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INHIBITORY CONTROL, IMPULSIVENESS, AND ATTENTION DEFICIT HYPERACTIVITY DISORDER

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ABSTRACT. *This article describes a new measure for studying inhibitory control, the stop signal paradigm, and the race model of inhibitory control of action (Logan & Cowan, 1984) on which it is based. This measure and model permit distinction and measurement of various processes which determine whether or not an action can be inhibited. Three studies are described that find a deficit in attention deficit hyperactivity disorder (ADHD) in inhibitory control of an ongoing action and in the processes involved in the reengagement of an alternative action following inhibition of an ongoing action. No evidence of deficient attentional capacity was found in ADHD that could account for these deficits. These deficits were most pronounced in children who had ADHD according to reports of both parent and teacher (pervasive ADHD) compared to those whose ADHD was reported by either parent or teacher but not both. No deficit was evident in children with conduct disorder (CD) or in those with a combined presentation of ADHD and CD despite the fact that these groups were characterized by clinical impulsiveness. The article discusses the implications of these findings for models of the relationship of cognitive deficit and behavior.*

The concept of impulsiveness is central to explanations of various problems of childhood including academic difficulties (Blackman & Goldstein, 1982; Walker, 1985), poor peer relationships (Milich & Landau, 1982; Pelham & Bender, 1982), and consequences of frontal lobe pathology (Shue & Douglas, 1992). In particular, impulsiveness has been important in defining and explaining the two most common psychiatric conditions of childhood, attention deficit hyperactivity disorder (ADHD; Douglas, 1983, 1988; Quay, 1988a, 1988c) and conduct disorder (CD; Loeber, 1990; Quay, 1988b, 1989). Both

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ADHD and CD are classified in DSM-III-R as disruptive behavior disorders (American Psychiatric Association, 1987) and both are characterized by impulsive behavior.

Although impulsiveness is only one of the features of ADHD, it seems to be particularly important in defining the syndrome (Frick et al., 1993). Compared to non-ADHD children, those with a diagnosis of ADHD exhibit various behaviors that suggest impulsiveness. They are thoughtless and disruptive in social situations, careless and inaccurate on academic tasks, and reckless and accident prone at play (Milich & Kramer, 1984; Pelham & Bender, 1982). Often ADHD children have great difficulty regulating their behavior in accord with the wishes or instructions of adults and are considered oppositional or defiant.

CD is characterized by a range of behavior in which the rights of others and major societal norms are violated. Behavioral impulsiveness is not a diagnostic requirement of CD. However, children with CD behave with apparent disregard for the consequences that their behavior might have on others or even on themselves despite their ability to verbalize the potentially negative consequences of their actions (Quay, 1986). Consequently, their behavior has been considered to be impulsive or disinhibited (e.g., Gorenstein & Newman, 1980).

THE MEANING OF IMPULSIVENESS

Despite the importance of impulsiveness to theories of child psychopathology, there is little agreement on the precise definition of the phenomenon or on the nature of the deficit or deficits that determine impulsive behavior (Milich & Kramer, 1984). Impulsiveness may refer to actions that are executed too quickly or in an unreasoned way, actions that cannot be withheld while deliberations proceed, behavior directed in a deliberate fashion toward obtaining immediate gratification at the expense of longer term goals, or actions that cannot be stopped or altered once they are initiated even if the consequences of the action might be undesirable or unpleasant.

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. Consequently, impulsiveness could arise under various circumstances. Individuals may appear impulsive if they respond before they have established the correct course of action due to lack of comprehension of a task; if they prepare, initiate, or execute their responses more quickly than others and possibly before a signal to stop their action has appeared; if they are overly attracted by rewards or insensitive to punishment; or if they are less able to stop or alter their actions once initiated.

SCOPE OF OUR RESEARCH

Our primary interest has been in the inhibitory control process involved in the stopping of an ongoing response. Inhibitory control is one of the executive control functions of the cognitive system that determine how various mental processes (e.g., encoding, recognition, retrieval) will work together in the performance of a task (Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984). Executive control is required in order to choose, construct, execute, and maintain optimal strategies for performing a task, as well as to inhibit and alter strategies that become inappropriate. Checking a swing at a bad pitch in baseball or stopping oneself from running into traffic are examples of situations requiring inhibitory control. In addition to changes in the external environment, individuals might

have to stop their action if they detect an error in their own performance. Detecting and correcting errors on academic tasks might provide an example of such a situation. In all of these circumstances, deficient inhibitory control will lead to a greater likelihood that a response will be executed rather than withheld. Consequently, it is reasonable to assume that individuals with deficient inhibitory control will appear impulsive in circumstances requiring stopping of action.

In this article, we describe a laboratory paradigm—the stop signal paradigm—which is our main method for measuring inhibitory control and explain how a particular model of inhibitory control known as the race model (Logan & Cowan, 1984) provides a theoretical framework for the analysis of performance on this task. Also, we describe two modifications of the stop signal paradigm, the change paradigm and the dual task paradigm, that permit measurement of response reengagement processes and attentional capacity. We review the results of an ongoing program of research into the nature of the cognitive deficit in ADHD (Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989; Schachar & Logan, 1990a, 1990b).

THE STOP SIGNAL PARADIGM

The stop signal paradigm provides a laboratory analogue of common, everyday situations requiring rapid and accurate execution of a thought or action and, on occasion, stopping of this action. In the stop signal paradigm, subjects are engaged in a computer-administered primary task (a forced-choice reaction time task). The subjects' task is to respond as quickly and as accurately as they are able. Occasionally and unpredictably (on 25% of trials), subjects are presented with a stop signal (a tone generated by the computer) that instructs them to withhold their motor response to the primary task. The main datum is whether or not subjects withhold their response to trials on which the stop signal occurred (details of the apparatus, stimuli, and procedures can be found in Schachar & Logan, 1990a).

The stop signal paradigm is advantageous because it affords clear definition of the conditions that trigger the act of control (i.e., presentation of the stop signal) and the changes that result from executing the act (i.e., inhibition of the response). Also, the stop signal paradigm permits direct investigation of the efficiency of the internally generated act of cognitive control involved in stopping of action. As will be explained below, the paradigm allows measurement of the latency of the act of control (i.e., stop signal reaction time).

THE RACE MODEL OF RESPONSE INHIBITION

According to the model of Logan and colleagues (Logan, in press; Logan & Cowan, 1984; Logan et al., 1984), the probability of inhibiting a response of a stop signal trial depends on the outcome of a race between two sets of processes—the go or primary task process and the stopping or inhibition process. The primary task processes begin with the presentation of the imperative or go stimulus and involve stimulus recognition, response choice, and the preparation and execution of the primary task response. The inhibition process commences with the presentation of the stop signal. The relative finishing times of these two processes determine the outcome of the race. 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 model assumes that the stopping and primary task processes are independent, and this seems to be the case when it has been investigated in adults (De Jong, Coles, Logan, & Gratton, 1990; Jennings, van der Molen, Brock, & Somsen, 1992; Logan & Cowan, 1984).

The relative finishing time of the stopping and primary task processes depends upon the speed and variability of the primary task process, the speed and variability of the inhibition process, and the probability that the inhibition process will be triggered. The outcome of the race also depends on the interval between the presentation of the primary task stimulus and presentation of the stop signal. If the stop signal occurs early enough, the subject always inhibits. If the stop signal occurs late enough, the subject always responds. It follows from this model that processes involved in the primary task response and processes involved in stopping responses can compensate for one another. Independent of the speed of the stopping process, more responses will be inhibited if primary task reaction times are slow. The stop signal paradigm has the advantage of distinguishing between primary task and inhibition processes. Variation in primary task reaction times among individuals or groups is controlled experimentally in the stop signal paradigm by presenting stop signals at various intervals *before* subjects' mean primary task reaction time. We call this period of time between the presentation of the stop signal and the subject's mean primary task reaction time "the stop-signal interval." The stop signal paradigm traces each subject's mean reaction time to trials on which no stop signal is presented and presents stop signals at various intervals before the subject's mean primary task reaction time.

Presentation of the stop signal tone at various intervals rather than at a single interval prevents subjects from delaying their responses for a particular amount of time in order to check for the presentation of a stop signal. Also, presentation of multiple stop signal intervals permits plotting of the function relating stop signal interval to probability of inhibiting a response (see Logan & Cowan, 1984 for details). The slope of the inhibition functions relating the probability of inhibiting a response at each stop signal interval provides an index of the efficiency of the inhibition process. In general, steeper inhibition functions indicate better inhibitory control (i.e., greater probability that the stopping process won the race with the primary task process).

The speed and variability of the primary task processes can be measured directly from performance on trials in which no stop signal is presented. The speed of the stopping process cannot be observed directly but can be inferred from the observed distribution of primary task reaction times in no-signal trials and the probability of inhibition on signal trials (Logan & Cowan, 1984; Logan et al., 1984). Stop signal reaction time is the difference between the point at which the stop signal was presented and the point at which the stopping process finished. We know when the stop signal was presented from the experimental protocol. We have to estimate the point at which the stopping process finished from the observed distribution of primary task reaction times in no-signal trials and the observed probability of inhibiting given a stop signal (see Logan, in press; Logan & Cowan, 1984). This is done by rank-ordering the distribution of primary task reaction times on no-signal trials. The nth fastest value is determined, where n is the number of responses in the primary task distribution multiplied by one minus the probability of inhibiting on stop signal trials (see Logan, in press; Logan & Cowan, 1984 for details). This calculation is repeated for each stop signal interval and the mean of these values is calculated. We refer to the latency of the stopping process as the stop signal reaction time (SSRT). A longer SSRT reflects less efficient inhibitory control.

In addition to the speed of the go and stopping processes, the outcome of the race will be influenced by variability in primary task latencies, by variability in SSRT, and by variation in the probability that the stopping process will be triggered in response to the stop signal. Distinguishing among these factors is important because variable inhibition processes and inhibition processes that are irregularly triggered reflect aspects of deficient inhibitory control as does longer SSRT. By contrast, variable primary task processes do

not reflect deficient inhibition. Yet, the less variable the primary task latencies, the greater the probability of successful inhibition at any particular SSRT. Logan and Cowan (1984) provide a method for disentangling these factors. 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, using the standard deviation of the primary task reaction times to define the units (known as ZRFT). If the inhibition functions from different individuals, groups, or conditions are not equivalent when probability of 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 inhibition process is more variable or it is triggered less often. If inhibition functions are equivalent when plotted as a function of ZRFT, then differences in primary task variability along with any observed differences in SSRT account for differences in probability of inhibition.

In summary, the stop signal paradigm and the race model of inhibitory control provide a method for examining the efficiency and speed of an internally generated act of control. A major advantage of this approach is that it permits distinction of inhibitory control from primary task reaction processes which do not reflect inhibitory control but which do influence whether an individual will execute or withhold a response. Details of the model are presented in Logan and Cowan (1984) and in Logan et al. (1984), and a user's guide to the stop signal paradigm is available in Logan (in press).

INHIBITORY CONTROL AND DISRUPTIVE BEHAVIOR DISORDERS

The etiology of ADHD and CD has been the subject of considerable speculation and research. Gorenstein and Newman (1980), for example, hypothesized that ADHD and CD are manifestations of a similar underlying neuropathology that results in a deficit in inhibitory control. In the context of the stop signal paradigm, this hypothesis leads to the prediction that deficient inhibitory control (flatter inhibition slopes, longer SSRT) will be evident equally in ADHD and CD. A contrasting perspective is that ADHD and CD, although similar in many behavioral manifestations, differ in the nature of their underlying deficit. For example, CD may be attributed to overactivity in the neural systems controlling approach, escape, and active avoidance, whereas ADHD may be associated with deficient inhibition (Quay, 1988a). This hypothesis predicts that ADHD, but not CD, will be associated with deficient inhibitory control. However, CD might be associated with shorter primary task reaction times.

Concurrent ADHD and CD

Very often, ADHD and CD occur together in the same child. Approximately 50% of ADHD children present with CD and 50% of CD children present with ADHD (Szatmari, Offord, & Boyle, 1989). Children with a mixed presentation of ADHD+CD seem to be overrepresented in clinic samples (e.g., Stewart, Cummings, Singer, & DeBlois, 1981).

The relationship of pure ADHD (i.e., ADHD without concurrent CD), pure CD (CD without concurrent ADHD), and mixed ADHD+CD is unclear. There is ample evidence that the mixed ADHD+CD group differs from ADHD in family adversity (Schachar & Wachsmuth, 1990), rates of parental and familial psychopathology (Biederman et al., 1992; Schachar & Wachsmuth, 1990), early developmental history (Sanson et al., 1993), and natural history (Barkley, Fischer, Edelbrock, & Smallish, 1990). More important from the perspective of the current research, the group with concurrent ADHD+CD appears to have a pattern of cognitive deficit that is different from that of the pure

ADHD croup (e.g., Chee et al., 1989; van der Meere, Hughes, Burger, & Sallee, 1993). Consequently, the inclusion of subjects with mixed ADHD+CD in studies of ADHD could obscure the correlates of ADHD and CD. In contrast, the results of research on cognition in distinct ADHD, CD, and ADHD+CD groups could shed light on the nature of ADHD+CD. If ADHD+CD represents the interaction of a developmental risk factor (ADHD) and adverse environmental circumstances, then we would expect the mixed ADHD+CD group to exhibit the same cognitive deficits as the pure ADHD group. However, if ADHD were a nonspecific epiphenomenon of CD, we might predict that neither the ADHD+CD and CD groups would exhibit cognitive deficits. In order to pursue these hypotheses in the research described in this article, we distinguished among subjects with pure ADHD, pure CD, and those with concurrent ADHD and CD (mixed ADHD+CD).

DEFINING ADHD AND CD

Our experiments (Schachar & Logan, 1990a, 1990b) were conducted with children referred for assessment of disruptive behavior to the departments of psychiatry or pediatrics of an urban pediatric hospital. Children seen in the hospital for uncomplicated and transient medical problems served as normal controls if they had no learning or behavior problems. All subjects underwent extensive clinical assessment to confirm a diagnosis and to exclude subjects with severe, concurrent medical problems, history of seizures, evidence of psychosis, or with an estimated full-scale IQ of less than 80. Normal control subjects underwent the same assessments and did not meet criteria for any disorder or learning difficulties. The subjects in these experiments were 7–11 years of age (mean age of approximately 9 years). Subjects in the various diagnostic groups had a similar mean estimated full-scale IQ of approximately 105. Any subject who was receiving medication (in all cases methylphenidate) had to be free of medication for at least 48 h before testing.

Diagnostic Procedure and Criteria

Our approach to diagnosis was influenced by research evidence suggesting that the overlap of ADHD and CD might, in part, be a result of the halo effect that arises with subjective diagnostic instruments (Abikoff, Courtney, Koplewicz, & Pelham, 1991; Schachar, 1991; Schachar, Sandberg, & Rutter, 1986). In our studies, one or both parents of each subject were interviewed using the Parent Interview for Child Symptoms (PICS; Schachar & Wachsmuth, 1989), a semistructured interview designed to elicit symptoms relevant to DSM-III-R diagnoses.[1] The PICS interview was developed because we found that assessment based on a detailed description of child behavior was necessary to distinguish symptoms of ADHD from those of CD. In the PICS, parents were asked to describe child behavior in a variety of settings (e.g., at play out of doors, in stores, etc.). For each setting, parents were asked to describe a recent example of their child's behavior and to respond to a series of specified probes about the behavior. Parents's subjective statements about child behavior were not considered; instead, each symptom was rated by the

[1]In Schachar and Logan (1990a), subjects were diagnosed using DSM-III criteria. With the revisions of DSM-III-R, the diagnostic criteria for ADHD, ODD, and CD were changed from those in DSM-III. We were able to reclassify the subjects in Schachar and Logan (1990a) according to DSM-III-R criteria because we had collected comprehensive diagnostic information about each subject. The results described in this article are based on DSM-III-R diagnoses and differ slightly from the results presented in Schachar and Logan (1990a).

interviewer according to defined criteria and on the bases of its severity, age appropriateness, and resultant degree of handicap. Only behaviors that were severe, handicapping, and age-inappropriate were considered diagnostic symptoms. The interrater reliability of this interview has been assessed by having a second interviewer rate a videotaped interview and found to be high (e.g., Schachar & Logan, 1990a).

At the time of our first experiments, no equivalent of the PICS was available for interviewing teachers. Consequently, in the first experiment to be described, we based teacher diagnosis of ADHD on three questionnaires: the Abbreviated Conners Teacher Rating Scale (ACTRS; Conners, 1973; the Rutter B scale (Rutter, 1967); and the SNAP questionnaire (Pelham, Atkins, & Murphy, 1981). For a diagnosis of ADHD, a child had to meet DSM-III-R criteria on the PICS and/or on two of the three teachers questionnaires: (a) an ACTRS score of 15 or more, a score predictive of a clinical diagnosis of hyperactivity (Goyette, Conners, & Ulrich, 1978); (b) a rating of at least 5 out of 6 on the Rutter B scale, a score obtained by 3% of 10-year-old boys in the general population (Schachar, Rutter, & Smith, 1981); and (c) the presence of four inattentive, four impulsive, and three hyperactive symptoms on the SNAP questionnaire, a score obtained by 5% of 10-year-old boys (Pelham, et al., 1981).

There have been various attempts to subdivide the large group of argumentative, defiant, stubborn, rule-breaking children (Loeber, Lahey, & Thomas, 1991; Quay, 1986). For example, DSM-III distinguished between oppositional disorder (OD) and CD. OD was criticized on the grounds that it had not been a validated diagnostic category that could apply to many children with relatively minor difficulties (Rutter & Shaffer, 1980). DSM-III-R included a category of oppositional defiant disorder (ODD) that required a greater number of symptoms to be present for a diagnosis. In comparison, the *International Classification of Diseases*, 9th revision (World Health Organization, 1978), makes no similar distinction between ODD and CD. Currently, the validity of ODD is unclear, but it is thought to be a developmentally earlier, possibly less severe but qualitatively similar form of CD (Anderson, Williams, McGee, & Silva, 1987; Reeves, Werry, Elkind, & Zametkin, 1987; Rey et al., 1988; Schachar & Wachsmuth, 1990; Werry, Methven, Fitzpatrick, & Dixon, 1983; Werry, Reeves, & Elkind, 1987). In our experiments, we combined subjects with *severe* oppositional disorder and those with CD into a single group. For subjects to be considered as having a severe oppositional disorder they had to meet criteria for the disorder both in and out of the home, with school being the typical out-of-home situation in which children met criteria. In other words, a parent-child problem that was not associated with similar difficulties in the child's relationship with other adults was not considered diagnostic of ODD. Of course, the requirement of pervasiveness is only one way of limiting the diagnosis to the most severe cases.

EVIDENCE OF DEFICIENT INHIBITORY CONTROL IN ADHD

In the first study to be described, the stop signal paradigm was used to examine inhibitory control in children with ADHD, CD, and ADHD+CD and in normal controls. Details of the results can be found in Schachar and Logan (1990a). Only a portion of the data from that study is shown in Figure 1.

Figure 1 presents the mean latency of primary task responses and mean SSRT for each group as well as the mean probability of inhibition as a function of the interval between subjects' mean primary task reaction time and the presentation of the stop signal. The findings indicate that the normal control, CD, ADHD+CD, and ADHD groups did not differ in the speed of the primary task process. However, longer SSRT and flatter inhibition slopes in the ADHD group indicated deficient inhibitory control compared to normal

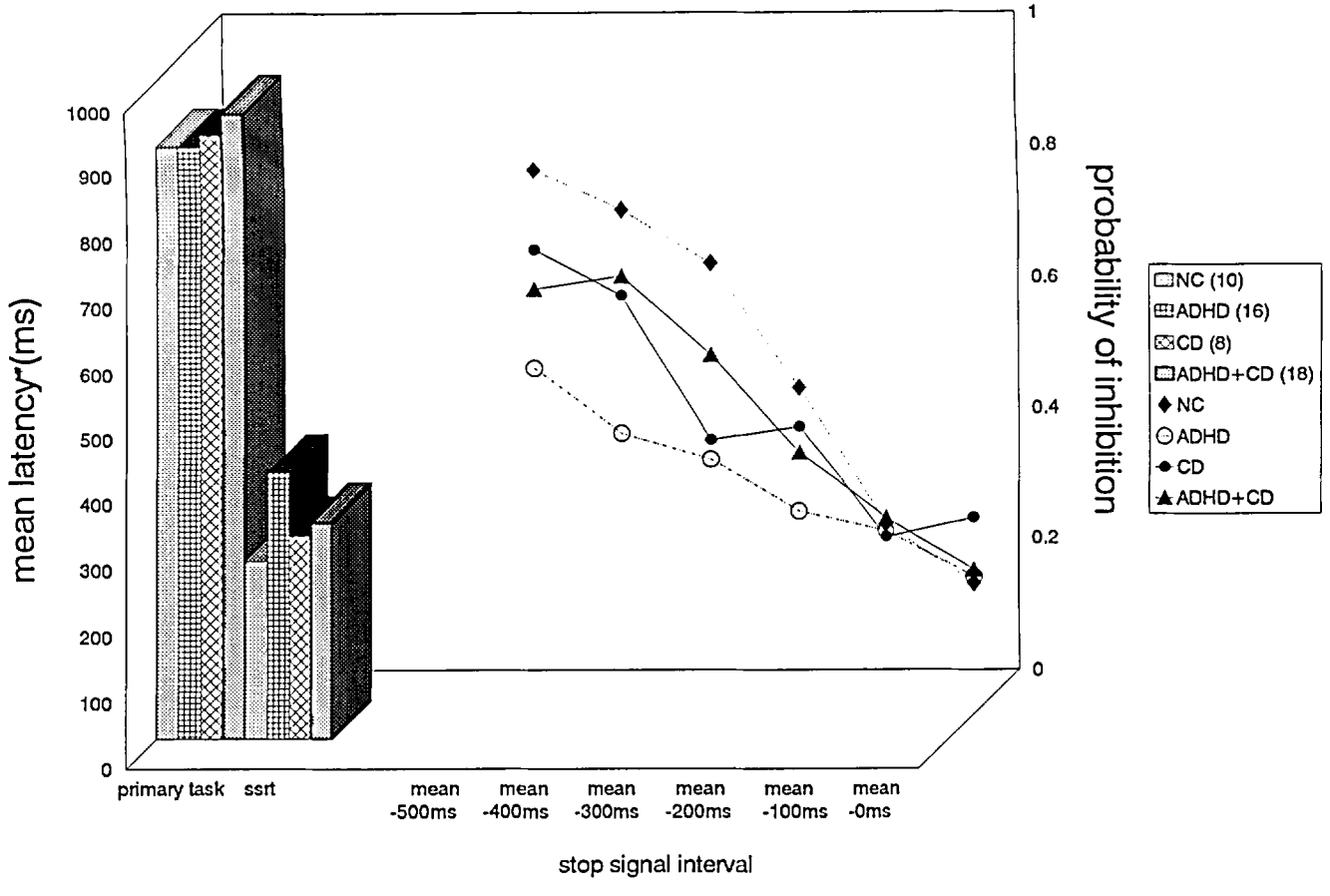

FIGURE 1. Mean Primary Task Reaction Time, Stop Signal Reaction Time (SSRT), and Probability of Inhibition at Each Stop Signal Interval for Normal Control, ADHD, ADHD + CD, and CD Groups.

controls. On average, subjects with ADHD had SSRT's that were over 100 ms longer than those of normal control, CD, and ADHD+CD subjects. As well, ADHD subjects had significantly flatter inhibition slopes than normal controls. When inhibition functions were plotted as a function of ZRFT, difference between groups in inhibition functions disappeared. This pattern of findings indicates that the ADHD group's inhibition processes were slower but not more variable or more likely to be triggered.

By comparison, the pure CD group and the mixed ADHD+CD group showed no evidence of deficient inhibitory control compared to normal controls. Neither mean SSRT, ZRFT, nor slopes of inhibition functions distinguished these groups from the normal control group.

INHIBITORY CONTROL AND RESPONSE REENGAGEMENT IN ADHD SUBTYPES

In a current experiment, we are examining the possibility that, in addition to deficient inhibitory control, the deficit in ADHD might involve a second aspect of executive control—response reengagement. Some acts of control involve alterations in action rather than or in addition to cessation of action; adjusting rather than checking a swing at a breaking ball, reacting to another player's feint in hockey or football, or changing to a new course of action following interruption of an unsuccessful or undesired one are examples of actions that require efficient reengagement of action following inhibition of an ongoing action.

In order to measure the response reengagement process of children with disruptive behavior disorders, we used a modification of the stop signal paradigm known as the change paradigm (Logan & Burkell, 1986). The change paradigm permits distinction and investigation of the processes involved in both inhibitory control and response reengagement. The change paradigm (Logan & Burkell, 1986) uses stimuli that are identical to those of the stop signal paradigm; only response requirements differ. In the change paradigm, subjects are required to inhibit their response to the stop signal tone and to execute immediately a secondary or change response using a separate response button. We call this response the secondary task response. The primary task and the stopping processes function pretty much the same in the stop signal and change paradigms. However, the additional requirement to reengage an alternative response following stopping of an ongoing action permits measurement of reengagement processes as well as response inhibition.

ADHD Subtypes

The second question addressed in the current experiment is the difference between subtypes of children with ADHD (e.g., Schachar, 1991; Schachar et al., 1981; Taylor, Sandberg, Thorley, & Giles, 1991). ADHD is not a homogeneous clinical entity. In addition to differences between ADHD and ADHD+CD, we have been interested in differences among children with a diagnosis of ADHD that arise from the pervasiveness of their symptoms. In the general population, only 16% of ADHD children meet criteria for ADHD both at home and at school (i.e., pervasive ADHD; Szatmari, et al., 1989). The vast majority (73%) of children meet ADHD criteria at school only or at home only (11%; Szatmari et al., 1989). Together, the latter two groups are referred to as the situational ADHD group. Previous research has demonstrated that pervasive ADHD subjects perform worse than situational ADHD subjects on a range of cognitive tasks (Boudreault et al., 1988; Sandberg, Rutter, & Taylor, 1978; Schachar et al., 1981; Taylor et al., 1991).

There were insufficient pervasive ADHD subjects in our first experiment to investigate

these differences adequately. Only 5 of the 16 subjects with pure ADHD had pervasive symptoms. However, these 5 subjects had vastly inferior inhibitory control compared to the remaining 8 situational ADHD subjects with mean SSRT of 570 ms compared with 289 ms for the situational subgroup.

Other 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 distinction is intuitively and clinically interesting. It is supported by previous studies that show a stronger relationship of teacher ADHD ratings than parent ADHD ratings with behavioral inattentiveness and cognitive impairment (Szatmari, Offord, Siegel, Finlayson, & Tuff, 1990). By contrast, parents' ratings correlate with behavioral hyperactivity more strongly than do teacher ratings (Hinshaw, Morrison, Carte, & Cornswee, 1987). In the second experiment, we distinguished among pervasive, home-situational, and school-situational ADHD in order to investigate potential differences in inhibitory control and response reengagement. Pervasive ADHD subjects met diagnostic criteria for ADHD both at home on parental report and at school on teacher report. Home-only ADHD subjects met criteria for ADHD on parental report but not on teacher report, and school-only ADHD met criteria on teacher report but not on parental report.

In order to investigate ADHD symptoms in the school context more thoroughly than would be possible with behavior rating scales, we developed a semistructured telephone interview for use with each child's teacher (Teacher Telephone Interview, TTI; Schachar & Tannock, 1990). The TTI interview follows a format similar to the PICS. Reliability of the TTI was calculated by having a child psychiatrist rate audio recordings of telephone interviews and was high.

Figure 2 shows mean reaction time of the primary task responses, mean reaction time of secondary task responses, and mean SSRT for each group as well as plots of the probability of inhibition at each stop signal interval for each diagnostic group. We examined group differences in several ways. We compared all three ADHD groups with normal controls, pervasive ADHD with normal controls, and home-only ADHD with school-only ADHD. There were only six subjects with pure CD. The data from these subjects are presented for comparison but were not included in the analyses. The performance of ADHD + CD subjects has not yet been analyzed.

The results of this experiment replicated the executive control deficits in pervasive ADHD found in the first study. Compared to normal controls, the pervasive ADHD group had significantly flatter inhibition slopes and a longer mean SSRT, which indicate deficient inhibitory control. The pervasive subjects were, on average, 120 ms slower in their mean SSRT than normal controls. The fact that differences in inhibition slopes were eliminated by plotting probability of inhibition at each stop signal interval against ZRFT indicates that the inhibition processes of pervasive ADHD subjects were slower but not any more variable or any less likely to be triggered.

In addition, the pervasive ADHD group was 100 ms slower and more variable in mean secondary task latencies, which indicates deficient response reengagement processes, and was slower by 100 ms and more variable in the execution of their primary task responses.

These deficits were most pronounced in the pervasive ADHD group. When all ADHD groups (pervasive, home-only and school-only) were combined, no differences were found in primary task processes or inhibitory control processes; only the difference in secondary task latency remained. In addition, no significant differences between school only and home only ADHD were noted, although the school-only ADHD group showed a trend to a greater deficit in inhibitory control than the home-only group and performed much like the pervasive subgroup.

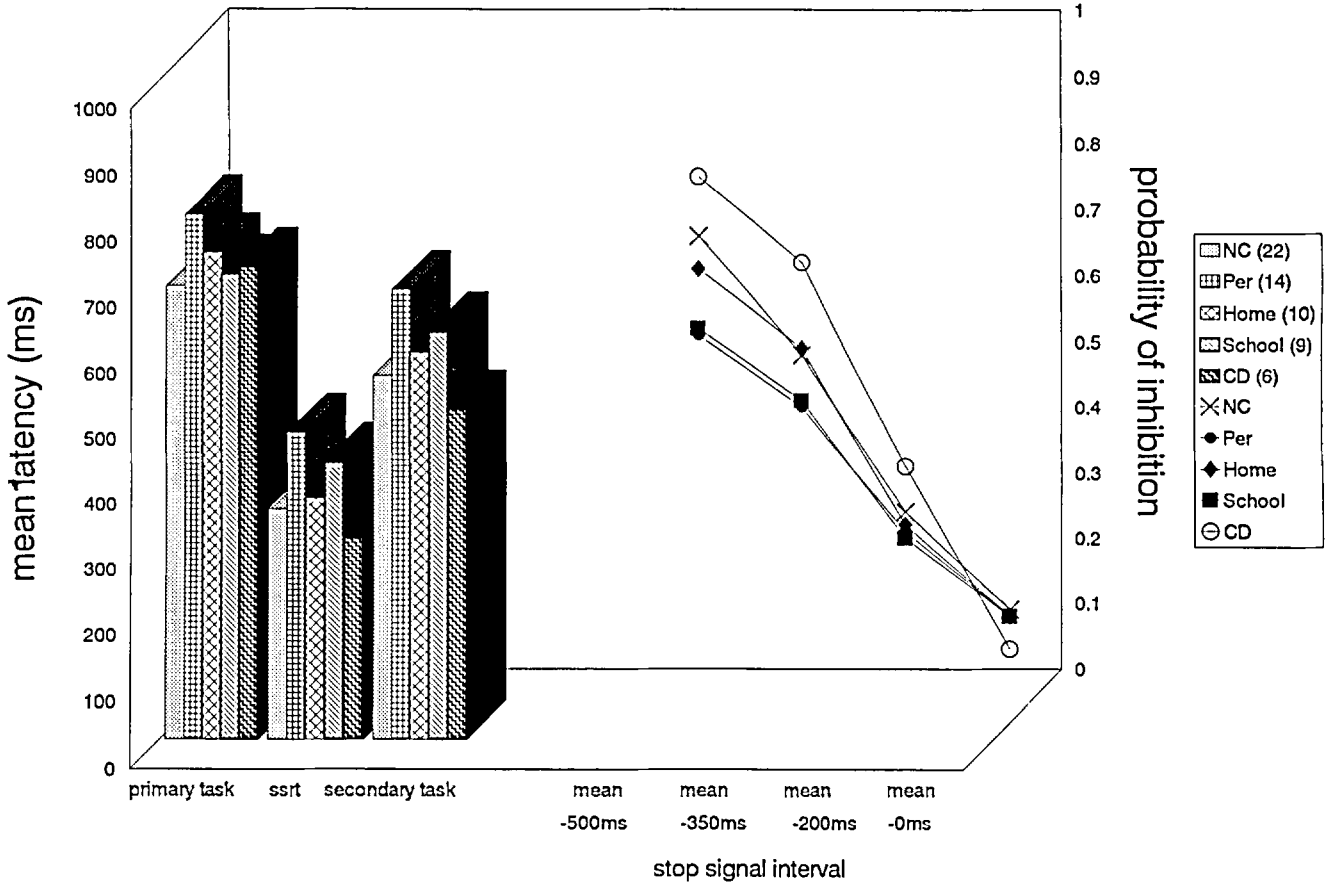

FIGURE 2. Mean Primary Task Reaction Time, Stop Signal Reaction Time (SSRT), and Probability of Inhibition at Each Stop Signal Delay for Normal Control, Pervasive ADHD, Home-Only ADHD, School-Only ADHD, and CD Groups.

The small group of six children with pure CD showed no evidence of deficient inhibitory control or response reengagement, and they did not differ in the mean latency of their primary task responses.

ATTENTIONAL CAPACITY AND INHIBITORY CONTROL

One possible explanation for the observed problems in inhibitory control could be that ADHD subjects have a deficit in attentional capacity. The processing of most or all mental tasks requires a certain amount of attentional capacity (Kahneman, 1973). If more capacity is available than is necessary to support the maximum rate of processing of a current task, that excess is *spare capacity* that can be used to support the performance of other tasks (e.g., monitoring for and responding to the presentation of a secondary task tone as in the stop signal paradigm). The amount of spare capacity determines the rate at which any secondary task is processed during the period when both tasks are being processed simultaneously; the more spare capacity, the greater the rate of secondary task processing. Rather than reflecting a specific deficit in inhibitory control, a potential explanation for the poorer performance of ADHD children on the stop signal paradigm is that they exhibit a relative inability to detect the stop signal because of deficient attentional resources.

Previously, we examined this hypothesis using a second modification of the stop signal paradigm. In the dual task version of the stop signal paradigm subjects responded to *both* the primary task and the secondary task (Schachar & Logan, 1990b for details). In the dual task, subjects were presented with stimuli that were identical to those of the stop signal paradigm. However, response requirements differed. In the dual-task paradigm, subjects were required to respond to *both* the primary task stimuli *and* the tone that served as the stop signal in the stop signal paradigm.

No difference was found among groups in the rate of secondary task processing in the dual task, indicating that ADHD was not associated with deficient attentional capacity. Apparently, deficient attentional capacity could not account for the inhibitory control deficit observed in the first two studies. There were too few subjects with pervasive, home-only, and school-only ADHD or with pure CD to draw conclusions about differences among subgroups.

IMPLICATIONS

Deficit in Executive Control

The results of these two experiments indicate that ADHD is associated with a deficit in executive control of action. ADHD subjects had flatter inhibition functions and longer SSRTs in both experiments, indicating deficiency in the inhibition of action. The absence of any group differences in ZRFT in either experiment indicates that the inhibitory control deficit in ADHD is not a result of a lesser tendency to trigger the response inhibition process, but rather a result of a slower inhibition process.

In the second experiment we found that ADHD children were slower in their responses to the secondary task, which indicates the presence of a deficit in the processes involved in the reengagement of alternative responses following inhibition of an ongoing action. According to theory, there is no interference between the processes involved in inhibition and those involved in reengagement of subsequent responses (Logan & Burkell, 1986). Therefore, we conclude that ADHD is associated with deficits in both response inhibition and response reengagement processes.

The fact that the deficit in ADHD involved both inhibitory control and the processes

involved in response reengagement indicates that the deficit in ADHD may affect executive control in a general way, as has been previously argued (c.f. Douglas, 1988; Shue & Douglas, 1992). Chee et al. (1989) found additional support for this hypothesis. They found that ADHD, in particular pervasive ADHD, was associated with a deficit in another aspect of executive control, the ability to sustain attention over short periods of time.

ADHD Subtypes and Executive Control

Taken together, these two experiments indicate important differences between pervasive ADHD and ADHD evident in the home context only or in the school context only. Pervasive ADHD subjects exhibited a severe deficit in inhibitory control and response reengagement. Home-only ADHD subjects exhibited no such deficit, whereas school-only ADHD subjects tended to look like they had a milder form of pervasive ADHD. We checked to see if these differences could be artifacts of severity or type of symptoms, but groups did not differ in nature or severity of symptoms. Some clue to the nature of the problem in home-only ADHD subjects may be found in the observation that they came from families with significantly more social adversity, characterized by broken families, overcrowding, and poverty (Schachar, Marriott, & Tannock, 1993). In general, these findings support the conclusion that teacher ratings of ADHD are more sensitive to cognitive deficit than are parent ratings of ADHD (c.f. Szatmari et al., 1990).

CD, ADHD + CD, and Executive Control

Although pure CD subjects were too few in number in the second study to permit definitive conclusions, our results did not confirm the presence of a cognitive deficit in CD. There was no evidence of deficient inhibitory control, deficient response reengagement, or differences in primary task response processes. Apparently, children with a diagnosis of CD had no difficulty with the fundamental processes involved in inhibiting their action or switching to alternative actions following inhibition of a current response. By comparison, both ADHD and CD have been associated with difficulties inhibiting responses in situations such as the Card Playing Task (Shapiro, Quay, Hogan, Schwartz, 1988). The obvious difference between these two tasks is the absence of a reward in the stop signal paradigm. The difference between tasks supports the hypothesis that the problems of children with CD are different from those of children with ADHD and may involve the possibility of reward. Deficient inhibitory control did not seem to be central to the difficulties of children with CD in our studies.

The finding of deficient response inhibition and reengagement in ADHD but not in CD suggests a useful strategy for determining the nature of the deficit in those children with concurrent ADHD and CD. The only finding from our program of research on this issue that is available so far indicates that subjects with ADHD and CD in our first experiment did not show an inhibitory control deficit, as did subjects in the pure ADHD group (Schachar & Logan, 1990a). If confirmed, these findings would indicate that ADHD + CD constitutes a variant of CD. Before this conclusion is accepted, replication is required, as is examination of the distinction between ADHD + ODD and ADHD + CD.

Implications of Differences Among ADHD Subgroups

The findings concerning differences among ADHD subtypes carry implications for research methodology and clinical practice. From a research perspective, it is likely that combination of context-dependent ADHD subtypes may have obscured important differ-

ences in cognitive function. Limiting study samples to pervasively ADHD children as has been done in many previous studies (e.g., Seidel & Joschko, 1990; Sergeant & Scholten, 1985) is a useful strategy but would exclude the majority of ADHD. Selection of study subjects based solely on the reports of a single informant would exclude some ADHD children who are symptomatic in one situation only and would not permit distinction between situational and pervasive ADHD (e.g., Atkins, Pelham, & Licht, 1985; Dykman, Ackerman, & Oglesby, 1979; Goldstein, 1987; Rosenthal & Allen, 1980). The current results argue for the examination of context-specific ADHD subtypes in studies of cognition.

Also, these results should alert clinicians to the possible differences between children whose symptoms are limited to the home context and those whose behavioral problems are manifest at school. Both groups had significant and equal levels of behavioral disturbance. However, the two situational subtypes might have distinct implications. These results partially support the proposal of DSM-IV to require evidence of pervasive symptoms for an ADHD diagnosis. The major problem with limiting the diagnosis to pervasive ADHD is that the criteria proposed in DSM-IV could exclude the vast majority of ADHD children who have symptoms evident in school but not at home. The current data suggest that the school-only group has a significant deficit in executive control. The stop signal paradigm is appropriate for the investigation of differences arising from other approaches to subtyping ADHD, (e.g., ADD with and without concurrent hyperactivity or ADHD with and without concurrent emotional disorder).

Advantages of the Stop Signal Paradigm

In the experiments reviewed in this article, we employed the stop signal paradigm and two modifications of the stop signal paradigm known as the change and dual task paradigms to investigate response inhibition, reengagement, and attentional capacity in ADHD and CD. These measures have several advantages over other measures of impulsiveness or inhibitory control. They are based on a specific model of inhibitory control of action and allow direct examination of the internally generated act of control involved in inhibition of action. Moreover, the measure and the race model upon which the stop signal paradigm is based permit distinction between inhibitory control and the processes involved in the execution of ongoing action (primary task processes). Primary task processes influence whether a response will be executed or not, but do not reflect inhibitory control. Fewer responses will be inhibited with faster primary task reaction times in most circumstances independent of the latency of the stopping process. The fact that pervasive ADHD was associated with longer primary task reaction times highlights the need to control for primary task processes when examining inhibitory control. Longer latency of response is observed commonly in ADHD (e.g., Sergeant & Sholten, 1985). If primary task reaction time is not controlled as it is in the stop signal paradigm, there is a possibility that the extent of the inhibitory control deficit may be underestimated. Failure to distinguish between stopping processes and other factors such as primary task response processes which affect the probability of withholding a response is a problem in many current measures of impulsiveness (e.g., go-no go paradigm, Trommer et al., 1988; continuous performance task, Halperin, Matier, Bedi, Sharma, & Newcorn, 1992; Halpern et al., 1988). Milich and Kramer (1984) present a discussion of other methodological problems in existing measures of impulsiveness.

The results of two trials of methylphenidate in children with ADHD (Tannock, Schachar, Carr, Chajczyk, & Logan, 1989; Tannock, Schachar, & Logan, 1992) highlight the usefulness of the stop signal paradigm in the study of the effects of medication on response

execution and response inhibition. In both of those trials, methylphenidate engendered improvement in the clinical manifestations of ADHD at the same time as speeding primary task responses. Moreover, the association was generally linear—greater improvement in behavior was associated with faster reaction times. By contrast, the effects of methylphenidate on inhibitory control were more complex and more interesting. Methylphenidate improved inhibitory control in both the experiment using the stopping task and in the experiment using the change task. However, there was a suggestion that the dose–response curves were different in the two tasks: Whereas inhibition improved with increasing dose on the stop signal paradigm, it improved and then declined as dose increased on the more complex change task. This pattern of results suggests that methylphenidate effects on cognition are specific (i.e., increasing dose does not simply result in improvement in all measures of cognitive function; instead, methylphenidate seems to affect response and inhibition processes in distinct ways).

Other Processes Involved in Impulsiveness

Although the stop signal paradigm can detect differences among groups in the speed of the primary task or go process, the race model does not provide an analysis of the factors which determine primary task processing. These processes are likely to include stimulus recognition, response choice, response preparation, and response execution. Moreover, the race model does not attempt a complete explanation of all of the processes that might be implicated in impulsive behavior. For example, the stop signal paradigm differs from those measures typically used to assess approach tendency in that there is no trade off between probability of reward and probability of punishment as there is, for example, in the Card Playing Task (Newman & Kosson, 1986; Newman, Patterson, & Kosson, 1987). Also, the stop signal paradigm differs from tasks such as the Differential Reinforcement of Low Rate Responding Task (Gordon, 1979; Gordon & Mettelman, 1988) in that no obvious delay is imposed on subjects before executing their response. It is reasonable to assume that the process of inhibitory control that is involved in the stopping or alteration of action plays a role in the performance on all of these tasks. However, these tasks involve many factors in addition to processes involved in response inhibition and response reengagement.

Generality of Findings

The stopping and change paradigms present conditions that are similar to a wide variety of everyday situations requiring stopping and subsequent alteration of action in response to an error in performance or to changing environmental circumstances. Consequently, inferences about the control required in these paradigms may be generalized to a range of similar circumstances in the life of a child with ADHD or CD. Among adults, a wide variety of continuous and discrete actions, such as arm movements (Henry & Harrison, 1961), speaking (Levelt, 1983), and typing (Logan, 1982), can be stopped in a similar amount of time, suggesting a similar mechanism for the inhibition of a range of actions. The generality of these results to other discrete and continuous acts requires study in children.

A Model of Impulsiveness and Inhibitory Control

These results draw attention to the importance of the distinction between inhibitory control as a cognitive construct and impulsiveness as a behavioral construct. We believe that there is a link between the two, but that deficits in inhibitory control cannot account

for all manifestations of impulsiveness. Deficient inhibitory control results in a greater likelihood that a response will escape control and be executed. However, impulsiveness refers to behavior under a very wide range of circumstances, including execution of socially inappropriate acts such as shouting out in class, thoughtless aggression, or choosing incorrect responses on academic tasks. Deficient inhibitory control might contribute to these behaviors under some circumstances, but not all. A range of other cognitive and noncognitive factors may contribute to these manifestations. Comprehensive models of impulsiveness must take all these factors into account.

Nature and Locus of Inhibitory Control Deficit

One of the main questions for further research concerns the nature and locus of the processes involved in response inhibition and reengagement. Our studies address a few of these issues. For example, we did not find evidence that a general deficit such as deficient attentional capacity accounted for the poorer performance of ADHD children on these tasks. We did find an association of age and inhibitory control which suggests that inhibitory control develops with age (Schachar & Logan, 1990a, Experiment 1).

Our findings do argue against the conclusion that the specific and exclusive deficit in ADHD is an inability to delay responding, as has been argued (Barkley, in press; Sonuga-Barke, Taylor, & Heptinstall, 1992; Sonuga-Barke, Taylor, Sembi, & Smith, 1992). These experiments demonstrate that ADHD is associated with deficient inhibitory control even in a situation that involves no delay and that a second deficit in response reengagement is evident.

Research in adults (De Jong et al., 1990) indicates that there may be two inhibition mechanisms involved in the regulation of action. One may involve prevention of central response activation from reaching criterion level. A second may involve interruption of the overt response by prevention of transmission of central motor outflow to peripheral motor structures. Jennings et al. (1992) linked inhibition of motor response to the midbrain structures involved in coordination of heartbeat. Shue and Douglas (1992) argue for a lateral frontal locus of the self-regulatory processes involved in inhibition based on an analogy between the performance deficits of ADHD children and patients with frontal lobe damage. The stop signal paradigm is an appropriate measure for future studies of the deficit in patients with frontal lobe damage or other clinical conditions characterized by impulsiveness.

CONCLUSIONS

The DSM-IV field trials highlighted the importance that clinicians place on behavioral impulsiveness in making a diagnosis of ADHD (Frick et al., 1993). Consequently, it is likely that in the future the constructs of behavioral impulsiveness and inhibitory control will receive greater attention in the study of child psychopathology. The development of theoretically informed, valid, and reliable measures of these constructs will be an important focus of research in child psychopathology. The race model of inhibitory control and the stop signal paradigm have provided a useful model and method for analyzing executive control of action in children with a diagnosis of ADHD.

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