POST-ERROR SLOWING IN PRESCHOOL CHILDREN AND ADOLESCENTS WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

by

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A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy,

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ABSTRACT

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by

OLGA G. BERWID

Advisor: Jeffrey M. Halperin, Ph.D.

The objective of this dissertation was to examine whether post-error reaction time slowing, an index of self-regulation, is impaired in individuals with Attention-Deficit/Hyperactivity Disorder (ADHD) at two separate developmental time points: preschool and adolescence. Two studies were conducted with separate cohorts. Study 1 examined post-error slowing in a sample of preschool children rated by parents, teachers, and clinicians as exhibiting high levels of ADHD symptoms. In addition to group comparisons based on symptom status, a cross-sectional examination of age-related changes in post-error slowing in typically developing preschoolers (controls) was also conducted. Study 2 compared post-error slowing in individuals diagnosed with ADHD in childhood, who were diagnostically reassessed in adolescence for persistence versus remission of the disorder, relative to a well-matched comparison group. Post-error slowing was examined as a function of both childhood and adolescent diagnostic status. The results of Study 1 indicated that, although children as young as 3 years of age

exhibited post-error slowing on a computerized reaction time task, the expected increases in post-error slowing with age were not found. Further, hyperactive/inattentive preschool children exhibited reduced levels of post-error slowing relative to controls. Thus, symptomatic preschool children appeared to be impaired on this index of self-regulation. Post hoc exploratory analyses suggested some support for greater attentional difficulties in hyperactive/inattentive children who failed to exhibit post-error slowing. Study 2 did not yield any significant results. Contrary to expectations, individuals with ADHD did not exhibit reduced levels of post-error slowing whether examined as a function of either childhood (ADHD, Control) or adolescent diagnostic status (Persisters, Remitters, Controls). Findings from Study 1 are discussed in the context of developmental changes in the complex neural circuitry underlying both post-error slowing and ADHD. Further investigation of the contributions of component cognitive processes (i.e., error detection, affect/motivation, attention, self-regulation) and their neural bases is recommended. In addition, consideration of post-error slowing as a potential endophenotype may be of benefit to research regarding the genetic underpinnings of ADHD.

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Specific Aims

Attention-Deficit/Hyperactivity Disorder (ADHD) is an impairing developmental disorder characterized by age-inappropriate levels of hyperactivity/impulsivity and inattention with onset prior to 7 years of age (American Psychiatric Association, 1994). Symptoms of ADHD typically arise during the preschool age range, and often earlier in development, and tend to remit with age, such that about half of preschool children naturally outgrow symptoms as they mature (Campbell, 1995). However, ADHD persists into adolescence and adulthood in about 50 to 70% of cases diagnosed in school-age children (Lahey, Miller, Gordon, & Riley, 1999).

A variety of deficits in cognitive function have been associated with ADHD, including various executive functions (e.g., inhibitory control, working memory, ability to delay gratification), state regulation (e.g., arousal, activation), motivation, sensitivity to reinforcement, and emotional regulation. Since the 1970s, when ADHD began to be seen as a neuro-cognitive disorder rather than a syndrome characterized solely by hyperactivity, there has been a debate regarding which of these cognitive functions lies at the core of the ADHD syndrome. There has been particular interest in an etiologic role of difficulties in executive functioning (Barkley, 1997; Berlin, Bohlin, & Rydell, 2003; Castellanos et al., 2000; Hughes, Dunn, & White, 1998; Nigg, 2001; Pennington & Ozonoff, 1996; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Shallice et al., 2002).

Error-related processes, including error monitoring, detection, correction, and ensuing task-strategy adjustment, have been attracting increasing attention from investigators in ADHD research (e.g., Krusch et al., 1996; O'Connell, Bellgrove, Dockree, & Robertson, 2004; Sergeant & van der Meere, 1988; Wiersema, van der Meere, & Roeyers, 2005). During choice reaction time (RT) tasks, healthy individuals typically slow their first correct responses following errors – a phenomenon known as post-error slowing (Rabbitt & Rodgers, 1977). This phenomenon is an indication of conscious response-strategy adjustment to compensate for suboptimal task performance (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). It arises early in development (i.e., by approximately 3.5 years of age) and, due to its conscious, effortful, and goal-related nature, is thought of as an executive function (Jones, Rothbart, & Posner, 2003).

There is evidence from behavioral, electrophysiological, and functional brain imaging studies that error-related processes are aberrant in ADHD (Burgio-Murphy et al., 2007; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; O'Connell et al., 2004; Overtoom et al., 2002; Rubia, Smith, Brammer, Toone, & Taylor, 2005; Schachar et al., 2004; van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007; Wiersema et al., 2005). Post-error slowing, in particular, has been shown to be attenuated in school-aged individuals with ADHD (Krusch et al., 1996; O'Connell et al., 2004; Schachar et al., 2004; Sergeant et al., 1988; Wiersema et al., 2005). However, this ability has not been investigated in preschool children or older adolescents with ADHD.

This dissertation is comprised of two studies designed to examine post-error slowing in 1) preschool children and 2) adolescents who had ADHD during childhood, using a computerized two-choice, self-paced RT task. The primary aim of Study 1 was to compare preschool children with high and low levels of ADHD symptoms in an effort to determine whether, similar to school-aged children with ADHD, symptomatic preschoolers exhibit attenuated post-error slowing. However, because post-error slowing has not been examined in preschool children using computerized tasks (as is typically done in older individuals), we first needed to determine whether such a task can be used with children within this age range, and if so, whether post-error slowing becomes more pronounced in healthy children between 3 and 6 years of age. It was hypothesized that, (1) post-error slowing would be observable in preschool children as young as 3 years of age; (2) the degree of post-error slowing would increase with age; and (3) preschool children exhibiting high levels of ADHD symptoms would not slow as much after making errors as children exhibiting low symptom levels. The presence of error processing deficits early in the course of the disorder could suggest lags in the development of systems including early developing mesolimbic dopaminergic/anterior cingulate/superior parietal brain regions, concurrently developing regions such as dorsolateral prefrontal cortex (PFC), or developing white matter tracts connecting all these regions with one another (Holroyd & Coles, 2002).

Study 2 examined adolescents recruited and diagnosed with ADHD as children between 7 and 11 years of age in the mid-90's as part of a longitudinal

study (Halperin et al., 1997). These individuals were recently seen for follow-up at which time they were diagnostically reassessed. Approximately 44% of those diagnosed in childhood continued to meet criteria for ADHD (persisters) while 30% were in remission, exhibiting few residual symptoms of ADHD (remitters). They were administered an RT task at the time of this follow-up assessment and the degree of post-error slowing in these two groups was compared with that of a never-ADHD comparison group as well as with one another. It was hypothesized that post-error slowing would parallel current ADHD status such that persisters would exhibit reduced levels of post-error slowing relative to remitters, who would more closely resemble controls.

Background and Significance

Attention-Deficit/Hyperactivity Disorder: Phenomenology and Development

Attention-Deficit/Hyperactivity Disorder (ADHD) is an impairing neurodevelopmental disorder characterized by age-inappropriate levels of inattention, and/or hyperactivity-impulsivity that cause significant impairment in day-to-day functioning. Rates of prevalence have been estimated to be between 3% and 7% in school-aged children. Boys and girls are differentially affected with an approximate 3:1 male-to-female ratio. The most current diagnostic classification system (Diagnostic and Statistical Manual of Mental Disorders – 4th Edition; DSM-IV) specifies that impairing symptoms must be present prior to 7 years of age, last for at least 6 months, and occur in at least two settings (e.g., home, school, work; American Psychiatric Association, 1994).

ADHD puts children at substantial risk for poor outcome later in life. These children's dysregulation, disruptiveness, and propensity for risk-taking behavior interfere with the development of self-regulation and social skills necessary for satisfactory academic progress and future occupational success (Mannuzza, Klein, Bessler, Malloy, & Hynes, 1997). Children with ADHD are frequently diagnosed with comorbid anxiety, mood disorders, disruptive behavior, and personality disorders and often grow into individuals who are frequently truant from school, have significant alcohol and/or drug abuse problems, and may become involved in legal entanglements due to criminal misconduct (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Wilens, Biederman, & Spencer, 2002). Needless to say, in addition to impairing the quality of life of

those affected, ADHD and its associated risks bear a substantial burden on society.

As a diagnostic category, ADHD is quite heterogeneous and comprises multiple subtypes based on two symptom clusters (i.e., hyperactivity/impulsivity and inattention). In addition to difficulty sustaining attention, symptoms in the inattention domain include such behaviors as difficulty with messiness, failure to give close attention to detail, disorganization, excessive forgetfulness, difficulty listening, and following instructions. The hyperactive/impulsive symptoms include excessive activity, fidgetiness, restlessness, and difficulties with self-control (e.g., difficulty awaiting one's turn, intrusiveness). Subtypes based on these symptom clusters include the Predominantly Inattentive; Predominantly Hyperactive/Impulsive; and Combined Types based on the prominence of symptoms in one cluster or both (American Psychiatric Association, 1994).

Complicating the phenotypic heterogeneity inherent in this diagnostic scheme, there is substantial variation in presentation across development. Overall, ADHD symptoms tend to decline with age. While high numbers of preschool children are considered to be overactive and hard-to-manage, a large proportion desist in exhibiting high levels of disruptive behaviors by the time they reach school age (Campbell, 1995). Studies also indicate that ADHD persists from childhood into adolescence and adulthood in approximately half of cases, while remitting in many individuals with age (Lahey et al., 1999). Further, age of onset tends to vary by ADHD subtype. Symptoms of hyperactivity and impulsivity typically emerge during the preschool years and decline with age after

the school-age years, while symptoms of inattention usually emerge early in school-age and tend to stay more stable throughout life (Applegate et al., 1997; Hart, Lahey, Loeber, Applegate, & Frick, 1995).

ADHD has a long history. The syndrome that characterizes ADHD has been subsumed under various diagnostic labels since 1902 when it was first described in the medical literature as a “defect of moral control” (Still, 1902). Research indicating that the ADHD syndrome often arose from the developmental effects of early brain insult gave rise to the concept of “minimal brain damage/dysfunction (MBD) in the 1920s to the 1950s. In the middle of the 20th century, research on “hyperkinetic impulse disorder” and “hyperactive child syndrome” led to the notion that the constellation of behaviors currently labeled as ADHD (including difficulties with attention and concentration) were attributable to organic neurologic dysfunction; specifically, cortical overstimulation resulting from insufficient thalamic filtering. Although the specific mechanisms of dysfunction are still a matter of considerable empirical inquiry and debate, the idea of ADHD as a neurological disorder still holds considerable weight (Barkley, 1996).

Since the 1970’s, the view of ADHD has changed from being considered a primary disorder of motor activity ("hyperkinetic reaction of childhood" in DSM-II, American Psychiatric Association, 1968) to being thought of as a primary disorder of attention and cognition with hyperactivity as a resultant epiphenomenon. This manifested as changes to the diagnostic schema to include “attention” in the title of the disorder (i.e., Attention Deficit Disorder in

DSM-III, Attention-Deficit/Hyperactivity Disorder in DSM-IV). The major implication of this change in the view of ADHD has been that impaired cognition is now thought of as the principal driving force behind the behavioral presentation of ADHD. Thus, over the past 40 years, much work has been done to try to elucidate the cognitive profile of ADHD (Barkley, 1996).

Recent Theoretical Accounts of ADHD Pathology: Top-Down or Bottom-Up Deficits? Early Lesion or Developmental Delay?

The extant literature since the 1970's has associated ADHD with deficient performance on tasks measuring a wide variety of cognitive domains, including various executive functions, such as working memory, sustained attention, organization and planning, interference control, inhibitory control, and emotional regulation (Berlin & Bohlin, 2002; Berlin et al., 2003; Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995; Halperin et al., 1988; Halperin et al., 1990; Houghton et al., 1999; Losier, McGrath, & Klein, 1996; Nigg, 2001; Oosterlaan, Logan, & Sergeant, 1998; Oosterlaan & Sergeant, 1998; Pennington et al., 1996; Rucklidge & Tannock, 2002; Stevens, Quittner, Zuckerman, & Moore, 2002; Wright, Waterman, Prescott, & Murdoch-Eaton, 2003); intrinsic motivation (Crone, Jennings, & van der Molen, 2003; Jennings, van der Molen, Pelham, Debski, & Hoza, 1997; Scheres, Oosterlaan, & Sergeant, 2001b; Slusarek, Velling, Bunk, & Eggers, 2001; Sonuga-Barke, De Houwer, De Ruiter, Ajzenstzen, & Holland, 2004) and the ability to tolerate delay of gratification (Kuntsi, Oosterlaan, & Stevenson, 2001; Sonuga-Barke, Taylor, & Heptinstall,

1992a; Sonuga-Barke, Taylor, Sembi, & Smith, 1992b; Sonuga-Barke, Williams, Hall, & Saxton, 1996); allocation of attention (Banaschewski et al., 2003; Crone et al., 2003); and time perception (Smith, Taylor, Rogers, Newman, & Rubia, 2002). In addition, studies of the performance of children with ADHD on computerized laboratory measures of inhibitory control (e.g., stop task, go/no-go task), response re-engagement (e.g., change task), sustained attention (e.g., continuous performance test; CPT), and warned RT have shown that children with ADHD consistently show longer and more variable RTs (Banaschewski et al., 2003; Borger et al., 1999; Borger & van der Meere, 2000; Kuntsi et al., 2001; Leth-Steensen, Elbaz, & Douglas, 2000; Oosterlaan et al., 1998; van der Meere, Shalev, Borger, & Gross-Tsur, 1995), which vary as a function of event rate (Borger et al., 2000; Scheres, Oosterlaan, & Sergeant, 2001a; van der Meere, Stemerdink, & Gunning, 1995; van der Meere, Vreeling, & Sergeant, 1992). This elevated RT variability has more recently been taken as evidence of lapses in sustained attention (Hervey et al., 2006; Leth-Steensen et al., 2000) and some recent data suggest that it might account for observed deficiencies in other areas of cognitive functioning such as inhibitory control (Lijffijt, Kenemans, Verbaten, & Van Engeland, 2005).

The theoretical literature is marked by a longstanding debate regarding which of the cognitive deficits observed to be associated with ADHD are responsible for the behavioral symptoms of the disorder. Virginia Douglas was the first to comprehensively describe ADHD as comprising deficiencies in the ability to 1) invest, organize, and maintain attention and effort, 2) control

behavior, 3) regulate arousal, and 4) resist the inclination to pursue immediate reinforcement (Douglas, 1983; Douglas & Peters, 1979). A more recent prominent theoretical account of the disorder posits that ADHD results from a delay in the development of inhibitory control which underlies the efficient use of several other executive abilities (i.e., working memory; self-regulation of affect, motivation, and arousal; internalization of speech; and reconstitution; (Barkley, 1997). Consistent with this theory, numerous studies have reported that children, adolescents, and adults with ADHD perform more poorly on measures of inhibitory control and executive functions than do controls.

In contrast to theories proposing core deficits in executive functions, others propose difficulties in affective/motivational systems as central to ADHD symptomatology. Sonuga-Barke identified a deficit in the ability of children with ADHD to delay gratification which led to his view that children with ADHD are delay-averse (Sonuga-Barke & Taylor, 1992; Sonuga-Barke et al., 1992a; Sonuga-Barke et al., 1992b; Sonuga-Barke et al., 1996). Since this theory was proposed, much work has identified differences in reward processing between children with ADHD and controls. A recent review concluded that, while children with ADHD do indeed seem abnormally sensitive to immediate and continuous reinforcement, this model could not account for the findings from one study (Daugherty & Quay, 1991) regarding normal-equivalent performance under conditions of non-continuous reinforcement (Luman, Oosterlaan, & Sergeant, 2005). In a similar vein, another theory proposes that children with ADHD have a steepened reward gradient, such that reward devaluation as a function of prior

delay is steeper in children with ADHD relative to controls (Sagvolden, Johansen, Aase, & Russell, 2005).

It seems increasingly clear that none of these models accounts for all of the variance in performance deficiency in children with ADHD. For example, with respect to Barkley's deficient inhibitory control hypothesis, results from studies of executive function in ADHD patients have been somewhat inconsistent. Recent meta-analyses indicated only moderate effect sizes for the differences between patients with ADHD and controls (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) and that only about 50% of individuals with ADHD are impaired on tests of inhibitory control and executive functions (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005). Furthermore, groups with ADHD perform equally poorly on many measures typically not considered to reflect executive function (Frazier, Demaree, & Youngstrom, 2004; van Mourik, Oosterlaan, & Sergeant, 2005).

Other studies pitting various models against one another have suggested that some individuals have difficulties with inhibitory control, while others have difficulties with delay aversion (Solanto et al., 2001; Sonuga-Barke, Dalen, & Remington, 2003). These studies have given rise to more comprehensive multiple-pathway models proposing that different core cognitive deficits can lead to the same diagnostic outcome (Sonuga-Barke, 2003).

Another more comprehensive model of cognitive dysfunction in ADHD is the Cognitive-Energetic Model (Sergeant, 2000; CEM; Sergeant, Oosterlaan, & van der Meere, 1999). This alternative account of deficient cognitive functioning in ADHD takes both "top-down" executive functioning deficits as well as the

increased performance variability under suboptimal conditions (i.e., very slow and very fast event rates, unrewarding, boring tasks) into account. These theorists acknowledge the centrality of deficient inhibition in the neuropathology of ADHD. However, they also posit that difficulty with the regulation of levels of arousal, activation, and effort in response to changing task demands – especially during effortful executive function (i.e., inhibitory control) tasks, is likely to be central in the neuropathology of ADHD. They thus leave a place for deficiencies in these "bottom-up" cognitive processes in their theoretical account of ADHD (Sergeant, 2005; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003). This model has probably garnered the most convincing empirical support to date.

A common weakness with all of these theoretical models is that none account for the high rates of remission of ADHD with development. In addition, executive functioning theories of ADHD are particularly problematic when onset of ADHD is early because it is during the preschool age range that many executive functions begin to emerge. One way in which executive functioning deficits can account for ADHD symptoms is if there is lag in the development of cognitive control. This is surely possible in at least a subset of individuals with the disorder. On the other hand, a recent theoretical review (Halperin & Schulz, 2006) posited that ADHD is caused by functional deficiencies in neural systems that develop prior to preschool age, but persistence/remission of the disorder is determined by the extent to which top-down executive neural systems can compensate for these early deficits and lead to the remission of the disorder. Thus, adolescents and adults with persistent ADHD are those with weakest

executive functioning, but it follows that young children with ADHD are not necessarily more impaired in these abilities than controls. The best way to investigate which of these scenarios is, indeed, the case is to conduct longitudinal investigations of ADHD from symptom emergence into late adolescence and adulthood with the goal of identifying factors associated with disorder persistence versus remission.

Error Processing: Bottom-Up and Top-Down Functions

Error detection is a preconscious, "bottom-up" process that is thought to involve anterior cingulate (ACC) brain regions. Error correction and post-error RT slowing (RTE+1; Rabbitt et al., 1977), where individuals typically exhibit longer RTs on correct trials following errors, are generally considered to be executive functions ("top-down" processes) involving conscious post-error strategic adjustments.

Event-related potential (ERP) studies of error processing during choice RT tasks have identified two medially-generated error-related signals (Falkenstein et al., 2000; see Holroyd et al., 2002 for review). The error-related negativity (ERN or Ne) is a negative deflection which usually peaks approximately 80 to 180 ms after an error occurs and has been associated with preconscious error detection and error correction (Falkenstein et al., 2000; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). The error positivity (Pe), a frequently, but not necessarily, occurring later positive deflection between 300 and 500 ms post-error, is reflective of further post-error processing and has been

associated with post-error slowing (Hajcak, McDonald, & Simons, 2003b; Nieuwenhuis et al., 2001). Several authors have suggested that this more poorly understood component reflects conscious recognition of the error, evaluation of the subjective emotional significance of errors, and/or performance adjustments after an error (Falkenstein et al., 2000; Nieuwenhuis et al., 2001; O'Connell et al., 2007). Functional magnetic resonance imaging (fMRI) and ERP source localization studies have suggested that activation in both dorsal and rostral ACC are responsible for the ERN (Debener et al., 2005; Hajcak, McDonald, & Simons, 2003a; Klein et al., 2007; Mathalon, Whitfield, & Ford, 2003), while the Pe component is thought to be generated in rostral ACC cortex and/or in more posterior brain regions (i.e., posterior cingulate/precuneus, superior parietal cortex; Herrmann, Rommler, Ehlis, Heidrich, & Fallgatter, 2004; O'Connell et al., 2007; van Veen & Carter, 2002).

Development of Error Processing

Post-error slowing has not been studied in young children using computerized tasks as it typically has been in adults and older children. While there is some evidence that computerized RT tasks can be used with preschool-aged children for the valid assessment of cognitive abilities (Weissberg, Ruff, & Lawson, 1990), perhaps due in part to the sparseness of such evidence, few studies have used RT in preschool children to assess the development of cognitive abilities. However, if shown to be valid measurement tools, the use of

RT tasks within the preschool age range would allow a greater degree of continuity in the study of cognitive development.

Error monitoring, detection, and correction, as well as post-error slowing have been shown to be intact during the late toddler and early preschool period and seem to emerge simultaneously with the ability to inhibit prepotent motor responses (Bullock & Lutkenhaus, 1988; DeLoache, Sugarman, & Brown, 1985; Jones et al., 2003). Using a highly structured, outcome-driven task (i.e., assembling a set of nesting cups by graded size), DeLoache, Sugarman, and Brown (1985) found that children as young as 18 months showed at least some awareness of errors, such that when they received feedback indicating that they had made an error, they often attempted to correct it.

However, findings from another study (Bullock et al., 1988) indicated that the ability to detect and correct errors did not appear until somewhat later in development. These authors used a series of less structured, goal-directed tasks (e.g., building a figure from blocks, wiping a blackboard clean) to study the development of volitional control in children between the ages of 15 and 35 months. Children were rated on their ability to work directly toward a stated goal without becoming derailed by engaging features of the tasks. More specifically, the extent to which children engaged in behavior directly related to achieving the goal, monitored and corrected errors, stopped when the task was completed, and showed positive affective responses to goal achievement were rated. Although by 25 months of age most children were able to spontaneously engage in intentional, goal-directed behavior and to show positive affective responses to

successful outcomes, the ability to monitor behavior for errors and to make the fine-tuned behavioral adjustments necessary to correct errors did not appear to develop until approximately 30 months of age.

Jones, Rothbart, & Posner (2003) employed the most direct examination of post-error slowing in children between 36 and 48 months of age. Using an age-appropriate, non-computerized go/no-go task (i.e., the Simple Simon task), they found that children between 36 and 38 months of age did not exhibit any slowing of responses after failed inhibitions, but children 39 months of age and older did. Further, their results indicated that post-error slowing increased with age: in children between 39 and 41 months of age, correct responses after errors were, on average, 26% longer than other correct responses, while the post-error RT increase in children between 46 and 48 months was 62% longer. In addition, post-error slowing and evidence of inhibitory control (i.e., increases in accuracy and slowing of RT for errors on inhibitory trials) began to appear at approximately the same age (i.e., 36 months).

Several studies examining the development of error processing from childhood and early adolescence into adulthood have employed electrophysiological recordings of performance during computerized choice RT tasks. These studies have generally shown that, while ERN amplitude increased with age from early childhood into adulthood, no age-related changes were observed in Pe amplitude or degree of post-error slowing (Davies, Segalowitz, & Gavin, 2004; Ladouceur, Dahl, & Carter, 2004; Santesso, Segalowitz, & Schmidt, 2006; Wiersema, van der Meere, & Roeyers, 2007). Taken together, these

studies suggest that post-error processes (i.e., Pe component and post-error slowing) mature earlier than indications of automatic error detection (i.e., ERN component). Wiersema et al. (2007) hypothesized that neural systems responsible for the ERN and Pe/post-error slowing are independent and develop according to different timelines. They proposed that a fully intact ERN is an indication of mature ACC-prefrontal cortical connections, while the Pe is generated by earlier maturing connections between posterior brain regions and rostral ACC.

Alternatively, Hogan, Vargha-Khadem, Kirkham, and Baldeweg (2005) proposed that the detection of age-related differences in ERN amplitude depends upon task difficulty and the sensitivity of tasks to maturation. This contention is supported by the lack of an observed association between Pe amplitude and post-error slowing (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Hajcak et al., 2003b; Luu, Flaisch, & Tucker, 2000; Nieuwenhuis et al., 2001) in the Santesso et al. (2006) and Wiersema et al. (2007) studies, possibly due to a restriction of range effect. These authors criticized previous studies for being confounded with age-related increases in accuracy and decreases in response times.

To address these weaknesses in the developmental literature, Hogan et al. (2005) made an effort to disentangle error-related changes from performance differences by manipulating task difficulty. They compared post-error slowing and changes in ERN and Pe amplitudes from adolescence (i.e., 12 to 18 years of age) to young adulthood (i.e., 18 to 22 years of age) in three different task conditions: a basic 2-choice RT task and a 4-choice spatial conflict task with

compatible and conflict conditions. Similar to the studies mentioned above, there were no age-related differences in Pe amplitude. However, they did find age-related increases in post-error slowing and ERN amplitude that were dependent upon task difficulty, such that the only significant differences found between the two age groups were in the most difficult task condition (i.e., 4-choice conflict condition).

Post-Error Slowing in ADHD

Error-related cognitive processes, including post-error slowing, have been shown to be relatively deficient in school-age children with ADHD using a variety of tasks. Sergeant & van der Meere (1988) administered a Sternberg memory paradigm with varying memory load conditions to a small sample of 8- to 12-year-old children with ADHD and age-matched controls. Children were instructed to correct their errors as soon as possible after committing them. Children with ADHD were generally slower, more inaccurate, and corrected errors less frequently than controls. Although all children in the study exhibited post-error slowing, controls exhibited an increase in post-error slowing with increasing load while children with ADHD did not, indicating that children with ADHD did not adjust their response strategies as the task became more difficult.

In a related study, Krusch et al. (1996) used the Sternberg memory task with varying memory loads with a larger sample of children with ADHD, including children with Oppositional Defiant Disorder (ODD) and children who were just short of meeting full diagnostic criteria for ADHD. Similar to Sergeant & van der

Meere's (1988b) findings, Krusch et al. found that, while children with ADHD exhibited post-error slowing, it did not increase with greater memory load. In addition, children meeting diagnostic criteria for ADHD and for ODD exhibited less slowing than children whose symptoms were subthreshold. Finally, post-error slowing increased with methylphenidate treatment in all diagnostic groups, but did not differentially increase for higher memory loads. Thus, although methylphenidate improved this self-regulatory ability in children with ADHD, it did not normalize their pattern of responding.

Several other studies have examined post-error slowing in children with ADHD using a variety of paradigms. Schachar et al. (2004) examined post-error slowing after failed inhibitions during a stop signal task and Wiersema, van der Meere, & Roeyers (2005) examined post-error slowing and event-related potentials (ERPs) related to error monitoring in school-aged children diagnosed with ADHD during go/no-go and warned choice RT tasks. In addition to higher rates of errors and more variable RTs in the ADHD groups, these two studies found diminished post-error slowing in children with ADHD (Schachar et al., 2004; Wiersema et al., 2005).

Two other studies failed to find evidence for reductions in post-error slowing in children with ADHD. O'Connell, Bellgrove, Dockree, & Robertson (2004) used the Sustained Attention to Response Test (SART), a go/no-go paradigm with a two-choice RT component, to investigate differences between controls and children with ADHD in post-error slowing and associated electrodermal activity (skin conductance response). Van Meel, Heslenfeld,

Oosterlaan, and Sergeant (2007) examined post-error slowing in children with ADHD using a flanker task (a computerized interference control task requiring participants to respond to the direction indicated by a centrally located stimulus while ignoring spatially incompatible flanking stimuli). However, van Meel et al. used a response deadline to exert time pressure. It is thus difficult to know whether differences in post-error slowing would have emerged in the absence of such a deadline.

In addition, a number of studies employing ERP measures have shown differences between children with ADHD and controls in error and post-error processing, but findings have varied with respect to the direction of differences, component processes, and associated brain areas affected. Wiersema, van der Meere, and Roeyers (2005) found that while the ERN was normal in children with ADHD when compared to control children during warned RT and go/no-go tasks, the Pe was significantly reduced. Overtoom et al. (2002) also found a reduced Pe in children with ADHD for failed inhibitions during a stop signal task. These studies suggest a deficit in conscious post-error strategy adjustments.

In contrast to these findings, Liotti et al. (2005) showed that children with ADHD did not exhibit any detectable ERN after errors during the choice RT component of the stop signal task while control children did, suggesting a lack of ability to detect errors. Similarly, van Meel and colleagues (2007) found that the ERN was marginally smaller in children with ADHD, but that the Pe did not differ across ADHD and control groups. Finally, yet another study (Burgio-Murphy et al., 2007) found that children with ADHD exhibited *larger* ERN amplitudes, but no

differences in Pe amplitude, during a simple 2-choice RT task with varied stimulus ratios. Notably, and in contrast to all other studies, there were no performance differences between children with ADHD and controls on this task. This study may indicate that, all else being equal, children with ADHD are more likely to detect errors.

Using a different electrophysiological indicator, O'Connell et al. (2004) examined differences in post-error slowing between school-aged children diagnosed with ADHD and age and sex-matched control children during the Sustained Attention to Response Task (SART) while recording skin conductance. Relative to controls, children with ADHD made more errors of commission and omission and exhibited greater RT variability for go stimuli. While the skin conductance response (SCR) was similar in the two groups for successfully withheld responses, there was a significant increase in SCR to commission errors in the control group that was absent in the ADHD group. However, both groups exhibited equivalent post-error slowing. The authors concluded that although children with ADHD appeared to show awareness of errors as indexed by post-error slowing, the reduced SCR suggests reduced processing of the emotional significance of errors.

Rubia, Smith, Brammer, Toone, & Taylor (2005) used rapid, mixed-trial, event-related fMRI to compare brain activation in children and medication-naïve adolescents with ADHD during a stop signal task. Performance on the task was individually adjusted to 50% accuracy so that performance was equalized across groups. They found increased activation in mesial prefrontal, ACC, and temporal

and mesial parietal cortex during failed inhibitions in control participants, but relative to controls, activation in posterior cingulate cortex and precuneus was reduced in those with ADHD. They interpreted these differences as indicative of failures in the attentional orienting system response to errors.

Summary and Conclusions

Studies suggest that an awareness of making errors is present early in childhood, by at least 2 ½ years of age, as indicated by observations of children's affective responses and slowing of correct responses after making errors during play activities. ERP studies (e.g., ERN) suggest that a preconscious error detection system associated with ACC functioning is functionally intact by the school-age years and that it likely continues to develop into late adolescence as indicated by age-related increases in ERN amplitude. Studies of post-error slowing and the associated Pe component also suggest that conscious motivational evaluation of errors and strategy adjustments in response to lapses in performance also develop from school-age to adolescence; however, the ability to detect improvements in these abilities is heavily contingent upon the use of sufficiently challenging tasks.

Although studies of error processing and post-error slowing in ADHD have consistently shown differences in some aspect of performance monitoring and adjustment, they are equivocal with respect to the exact component process(es) affected. Whereas some studies indicate that the ACC-mediated error detection system functions less efficiently, one study suggested that this system in children

with ADHD is more sensitive to error commission, and others found no differences. With respect to post-error processes, while some studies assaying the conscious, emotional evaluation of error significance and/or post-error performance strategy adjustment suggested deficiencies in ADHD, others found no differences. Thus, while investigators generally seem to agree that there are performance monitoring and adjustment anomalies in children with ADHD, the nature of these anomalies is still a matter for further investigation.

Rationale for the Current Studies

The studies comprising this dissertation were meant to address the following gaps in the literature:

(1) To date, there are no studies of post-error slowing using measures analogous to those used with older children and adolescents (i.e., computerized RT tasks) in early childhood. The successful use of computerized RT tasks with preschool children would indicate that their use is appropriate in this age range, and that they could, thus, be used to lend a greater degree of continuity within the developmental literature on error processing.

(2) Although deficiencies in error processing have been identified in school-aged children with ADHD, error processing has not been examined early and late in the course of ADHD. Thus, there is not only a lack of clarity regarding which component error-related processes are affected, but also when in the course of development these differences emerge and when (and if) they remit.

Given that the majority of cognitive theories of ADHD specify a role for deficient executive functioning that, if etiologic, should be present at symptom onset or very shortly thereafter, determining whether there are deficits in error processing early on would have heuristic value regarding the pathophysiology of ADHD. The presence of error processing deficits early in the ontogeny of the disorder could suggest lags in the development of systems including early developing mesolimbic dopaminergic/anterior cingulate/superior parietal brain regions, concurrently developing regions such as dorsolateral PFC, and/or the developing white matter tracts connecting all these regions with one another.

Executive functioning has also been theorized to play a role in the remission of ADHD with development. Thus, much can be learned by examining executive functioning in individuals prospectively diagnosed with ADHD in childhood, in adolescence, and adulthood, many of whom no longer meet criteria for ADHD. For example, Halperin, Trampush, Miller, Marks and Newcorn (in review) compared individuals with persistent ADHD with those in remission on various measures of neuropsychological functioning. In support of Halperin and Schulz's (2006) theory, they found that ADHD persisters and remitters both showed relative deficiencies on non-executive functioning measures (i.e., greater RT variability, indicating deficient arousal; lower signal detectability (*d'*); and higher movement counts on solid-state actigraph measures). However, on measures of executive functions, while the performance of persisters was deficient, that of remitters was similar to controls.

This dissertation is comprised of two studies designed to examine post-error slowing in (1) preschool children exhibiting high levels of ADHD symptoms and (2) adolescents diagnosed in childhood with ADHD, whose symptoms have either persisted or remitted in late adolescence/early adulthood.

Study 1: Post-Error Slowing in Hyperactive/Inattentive Preschoolers

There were three main objectives of this study. First, we attempted to determine whether post-error slowing can be measured reliably in preschool children between the ages of 3 and 6 years using a computerized choice RT task. If post-error slowing can be demonstrated in this population using such a task, it would provide evidence for the valid use of computerized RT measures to examine cognitive processes in children in this age range. The second objective was a cross-sectional investigation of the developmental time course of post-error slowing in this age range. The third objective was an examination of post-error slowing in children exhibiting high levels of ADHD symptoms.

Hypotheses for Study 1

1. Based on research indicating the presence of post-error slowing in children as young as 36 months of age (Jones et al., 2003), we predicted that post-error slowing would be present in typically-developing children (i.e., those exhibiting low levels of hyperactivity/impulsivity and inattention) between the ages of 3 and 6 years. Further, we hypothesized that post-error slowing would be observed in even the youngest children.

2. Consistent with findings suggesting that post-error slowing increases with age (Hogan, Vargha-Khadem, Kirkham, & Baldeweg, 2005), we expected to find a pronouncement of post-error slowing with age in control children.
3. Based on previous findings of attenuated post-error slowing in school-aged children with ADHD (Krusch et al., 1996; Schachar et al., 2004; Sergeant et al., 1988; Wiersema et al., 2005), preschool children exhibiting high levels of ADHD symptoms were expected to exhibit reduced post-error slowing relative to control children.

Study 2: Post-Error Slowing in Adolescents Diagnosed with ADHD in Childhood

The main objective of this study was to determine whether individuals diagnosed with ADHD in childhood exhibit deficiencies in post-error slowing in adolescence. Further, adolescents were divided into those with persistent ADHD and those in remission, who exhibited only low levels of residual symptoms, to enable further examination of post-error slowing differences between these two groups.

Hypotheses for Study 2

1. Based on studies showing reductions in post-error slowing in children with ADHD (Krusch et al., 1996; Schachar et al., 2004; Sergeant et al., 1988; Wiersema et al., 2005), we expected that adolescents diagnosed with ADHD in childhood would exhibit reduced levels of post-error slowing relative to same-aged controls.

2. Based on Halperin and Schulz's (2006) theory, we predicted that post-error slowing in adolescents with persistent ADHD would be reduced relative to those adolescents in remission, who would exhibit levels of post-error slowing more similar to controls.

Study 1: Post-Error Slowing in Hyperactive/Inattentive Preschool Children

Method

Participants and Recruitment

Children 3 through 6 years of age were recruited between 2000 and 2006 from local preschools in Queens, New York or by direct clinical referral from parents, teachers, school psychologists, and school social workers. All participants were free of prior diagnoses of mental retardation, neurological disorder (e.g., seizure disorder), and pervasive developmental disorder. The study was approved by the Institutional Review Board of Queens College of the City University of New York. Written informed consent was obtained from the parents of all participants. All participants were compensated for their time and travel expenses associated with participation in the study.

Prior to an extensive laboratory assessment, all children were rated by both parents and teachers on the 18 DSM-IV symptoms of ADHD using either the ADHD-RS-IV, Home & School Versions (DuPaul, Power, Anastopoulos, & Reid, 1998; children recruited from 2003 through 2006) or a very similar DSM-IV symptom checklist (children recruited from 2000 through 2003), which, like the ADHD-RS-IV, asks informants to rate each symptom on a 4-point scale. Both of these scales have been found to have adequate reliability and validity in assessing the presence of ADHD symptoms in young children (DuPaul, 1991; DuPaul et al., 1998; Faries, Yalcin, Harder, & Heiligenstein, 2001). One recent study indicated that there were moderate levels of agreement between parents and teachers on ADHD-RS-IV ratings (r = .554; Healey, Miller, Castelli, Marks, &

Halperin, 2007). After children were seen in the laboratory, they were also rated by the examiners on a clinical rating scale developed in our laboratory called the Behavioral Rating Inventory for Children (BRIC), where children are rated on a 5-point scale in 5 domains: Attention, Activity Level, Impulsivity, Affect, and Sociability. Ratings of 3 and above in the Attention, Activity Level, and Impulsivity domains were considered to be indicative of the presence of problematic levels of ADHD symptoms within the clinical laboratory setting. This scale has been shown to have excellent construct- and criterion-related validity as well as strong test-retest reliability (Gopin, Policaro, Healey, Marks, & Halperin, 2006).

Based on parent and teacher symptom ratings, as well as the BRIC ratings, children were placed in either the “hyperactive/inattentive” (HI) group or control group. Control children were those who were rated as exhibiting low levels of ADHD symptoms (i.e., fewer than 3 in each domain) on the ADHD-RS-IV or DSM-IV checklist by both parent and teachers. Although not formally clinically diagnosed with ADHD, these children exhibited at least 6 symptoms in either the hyperactivity/impulsivity or inattention domain according to one rater (e.g., parent), and some symptoms of ADHD according to another rater (e.g., at least 2 symptoms in any domain by teacher or a rating of 3 in any of the symptoms-relevant domains on the BRIC). Thus, children in the HI group met the symptom count, age-of-onset, and cross-situationality criteria for a diagnosis of ADHD as outlined in the DSM-IV (American Psychiatric Association, 1994).

Full Scale IQ (FSIQ) was estimated using the Information subtest scaled

score from the Wechsler Primary and Preschool Scale of Intelligence (WPPSI). Different versions of this subtest were administered depending on time of entry into the study (i.e., Revised Edition (WPPSI-R; Wechsler, 1989) prior to 2004; Third Edition (WPPSI-III; Wechsler, 2002) for 2004 and later). Scores on the Information subtest correlate highly with FSIQ in both the WPPSI-R ($r = 0.71$) and WPPSI-III ($r = 0.82$-0.83).

An ethnically diverse sample of 165 children (95 controls, 70 HI) between the ages of 3.2 and 5.8 years (mean age = 4.43 years; SD = 0.54 years) met criteria for one of the two groups, with 8.5% being African American; 16.4% Asian; 36.4% Caucasian; 15.8% Hispanic; and 23.0% of mixed ethnicity. Not unexpectedly, the proportion of males was unequally distributed across the control and HI groups, with 51% of controls and 76% of HI children being male ($\chi^2 = 10.77$, $p = .001$). Mean WPPSI Information subtest scaled score was significantly higher among control children (mean = 11.57; SD = 3.02) than HI children (mean = 10.43; SD = 3.14; $t = 2.33$; $p < .05$). See **Table 1** for the demographic characteristics of this sample.

Table 1.

Preschool Sample Characteristics: Full Sample

	Control (n=95)		HI (n=70)			
Characteristic	Mean	SD	Mean	SD	t/χ^2	*p*
% Boys	50.5%		71.7%		10.77	**.001**
Age	4.39	0.59	4.47	0.46	1.00	.32
WPPSI Information	11.57	3.02	10.43	3.14	2.33	**.02**
Parent DSM-IV Ratings						
Inattention	4.24	3.05	11.00	5.52	9.26	**<.001**
Hyperactivity/Impulsivity	4.38	2.65	15.50	6.16	14.16	**<.001**
Teacher DSM-IV Ratings						
Inattention	2.05	2.44	14.70	7.36	13.83	**<.001**
Hyperactivity/Impulsivity	2.04	2.59	17.49	7.81	15.91	**<.001**
Clinician BRIC Ratings						
Activity	2.36	1.18	3.71	1.12	7.27	**<.001**
Impulsivity	1.93	.98	3.37	1.40	7.56	**<.001**
Attention	2.04	1.15	3.53	1.20	7.88	**<.001**

Reaction Time Task

Sixty trials of a two-choice, self-paced, computerized, serial RT task were administered in three blocks of 20 trials each. These trial blocks originally served as control conditions for a version of Nassauer & Halperin's (2003) computerized Perceptual and Motor Conflict Test, which was modified for use with young children (Marks et al., 2005).

To maintain interest and prevent fatigue, each RT block was administered interspersed with the experimental conditions of the task as well as other cognitive tasks in a preset order that was the same for all participants. For all three blocks, instructions were given to children using a demonstration booklet followed by 10 practice trials on the computer prior to task administration.

Children received verbal feedback during practice trials to ensure that they understood the instructions and to correct erroneous performance.

During Blocks 1 and 3 of the task, 10 left-pointing and 10 right-pointing arrows appeared in random order on the computer screen. During Block 2, rectangles appeared on either the right or the left side of the screen (10 on each side) in quasi-random order. Stimuli remained on the screen until the child responded. A 1s ISI followed each response. For all three blocks children were instructed to make the response that was most compatible with stimulus direction (in the case of arrows) or location (in the case of rectangles), with the corresponding hand, as fast as possible without making errors. For example, children were asked to press the right button for a right-pointing arrow with their right hand. The response device was a 2-button stationary mouse placed at midline midway between the child and the computer screen. The mouse was large enough to accommodate the difficulties with fine motor coordination characteristic of young children. The buttons on the mouse were clearly separated, with one on each side of the device, so that children could press each of the buttons with a different hand.

The examiner was present for the duration of the task and gave children reminders during off-task behavior and obvious attentional lapses to “keep playing.” To minimize fatigue and maximize effort allocation during task performance, the task battery was administered to children by examiners well trained in study procedures to ensure smooth transitions between tasks. Children were given frequent breaks for rest, bathroom use, and snacks. They

were also regularly given verbal encouragement, praise, and sticker rewards contingent upon effort, not performance.

Data Analysis

The objectives of the analyses were to examine (1) whether children's correct RTs following errors (E+1 responses) were indeed slower than their correct RTs following other correct responses (C+1 responses); (2) whether this slowing became more pronounced with age; and (3) whether children exhibiting high levels of ADHD symptoms showed reduced slowing compared to those with low levels of ADHD symptoms. The first two objectives were addressed by dividing the control group into 3 equally sized age groups so that age could be included in a 3-way Age (Young vs. Middle vs. Old) x Response Type (C+1 vs. E+1) x Sex (male vs. female) mixed factorial analysis of covariance, controlling for estimated IQ using the WPPSI Information subtest scaled score. The third objective was addressed by a 3-way Group (HI vs. control) x Response Type (C+1 vs. E+1) x Sex (male vs. female) mixed-factorial analysis of covariance, again controlling for estimated IQ. These analyses were repeated without covarying for IQ to examine the stability of effects as a function of this variable.

Mean RT (MRT) was used as the dependent variable in each of these analyses. This is the dependent measure typically used in studies of RT in ADHD. In addition, the mean, rather than the median, was used to prevent the exclusion of elongated RTs that, although outlying, may be associated with post-error slowing. Similar to the methods used by Krusch et al. (1996), the selection

of trials for each of the two Response Type conditions was constrained to control for the effects of extraneous variables on correct RTs (e.g., random, unintentional, or impulsive responding, lapses in attention or effort lasting several trials). First, correct RTs were only included if they were between 300 ms (i.e., below which children were empirically determined to perform at chance levels) and 2188 ms (i.e., the RT representing the 90th percentile of all correct responses).

Second, for C+1 trials, the correct response preceding the target correct response could not follow an error. Thus, C+1 responses were third in a correct-correct-correct sequence. This was primarily done to control for chance responding, since it is less likely that children would respond correctly by chance three times consecutively rather than twice.

Finally, because errors that follow other errors may be different in nature than errors that occur in isolation (i.e., may be reflective of longer lapses in attention rather than erroneous encoding or response selection), for E+1 trials, the error preceding the target correct response could not follow another error. Thus, E+1 responses were third response in a correct-error-correct sequence. Children had to make a minimum of two eligible C+1 and E+1 responses to be included in the analyses.

Results

Sample Characteristics

One child in the HI group was unable to perform the task. Twenty-three (24.2%) controls and 7 (10.0%) HI children made fewer than 2 errors. Of those who made 2 or more errors, 17 (17.9%) control children and 6 (8.6%) HI children made fewer than 2 eligible E+1 responses; one HI child made fewer than 2 eligible C+1 responses. After excluding these children, there were 55 children in each group. **Table 2** lists overall mean accuracy rates, number of C+1 and E+1 responses, mean overall RT, and MRT for correct and erroneous responses separately. Controls exhibited significantly higher accuracy rates and a significantly higher number of C+1 responses. There were no differences in mean correct RT as a function of response hand in either group.

Table 2.

Performance Statistics for the Preschool Sample: Partial Sample*

	Control (n=55)		HI (n=55)			
Statistic	Mean	SD	Mean	SD	*t*	*P*
% Correct	86.94	9.88	80.24	11.81	3.23	**<.01**
# C+1[a]	33.44	11.06	27.53	11.39	2.76	**<.01**
# E+1[b]	3.44	1.15	3.87	1.75	1.54	.13
$RT_{correct}$	1033	179	1011	179	.65	.52
RT_{error}	1170	764	1507	1771	1.30	.20

* Participants with fewer than 2 eligible C+1 or E+1 responses excluded
[a] E+1 = correct responses made following isolated errors
[b] C+1 = number of correct responses made following 2 consecutive correct responses

Age-Related Effects

For age-related analyses, the control group was divided into 3 equal-sized age groups. The Young group consisted of children between the ages of 3.3 and 4.0 years of age; the Middle Group comprised children between 4.0 and 4.6 years old; and the Old Group, children between 4.6 and 5.4 years old. The mean estimated IQ of each of these groups differed significantly, with posthoc Tukey HSD comparisons indicating that the Old group exhibited a significantly lower mean estimated IQ than the two younger groups. **Table 3** shows the mean ages, WPPSI Information subtest scaled scores, sex distributions, and performance data for each of the 3 age groups. There were no differences in mean correct RT as a function of response hand in any of the age groups.

Table 3.

Preschool Age Group Characteristics

	Young (n = 18)		Middle (n = 19)		Old (n = 18)			
Statistic	Mean	SD	Mean	SD	Mean	SD	F/χ^2	*p*
% Boys	33.3%		47.4%		66.7%		4.04	>.10
Age[a]	3.66	.26	4.31	.14	4.96	.16	200.85	**<.001**
WPPSI Info[b]	13.06	2.18	12.21	2.53	10.22	2.65	6.30	**<.005**
% Correct	83.89	11.15	85.18	10.51	91.85	5.45	3.73	**<.05**
# C+1	29.00	12.16	32.21	11.51	39.17	6.58	4.50	**<.05**
# E+1	3.56	1.20	3.58	.96	3.17	1.30	.73	.49
$RT_{correct}$[c]	1104	175	991	157	1006	195	2.20	.12
RT_{error}	1310	837	1118	774	1083	697	.46	.64
RT_{C+1}[c]	1060	186	938	162	948	205	2.45	.10
RT_{E+1}[c]	1257	354	1197	339	1274	334	.26	.77

[a] Young 3.3 – 4.0 years; Middle 4.0 – 4.6 years; Old 4.6 – 5.4 years
[b] WPPSI Info = Information subtest from the WPPSI-R/III
[c] RTs under 300 ms and over 2188 ms excluded;

The results of the 3-way Trial Type (C+1 vs. E+1) x Age (Young vs. Middle vs. Old) x Sex (Male vs. Female) ANCOVA comparing the differences in MRT for the two response types (C+1 vs. E+1) across the 3 age groups and as a function of sex, covarying for WPPSI Information subtest scaled score, are shown in **Figure 1**. As expected, there was a main effect of Trial Type, such that MRT_{E+1} was slower than MRT_{C+1} ($F = 6.59$; $p = .01$; partial $\eta^2 = .12$), indicating that these children significantly slowed following errors. Furthermore, *post hoc* paired *t*-tests showed that all three groups exhibited post-error slowing, indicating that post-error slowing occurs at an early age. On average, children in the Young group slowed by 197 ms ($t = 2.76$, $p = .01$); those in the Middle group slowed 258 ms ($t = 4.24$, $p < .001$); and those in the Old group slowed 326 ms ($t = 4.13$, $p = .001$). However, the Trial Type x Age interaction was not significant, suggesting that post-error slowing did not become significantly more pronounced with age in the control group ($F = .12$; $p > .10$; partial $\eta^2 = .005$). Finally, there was no impact of sex on post-error slowing ($F = .15$; $p > .10$; partial $\eta^2 = .003$).

When this analysis was repeated without covarying for estimated IQ, the results were virtually identical; however, the size of the post-error slowing main effect was much larger ($F = 36.25$; $p < .001$; partial $\eta^2 = .43$).

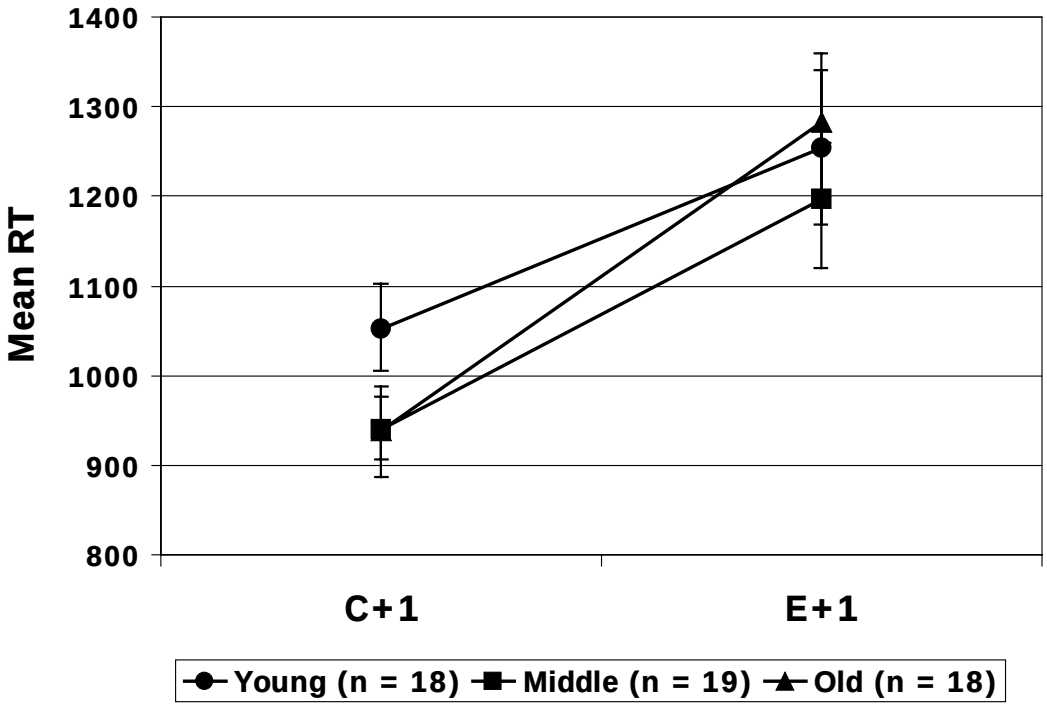

Figure 1. Age effects on post-error RT slowing in preschool children (error bars indicate standard error of the mean).

Effect of ADHD Symptoms

Table 4 shows the demographic characteristics and estimated IQ, using the WPPSI Information subtest scaled score, for the included sample by symptom group. As designed, the HI and control groups differed from one another on parent and teacher ratings of inattention and hyperactivity and on the BRIC ratings of Attention, Activity Level, and Impulsivity. The two groups also differed significantly on WPPSI Information subtest scaled score. Finally, the sex distribution was unequal across the two groups, with a higher proportion of boys making up the HI group.

Table 4.

Preschool Sample Characteristics: Partial Sample

	Control (n=55)		HI (n=55)			
Characteristic	Mean	SD	Mean	SD	t/χ^2	*p*
% Boys	49.1%		72.7%		6.45	**.01**
Age	4.31	0.56	4.48	0.46	1.70	.09
WPPSI Information	11.84	2.69	10.33	3.29	2.62	**.01**
Parent DSM-IV Ratings						
Inattention	4.22	2.97	11.47	5.39	8.75	**<.001**
Hyperactivity/Impulsivity	4.38	2.72	15.67	6.38	12.07	**<.001**
Teacher DSM-IV Ratings						
Inattention	2.18	2.50	15.20	7.29	12.54	**<.001**
Hyperactivity/Impulsivity	2.45	2.77	17.82	7.62	14.06	**<.001**
Clinician BRIC Ratings						
Activity	2.67	1.24	3.72	1.12	4.57	**<.001**
Impulsivity	1.96	1.00	3.30	1.46	5.44	**<.001**
Attention	2.24	1.19	3.59	1.16	5.92	**<.001**

The results of the 3-way Trial Type (C+1 vs. E+1) x Group (Control vs. HI) x Sex (male vs. female) mixed factorial ANCOVA examining the effects of high levels of ADHD symptoms and sex on post-error slowing, covarying for estimated IQ are shown in **Figure 2**. Overall, the main effect of trial type was significant, with children exhibiting the expected slowing after errors ($F = 14.40$; $p < .001$; partial $\eta^2 = .12$). The Trial Type x Group interaction was also significant ($F = 6.75$; $p = .01$; partial $\eta^2 = .06$) with HI children slowing significantly less after error commission than controls (143 ms *versus* 261 ms, respectively). This effect is highlighted by the fact that, while MRT_{C+1} of both groups were virtually identical (mean difference = 7 ms), MRT_{E+1} significantly differed (mean difference = 111 ms; $t = .1.96$; $p = .05$). The Trial Type x Group x Sex interaction was not significant, indicating that sex did not interact with ADHD symptoms to impact the post-error slowing phenomenon ($F = .37$; $p > .10$; partial $\eta^2 = .004$), nor were

there any other sex related effects on MRT. Repeating the analysis without covarying for estimated IQ yielded comparable results, resulting only in a greater main effect for Trial Type ($F = 39.39$; $p < .001$; partial $\eta^2 = .27$).

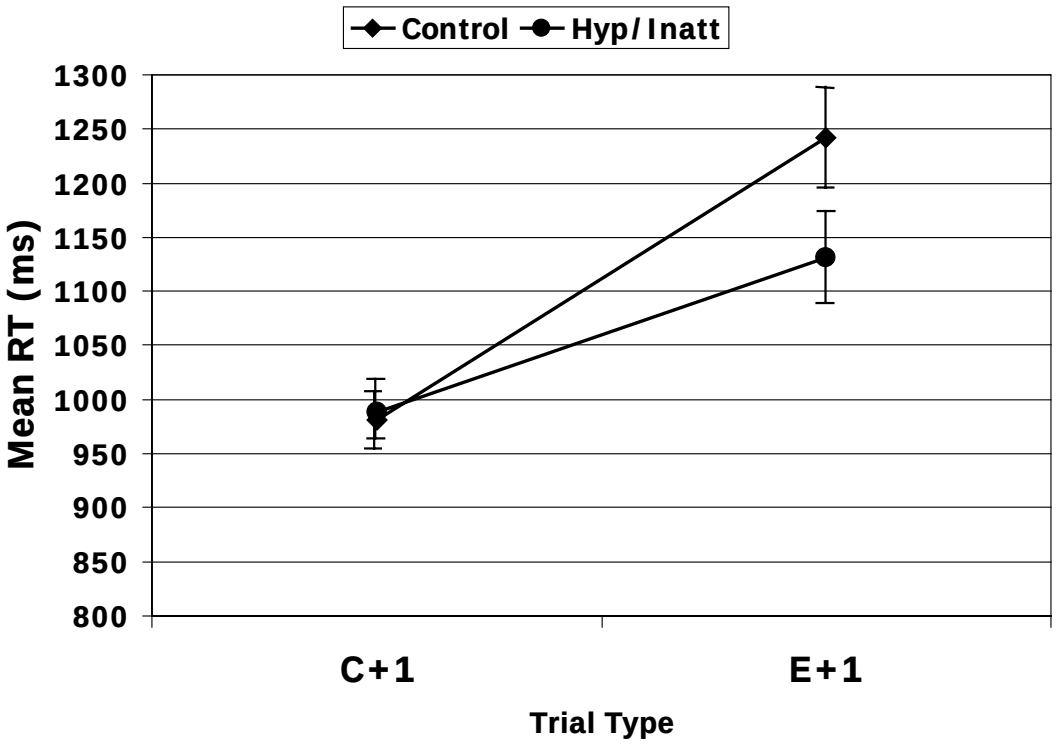

Figure 2. Effect of ADHD symptoms on post-error RT slowing in preschool children (error bars indicate standard error of the mean).

Post Hoc Exploratory Analyses

To further explore differences between preschool children who did and did not slow in each of the two groups, the sample was divided into those who

slowed after errors ($MRT_{C+1} < MRT_{E+1}$; $n = 84$) and those who did not ($MRT_{C+1} \geq MRT_{E+1}$; $n = 26$). The proportion of children in each group who failed to exhibit post-error slowing did not significantly differ (18.2% of controls, 29.1% of HI children; $\chi^2 = 1.81$; $p > .10$). These two groups were then compared in the HI sample and the control sample separately on a variety of variables that might be related to inattention, including parent and teacher rated symptoms (hyperactivity/impulsivity and inattention); clinician BRIC ratings of hyperactivity, inattention, and impulsivity, solid state actigraph measures of motor activity during the laboratory assessment; and the number of runs of errors they made during the RT task (i.e., the number of times 2 or more successive errors were made) using independent samples t tests (see **Table 5**). Within the HI sample, children who did not exhibit post-error slowing were rated as more inattentive by both parents and teachers, but *not* more hyperactive/impulsive. In contrast, although clinicians did not rate them as being more inattentive during the laboratory assessment, they did rate HI children who did not exhibit post-error slowing as being marginally more active and significantly more impulsive on the BRIC. HI children who did not slow after making errors also made more runs of successive errors, likely due to lapses in attention to the task. The only difference between controls who did and did not slow after errors was that, like those in the HI group, controls who did not slow were rated to be more active and more impulsive during the lab assessment by clinicians on the BRIC.

Table 5.

Differences Between Preschool HI Children Who Did and Did Not Exhibit Post-Error Slowing

	- Slowing		+ Slowing			
Characteristic	Mean	SD	Mean	SD	*t*	*p*
HI group						
N	16		39			
WPPSI Information	11.40	3.04	9.92	3.32	1.56	.13
Error Runs[a]	11.33	6.74	6.10	4.67	3.08	**<.005**
Actigraph[b]	591.96	666.54	497.90	367.59	.66	.51
Parent DSM-IV Ratings						
Inattention	13.81	5.53	10.51	5.09	2.13	**<.05**
Hyperactivity/Impulsivity	16.38	6.70	15.38	6.31	.52	.61
Teacher DSM-IV Ratings						
Inattention	18.50	6.30	13.85	7.30	2.23	**<.05**
Hyperactivity/Impulsivity	18.81	7.17	17.41	7.85	.62	.54
BRIC Ratings						
Activity	4.13	.99	3.56	1.14	1.67	.10
Impulsivity	4.07	1.22	3.00	1.45	2.52	**<.05**
Attention	3.93	.88	3.46	1.23	1.56	.13
Controls						
N	10		45			
WPPSI Information	12.30	2.45	11.73	2.76	.60	.55
Error Runs[c]	3.67	2.74	6.23	6.21	1.18	.25
Actigraph[d]	372.14	233.34	339.98	316.17	.26	.80
Parent DSM-IV Ratings						
Inattention	3.90	3.21	4.29	2.94	.37	.71
Hyperactivity/Impulsivity	4.20	2.20	4.42	2.84	.23	.82
Teacher DSM-IV Ratings						
Inattention	2.20	2.30	2.18	2.56	.03	.98
Hyperactivity/Impulsivity	3.20	3.55	2.29	2.59	.94	.35
BRIC Ratings						
Activity	3.56	1.01	2.48	1.22	2.48	**<.05**
Impulsivity	2.78	.97	1.79	.93	2.90	**<.01**
Attention	2.67	1.00	2.14	1.22	1.20	.24

[a] –Slowing HI *n* = 15; +Slowing HI *n* = 31

[b] Mean of waist and ankle medians

[c] –Slowing Control *n* = 9; +Slowing Control *n* = 22

Study 2: Post-Error Reaction Time Slowing in Adolescents/Young Adults with Childhood ADHD

Method

Participants and Recruitment

Eighty-seven adolescents/young adults with childhood ADHD and 84 controls participated in this study. Those with childhood ADHD were initially referred to a study investigating ADHD and other disruptive behavior disorders between 1990 and 1997, when they were between the ages of 7 and 11 years of age (Halperin et al., 1997). They were referred through local schools and medical care providers. This follow-up assessment took place on average 9.30 years (SD = 1.65 years) after the initial evaluation.

At baseline, all children were assessed for the presence of Axis I disorders using parent reports on the Diagnostic Interview Schedule for Children (DISC), version 2.1 or 2.3, depending on time of recruitment (Fisher et al., 1993; Shaffer et al., 1996) and the Child Behavior Checklist (Achenbach, 1991). Teacher reports on the IOWA Conners Rating Scale (Loney & Milich, 1982) were also used. Children with any chronic serious medical or neurological condition were excluded. All participants met diagnostic criteria for ADHD, Combined Type at the time of initial assessment. Those assessed at follow-up did not significantly differ from the baseline sample with respect to age at initial evaluation, rates of childhood comorbid diagnoses, Full Scale IQ (FSIQ), socio-economic status

(SES), or ADHD behavior ratings at initial assessment (all p's > .10). See **Table 6** for childhood characteristics of the ADHD sample.

Table 6.

Adolescent ADHD Sample Characteristics in Childhood

	ADHD (*n* = 87)	
Characteristic	Mean	*SD*
Age (years)	9.10	1.25
WISC-R/WISC-III FSIQ[a]	94.22	14.62
WISC-R/WISC-III VIQ[a]	95.40	16.31
WISC-R/WISC-III PIQ[a]	93.90	15.37
CBCL Attention problems[b]	71.83	10.13
IOWA Conners:		
Inattention/Overactivity	11.06	3.22
Aggression/Defiance	8.28	4.73

[a]WISC-R/WISC-III = Wechsler Intelligence Scale for Children, Revised/3rd Editions
[b]CBCL = Child Behavior Checklist.

The 84 never-ADHD healthy adolescent/young adult control participants were recruited at the time of the follow-up study from the same urban communities as the ADHD group. A variety of methods was used to recruit the Control group, although most came through targeted advertisement in neighborhoods matching those of the ADHD sample by zip code. Those interested in participating were screened by telephone for study eligibility. Criteria for exclusion included chronic medical, psychiatric, or neurological conditions.

The Institutional Review Boards of Queens College of the City University of New York and the Mount Sinai School of Medicine approved all study procedures. Written informed consent was obtained from all adolescents above the age of 18 years and the parents of those under age 18 years. Verbal assent

was obtained from youth under the age of 18 years. All participants were compensated for their time and travel expenses associated with participation.

ADHD diagnosis was assessed at follow-up using the Kiddie-SADS Present and Lifetime Version (K-SADS-PL; Kaufman et al 1997), which was administered to each adolescent and a parent separately. Evaluators were either Ph.D.-level psychologists or trained psychology graduate students and were blind to group membership for the interview. Symptoms were coded as present if either informant or the interviewing clinician endorsed the item as causing significant distress or impairment. Based on this interview, the childhood ADHD group was subdivided into those who continued to meet diagnostic criteria for ADHD ("Persisters" n = 36) and those who clearly did not ("Remitters" n = 28). Persistence was defined as meeting DSM-IV diagnostic criteria for ADHD (i.e., a minimum of six symptoms of inattention and/or six symptoms of hyperactivity-impulsivity). Among the Persisters, 28.6% continued to meet full DSM-IV criteria for ADHD – Combined Type (ADHD-C), 50.0% met symptom criteria for ADHD Predominantly Inattentive Type (ADHD-I), and 21.4% met criteria for ADHD-Hyperactive-Impulsive Type (ADHD-HI). Based on childhood status, the majority of those presenting as either ADHD-I or ADHD-HI had ADHD-C at baseline, likely being in partial remission from this subtype, rather than having shifted to another subtype.

The Remitters group included those individuals with three or fewer symptoms of inattention and three or fewer symptoms of hyperactivity-impulsivity. Those individuals with more than three symptoms in either domain, but fewer

than six symptoms in one domain were not included in analyses that focused on outcome status (n = 23).

To help verify the subgroup classification based on the K-SADS-PL interview, all adolescents and parents independently completed a DSM-IV ADHD symptom checklist consisting of all 18 DSM-IV inattentive and hyperactive/impulsive items. Possible scores for each item ranged from 0 (“not at all”) to 3 (“very much”).

The ADHD and Control groups did not significantly differ from one another on age, gender, ethnicity, SES, and FSIQ (all $p > .05$; see **Table 7**); however, adolescents diagnosed with ADHD in childhood did exhibit marginally lower FSIQ and VIQ. General intellectual functioning was estimated using the Wechsler Adult Scale of Intelligence, Third Edition (WAIS-III; Wechsler, 1997) at the time of follow-up assessment. Overall, the entire sample (N = 171) was predominantly male (87.7%) and racially and ethnically diverse (26.0% African American, 24.1% Caucasian, 30.6% Hispanic, and 19.3% mixed or other descent). All participants and their parents were proficient in English. At the time of follow-up assessment, the age of the sample ranged from 15 to 21 years (mean age = 18.41 years, SD = 1.64), with one outlying participant aged 26.31 years. Most participants in both groups lived in an urban environment within a major metropolitan area. SES was determined using parental occupation and education and was quantified using the socioeconomic prestige scale of Nakao and Treas (1994). The mean SES score for the follow-up study was 42.24 (SD = 17.00), with the full range of possible scores being represented (20 to 96). The modal score in the sample

was 20 (n = 30, 17.9%), the value given to persons unemployed or on welfare. Thus, the sample was primarily represented by lower to lower-middle SES individuals. See **Table 7** for adolescent characteristics.

Table 7.

Adolescent Follow-Up Sample Characteristics

	ADHD (*n* = 87)		Control (*n* = 84)			
Characteristic	Mean	*SD*	Mean	*SD*	*t*	*p*
Age (years)	18.32	1.63	18.49	1.65	.67	.50
SES[a]	43.79	17.35	44.69	16.60	1.18	.24
WAIS-III FSIQ	92.65	14.64	96.56	15.27	1.70	.09
WAIS-III VIQ	93.13	15.58	97.82	16.03	1.94	.06
WAIS-III PIQ	92.72	14.46	94.98	13.75	1.04	.30
Parent DSM-IV Report						
Inattention	14.44	7.44	3.82	4.45	10.75	**<.001**
Hyperactivity/Impulsivity	9.57	7.65	2.00	4.22	7.61	**<.001**
Adolescent DSM-IV Self-Report						
Inattention	9.58	6.57	2.78	3.34	8.22	**<.001**
Hyperactivity/Impulsivity	7.44	6.95	1.89	2.50	6.70	**<.001**

[a]SES range = 20 to 96 (Nakao and Treas 1994)

Reaction Time Task

All participants were administered Nassauer and Halperin's (2003) Motor Conflict Task. This is a computerized two-choice, self-paced RT task consisting of a total of 400 trials administered in 5 conditions of varying levels of conflict. The reader is referred to the original publication for a full-description of the task. Only the highest-conflict condition (Condition 5; *n* = 80 trials) was used for this study, during which 40 left- and 40 right-pointing arrows successively appeared in random order and in equal proportions on either the left or right side of the

computer screen. The same random order of stimuli was presented to each participant. Participants were instructed to ignore the location of the arrow and to press the mouse button on the side opposite that indicated by the arrow (e.g., to press the right mouse button in response to a left pointing arrow appearing anywhere on the screen.

The other four conditions, which were not used in this study, were either no-conflict (i.e., Conditions 1 and 3) or low-conflict (i.e., Conditions 2 and 4) conditions. In Condition 1, participants responded to rectangles appearing on either the left or the right side of the screen by pressing the mouse button compatible with the location of the stimulus (e.g., left button press for left-sided rectangle). In condition 2, participants responded with a button press incompatible with the stimulus location (e.g., left button press for right-sided rectangle). In Condition 3, centrally located left- or right-pointing arrows appeared on the screen and participants responded with the mouse button compatible with the direction indicated by the arrow. In Condition 4, participants responded with the mouse button incompatible with the direction indicated by the arrow. Each of these conditions consisted of 40 randomized trials with a 50% probability of either stimulus.

The response device was a standard two-button mouse for use with a desktop personal computer. Participants used the index and middle fingers of the dominant hand to respond. They were encouraged to respond as quickly as possible without sacrificing accuracy.

Data Analysis

The objectives of the analyses were to examine (1) whether adolescents diagnosed with ADHD in childhood showed reduced slowing compared to controls, and (2) to compare the degree of post-error slowing across adolescent/young adult Persisters, Remitters, and Controls. The first objective was addressed by a 2-way Childhood Diagnosis (Control vs. ADHD) x Trial Type (C+1 vs. E+1) mixed factorial analysis of covariance, controlling for WAIS-III FSIQ.

The second objective was addressed by a 2-way Adolescent Status (Persisters vs. Remitters vs. Controls) x Trial Type (C+1 vs. E+1) mixed-factorial analysis of covariance, again controlling for FSIQ. These analyses were repeated without covarying for IQ to examine the stability of effects as a function of this variable.

Mean RT (MRT) was used as the dependent variable in each of these analyses. Trials were selected for each of the two Trial Type conditions in a manner similar to that used for the Preschool study described above with the following differences. Only correct RTs between 200 and 2000 ms were included. As was the case in the preschool study, adolescents had to make a minimum of three C+1 and E+1 responses between the acceptable timeframes to be included in the analyses.

Results

Sample Characteristics

Seventeen participants (4 controls, 13 ADHD) did not attain accuracy rates of 70% or greater and were thus excluded. Of those achieving greater than 70% accuracy, 91 participants (51 Controls, 40 with ADHD) made fewer than 3 isolated errors (19 Controls, 10 with ADHD made no errors). Thus, the final analysis included 29 Controls and 34 individuals with ADHD. The proportion of males did not differ across the Control and ADHD groups (86.2% Controls, 88.2% ADHD). As with the full sample, the ADHD and control groups differed from one another on parent and self-reports of inattention and hyperactivity/impulsivity. Neither FSIQ nor SES differed significantly across the two groups (see **Table 8**).

Table 8.

Adolescent Follow-Up Sample Characteristics: Partial Sample

	Control (n = 29)		ADHD (n = 34)			
Characteristic	Mean	SD	Mean	SD	t/χ^2	p
Age (years)	18.22	1.56	17.89	1.25	.93	.35
SES[a]	38.17	14.90	42.56	14.52	1.18	.24
WAIS-III FSIQ	90.45	11.17	90.91	12.55	.15	.88
Parent Report						
DSM-IV Inattention	3.54	4.15	15.40	7.57	7.39	**<.001**
DSM-IV Hyperactivity/Impulsivity	2.04	24.81	10.63	8.08	4.91	**<.001**
Adolescent Self-Report						
DSM-IV Inattention	3.23	4.03	12.13	7.84	5.45	**<.001**
DSM-IV Hyperactivity/Impulsivity	2.27	3.00	10.23	8.29	4.91	**<.001**

[a]SES range = 20 to 96 (Nakao and Treas 1994)

Overall Task Performance

Figure 3 shows the increase in RT for both ADHD and Control groups in the high-conflict condition (Condition 5) relative to the other 4 low- and no-conflict conditions of the task. A two-way mixed-factorial ANOVA examining the effects of Condition and Childhood Diagnostic Group Status on mean correct RT indicated that Condition 5, the task block used for this study, was the most challenging for both groups ($F = 55.77$; $p < .001$; partial $\eta^2 = .48$). Follow-up paired samples *t* tests indicated that the mean correct RT for Condition 5 was significantly longer than that for all low- and no-conflict conditions. Although there was a marginal difference between the two diagnostic groups in mean correct RT on all conditions of the task ($F = 2.79$; $p = .10$), this difference did not vary significantly as a function of Condition ($p > .10$).

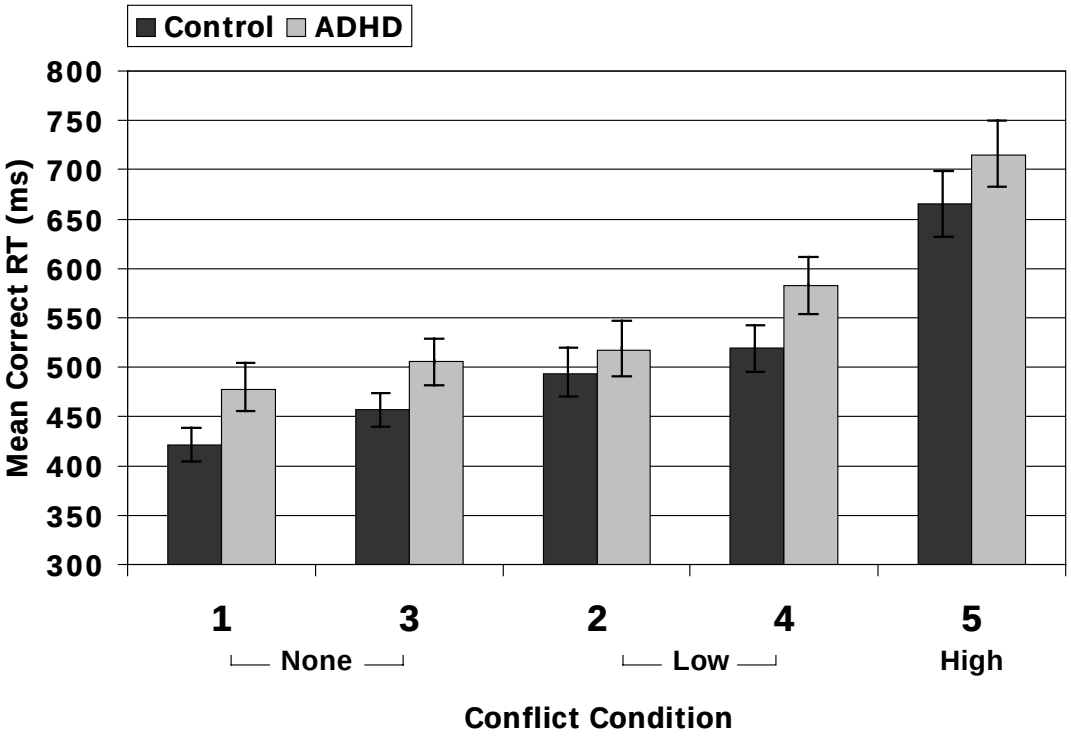

Figure 3. Effect of conflict on mean correct RT across the 5 conditions of the Motor Conflict Task in adolescents. Conditions 1 and 3 = no conflict; Conditions 2 and 4 = low conflict; Condition 5 = high conflict. Error bars represent standard error of the mean.

Effects of Diagnostic Status on Post-Error Slowing

Table 9 shows the task performance data for the Control and ADHD groups. In terms of overall task performance, Controls were slightly, but not significantly more accurate than those with ADHD and made marginally more C+1 responses. However, Controls and those with ADHD did not significantly differ in the number of E+1 responses or in MRT for correct responses, errors, C+1 responses, or E+1 responses

Table 9.

Adolescent Follow-Up Task Statistics: Partial Sample*

	Control (n = 29)		ADHD (n = 34)			
Statistic	Mean	*SD*	Mean	*SD*	*t*	*p*
% Correct	92.80	3.34	90.15	7.27	1.91	.06
# C+1[a]	62.03	5.79	58.06	10.72	1.87	.07
# E+1[b]	4.41	1.43	5.09	1.90	1.57	.12
$RT_{correct}$	665	170	715	183	1.11	.27
RT_{error}	662	453	596	209	.73	.47
RT_{C+1}	618	123	652	119	1.10	.28
RT_{E+1}	698	226	714	182	.31	.76
Mean Z (MRT_{E+1})[c]	.38	.62	.35	.81	.17	.87

*Participants with fewer than 3 eligible C+1 or E+1 responses and <70% accuracy excluded).
[a]C+1 = number of correct responses made following 2 consecutive correct responses
[b]E+1 = correct responses made following isolated errors
[c] Mean Z (MRT_{E+1}) = $\Sigma[(RT_{E+1} - MRT_{C+1}) / RTSD_{C+1}] / n_{E+1}$

In terms of post-error slowing, the two-way Trial Type (C+1, E+1) x Childhood Diagnostic Status (Control, ADHD) ANOVA showed that there was a main effect of Trial Type, indicating the presence of post-error slowing ($F = 13.97$; $p < .001$; partial $\eta^2 = .19$). However, there was no significant interaction between Trial Type and Group, suggesting that the degree of post-error slowing did not differ across the groups; the control group slowed by an average of 80 ms, while the ADHD group slowed by 62 ms ($p > .10$; see **Figure 4**). As indicated in **Table 9**, individuals in both groups slowed by an average of one-third of a standard deviation relative to their individual MRT_{C+1}.

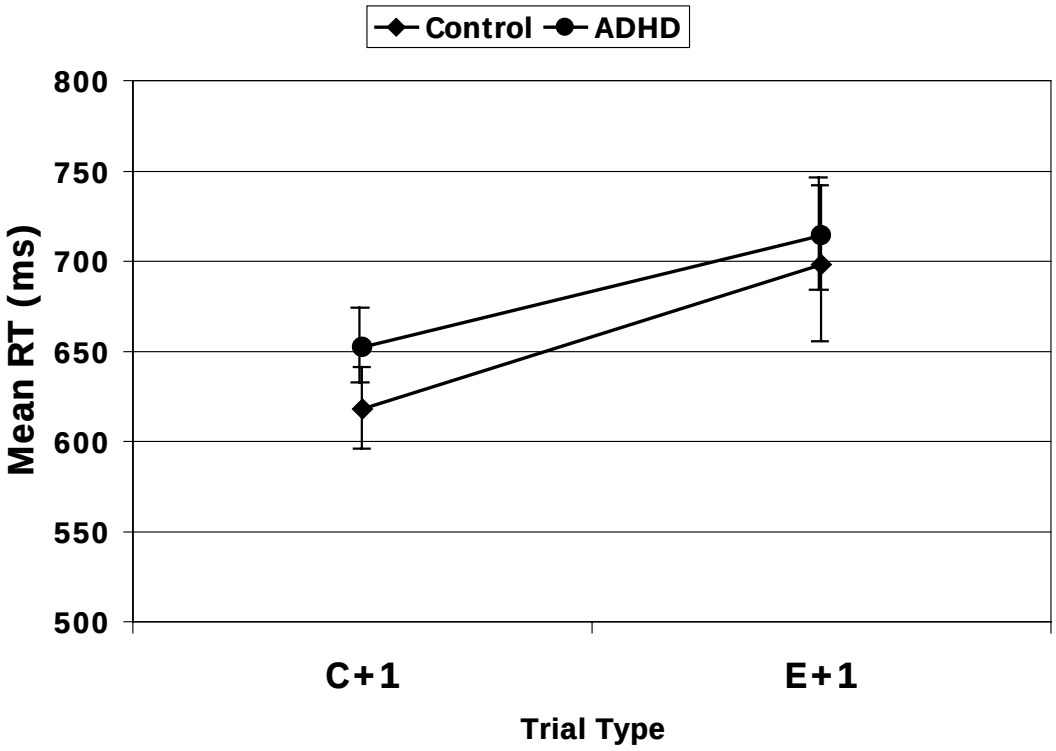

Figure 4.

Effect of childhood ADHD status on post-error slowing in adolescence/young adulthood (error bars indicate standard error of the mean).

When the Childhood ADHD Status group was separated by adolescent diagnostic status, there were 17 Persisters and 7 Remitters. Persisters and remitters did not differ significantly in overall accuracy rate, the number of C+1 or E+1 responses, or MRTs for correct, erroneous, C+1, or E+1 responses. However, when the degree of post-error slowing exhibited by the two groups was compared in the form of standard deviations from MRT_{C+1} (i.e., Z (MRT_{E+1})), while Persisters slowed by two-thirds of a standard deviation, Remitters did not slow at all (see **Table 10**).

Table 10.

Adolescent ADHD Group Task Statistics: Persisters vs. Remitters*

	Remitters (n = 7)		Persisters (n = 17)			
Statistic	Mean	*SD*	Mean	*SD*	*t*	*p*
% Correct	92.14	5.14	89.93	6.71	.78	.44
# C+1[a]	61.14	8.15	56.88	10.55	.95	.35
# E+1[b]	4.86	1.68	5.41	2.09	.37	.54
$RT_{correct}$	670	180	720	213	.54	.60
RT_{error}	656	348	556	134	1.04	.31
RT_{C+1}	634	145	643	122	.14	.89
RT_{E+1}	615	162	750	180	1.72	.10
Mean Z (MRT_{E+1})[c]	-.09	.44	.60	.08	2.66	**.02**

*Participants with fewer than 3 eligible C+1 or E+1 responses and <70% accuracy excluded
[a] C+1 = number of correct responses made following 2 consecutive correct responses
[b] E+1 = correct responses made following isolated errors
[c] $Z\ (MRT_{E+1}) = \Sigma[(RT_{E+1} - MRT_{C+1}) / RTSD_{C+1}] / n_{E+1}$

The examination of post-error slowing as a function of adolescent status using a two-way mixed factorial Trial Type (C+1, E+1) x Adolescent Diagnostic Status (Control, ADHD Persisters, ADHD Remitters) ANOVA indicated that, as a whole, individuals significantly slowed after errors ($F = 5.36$; $p < .05$; partial $\eta^2 = .10$). However, when broken down into groups by adolescent diagnostic status, despite the fact that Remitters did not exhibit post-error slowing while the other two groups did (see **Figure 5),** the groups did not differ significantly in the degree of post-error slowing they exhibited ($p > .10$).

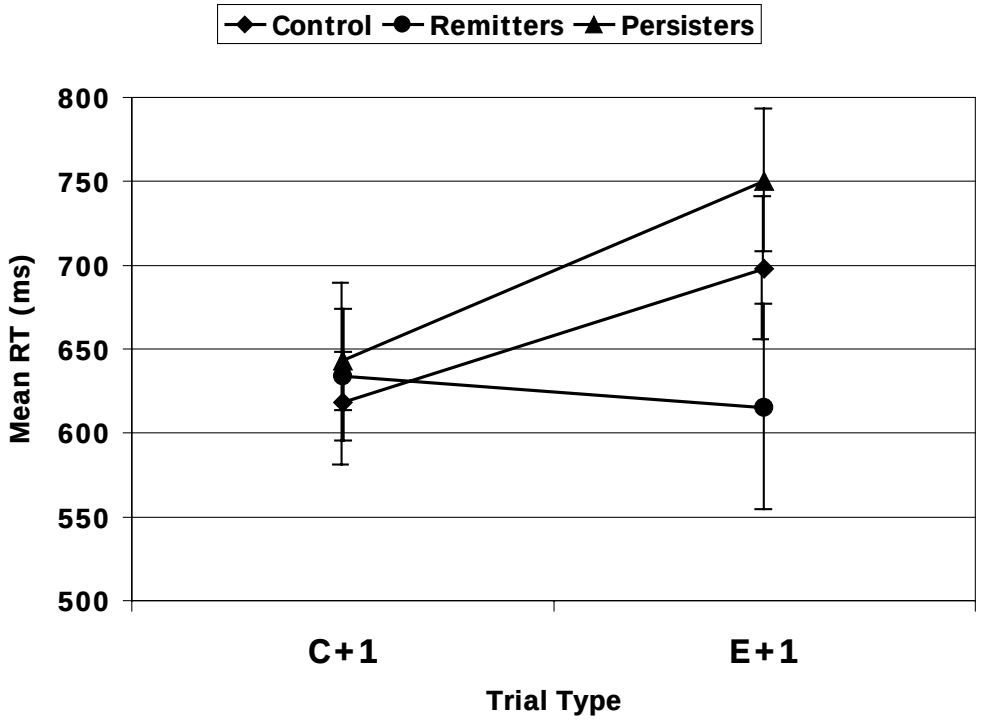

Figure 5.

Post-error RT slowing in adolescent ADHD Persisters and Remitters relative to controls (error bars indicate standard error of the mean).

Post-hoc Exploratory Analyses

Due to the increasing recognition that ADHD comprises clinically and neuropsychologically heterogeneous subgroups of individuals, the differences between childhood diagnostic groups was further explored. The sample was divided into individuals who slowed after errors ($MRT_{C+1} < MRT_{E+1}$; $n = 42$) and those who did not ($n = 21$). While 75.9% of Controls exhibited post-error slowing, only 58.8% of individuals with ADHD did. However, this difference was not significant ($\chi^2 = 2.05$; $p > .10$).

Among those diagnosed with ADHD in childhood, those who slowed (+Slowing) and those who did not (-Slowing) were compared with one another, as well as with Controls, on several other measures collected at the time of the follow-up assessment. These three groups were compared on measures of current ADHD symptom severity; activity levels measured during the laboratory assessment; and measures of attention/effort, response inhibition, and state regulation from an Identical-Pairs Continuous Performance Test (CPT-IP). Current ADHD-symptom severity was measured using the sum of DSM-IV symptom checklist Inattention and Hyperactivity/Impulsivity ratings.

Solid-state actigraphs were used to measure motor activity during the evaluation. Actigraphs are acceleration-sensitive devices with solid-state memory that store data in the form of number of movements per minute. Data collected from these devices have successfully differentiated boys with ADHD from controls (Porrino et al., 1983), have been shown to be reliable across testing sessions (Reichenbach, Halperin, Sharma, & Newcorn, 1992), and to correlate significantly with parent and teacher ratings of hyperactivity (Reichenbach et al., 1992). Actigraphs were worn on the waist and non-dominant ankle for the duration of the full six-hour assessment, of which this study was a small part. Median movement counts from waist and ankle actigraphs were averaged together and used as the dependent measure in these analyses.

The CPT-IP, similar to that used by (Cornblatt & Erlenmeyer-Kimling, 1985), required participants to monitor for and press a key in response to a rare

target signal (i.e., two successive series of 4-digit stimuli). Four hundred trials were administered, each with a duration of 200ms and a 1500ms inter-stimulus interval. Target frequency was 10%, and the entire task lasted approximately 12 minutes. The task measures processes related to attention/effort (i.e., hits, errors of omission), response inhibition (i.e., false alarms), and state regulation (i.e., MRT, RTSD). Data from all of these measures have consistently distinguished children and adults with ADHD from controls (Hervey et al., 2006; Losier et al., 1996).

For CPT-IP and actigraph measures all three groups were compared. For DSM-IV symptom ratings, only the two ADHD groups (those who exhibited post-error slowing and those who did not) were compared with one another. Each group of variables (i.e., DSM-IV symptoms, CPT-IP measures, actigraph means) were subjected to separate multivariate analyses of variance (MANOVAs) using Hotelling's Trace. Where significant multivariate F's were found, follow-up ANOVAs were conducted to look for significant differences among the groups. Finally, differences among the groups were characterized with the use of post-hoc Tukey HSD statistics (see **Table 11** for multivariate and univariate F statistics).

As shown in **Table 11**, the only measure on which controls who exhibited post-error slowing, and adolescents with childhood ADHD who did and did exhibit post-error slowing significantly differed was overall RTSD on the CPT-IP (partial η^2 = .19). On this measure, those with childhood ADHD who did not exhibit post-error slowing exhibited significantly more variable RT for correct responses on

the CPT-IP than Controls who exhibited post-error slowing (Tukey HSD = ; p < .05).

Table 11.

Comparison of Adolescent Controls with Participants Diagnosed with ADHD Who Exhibited Post-Error Slowing and Those Who Did Not on Measures of Current Symptom Severity, CPT-IP Measures, and Motor Activity During the Evaluation.

	ADHD				Controls			
	- Slowing n = 11		+ Slowing n = 18		+ Slowing *n* = 19			
Statistic	Mean	*SD*	Mean	*SD*	Mean	*SD*	*F*	*p*
DSM-IV Symptoms[a]	-	-	-	-	-	-	.68	.61
Child Inatt	11.17	8.63	12.78	7.45	-	-	.45	.51
Child H/I	10.08	8.31	10.33	8.51	-	-	.01	.92
Parent Inatt	15.00	8.64	15.63	7.13	-	-	.20	.66
Parent H/I	9.00	7.35	11.58	8.52	-	-	1.12	.30
CPT Measures[a]	-	-	-	-	-	-	2.10	**<.05**
Hits	26.91	7.71	29.67	4.47	29.95	5.79	1.06	.36
False Alarms	39.55	40.98	44.00	52.15	21.53	27.61	1.49	.24
MRT	623	75	598	120	635	91	.66	.52
RTSD[b]	271	76	223	68	193	48	5.27	**<.01**
Actigraph[a,c]	-	-	-	-	-	-	.74	.57
Mean Median[d]	5.80	10.66	6.12	12.41	2.54	2.86	.75	.48
Mean SD[e]	214.22	211.14	205.53	156.65	131.98	80.39	1.50	.24

[a] Hotelling's Trace Multivariate F
[b] ADHD – Slowing significantly more variable than ADHD + Slowing (p < .01)
[c] *n*'s for actigraph measures: 18 +Slowing Controls, 17 +Slowing ADHD, 9 –Slowing ADHD
[d] Average ankle and waist medians
[e] Average ankle and waist standard deviations

Discussion

Study 1

In accordance with the first hypothesis of Study 1, it was clearly demonstrated that preschool children as young as 3 years of age exhibited post-error slowing on a computerized choice RT task. The third hypothesis was also confirmed; consistent with results of past studies in older children with ADHD, hyperactive/inattentive preschool children exhibited a reduction in post-error slowing in comparison with controls. However, contrary to the expectation described in Hypothesis 2, we did not find evidence for an increase in post-error slowing between the ages of 3 and 6.

Further *post hoc* exploratory analyses in the preschool sample examined the differences between children who did and did not exhibit post-error slowing. The results of these analyses indicated that, overall, those who failed to exhibit post-error slowing exhibited more behavioral dysregulation during the laboratory assessment according to clinician ratings. Further, within the HI group, children who did not slow after errors exhibited elevated levels of parent- and teacher-rated inattention and made more runs of successive errors, which is suggestive of lapses in sustained attention during the task. These data suggest that lack of post-error slowing in hyperactive/inattentive children may be indicative of poorer sustained attention.

The finding that normally developing preschool children exhibit post-error slowing is consistent with previous literature indicating that children as young as 3.5 years exhibit evidence of intact error detection and awareness (Jones et al.,

2003). But more importantly, this phenomenon has not yet been demonstrated in children of this age using a computerized RT task. Thus, this finding is, to some degree, novel and adds additional support for the valid use of computerized tasks in preschool children. The use of such tasks in preschoolers could potentially lend a greater degree of continuity to research on normal cognitive development (Weissberg et al., 1990). Finally, while the results showing attenuated post-error slowing in HI children were expected based on past research in older children with ADHD (Krusch et al., 1996; Schachar et al., 2004; Sergeant et al., 1988; Wiersema et al., 2005), this is the first study that has shown error processing deficits in such young symptomatic children. Thus, the implications of this finding are substantial.

Post-error slowing has generally been presumed to reflect conscious strategy adjustments in response to suboptimal task performance. This idea is supported by one study which reported a significant correlation between post-error slowing and subsequent post-error accuracy ($r = .448$; Hajcak et al., 2003b) as well as by studies that have associated post-error slowing with activity in lateral PFC (Garavan, Ross, Murphy, Roche, & Stein, 2002; Kerns et al., 2004). However, a multitude of underlying processes may contribute to deficits in post-error slowing in addition to self-regulatory functions. These include basic error monitoring, affective/motivational, and attentional processes.

Although there is debate regarding the exact role of ACC in error detection (i.e., detection of error-produced conflict versus error detection *per se*), many ERP and fMRI studies have localized the error detection signal (i.e., the ERN) to

the ACC/rostral cingulate zone (RCZ; Debener et al., 2005; Kerns et al., 2004; Klein et al., 2007; Mathalon et al., 2003). Error-related activity in this region has been shown to correlate with subsequent post-error slowing (Debener et al., 2005; Garavan et al., 2002; Kerns et al., 2004), suggesting that ACC activity related to error detection is important to post-error behavioral adjustment. Studies also indicate that ACC activity increases with age into late adolescence/early adulthood (e.g., Davies et al., 2004; Hogan et al., 2005) suggesting that maturation of this region and/or its connectivity with regions implicated in self-regulatory functions is protracted.

Two recent theories have addressed the affective/motivational contribution to post-error slowing. Both of these theories posited that the ERN is a negative reinforcement learning signal mediated by mesolimbic dopaminergic innervation of PFC, which in turn, signals motor neurons in ACC (thought to generate the ERN) to up-regulate motor behavior in response to suboptimal task performance (Holroyd et al., 2002; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004). In support of this idea, one study found an inverse relationship between the ERN and subsequent error rates (Hajcak et al., 2003b). Also consistent with this hypothesis, studies have suggested that affective and motivational factors are associated with modifications in the ERN. For example, the ERN is larger when accuracy is emphasized over speed in task instructions (Falkenstein et al., 2000) and self-reported anxiety has been shown to be positively related to ERN amplitude (Hajcak et al., 2003a). Further, activity in

RCZ has been shown to be modified by violations in reward expectancies (see Ridderinkhof et al., 2004 for review).

Finally, several studies have indicated that attention influences post-error slowing, such that post-error slowing is absent when individuals are not aware of having made an error (Hester, Foxe, Molholm, Shpaner, & Garavan, 2005; Klein et al., 2007; Nieuwenhuis et al., 2001). Thus, four possible hypotheses for reduced post-error slowing include: (1) inefficient error detection; (2) decreased motivation to perform well on the task; (3) decreased ability to exert effortful control to compensate for suboptimal task performance; and (4) lack of awareness of error commission.

Unfortunately, data that could be used to test the first three hypotheses were not collected as part of this study. However, the fourth hypothesis was supported somewhat by the results of the *post hoc* exploratory analyses, which suggested that HI children who did not slow following errors were rated to be more behaviorally dysregulated in the laboratory and inattentive both at home and at school. In addition, HI children who did not slow made more runs of successive errors, suggestive of lapses in attention during the task itself. These findings provide some support for the idea that lack of awareness of errors due to lapses in attention during the task may be the cause of the reduced post-error slowing observed in hyperactive/inattentive children.

There were several limitations in this study that preclude unequivocal interpretation of the results. It is likely that the failure to detect any significant increase in post-error slowing with age resulted from the disproportionately high

number of control children excluded due to perfect or near perfect performance, an unfortunate side effect of using a task not specifically designed to assess error processing. Hogan et al. (2005) found that the degree to which increases in post-error slowing between 12 and 22 years of age were evident was largely dependent on task difficulty, with more challenging tasks eliciting greater differences in post-error slowing between younger and older individuals. In fact, 71% of children who were excluded from analyses due to excessively high accuracy (fewer than 2 total errors) were over the age of 4.6 years, the minimum age for inclusion in the oldest group. These are presumably the children who would have exhibited the greatest levels of self-regulation and thus, the greatest levels of post-error slowing on a sufficiently difficult task.

The creation of tasks for use with preschool children that are both sufficiently engaging and motivating and adequately sensitive to the rapid development and wide range of ability within this age range is a formidable challenge in research with children during this developmental period, particularly when behaviorally disordered children are included. The ideal task would have calibrated accuracy to about 80% in the vast majority of individuals. However, in this study, only 35% of controls and 54% of HI children had rates of accuracy of between 70 and 90%. In addition, preschool children tend to experience longer and more frequent lapses in attention resulting in runs of successive errors; even among those children who made more than one error, there were also many children who did not produce the requisite number of *isolated* errors to be included in the analyses. Thus, in sum, the substantial number of children

excluded from these analyses limits the generalizability of the findings from this study.

Study 2

In contrast to the findings of Study 1, those of Study 2 were much more equivocal, failing to support either of the two main hypotheses. Post-error slowing did not vary as a function of childhood or adolescent ADHD diagnostic status. However, *post hoc* exploratory analyses indicated that adolescents diagnosed with ADHD in childhood who did not slow their RTs after errors exhibited higher RT variability on a separate computerized task (i.e., identical-pairs CPT). This finding suggested weak support for poorer state regulation and possible lapses in attention in these individuals (Hervey et al., 2006; Leth-Steensen et al., 2000).

The null findings of this study may be reflective of the substantial limitations of this study. Although post-error slowing was evident in the adolescents both with and without childhood ADHD, similar to the case in the preschool study where we failed to find significant age-related increases in post-error slowing, the failure to find significant differences between the groups may have been due to inadequate task difficulty and/or a related sampling bias. The typical differences in accuracy rates between the groups were very small, with accuracy rates over 90% in both groups, and only marginally significant. In addition, the task used in this study consisted of only 80 trials. Post-error slowing

averages were based on very few errors, atypical for a study of this kind. This may have precluded a meaningful analysis of errors.

As with the task used in the preschool study, this RT task was not intended to be used for analysis of errors; rather, it was intended to analyze increases in mean correct RT as a function of increasing perceptual and motor conflict (Nassauer & Halperin, 2003). High accuracy rates were desirable and the task was created to minimize errors. Thus, relatively few participants made the requisite numbers of errors to be included in the analysis (i.e., 36% of Controls, 29% of Remitters, and 57% of Persisters). This fact calls into question the extent to which the participants (particularly controls) included in this analysis, who were selected because of relatively high error rates, are representative of the populations from which they were drawn. The equivalent levels of post-error slowing exhibited by both childhood diagnostic groups may be due to the fact that the included controls are those with the poorest cognitive functioning.

Nevertheless, despite the fact that the results of the exploratory analyses must be interpreted with extreme caution, the finding that those adolescents with childhood ADHD who did not exhibit post-error slowing exhibited more variable RT during the CPT-IP than controls may converge somewhat with the results of the exploratory analyses in the preschool study. They may point to the lack of awareness of errors as a possible cause of the post-error slowing absence in these individuals. Recent research using ex-Gaussian analysis to examine RT distributions has shown that the elevated RT variability typically found in

individuals with ADHD can be accounted for by lapses in sustained attention that cause longer RTs (Hervey et al., 2006; Leth-Steensen et al., 2000).

General Discussion and Theoretical Significance

The main objective of this dissertation was to examine post-error slowing in ADHD at two developmental time points, preschool age and late adolescence/early adulthood. As mentioned earlier, studies have suggested that error processes, including post-error slowing, are dependent upon a complex network of interacting brain areas, including ACC, lateral PFC, posterior cingulate/superior-parietal cortex/precuneus, and the mesolimbic dopaminergic system. To summarize, mesolimbic dopaminergic influences on PFC and ACC have been suggested to underlie performance monitoring and error detection (Holroyd et al., 2002; Ridderinkhof et al., 2004), while rostral ACC, lateral PFC, and superior/mesial parietal and posterior cingulate areas are thought to mediate the up-regulation of performance in response to errors and, perhaps, the conflict they produce (Hajcak et al., 2003a; Herrmann et al., 2004; Hester et al., 2005; Kerns et al., 2004; Kiehl, Liddle, & Hopfinger, 2000; Mathalon et al., 2003; O'Connell et al., 2007; van Veen et al., 2002). In addition, associated interconnections of these regions with the limbic circuitry (Falkenstein et al., 2000; Mathalon et al., 2003; Nieuwenhuis et al., 2001; van Veen et al., 2002), and locus coeruleus (Aston-Jones & Cohen, 2005) have been proposed to play valuable roles in the affective evaluation of errors as well as the motivated

modulation of cognitive energetic resources (e.g., arousal) in response to suboptimal performance.

Abnormal activity in virtually all of these regions has been demonstrated in ADHD (Bush et al., 1999; Casey et al., 1997; Durston, 2003; Durston et al., 2003; Fallgatter et al., 2004; Rubia et al., 2005; Schulz et al., 2004; Schulz, Newcorn, Fan, Tang, & Halperin, 2005; Swanson et al., 2007; Tamm, Menon, & Reiss, 2006). Fassbender and Schweitzer (2006) recently posited that ACC dysfunction may be primary in ADHD and that activation in more posterior cortical areas on tasks of cognitive control (e.g., inhibitory control) in place of normal levels of activation in ACC and PFC may be a compensatory mechanism. However, a recent meta-analysis suggested that imaging studies of tasks other than inhibitory control tasks, found decreases in parietal activation as well as abnormal activation in PFC and ACC (Dickstein, Bannon, Castellanos, & Milham, 2006). In light of this literature, post-error slowing abnormalities in ADHD could be a result of dysfunction in any number of involved brain regions (e.g., PFC, ACC, superior parietal cortex, mesolimbic dopaminergic pathways, locus coeruleus) or disruption in the connectivity between them.

The fact that post-error slowing is compromised in ADHD so early in the course of the disorder, and long before other executive functioning deficits are detectable (Berwid, Curko, Santra, Bender, & Halperin, 2005; Marks et al., 2005) may suggest that post-error slowing deficits lie at the heart of the disorder and that there are deficiencies or delays in the development of brain systems that are ontogenetically older, like ACC, or dopaminergic and/or noradrenergic

innervation of frontal cortex. Thus, this early deficit is more supportive of bottom-up models of cognitive dysfunction in ADHD rather than deficiencies in PFC alone, as executive function models have suggested.

Future directions

Future studies should explore the relative contributions of deficient error monitoring (i.e., reduced ERN), prefrontally mediated self-regulation, affect/motivation, and/or attention to post-error slowing reductions in young hyperactive/inattentive children. The hypothesis that a deficit in effortful control in response to task demands could be explored using manipulations of task difficulty, similar to those done in the studies of Sergeant and van der Meere (1988) and Krusch et al. (1996). Both these studies showed that whereas effortful control (indexed by post-error slowing) increased as a function of increasing task demands in typically developing children, children with ADHD did not adjust in this way. Correlations between post-error slowing and other measures of self-regulation, such as motor inhibition and interference control, and neural activation in areas considered to be involved in cognitive control may also be enlightening.

The question of whether post-error slowing in HI preschoolers is attenuated due to reduced affective response to errors could be investigated by examining changes in autonomic activity following error commission via skin conductance responses (SCR) and/or heart rate changes. Hajcak et al., (2003b) showed a positive relationship between electrodermal activity and the degree of

post-error slowing on a Stroop-like task. They also found significant heart rate deceleration in response to error commission; however this measure was not related to post-error slowing. In a separate study, this same research group found a relationship between the magnitudes of the error-related SCR and self-reported negative affect (Hajcak, McDonald, & Simons, 2004). Finally, O'Connell and colleagues (2004) found that while older children with ADHD exhibited levels of post-error slowing equivalent to controls (which they used as an indication of error awareness), the SCR following errors was reduced, suggesting that individuals with ADHD did not ascribe the same level of affective significance to errors as controls. However, it should be noted that Hajcak et al. (2003b) argued that due to the long time course of the ANS response, post-error slowing could not be solely dependent upon autonomic activation.

Finally, the hypothesis that lack of awareness of errors is primarily responsible for reductions in post-error slowing could be tested using a paradigm similar to that used in a recent study by O'Connell et al. (2007), which specifically required participants to indicate when they have committed an error.

Another interesting direction to pursue would be to determine whether the degree of post-error slowing in hyperactive/impulsive preschool children relates in any way to symptom exacerbation/abatement. Studies have indicated that large numbers of preschool children exhibit hyperactive/inattentive behaviors and a high proportion of hyperactive/inattentive preschool children tend to outgrow their disruptive behaviors with age (Campbell, 1995). These observations would make examining the parallel longitudinal courses of symptoms and post-error

slowing potentially elucidating in terms of the mechanisms at work in the etiology of ADHD. In addition, in light of the fact that ADHD has been shown to be strongly heritable, the early presence of this post-error slowing deficit may make it an appropriate candidate for an endophenotype (Castellanos & Tannock, 2002).

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