Journal of Abnormal Child Psychology, Vol. 32, No. 3, June 2004, pp. 285–293 (© 2004)

Evidence for an Error Monitoring Deficit in Attention Deficit Hyperactivity Disorder

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Received May 14, 2003; revision received November 1, 2003; accepted November 20, 2003

We studied error monitoring in ADHD and control children in a task requiring inhibition of a motor response. The extent of slowing following successful (stopped) and failed (nonstopped) inhibition was compared across groups. We also measured the time required to inhibit a response (stop signal reaction time, SSRT). Compared to controls, ADHD participants slowed less following nonstopped responses. Slowing did not vary with comorbid reading, oppositional, conduct or anxiety disorder, sex or ADHD subtype. Slowing after nonstopped responses was marginally, although significantly correlated with total ADHD symptoms and with age. ADHD participants had significantly longer SSRT than controls, but SSRT was not significantly correlated with slowing. The apparent deficit in error monitoring in ADHD and its independence from the inhibition deficit observed in ADHD has implications for executive control models of ADHD, performance problems associated with the disorder and for component theories of executive control.

KEY WORDS: error monitoring; ADHD; inhibition; cognition.

INTRODUCTION

Error monitoring refers to online detection of errors and subsequent adjustment of performance. It is one of the executive control processes that provide top–down adjustment of elementary mental operations (Logan, 1985; Norman & Shallice, 1986). Error monitoring is evident in slowing of responses following errors of the type typical of speeded reaction time tasks, reasoning tasks, verbal analogies, and memory search tasks (Rabbitt, 1966a, 1966b, 1968). As well, slowing occurs after failed attempts to inhibit a response (Reiger & Gauggel, 1999b).

Error monitoring has been studied far less in children than in adults. Normal children slow following errors in speeded choice reaction time tasks (Krusch et al., 1996; Sergeant & van der Meere, 1988; Shallice et al., 2002), but it is not known whether they slow following failed attempts to inhibit a response. Therefore, the first aim of this study was to determine whether children slow following inhibition errors.

The second aim of the study was to determine whether children with a diagnosis of attention deficit hyperactivity disorder (ADHD) have a deficit in error monitoring. Given that ADHD children display poorly regulated behavior, inaccurate and variable task performance (Leth-Steensen, Elbaz, & Douglas, 2000), and are thought to have a generalized executive control deficit (Pennington & Ozonoff, 1996; Sergeant, 2000), we predicted that they would exhibit deficient error monitoring compared with normally developing children. We examined the correlation of error monitoring and common ADHD comorbidities (reading disability, oppositional, conduct and anxiety disorders) to assess the specificity of the relationship between ADHD and error monitoring.

The third aim of the study was to determine the relationship between response inhibition and error monitoring. Response inhibition is a key executive control

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process that comes into play when intended responses are delayed or already initiated responses are stopped. Inhibition processes are triggered by changing intentions or an external signal to stop what one is doing. Once initiated, the inhibition process races with the response execution process. Whichever process finishes first determines whether a response will be executed or not (Logan, 1994).

Some theories posit that executive control consists of a number of distinct but interacting components such as inhibition and error monitoring. These component theories are supported by the weak correlation in performance that is observed on various executive control tasks (Goldman-Rakic, 1995; Miyake et al., 2000; Shallice & Burgess, 1996; van der Molen, 2000). Component theories are also supported by the dissociation of the neural systems involved in inhibition and error monitoring (Garavan, Ross, Murphy, Rochi, & Stein, 2002). Component theories predict weak correlation between inhibition and error monitoring. By contrast, unitary theories of executive control posit a unified mechanism or a common resource underlying various aspects of executive control (Duncan, 1995). Unitary theories predict a substantial correlation between error monitoring and inhibition.

Similarly, there are unitary and component views of the executive control deficit in ADHD. According to unitary theories, a generalized cognitive-energetic deficit will affect a range of executive control processes in ADHD (Sergeant, 2000). Variations of the unitary theory identify deficient inhibition as the "up stream" cognitive deficit in ADHD from which other executive control deficits arise (Barkley, 1997). Unitary theories predict a strong association in ADHD between error monitoring and inhibitory control. By contrast, component models of the deficit in ADHD hold that there are multiple pathways to ADHD, each associated with distinct cognitive deficits (Sonuga-Barke, 2002). These theories do not predict a strong correlation among executive processing deficits.

We studied error monitoring and response inhibition using the stop signal paradigm. The stop signal paradigm consists of two concurrent tasks: a go task and a stop task (Logan, Schachar, & Tannock, 1997). The go task is a speeded choice reaction time task. The stop task involves the random presentation on 25% of trials of a signal following the go signal, which instructs the participant to stop their ongoing response. The stop signal paradigm affords an opportunity to assess slowing following nonstopped responses (inhibition errors). The stop signal paradigm also permits estimation of the latency of the inhibition process (Logan, 1994; Logan & Cowan, 1984).

METHODS

Participants

One hundred and fifty-one ADHD participants were drawn from referrals to a clinic specializing in children with attention, learning, and behaviour problems in a large urban pediatric hospital. Participants were 7–16 years of age, and attending a primary or high school ensuring that a teacher could serve as informant in addition to a parent. The sample was similar in socioeconomic status and ethnicity to that of the community from which it was drawn. Forty-one normal controls were recruited through advertisement in the newspaper. They were assessed in the same way as the ADHD cases. We used the Parent Interview for Child Symptoms (PICS-IV; Ickowicz et al., submitted) for our parent interview. The PICS-IV covers *DSM-IV* criteria for ADHD, oppositional defiant disorder (ODD), conduct disorder (CD), and all the other axis I diagnoses that were necessary to establish inclusion and exclusion criteria for ADHD, as well as covering developmental, medical, and social history. The reliability of this interview is high (e.g., $\kappa = 80\%$ for ADHD; intraclass correlation $\gamma > .90$). The Teacher Telephone Interview (TTI-IV; Tannock, Hum, Masellis, Humphries, & Schachar, 2002) is a 30 min interview administered to teachers over the telephone by a trained interviewer. The TTI-IV covers ADHD, ODD, CD, and screens for other disorders. For both of these instruments, symptom presence was rated using specific criteria in an extensive manual. All interviewers were trained to a criterion of 90% symptom agreement before the study began and all interviews were recorded so that we could maintain surveillance, assess reliability, and prevent criterion shift. One of four social workers with extensive experience in the clinic conducted the parent interview and a research technologist with a Master's degree in clinical psychology conducted the teacher interview.

A psychological assistant, supervised by a registered clinical psychologist, assessed intellectual ability (Wechsler Intelligence Scale for Children 3rd ed., WISC-III; Wechsler, 1991b), reading (Woodcock Reading Mastery Test-R Word Identification and Word Attack subtests; Woodcock, 1987), and achievement in reading and mathematics (Wide Range Achievement Test 3rd ed., WRAT-III; Wilkinson, 1993; Wechsler Individual Achievement Test, WIAT; Wechsler, 1991a). A registered speech pathologist conducted language (Clinical Evaluation of Language Fundamentals 3rd; ed., CELF-III; Semel, Wiig, & Secord, 1995), hearing (pure tone audiometric screening) and vision (screening of visual acuity) assessments. Children completed the Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, &

Connors, 1997) as a self-report measure of comorbid anxiety symptoms.

A global measure of impairment was obtained by having parents and teachers rate each participant on the Ontario Child Health Survey Scales (OCHS; Boyle et al., 1993). Impairment scores were standardized by age and gender using general population norms.

To be classified as ADHD for research purposes, children had to meet *DSM-IV* criteria for ADHD (American Psychiatric Association, 1994) defined as at least 6 of 9 inattentive, 6 of 9 hyperactive–impulsive symptoms, or both. To ensure that children were at least moderately impaired in two settings as per *DSM-IV*, we required that they met criteria for ADHD in the parent or the teacher interview, exhibited a minimum of 4 ADHD symptoms according to the *second* informant and had at least a "moderately impaired" rating on the parent and teacher impairment scale. Participants were categorized into ADHD subtypes (inattentive, hyperactive–impulsive, or combined) based on all information using *DSM-IV* criteria. Participants were excluded if they fulfilled any of the following criteria: (a) IQ below 80 on both verbal and performance scales of the WISC-III, (b) presence of pervasive developmental disorder, psychosis, obsessive compulsive disorder (OCD), Tourette syndrome, serious medical problem, substance abuse, or history of traumatic brain injury, (c) concurrent treatment with medication other than a stimulant, (d) specific language impairment (CELF total language score <85) as it may interfere with assessment and cognitive testing, or (e) hearing or visual impairment. About 10% of cases had one of these exclusions.

Children were categorized into those with and without reading disorder (RD). We used an IQ-nondiscrepant definition of decoding problems, because extensive research has shown that both IQ-discrepant and IQ-nondiscrepant definitions validly identify children as reading disabled, with little evidence that these definitions differ in chronicity of problems (Fletcher, Francis, Shaywitz, & Lyon, 1998; Shaywitz, Fletcher, Holahan, & Shaywitz, 1992). RD was assessed using a definition of low achievement in standardized tests of single word and nonword reading (WRMT-R Word Attack, Word Identification, WRAT-3 Reading; Fletcher et al., 1998). RD was defined by scores of at least 1.5 (*SD*) below the mean for age on at least one of the three tests or by scores that were at least 1.0 (*SD*) below the mean for age on at least two of the three tests. Oppositional defiant disorder (ODD) was diagnosed if there were four or more *DSM-IV* symptoms. Conduct disorder (CD) was diagnosed if the number of *DSM-IV* CD symptoms in the parent and teacher interviews totalled three or more. CD, and ODD were combined into a single entity for analysis (CD/ODD). Anxiety disorder was diagnosed if the child met criteria for generalized or separation anxiety disorder on the parent interview or scored above threshold on the MASC. No other anxiety disorders were identified. We calculated quantitative scores for ADHD, CD and ODD symptoms by summing appropriate PICS and TTI items, and for anxiety by summing symptoms of generalized and separation anxiety in the PICS and TTI and by using the MASC total score.

Stimuli and Apparatus

The stop task involved two concurrent tasks (Logan et al., 1997). The primary or go task was a choice reaction time task involving discrimination between an X and an O presented in the centre of a computer screen for 1000 ms following a 500 ms fixation point. The go stimulus was followed by a blank screen for 2000 ms allowing 3000 ms for response and a total trial duration of 3500 ms. Participants were asked to respond as quickly and as accurately as possible. The secondary or stop task involved a 1000 Hz tone emitted from the computer. This tone followed the presentation of the go task stimulus and instructed participants to withhold their response on that particular trial. Tones occurred randomly on 25% of trials. We used a dynamic tracking procedure to set the timing of the tone (stop signal delay). The earlier the tone is presented, the less difficult it is to stop their response. The later the tone is presented, the more difficult it is for participants to stop their responses to the go stimulus. At the beginning of the task, stop delay was set at 250 ms. If a participant was able to stop successfully, the delay was lengthened (by 50 ms) on the succeeding stop trial. If the individual was unable to stop, the delay was shortened by the same amount on the succeeding trial. This "tracking" procedure converged on the delay at which individuals were able to stop 50% of the time. At this delay, the outcome of the "race" between the go process (go reaction time; go RT) was tied with the outcome of the stopping process. The mean latency of the go RT was observable from the 75% of trials in which no stop signal was presented. The latency of the stop process was unobservable—if the participant stops, no response was evident. The stop process had finished before the go process, but how much before was not known. If the go process finished before the stop process, the individual responded much as if no stop had been presented. We could calculate the latency of the unobserved stop process, known as stop signal reaction time (SSRT), by subtracting mean delay (at which the participant inhibits 50% of the time) from mean go reaction time (Logan, 1994). Longer SSRT reflects poorer inhibition. The stop task is based

on an established model of inhibition (Logan & Cowan, 1984).

The task was presented in four blocks of 24 trials. Sixteen trials were go trials without stop signals and eight included a stop signal. Participants responded with the right index finger to one go stimulus and with the index finger of the other hand to the other stimulus. X and O appeared with equal frequency in each block. Stop signals were presented randomly and with equal frequency with the right and left hand response. Participants were instructed to respond as quickly as possible without making errors (such as pressing an X for an O).

To measure error monitoring, we identified trials in which a stop signal was presented, but the participant failed to stop. We calculated mean RT for the first correct go task response that immediately followed each inhibition failure (Error + 1 RT). We calculated posterror slowing as the difference between mean Error + 1 RT and mean go RT.

The model and the measure have been validated using a range of responses (typing, button presses, eye movement), and in several populations (children, monkeys, seniors, ADHD), using Monte Carlo simulation (which shows that the task and model are robust over substantial variation in performance and task parameters), and various measurement techniques (psychophysiological, reaction time, single cell recording). Performance is sensitive to the effects of drugs (methylphenidate in children, alcohol in adults; Logan, 1994). SSRT is stable within session (split-half reliability of $\kappa > .9$; Logan et al., 1997) and over time (Kindlon, Mezzacappa, & Earls, 1995; Schachar et al., 2001).

Procedure

All procedures were explicitly outlined in our information and consent forms and were approved by our research ethics board. The parent, teacher, and child assessments were conducted without knowledge of the screening diagnosis or the results of other portions of the assessment including the results of the stop signal paradigm. All children were free of medication for at least 24 hr on the day of assessment. A drug free trial of at least 3 days was arranged before teacher ratings and interviews when the child's teacher had not observed the child without medication within the preceding 6 months.

Analysis

We examined error monitoring and group differences in several ways. First, we compared the proportion of the ADHD group and the control group that slowed to any extent following nonstopped responses using chi-square test. Second, we compared slowing after nonstopped responses in the ADHD group and the control groups using a two-group (ADHD vs. control) by two-condition (Go RT vs error + 1 RT) Analysis of Variance (ANOVA). This analysis allowed us to determine whether participants slowed significantly and to determine whether groups differed in slowing. Third, we used linear regression analysis to assess the effect of potential covariates (age, IQ, sex, RD, CD/ODD, and anxiety) on slowing and to examine the interaction between covariates and diagnostic group. We included interactions to detect any differential effects of these covariates across groups. The regression analyses began with a saturated model and proceeded with backward elimination of nonsignificant terms. The goodness of fit of the resulting model was evaluated with the R-squared coefficient (R^2). In addition, we examined the association of SSRT and posterror slowing in the ADHD and control groups.

RESULTS

Demographic characteristics, mean reaction time, and posterror slowing for the ADHD groups and controls are shown in Table I. The ADHD group had average IQ scores, but the control group had significantly higher scores, $F(1, 190) = 60.9$, $p < .001$. The ADHD group included a higher percentage of boys, $\chi^2 = 20.5$, $p < .01$, but the groups were comparable in age, $F(1, 190) = 1.3$, *ns*. The majority of ADHD participants were combined subtype (55%); 26% were inattentive and 19% were hyperactive–impulsive subtype. Of the ADHD participants, 24% met criteria for RD, 40% met criteria for CD or ODD, and 21.9% for anxiety disorder.

Table I. Demographic Characteristics and Stop Signal Paradigm Performance in ADHD and Controls Groups

Mean	ADHD, $N = 151$	Controls, $N = 41$	F/X^2	p
Age (years)	8.7 (1.7)	9.0 (1.8)	1.31	*ns*
Sex (% male)	76.2	39.0	20.51	<.001
WISC IQ	102.1 (12.1)	118.3 (10.5)	60.90	<.001
Go accuracy (%)	93.1 (5.1)	94.3 (5.9)	1.74	*ns*
Go RT (ms)	634.5 (135.4)	577.7 (138.3)	5.64	<.05
SSRT (ms)	313.5 (167.7)	233.7 (97.8)	8.49	<.01
E + 1 RT (ms)	677.1 (166.3)	652.4 (160.3)	0.72	*ns*
Posterror slowing (ms)	42.6 (97.0)	74.4 (49.7)	4.20	<.05

Note. RT = reaction time; SSRT = stop signal reaction time; E + 1 RT = reaction time on go trials following nonstopped responses; *ns* = nonsignificant.

Groups did not differ in accuracy of the go task, but the ADHD group had slower go RT, $F(1, 190) = 5.6$, $p < .05$, and longer mean SSRT, $F(1, 190) = 8.5$, $p < .01$.

Although on average the majority of participants slowed to some degree following nonstopped responses, ADHD participants were significantly less likely to slow after nonstopped responses (95.1% for controls and 72.2% for ADHD, $\chi^2 = 9.6$, $p < .01$).

Participants slowed significantly after nonstopped responses (49.5 ms), $F(1, 190) = 55.9$, $p < .0001$, but ADHD participants slowed significantly less (42.6 ms) than controls (74.4 ms), $F(1, 190) = 4.2$, $p < .05$. Regression analysis revealed that slowing after nonstopped responses did not vary with sex, IQ, ADHD subtype, comorbid CD/ODD, anxiety, or RD. Moreover, the effect of slowing was not qualified by any interactions between group and these comorbid conditions or between group and sex. However, age affected performance. Following nonstopped responses, older participants slowed less than younger participants ($\beta = -.15$, $p < .05$). Even after taking any nonsignificant differences in age into account, ADHD participants slowed less than controls ($\beta = -.16$, $p < .05$). SSRT was not significantly correlated with slowing after nonstopped responses ($r = -.13$, *ns*). Slowing after nonstopped responses was not correlated with Go RT, variability in Go RT or accuracy in the go task

Less slowing after nonstopped responses was significantly correlated with greater number of ADHD symptoms ($r = -.15$, $p < .05$) although the magnitude of the association was not great. Slowing was not significantly correlated with either teacher-rated or parent-rated impairment.

DISCUSSION

The aims of this experiment were three-fold. First, we sought to determine whether the error monitoring system in children reacted to failed inhibition the way it does to other kinds of errors. Second, we wanted to know whether there was a deficit in error monitoring among children with ADHD compared with normally developing children. Third, we wanted to assess whether error monitoring and response inhibition were independent components of executive control to inform both cognitive theories and theories of the deficits among individuals with ADHD. Results indicate that children slow following failed inhibition; that ADHD children slow less than their normally developing peers following nonstopped responses; and that error monitoring as indexed by slowing after inhibition errors is not significantly correlated with response inhibition (as indexed by the latency of the stopping process).

The current result confirms that children, like adults (Kleiter & Schwarzenbacher, 1989; Rabbitt, 1966a, 1966b) slow following performance errors when errors are defined as failed attempts to inhibit a speeded motor response. Error monitoring as indexed by posterror slowing varied with age within the range of 7–16 years: Older children slowed less than younger ones. Kramer, Humphrey, Larish, Logan, and Strayer (1994), found that elderly participants slowed more than younger ones following nonstopped responses (50 ms vs. 21 ms) and Rabbitt (1966a) showed that elderly individuals slowed more than younger ones in a choice reaction time task that did not involve response inhibition. Together, these studies begin to map the developmental trajectory of error monitoring, indicating that in normal individuals, the behavioral response to errors decreases as individuals get older and then increases in older age. A similar curvilinear pattern of development in executive control has been found for other aspects of executive control such as inhibition (Bedard et al., 2002; Luna et al., 2001; Luna & Sweeney, 2001; Williams, Ponesse, Schachar, Logan, & Tannock, 1999).

The current study confirms previous reports of abnormal error monitoring in ADHD children (Krusch et al., 1996; Sergeant & van der Meere, 1988) and extends the observation of an error monitoring deficits in ADHD children after failed inhibition of a motor response. Individuals with a diagnosis of ADHD slowed after fewer inhibition failures than normal controls and when they did slow, they slowed to a lesser extent. This pattern suggests that ADHD individuals differ from normally developing individuals both in terms of error detection, as reflected in posterror slowing, and in behavioral adjustment to errors as reflected in the extent of slowing after errors.

Differential error monitoring in ADHD children was not attributable to differences in IQ, age, gender, or response speed (go RT). Slowing after inhibition errors was not associated with go reaction time indicating that slowing in ADHD children is not simply a manifestation of slower responses of all kinds. Interestingly, stimulant medication increases the extent of slowing following errors even though it tends, in general, to speed responses (Krusch et al., 1996). This apparent dissociation suggests that drug induced changes in error monitoring may play a role in improved task performance. The results suggest the predictive validity of error monitoring deficit in ADHD children: Less slowing after nonstopped responses was correlated with a greater number of ADHD symptoms.

The relationship between error monitoring deficit and ADHD appears to be specific in that no association was found with RD, CD, ODD or anxious behaviors, or with a measure of generalized impairment. Given the known association of the error monitoring and the limbic system

one might have expected that anxiety would be linked to defective error monitoring (Luu, Flaisch, & Tucker, 2000). This was not the case suggesting that there is not a distinct mechanism underlying ADHD with comorbid anxiety. It will be important to examine groups of ADHD children with and without comorbid anxiety as well as those with anxiety in the absence of ADHD to confirm these findings. These studies should employ more extensive measures of mood and anxiety than were used in the current study.

In addition to a deficit in error monitoring, the current study replicates the well-established finding of an inhibition deficit in ADHD (e.g., Schachar, Mota, Logan, Tannock, & Klim, 2000). The results indicate that an important distinction can be drawn between error monitoring and response inhibition in normal individuals and in ADHD. Although error monitoring and inhibition were both deficient in ADHD, the two deficits were not significantly correlated. The independence of inhibition and error monitoring deficits in ADHD argues for a multiple pathway model of ADHD (Sonuga-Barke, 2002) rather than unified models that claim that inhibition is the central "up-stream" deficit from which other executive deficits are derived (Barkley, 1997). These two processes may be distinct and under the control of different genetic, neural, or psychological mechanisms (Crosbie & Schachar, 2001) yet each could result in the same or very similar ADHD phenotype.

The lack of a strong association of inhibition and posterror slowing suggests that failures to slow after an error are not simply a reflection of deficient response inhibition (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Gehring & Fencsik, 2001). If that were the case, less efficient response inhibition (i.e., longer SSRT) would have been associated with less posterror slowing.

The observed independence of inhibition and error monitoring is consistent with several lines of research. In electrophysiological studies, response inhibition is evident in a negative deflection, N200, occurring about 200 ms following a signal to stop a response (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Van Boxtel, Van der Molen, Jennings, & Brunia, 2001). The latency of the N200 is not correlated with the latency of the error-related negativity (ERN) which is a negative wave occurring at the time of an error and peaking at about 80–100 ms after the error (Falkenstein et al., 2000; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Coles, Meyers, & Donchin, 1990). The ERN is greatest in conditions when the N200 is least, suggesting that the two processes are separable (Kopp & Rist, 1999; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). Event-related functional magnetic resonance imaging indicates that distinct brain regions are activated during inhibition and error processes (Braver, Barch, Gray, Molfese, & Snyder, 2001; Chevrier, Noseworthy & Schachar, 2003). Studies using single cell recording (Gemba, Sasaki, & Brooks, 1986; Niki & Watanabe, 1979; Stuphorn et al., 2000) have identified a set of neurons in the anterior cingulate gyrus and supplementary eye fields in animals that are active specifically after errors and that are distinct from a group of neurons activated by withholding a response. In summary, these observations support the cognitive and neural dissociation of inhibition and error monitoring and the component theory of executive control in normal individuals (Shallice & Burgess, 1996).

There is considerable debate about the mechanism of error monitoring. One hypothesis is that slowing results from comparison of actual responses with the representation of intended responses (Bernstein, Scheffers, & Coles, 1995; Dehaene, Posner, & Tucker, 1994; Holroyd & Coles, 2002). According to comparator theories of error monitoring, posterror slowing indicates that representations of recently executed responses are stored temporarily and compared with memory of the instruction set for the task (Gehring, Gross, Coles, Meyers, & Donchin, 1993; Kleiter & Schwarzenbacher, 1989; Scheffers et al., 1996; Scheffers & Coles, 2000). This comparison process takes time and delays the response on subsequent trials. In addition, slowing after errors in choice reaction time tasks might reflect suppression of an attempt to correct the erroneous response (Dehaene et al., 1994; Rabbitt & Roger, 1977). Suppression of corrected responses is unlikely to account for slowing after failed inhibition in the current study because there is no possibility of this type of correction in the stop signal task: One cannot stop a response once it is executed. Another possibility is that participants deliberately increase their decision criterion after a stop signal to increase the probability of stopping on successive trials. Therefore, it is possible that ADHD is associated with an insensitivity to errors or punishment, or with a failure to maintain an appropriate response set in working memory, or with a desire to avoid lengthening the task (Songua-Barke, 2002; Songua-Barke, Williams, Hall, & Saxton, 1996).

Several lines of research link the error monitoring system to a neural network involving the medial frontal system, in particular the anterior cingulate gyrus (ACC) and lateral frontal areas (Dehaene et al., 1994; Gehring & Knight, 2000; Holroyd & Coles, 2002). Studies of patients with medial prefrontal lesions are consistent with the mediating role of these regions in error monitoring. These patients often appear unconcerned with the negative consequences of their actions (Rylander, 1947; Tow & Whitty, 1953). They continue to make mistakes despite the fact that actions are obviously detrimental to their goals

(Eslinger & Damasio, 1985). In addition to the observed deficits in patients with medial frontal lesions, those with more lateral prefrontal lesions also show alteration in ERN and impaired posterror monitoring (Gehring & Knight, 2000).

Abnormal error monitoring is evident in other pathological conditions. Gehring and colleagues (Gehring, Himle, & Nisenson, 2000) showed that the amplitude of the ERN was larger in patients with OCD than in normal controls, and among OCD patients, was larger in those with more severe symptoms despite absence of difference in overall task performance (see also, Hajcak & Simons, 2002). By contrast, individuals with low-trait socialization (an analog of psychopathy) exhibit smaller ERN (Dikman & Allen, 2000). It is noteworthy that the size of the right ACC is related to a temperamental disposition to fear and anticipatory worry (Pujol et al., 2002). The fact that abnormality in error monitoring is associated with several psychopathological conditions suggests that they may share a common neural substrate. On the other hand, the fact that OCD shows exaggerated error monitoring (as gauged by the ERN) and ADHD shows reduced error monitoring (as gauged by slowing in posterror reaction time) suggests that the two conditions may differ in the specific effect on a common substrate.

In summary, this study demonstrates for the first time that failure to successfully stop a response triggers error monitoring as evident in slowing of subsequent responses. ADHD children slow less than normal children. This failure to slow may reflect faulty error detection and/or faulty behavioral adjustment to errors. Deficient error monitoring is an important executive control process that appears to be independent of the inhibitory control deficit in ADHD. Error monitoring is dependent on intact fronto-subcortical circuits and the ACC, in particular. Alteration in error monitoring could result in inconsistent, inaccurate, and poorly regulated behavior as well as deficits in self-regulated learning.

ACKNOWLEDGEMENT

This research was supported by a grant from Canadian Institutes of Health Research (MOP44070; New Emerging Team, NET: KIDNET, Grant 54016).

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