhibition and response re-engagement (secondary-task process) are independent. Although the observation of independence between these processes needs to be confirmed in children, it is reasonable to conclude that the deficits in inhibition and re-engagement represent distinct deficits rather than a common one.

Assuming that these differences among subjects with ADHD context-specific subtypes are confirmed, these observations have several implications for research practice. It is likely that the combination of context-dependent ADHD subtypes may obscure important differences in inhibitory control or in other aspects of cognitive performance. As noted in these results, if all ADHD subtypes are combined, the combined ADHD groups do not differ in inhibitory control from normal control children.

One solution would be to limit study samples to pervasively ADHD children as has been done in many previous studies (e.g., Abikoff, Gittelman, & Klein, 1980; Aman & Turbott, 1986; Seidel & Joschko, 1990; van der Meere & Sergeant, 1987, 1998a, 1988b; van der Meere, Wekking, & Sergeant, 1991). This solution ignores the large proportion of children with ADHD who exhibit school-only ADHD (Szatmari *et al.*, 1989). Based on current results, it may be premature to conclude that school-only ADHD is not associated with deficient executive control. Alternatively, investigators could select subjects basely solely on the reports of a single informant. This strategy would exclude some children with ADHD who are symptomatic in one situation only and would not permit a distinction between situational and pervasive ADHD (e.g., Atkins, Pelham, & Licht, 1985; Dykman, Ackerman, & Oglesby, 1979; Goldstein, 1987a, 1987b; Rosenthal & Allen, 1980; Sergeant & Scholten, 1985a, 1985b).

The executive control deficits observed in this study provide a potential cognitive model of the deficit underlying pervasive ADHD. Deficient inhibitory control results in a greater likelihood that a response will escape control and be executed. As a result, it is reasonable to assume that persons with deficient inhibitory control, as measured by the change or stop-signal paradigm, will appear impulsive in a range of circumstances that demand that an action be stopped. Deficient ability to execute an action after the inhibition of an ongoing action should contribute to the appearance of inattentiveness in situations requiring flexible and rapid shifting of attention from one response to another as the situation demands. Typically, these situations are problematic for children with ADHD.

However, deficient inhibitory control cannot account for the impulsive behavior of all ADHD children. All three subgroups of ADHD — pervasive, school-only, and home-only situational ADHD — were equally symptomatic, and children in all three groups met current diagnostic criteria for ADHD. Yet only the pervasive group and, to a lesser extent, the school-only situational group exhibited deficient inhibitory control. Apparently, it is possible to exhibit the symptoms of ADHD but not a deficit in inhibitory control. Presumably, many factors are implicated in the genesis of impulsive behavior including task comprehension; processes involved in delaying, preparing, initiating, and executing ongoing responses; processes involved in interrupting and altering an ongoing response; and effects of reward and punishment. Impulsiveness could arise if subjects respond before they have established the correct course of action because of a lack of comprehension of a task. Impulsiveness could occur as well if subjects prepare, initiate, or execute their responses more quickly than others; if

they are less able to stop or alter their actions once initiated; if they are overly attracted by rewards; or if they are insensitive to punishment.

These results confirm the conclusions based on the performance of children with ADHD on a range of measures of inhibitory control. For example, subjects with ADHD make more fast errors on the Matching Familiar Figures task (i.e., more false-positive errors on the Continuous Performance Task; Halperin *et al.*, 1988) and on the go–no-go task (Trommer, Hoeppner, Lorber, & Armstrong, 1988). Subjects with ADHD also make more perseverative errors in the face of changing task demands on the WCST (Chelune *et al.*, 1986; Dyme *et al.*, 1982; Gorenstein *et al.*, 1989). It is possible that deficient inhibitory control is the factor that underlies the performance deficits in these diverse tasks.

The race model of inhibitory control proposed by Logan and Cowan (1984) highlights the limitations of these traditional methods of measuring inhibitory control. The race model demonstrates that the probability that a response will be stopped or controlled depends not only on the efficiency of the inhibitory control processes (speed, variability, and probability of triggering) but also on the speed and variability of the ongoing action. Traditional measures do not take into account the speed and variability of primary-task processing the way that the change and stop-signal paradigms do. According to the race model, the slower primary-task responses associated with ADHD will result in an underestimation of the inhibitory control deficit of some children. Consequently, traditional measures can be descriptive only and cannot clarify the nature of underlying deficits.

One of the main questions for further research in inhibitory control in subjects with ADHD concerns the nature and locus of the response inhibition process (see De Jong *et al.*, 1990; Jennings *et al.*, 1992; Shue & Douglas, 1992).

There has been a shift in the literature from support for an attention or cognitive deficit to the postulation of a deficit in motivation underlying the problems of children with ADHD (Barkley, 1990; Douglas, 1988; Draeger, Prior, & Sanson, 1986; Henker & Whalen, 1989; Prior & Sanson, 1986; Sergeant, 1988; Sergeant & van der Meere, 1990). Although factors such as poor motivation undoubtedly play a part in determining the clinical manifestations of the disorder, the results of the current study are in accord with the argument that suggests that it is premature to abandon the hypothesis that ADHD is characterized by a deficit in higher-order cognitive functions and endorse the deficient-motivation hypothesis (e.g., Barkley, 1994; Douglas, 1988; Quay, 1988).

APPENDIX

Setting Stop-Signal Delay

In the change paradigm, differences between individuals, conditions, or groups in the speed of performing the primary task are controlled by presenting the stop-signal in relation to each subject's mean primary-task reaction times (see Logan, in press, for alternative methods of setting the stop-signal interval). Each subject's mean reaction time is calculated and stop-change signals are presented at fixed intervals before this point in time. No matter how slow or how fast a subject's mean primary-task response times, the race between the primary-task and the stopping response is biased to an equivalent extent. With this method, we can equate the opportunity or time available to inhibit for each subject. Consequently, the probability of successfully withholding a response when a stop-change signal occurs reflects inhibitory control rather than primary-task latencies. Any response that was stopped after the presentation of the stop signal is considered a successful inhibition of response. To control for differences between subjects in strategy (e.g., a subject may have held back a response in an attempt to increase the probability of inhibiting), a stop-signal delay was defined as one of four present intervals inserted between the stop-change signals stimulus and the subject's expected primary-task response time.

The estimate of the expected primary-task response time for a given trial was calculated from the average of the response times in the immediately preceding correct-response, nonsignal trials. More specifically, a queue of 36 primary-task reaction times was kept to generate a running calculation of expected response time. As each new primary-task response time was recorded, the last one in the queue was dropped from the calculation. On a given stop-signal trial, the beep was presented either 500, 350, 200, or 50 ms before the expected primary-task response time. Thus, there was an interval of 50 to 500 ms between the stop-change signal and the subject's typical response.

P-Inhibition Slope

The p-inhibition slope is generated when p-inhibition is plotted against the stop-signal interval. In this study, we plotted the corrected p-inhibition by stop-signal interval. The correction is necessary because children in general, particularly children with a diagnosis of ADHD, fail to respond some of the time, either as a result of an active strategy or inattentiveness. For

example, subjects might try to increase the p-inhibition by deciding before the trial not to respond, independent of stimulus events. Since some of these omissions may occur on stop-signal trials, the observed p-inhibition may reflect both omissions and true response inhibition. This effect extended across all stop-signal intervals would cause an artifactual increase in the height and steepness of inhibition functions. Consequently, we corrected the p-inhibition at each interval for the percent of omissions observed on nonsignal trials, using the following formula:

$$y = \frac{x - o}{(N - q) - o}$$

where y is the corrected number of inhibited trials at a specific interval, x is the observed number of inhibited trials at that interval, and o is the correction for the number of omissions on no-signal trials. N is the total number of stop-signal trials at each interval ($N = 18$ in the current study), and q is a correction for the number of trials at each interval that were executed but in an inappropriate fashion (e.g., early responses, response to the tone followed by response to the primary-task stimulus; response to tone before tone presentation but no response to primary-task stimulus).

We calculated the correction for the number of omissions, o, according to the following formula:

$$o = \frac{n}{p - t}$$

where n is the number of no responses on no-signal primary-task trials, p is the total number of no-signal primary-task trials ($p = 216$ in this study), and t is the number of responses to the secondary task on no-signal primary-task trials. We subtracted t from the total number of primary-task because t indicates that subjects perceived the primary-task stimulus but incorrectly responded to the secondary task. All subsequent analyses were conducted on the corrected probability of inhibition.

SSRT

Acts of control, like any other acts, must take time. Even though it cannot be observed directly, the latency of the response inhibition process — the SSRT — can be calculated from the observed distribution of primary-task reaction times (on trials without stop-change signals) and the probability

of inhibition (see Logan & Cowan, 1984). More specifically, the distribution of primary-task reaction times on no-signal trials was rank ordered and the *n*th fastest value was determined; *n* is the number of responses in the primary-task distribution multiplied by 1 minus the corrected probability of inhibiting on stop-signal trials. The *n*th value provided an estimate of the reaction time to the stop signal relative to the onset of the primary-task stimulus. We obtained an estimate of stop-signal reaction time by subtracting out the stop-signal interval. SSRT was estimated at each of the four stop-signal intervals and we took the median of these four values as the SSRT.

ZRFT Slope

In addition to being slow, a response inhibition process also may be variable or the inhibition process may not be triggered. Logan and Cowan (1984) described a correction that permits examination of the effect of variability in the SSRT and of the probability of triggering a stopping response on the probability inhibition while controlling for the effect of variability in primary-task reaction time (see Logan & Cowan, 1984). The probability of inhibition is plotted as a function of a *Z* score that represents the relative finishing time of the primary-task and inhibition processes in standard deviation units, and uses the primary-task reaction times to define the units delay minus SSRT:

$$ZRFT = \frac{interval - SSRT}{SDRT}$$

where ZRFT is the relative finishing times of the stopping and the primary-task processes, expressed as a *Z* score; interval is the period between the presentation of the stop signal and the subject's mean primary-task reaction time; SSRT is the estimated stop-signal response time; and SDRT is the standard deviation of the primary-task response times. ZRFT is calculated for each interval. This procedure is repeated for every stop-signal interval.

If inhibition functions from different individuals, groups, or conditions are not equivalent when the p-inhibition is plotted as a function of ZRFT at each interval, then we conclude that the shallower functions represent deficiencies in inhibitory control: Either the inhibitory process has more variability or it is triggered less often. If inhibition functions are equivalent when plotted by ZRFT, any differences in the latency of the inhibition process (SSRT) or in primary-task variability account for differences in inhibition functions.

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