EXAMINING PERFORMANCE MONITORING IN ATTENTION DEFICIT HYPERACTIVITY DISORDER

by

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A thesis submitted in conformity with the requirements for the degree of Master of Arts
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Abstract

Behavioural symptoms, cognitive deficits, and findings from electrophysiological, neuroimaging and genetic studies all suggest atypical performance monitoring in ADHD. Performance monitoring involves error detection and post-error behavioural adjustment and is crucial to behavioural self-regulation and reinforcement learning, both of which are dysfunctional in ADHD. Therefore, post-error slowing was examined in children with ADHD and controls using a modified flanker task both with, and without, error detection provided. There was a significant main effect of group on post-error slowing across conditions and when error-detection was provided, significant post-error slowing deficits were found in children with ADHD. These findings suggest that the performance monitoring deficit in ADHD is specific to post-error behavioural adjustment and supports the inclusion of this deficit in the neurocognitive profile of ADHD. Findings are discussed in terms of current neurocognitive reinforcement learning models of ADHD.
Acknowledgements

Firstly, I would like to acknowledge my husband Dave, for his constant and tireless love and support throughout our life together, but especially during these past two years of graduate school and thesis research. I have only achieved so much and am pursuing my dreams because of his never ending encouragement, strength and optimism. Without his unwavering love and support, none of this would be possible.

I would also like to acknowledge my parents for their love, support, wisdom, guidance, and belief in me throughout my life. I would like to thank them for making me believe that anything I put my mind to is possible and for always setting the bar very high and giving me something to reach for.

I would especially like to acknowledge the generous time and guidance that Dr. Russell Schachar has given in assisting me with this thesis and in other endeavors over the past two years. Not only have I learned a tremendous amount from his vast knowledge, but I have also immensely enjoyed working with such an amiable and compassionate individual as well. He has guided me to be able to think and reason like a scientist, a skill that I hope to continue to hone with his mentorship. I would also like to thank Dr. Schachar for his immense trust and confidence in me and in my research capabilities and potential.
I would also like to acknowledge the support of my SCCP friends, my family, and the staff of Dr. Schachar’s ADHD lab at SickKids (especially Dr. Lisa Goos and Dr. Jennifer Crosbie). Thank you all for supporting me and for always putting a smile on my face.

Lastly, but most importantly with respect to my thesis research, I would like to thank the children and their families who have volunteered to engage in our research, and in doing so, have become our partners on the journey to find answers and help for children with ADHD.
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Chapter 1

Introduction

“To err is human, to perfect is divine”
- Anonymous

Behavioural and diagnostic features of Attention Deficit Hyperactivity Disorder

Attention Deficit Hyperactivity Disorder (ADHD) is a common psychiatric disorder of childhood that affects 3 to 7% of children (American Psychiatric Association, 2000). ADHD is characterized by chronic levels of either inattention, impulsive hyperactivity, or both (see Appendix A for Diagnostic and Statistical Manual of Mental Disorders Fourth Edition Text Revision (DSM-IV-TR) criteria, American Psychiatric Association, 2000). In addition, symptom severity surpasses what is expected in normal development and compromises daily functioning in important areas of life including academic performance and peer and family relationships (Smith, Barkley, Shapiro, 2006). As many as 50% of children referred to mental health clinics are diagnosed with ADHD (Offord, 1985). Up to 87% of those children may also be diagnosed with another disorder, and a further 67% have at least two comorbid disorders (Kadesjo & Gillberg, 2001). For example, children with ADHD are more likely to have coexisting symptoms and a diagnosis of oppositional defiant disorder (ODD) or conduct disorder (CD) than are children without the disorder (Angold, Costello, & Erkanli, 1999). In addition, children with ADHD are at increased risk of comorbid psychopathologies such as anxiety, mood and learning disorders (Biederman et al., 1992; Mayes, Calhoun, & Crowell, 2000; Seidman, Biederman, Monuteaux, Doyle, & Faraone, 2001). ADHD is also diagnosed approximately three
times more often in boys than in girls (Smith et al., 2006). Research indicates that ADHD persists into adolescence and adulthood in up to 66% of cases (Barkley, Fischer, Smallish, & Fletcher, 2002). ADHD is also associated with considerable impairment across multiple domains of functioning. Children with ADHD consistently display poor behavioural judgment and are shown to be at increased risk for the development of substance use, academic underachievement and school failure, low occupational status, difficulties with social relationships, and overall poor social adjustment (Mannuzza, Klein, Bessler, Malloy, & Hynes, 1997; Smith et al., 2006; Barkley, 1997). These learning, social, and behavioural problems all suggest that individuals with ADHD have some degree of difficulty with behavioural self-regulation and monitoring of their behaviour and/or performance.

**Cognitive features of ADHD**

Current theories of ADHD postulate that core symptoms of the disorder arise from cognitive deficits in executive functioning (Barkley, 1997; Pennington & Ozonoff, 1996) which, in turn arise largely from underlying genetic and neural variation. Executive functions exert cognitive control on processes that enable efficient planning, execution of responses and regulation of goal-directed behaviour (Barkley, 1997). Examples of situations in which executive functioning is required are those where an individual inhibits their behaviour, manages their behaviour when an associated reinforcement is delayed, holds and manipulates information in mind, and self-regulates the accuracy of their behaviour.
As such, children with ADHD exhibit deficits in a variety of cognitive processes, including altered responses to reward (Solanto et al., 2001; Douglas & Parry, 1994, 1983), response inhibition (Schachar et al., Mota, Logan, Tannock, & Klim, 2000; Barkley, 1997), reinforcement delay aversion (Sonuga-Barke, 2003) and working memory (Barnett et al., 2001). In addition, research has found that these children display significantly greater variability in cognitive task performance compared with other children suggesting that symptoms of inattention and hyperactive impulsivity play a role in their responding (Douglas & Parry, 1983). Combined, these cognitive deficits point to difficulties with self-regulation, which is defined as self-directed actions, and which is used to adjust one’s ongoing behaviour for a delayed future consequence. A key self-regulatory cognition actively being investigated in current ADHD research is performance monitoring.

**Performance monitoring**

Performance monitoring refers to the monitoring of one’s performance (or behaviour) for errors, the detection of errors, and the subsequent adjustment of performance. It is one of the executive control processes that provide top-down regulation of elementary mental operations (Logan, 1985; Norman & Shallice, 1986). Errors indicate any type of deviation from originally intended actions and may hinder the achievement of particular goals for an individual. However, errors can also lead to adaptation of behaviour, learning, and subsequent skill acquisition. This process is crucial to the reinforcement learning process, in that erroneous responses have the potential to lead to either positive
or negative outcomes. In fact, goal-directed behaviour requires continuous and appropriate performance monitoring and the process of skill acquisition can be described as the successive elimination of errors (Ohlsson, 1996). The importance of learning from errors is emphasized in education (Borasi, 1994) which is often an area of concern for children with ADHD. A person may possess the knowledge that is required to determine whether a response is incorrect (i.e. detect the error), but lack the knowledge that is required to perform better on subsequent trials (i.e. correct the error; Ohlsson, 1996). Performance monitoring is therefore a critical component for any feedback learning system and is important for self-regulation of behaviour. When individuals effectively monitor their performance for errors, they are accurately detecting when they have made an error and modifying their actions in order to reduce the likelihood of future errors, a process that results in learning. Given an understanding of what performance monitoring is, it is important to describe the cognitive mechanisms that enable people to detect and correct their own errors.

**Sub-processes of performance monitoring: Error detection and post-error behavioural adjustment**

Performance monitoring is commonly considered to consist of the sub-processes of error detection and behavioural adjustment following an error. It is important to note that they do not account for the only components of the process as, for example, the detection of correct responses is also involved in performance monitoring. However, for the purposes of this study only these two components will be discussed.
Error detection is thought of as the knowledge required for the recognition that one has committed an error, whereas post-error behavioural adjustment or strategies, is thought of as the knowledge required for appropriately altering one’s behaviour or performance to reduce the future occurrence of errors. In order to enable error detection and any subsequent behavioural adjustment, ongoing cognitive processes designed to monitor one’s performance must taking place (Yordanova, Falkenstein, Hobnsbein, & Kolev, 2004). Gehring and colleagues (1993) suggested that it is plausible to assume that the prevalence of errors as well as their potential high cost has led to the evolution of cognitive mechanisms aimed at monitoring the accuracy of one’s actions and also that attempt to correct, or compensate for errors (Gehring, Gross, Coles, Meyers, & Donchin, 1993). The notion that a performance monitoring system exists is evident in many theories of cognition. For example, the concept of performance monitoring is included in theories of action (MacKay, 1987), learning (Adams, 1971), speech (Levelt, 1983), consciousness (Kosslyn & Koenig, 1992), and perhaps of most relevance to ADHD, in theories of executive control systems (Logan, 1985; Shallice, 1988).

Error detection

The investigation of performance monitoring began with behavioural studies (Rabbitt, 1966), but recent research in the area has been conducted using electrophysiological (ERP) and neuroimaging paradigms. Although the current study explores performance monitoring at the behavioural level, a review of the literature from ERP and neuroimaging paradigms is important to a theoretical understanding of this phenomena.
Recently, direct evidence for a sub-process of performance monitoring, error detection, has come from ERP studies that delineate a specific brain wave that is generated during erroneous trials, termed the error-related negativity (ERN; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring et al., 1993; Ullsperger & von Cramon, 2004). The ERN is represented by a sharp, negative deflection, peaking approximately 50 to 100 ms following an error (Ullsperger & von Cramon, 2004; Gehring et al., 1993). The ERN is believed to represent the cognitive processes that are engaged when an individual recognizes that they have made an error. Therefore, error detection can be thought of as a within-trial process whereby individuals evaluate whether their actions or responses on each trial are correct or incorrect. The consistency with which the ERN has been found on error trials has led to it being conceptualized as an electrophysiological index of error detection (Bernstein, Scheffers, & Coles, 1995; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Holroyd, Dien, & Coles, 1998; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). ERN appears to depend specifically on the detection of errors and not on the execution of an erroneous motor response, as ERN is linked not only to overt errors in discrimination tasks (Falkenstein, Hohnbein, & Hoornan, 1995), but also to feedback on the commission of errors (Luu, Tucker, Derryberry, Reed & Poulsen, 2003; Miltner, Braun, & Coles, 1997). Studies investigating the generation of the ERN have found it to be localized to, or generated by, a medial frontal region in the brain, particularly in the anterior cingulate cortex (ACC; Gehring & Fencsik, 2001; Luu et al., 2003; Miltner et al., 2003). These findings are supported by hemodynamic methods employing functional magnetic resonance imaging (fMRI) techniques, which indicate activations of the ACC during erroneous trials (Gehring & Knight, 2000; Kiehl, Liddle, & Hopfinger, 2000).
The ACC is believed to be highly flexible as it is capable of detecting errors using information from a variety of contexts. For example, the generation of the ERN is found to occur in several different tasks, including the Eriksen Flanker task, the Sternberg Memory Scanning task, as well as several auditory and visual focused and divided attention tasks (for a review see Falkenstein et al., 1995; Gehring, Coles, Meyer, & Donchin, 1995), and is also found when errors are committed using the feet, eyes, and hands (Holroyd et al., 1998; Nieuwenhuis et al., 2001).

It is suggested that the ERN reflects a general error detection mechanism (Coles, Scheffers, & Fournier, 1995) and several psychological theories exist regarding the cognitive processes involved in that error detection mechanism. Comparator theories hypothesize that the ERN reflects the functioning of a mechanism that compares a representation of the intended response to a representation of the actual response in order to determine whether an error has been made (Bernstein et al., 1995). These comparator theories contend that an error detection mechanism updates on-line representations of goals in an effort to improve performance. The conflict theory (Botvinick, Braver, Barch, Carter, & Cohen, 2001) suggests that ACC activity during error trials is generated immediately during and following the error and is reflected in the ERN. This activity is believed to be the result of the cognitive conflict between the error and the intended response as they are incompatible with each other (Van Veen & Carter, 2002; Botvinick et al., 2001). Holroyd and Coles (2002) propose a model of ERN generation that adopts a reinforcement learning algorithm that has been previously used to describe activity of the mesolimbic dopamine system, termed the method of temporal differences (TD) error
signal. Within the framework of this model, Holroyd and Coles (2002) posit that the ERN is generated when the neural system detects that the consequences of a response are worse than intended. The system produces an error signal (i.e. the TD error signal) which is then used to train the motor system in order to improve performance. Therefore, individuals engage in learning appropriate responses through the influence of reinforcement principles.

*Behavioural adjustment after errors: Post-error slowing*

Research examining the functioning of the performance monitoring system began several decades ago with behavioural studies investigating individual’s responses and reaction times during speeded choice reaction time tasks. Specifically, early studies discovered that individuals tend to slow their reaction times on trials following an error (Rabbitt, 1966). More recently, Rabbitt (2002) demonstrated that despite instructing young adults to ignore errors in their performance, they still continued to register them as their reaction times following errors tended to be slow. Therefore, the second sub-process in performance monitoring, behavioural adjustment following errors, is reflected in specific post-error strategies such as changes in one’s reaction times following errors. For example, within tasks in which accuracy is emphasized over speed, the appropriate form of behavioural adjustment is to slow one’s reaction time following the commission of an error, in order to reduce the likelihood of committing future errors. This strategy is referred to as post-error slowing and is revealed by the slowing of reaction time on correct trials following the error (Rabbitt, 1966). The usefulness of employing post-error
strategies is related to the learning process in that the reduction in one’s speed of responding strategically increases the likelihood of making a correct response on the following trials, and thereby reduces the number of errors made. Post-error slowing has also been found within the context of failed inhibition errors (Schachar et al., 2004), as well as on a variety of other cognitive tasks (Gebring & Fencik, 2001; Botvinick et al., 2001). These reports are consistent with the hypothesis that participants adjust their speed in response to an error (Rabbitt & Rodgers, 1977; Laming, 1979). Furthermore, the post-error slowing revealed from these studies is postulated as a behavioural index of the functioning of the performance monitoring system. Although it is clear that individuals slow their reaction time after errors, the specific mechanism of this post-error slowing is not yet known.

Data obtained from ERP and behavioural studies of performance monitoring are believed to be complementary with each other. For example, research shows that the degree of post-error slowing is directly related to the size of the ERN amplitude on the error trial (Coles et al., 1995; Botvinick et al., 2001). Thus, the larger the ERN amplitude, the more participants slow their reaction times on trials immediately following errors (Gehring et al., 1993; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). Furthermore, larger ERNs have been elicited when there is a greater discrepancy between the desired/correct and actual/incorrect response, thereby eliciting a greater amount of post-error adjustment (Bernstein et al., 1995). Another ERP component related to performance monitoring is the error positivity (Pe; Nieuwenhuis et al., 2001). The Pe is a positive wave that usually follows the ERN approximately 200 ms to 500 ms after erroneous responses (Ullsperger
& von Cramon, 2004). However, the functional significance of the Pe is much less clear. It seems to vary independently of the ERN and shows a high variance across subjects and tasks (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). In their review, Falkenstein et al. (2000) considered several hypotheses regarding the Pe including that it could be a correlate of immediate error correction, or rather that it may be elicited by the error event itself and reflect additional processing that becomes necessary when an error is encountered. Therefore, it has been posited that the ERN specifically reflects the detection or processing of errors and that the Pe is elicited by the evaluation of the incorrect response (Falkenstein et al., 2000; Leuthold & Sommer, 1999; Overbeek, Nieuwenhuis, Ridderinkhof, 2005). This hypothesis is supported by Vidal, Hasbrouco, Grapperon, and Bonnet (2000) who reported that the Pe was only observed after errors and by other studies that have notably found that the Pe was only present for conscious, rather than unconscious errors (Nieuwenhuis et al., 2001). Furthermore, the Pe has been thought to reflect the subjective or emotional processing of errors (Falkenstein et al., 2000; Nieuwenhuis et al., 2001) as well as the adjustment of response strategies following error detection, such as post-error slowing (Nieuwenhuis et al., 2001; Hajcak, McDonald, & Simons, 2003; Luu, Collins, & Tucker, 2000). As a result of this proposed association of the ERN with error detection and the Pe with post-error slowing, findings from ERP studies (which represent the majority of studies in performance monitoring) can be inferred to apply to behavioural processes of performance monitoring as well.

In addition to ERP studies, the current understanding of behavioural adjustment following errors involves other components, including psychological and neurochemical
Theories drawn from cognitive psychology suggest that slowing following an error results from the comparison of actual responses with representations of intended responses (Bernstein et al., 1995; Dehaene, Posner, & Tucker, 1994; Holroyd & Coles, 2002). These comparator theories posit that post-error slowing indicates that representations of recently executed responses are stored in short-term memory and are compared with the individual’s working memory that holds the instruction set for the given task (Gebring et al., 1993; Scheffers et al., 1996; Scheffers & Coles, 2000). It is proposed that this process of comparison takes time and would delay the individual’s response on subsequent trials. Another cognitive theory of performance monitoring suggests that slowing of responses following errors reflects an attempt to correct the erroneous response (Dehaene et al., 1994; Rabbitt & Rodgers, 1977). Given that the performance monitoring process is intimately linked with learning, it is possible that participants are increasing the amount of time they take in choosing and executing a response following an error in order to decrease the probability of future errors occurring. This in turn may reflect a more intrinsic or motivational influence on the performance monitoring process. Therefore, post-error slowing may reflect more of a conscious response rather than an automatic one. For example, the more sensitive one is to making errors and the more sensitive one is to getting a correct response, the larger the ERN and the corresponding post-error slowing will be. This hypothesis is supported by studies which have found motivational influences can affect ERN generation (see Chapter 4: Discussion for a brief review of the possible influences on the ERN).
As the two sub-processes involved in performance monitoring have been fully described, the interaction and relationship between these two processes must be understood in order to fully comprehend the overall mechanism of performance monitoring and how it results in reinforced learning.

**Proposed cognitive model of performance monitoring**

A cognitive model of performance monitoring is presented in Figure 1. The model is a modified version of a cognitive model of the flanker task developed by Holroyd, Yeung, Coles, and Cohen (2005) and incorporates the specific roles of error detection, behavioural adjustment, reinforcement learning, and the hypothesized impact on the mesolimbic dopamine system, specifically the striatum. In the model (see Figure 1), performance monitoring consists of two modules, the task module, which is responsible for coordinating the necessary processes for successful completion of the task; and the monitor module, whose function is to monitor responses and inform the task module about performance.

There are several processes that occur within each of the modules. Within the task module, the instructions for a given task are processed and stored in short-term and working memory. A perception layer encodes the external input coming into the module, such as information about stimuli and task parameters such as timing. The attention layer facilitates the process of target recognition by increasing activity in the perception layer associated with the recognition of target stimuli, and by inhibiting activity associated
with the processing of the flanking stimuli. Information from the perception and attention layers is then used to formulate an initial representation of the desired responses, which is transferred to working memory for future access and response modification. The representation of the desired response is also sent to the monitor module, where such information is used to produce comparisons of actual responses with the desired responses. The representation of the desired response serves to parameterize the functions of both the task module and the monitor module. The final process within the task module is the response layer, which generates a response command by applying appropriate stimulus-response mappings.

The monitor module uses the representation of desired responses sent by the task module to evaluate performance. Within the monitor module, the value layer assigns levels of salience to errors committed, which are relayed to the TD (temporal differences) unit layer. The TD unit utilizes the information sent from the value layer and produces temporal difference error signals (TD error; Sutton, 1988; Holroyd et al., 2005). These error signals are predictions about task performance, in that positive values indicate that future responses will be favorable for the individual (i.e., a correct response), whereas negative values indicate the occurrence of unfavorable responses (i.e., errors). The TD unit produces enhanced error signals for errors that are deemed to be more salient based on information from the value layer. The TD error signals are then sent to the task module where the information is used to improve performance. For example, negative TD signals indicate that behavioural adjustments are required in order to improve performance and reduce future errors. Information from the error signal is then processed
by the task module and used to form new representations of desired responses, thereby updating the representation held in working memory. Decisions are then made regarding modifications to response output, such as slowing one’s reaction time. At the behavioural level, these improved post-error strategies lead to improved task performance. Task appropriate responses are then reinforced by the generation of positive TD signals and the elimination of inappropriate responses is attempted by the generation of negative TD error signals. If external feedback is provided (e.g., knowledge of performance), information about the feedback on performance is gathered by the monitor module and used to generate TD error signals (Holroyd et al., 2005). This system continues its feedback loop until no further errors occur, resulting in reinforced learning.

This cognitive model of performance monitoring proposes a strong link between performance monitoring and reinforcement learning and a mechanism through which this is achieved. Given that this is a theoretical model, it is important to address if this proposed link and mechanism of reinforced learning is supported by neurological studies, especially those on children with ADHD.

**The mesolimbic dopamine pathway: Linking performance monitoring and reinforcement learning**

To date, most neurological research has presented ADHD as a disorder in which dopamine functioning plays a critical role in its development and expression. More
specifically, dopamine dysfunction in ADHD has implications for cognitive processes such as performance monitoring and reinforcement learning. However, dopamine is wide-spread throughout the brain, therefore affecting numerous systems. Although an imbalance in one or more of these systems will pose difficulties for the organization and regulation of behaviour, for the investigation of performance monitoring and reinforcement processes, the functioning of the mesolimbic dopamine pathway is emphasized. This particular pathway has been implicated in the behavioural and cognitive features of ADHD and specifically in the performance of individuals with the disorder under conditions of reinforcement learning (for a review see Robbins & Everitt, 1996). As such, it is hypothesized that dysfunctional dopamine neurotransmission can produce alterations in reinforcement learning, thereby generating key behavioural aspects of ADHD.

Holroyd and Coles (2002) have provided a theory which links performance monitoring and reinforcement learning. The reinforcement learning model of performance monitoring suggests that when participants commit errors, the mesolimbic dopamine pathway in the brain conveys a reinforcement learning signal to the frontal cortex where it is used to facilitate the development of adaptive motor strategies and responses. The theory contends that the performance monitoring system evaluates ongoing events or performance and predicts whether the events or outcomes are favorable or unfavorable. A positive dopamine signal is elicited by the system when an event or outcome is better than predicted, and a negative dopamine signal is elicited when it is worse than predicted. These signals serve as error signals (TD error signals) which the system uses to improve
performance (Holroyd & Coles, 2002), thereby contributing to learning through the
effects of reinforcement. The mesolimbic dopamine pathway is suggested to play an
integral role in reinforcement learning as the system is highly involved in the processes of
motivation, reward and learning, as demonstrated by studies using ERP and single cell
recordings (Schultz, 2000, 2002; Holroyd & Coles, 2002; Holroyd, Larsen, & Cohen,
2004). The mesolimbic pathway originates in the ventral tegmental area and terminates
in the nucleus accumbens and associated limbic foci. Studies point to the specific role of
this circuit in signaling rewards, coding incentive value, and regulating other behavioural
processes involved in maintaining responding under conditions of delayed reward
(Richardson & Gratton, 1998).

Research supporting the role of the mesolimbic pathway in reinforcement learning has
revealed that it is under-active in ADHD resulting in abnormal functioning of the cortico-
striatal-thalamic loops involved in the control of sensory motor and cognitive functions
(Solanto, 2001). In addition, neuroimaging research has reported reduced striatal
activation in individuals with ADHD both at rest and during the performance of cognitive
tasks (Lou, Henriksen, Bruhn, Borner, & Nielsen, 1990; Vaidya et al., 1998; Rubia, et al.,
1999). In recording spike activity from primate mesolimbic dopamine cells as they learn
to perform various tasks (Schultz, Apicella, Scarnati, & Ljungberg, 1992), researchers
reported that both the presentation of a tangible reward as well as the anticipation of such
reward engaged the system and produced phasic dopamine responses (Schultz, Apicella,
& Ljungberg, 1993). When a reward outcome was less than expected, there was a
 corresponding phasic decrease in DA activity in the striatum. Therefore, Schultz and
colleagues suggest that the phasic responses seen in the dopamine neurons may serve as error signals (Schultz et al., 1992). Furthermore, research indicates that the type of manipulation that influences the mesolimbic dopamine system can be concrete, for example rewards, or more abstract, such as the knowledge that one has performed well (Holroyd & Coles, 2002).

Recent neuroimaging studies with humans implicate the ventral striatum (within the mesolimbic pathway) in reinforcement processing and reinforcement-based learning (Delgado, Nystrom, Fissell, Noll, & Fiez, 2000; Elliott, Friston, & Dolan, 2000; O’Doherty, 2004). Specifically, positron emission tomography (PET) studies reveal dopamine release in the striatum during reinforcement prediction and tracking reinforcement prediction errors (Zald et al., 2004; Knutson, Fong, Adams, Varner, & Hommer, 2001; O’Doherty, Dayan, Friston, Critchley, & Dolan, 2003).

To date, methylphenidate, the stimulant most commonly used to treat ADHD, has been found to normalize Pe amplitudes in children with ADHD (Jonkman, van Melis, Kemner, & Markus, 2007; Groen et al., 2008). This further supports the link between mesolimbic dopamine dysfunction and performance monitoring deficits as methylphenidates acts on dopamine within the brain. Further support for the role of dopamine signaling in performance monitoring was found in an important study where participants were grouped according to a dopamine receptor gene polymorphism that was linked to a reduction in neural D2 dopamine receptors (Klein et al. 2007). The participants that carried the gene responded less to negative feedback about performance than those
without the gene. This study is the first to demonstrate that learning from errors required appropriate dopaminergic signaling as dopamine D2 receptor reduction decreased sensitivity to errors in performance.

In summary, the role of dopamine in the mesolimbic pathway and its association with dysfunction in ADHD, coupled with its link to the cognitive functioning and neural activation found in reinforcement learning, points to the need to investigate performance monitoring processes within ADHD.

**Why study performance monitoring in ADHD?**

Performance monitoring is an important executive control process and alterations in it could result in inconsistent, inaccurate, and poorly regulated behaviour as well as deficits in self-regulated learning (Ullsperger, & von Cramon, 2004). Errors result from deviations from originally intended goals, which mean that they hinder the achievement of the goals for an individual. Despite this initial negative consequence, from a greater perspective, errors lead to adaptation of behaviour, learning and skill acquisition (Ullsperger, & von Cramon, 2004). Therefore, the ability to monitor actual performance and desired goals is crucial for self-directed and adaptive behaviour in an ever-changing environment.

Given the importance of monitoring one’s behaviour for errors in reinforcement learning, deficits in performance monitoring likely underlie the behavioural, learning and cognitive
difficulties observed in ADHD. Furthermore, theoretical models of the disorder propose altered reinforcement learning processes as a core deficit in ADHD. Therefore the importance of the performance monitoring process to learning and behavioural regulation indicate that the investigation of performance monitoring within ADHD is warranted.

To date, only six behavioural studies have examined performance monitoring in children with ADHD (Groen et al., 2008; Jonkman et al., 2007; Wiersema, van der Meere, & Roeyers, 2005; Krusch et al., 1996; Schachar et al., 2004; Sergeant & van der Meere, 1988). The results generated by the studies are consistent and indicate a deficit in performance monitoring in children with ADHD. For example, the most recent study used ERP techniques and compared children with ADHD to normal children on a feedback-based learning task (Groen et al., 2008). ADHD children showed a decreased ERN and Pe throughout the task, but especially in the latter stages of the feedback-based learning. Another study employing ERP methodology compared children with ADHD and healthy controls during performance of a flanker task (Jonkman et al., 2007). ADHD children made more errors, especially in high-response-conflict conditions, and did not show post-error slowing. It was found that ERN amplitude was unaffected; however Pe amplitude was reduced in the ADHD group. Schachar et al. (2004) found that children with ADHD slow significantly less in their reaction times following errors of failed inhibition compared with normal control children. This difference in post-error slowing was not attributable to differences in IQ, age, gender, or response speed (i.e. the extent of post-error slowing was not simply a function of the participant’s overall response time.) In addition, this study found that post-error slowing was significantly correlated with a
greater number of ADHD symptoms, indicating that children in the ADHD group with
greater severity slowed the least following errors. Results from this study are consistent
with other reports of a performance monitoring deficit in children with ADHD
(Wiersema et al., 2005; Krusch et al., 1996; Sergeant & van der Meere, 1988).

Despite the consistency in findings of a performance monitoring deficit, the specific sub-
process of the performance monitoring deficit in ADHD remains unclear. This is because
the two sub-processes of performance monitoring (error detection and post-error
behavioural adjustments) were not always explicitly investigated by isolating one from
another in each of these studies, thereby limiting the conclusions that can be drawn
regarding error detection ability and behavioural adjustment independently within
ADHD.

Therefore, the current study aims to examine the sub-processes of error monitoring in
children with ADHD with greater precision. Specifically, this study will explore the sub-
process of post-error slowing in children with ADHD under baseline conditions and
whether it is altered or not when they are provided with overt and immediate error
detection. As an overarching goal, the current study also aims to broaden the
conceptualization and understanding of executive function deficits in ADHD to include
performance monitoring, enabling a more comprehensive exploration of the cognitive
underpinnings of this disorder and its possible etiologies.
Study aims and hypotheses

The first aim of this study is to examine the post-error slowing of children with ADHD and control children. In order to achieve this goal, the development of a task measuring performance monitoring was required. Therefore, a modified flanker task (Eriksen & Eriksen, 1974) was designed to elicit a high error rate among participants. The neutral condition was employed to determine whether children with ADHD possess a deficit in behavioural adjustment following errors, operationalized as less post-error slowing compared to controls. The term “neutral” was used in this condition as simply meaning that no feedback about performance was presented to participants, but it does not imply any other interpretation. It is predicted that children in the ADHD group will demonstrate a deficit in post-error slowing (i.e. less post-error slowing) compared with children in the control group, thereby confirming the deficit previously reported within this population.

The second aim of this study is to isolate the sub-process of post-error slowing involved in performance monitoring. The detection of one’s errors is crucial to learning and to employing appropriate behavioural adjustment following the commission of errors. However, some previous ERP research on children with ADHD has demonstrated reduced ERN amplitude on error trials compared with control children, suggesting a possible deficit in error detection mechanisms and therefore, by association, a deficit in any post-error behavioural adjustment dependent on it (Liotti, Pliszka, Perez, Kotbmann, & Woldoroff, 2005). In contrast, other studies have found that there is no error detection
deficit in ADHD (Sergeant & van der Meere, 1988; Wieserma, et al., 2005; Jonkman et al., 2007; Burgio-Murphy et al., 2007). Therefore, manipulating the reliance of participants on the error detection component of performance monitoring, where it remains unclear if there is a deficit or not for children with ADHD compared to controls, will allow for a possible deficit in the post-error slowing sub-process to be isolated. This is important as past research indicates that the likely location of the purported performance monitoring deficit in children with ADHD may lie in the behavioural adjustment following the commission and detection of errors. As such, the feedback condition was designed to investigate post-error slowing when knowledge of performance on every trial was provided, thereby manipulating the reliance on the self-detection of errors required for any behavioural adjustment following an error. Participants are provided with feedback tones emitted from computer speakers. These tones reflect the accuracy of performance by indicating explicitly when participants have made a correct or incorrect response. A high pitched tone indicates a correct response has been made and a low pitched tone indicates an incorrect response has been made. This experimental manipulation was chosen as it was able to capture both sub-processes of performance monitoring within the same task, while attempting to isolate one from the other as much as possible.

Examining behavioural adjustment processes without requiring self-detection of errors is a novel approach in research on children with ADHD. Previous studies did not isolate post-error slowing from error detection in their investigation of performance monitoring.
and as a result, when deficits have been reported, the mechanism(s) of these deficits remain unclear.

Therefore, it is hypothesized that children with ADHD will demonstrate decreased post error slowing across conditions compared with control children. This is hypothesized because, based on the majority of research to date, it is predicted that children with ADHD do not have an error detection deficit, and rather that their purported performance monitoring deficit is localized to a deficit in post-error behavioural adjustments. In addition, it is hypothesized that children with ADHD and control children will not demonstrate a significant difference in post-error slowing between the feedback and neutral conditions. This is hypothesized because it is posited that like controls, children with ADHD do not have a deficit in error detection and so any post-error slowing deficit cannot be mitigated by providing external feedback of error detection.
Chapter 2

Method

Participants

Seventy-four ADHD participants were drawn from referrals to a clinic specializing in children with attention, learning and behaviour problems in a large urban pediatric hospital. Forty normal controls were recruited through advertisement within the hospital. Participants ranged in age from 6 to 16 years of age and were attending a primary or secondary school, ensuring that both a parent and a teacher could serve as informants. The sample was similar in socioeconomic status and ethnicity to that of the community from which it was drawn. All children were required to be free of stimulant medication for at least 24 hours prior to testing. Participants who were taking non-stimulant medications were excluded from the study.

The Parent Interview for Child Symptoms (PICS-IV; Ickowicz et al., 2006) was used for the parent interview of all ADHD cases. The PICS-IV covers DSM-IV-TR (American Psychiatric Association, 2000) criteria for ADHD, Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), and all other Axis I diagnoses that were necessary to establish inclusion and exclusion criteria for ADHD, as well as covering developmental, medical, and social history. The reliability of this interview is high (e.g., $\kappa = 80\%$ for ADHD; intraclass correlation $\gamma > .90$). The Teacher Telephone Interview (TTI-IV; Tannock,
Hum, Masellis, Humphries, & Schachar, 2002) is a 30 min interview that was administered to teachers over the telephone by a trained interviewer. The TTI-IV covers symptoms of ADHD, ODD, CD, and screens for other disorders. For both of these instruments, symptom presence was rated using specific criteria in an extensive manual. All interviewers were also trained to a criterion of 90% symptom agreement before interviewing parents or teachers. A social worker or nurse with extensive experience conducted the parent interview and a research technologist with a Master’s degree in psychology conducted the teacher interview. Normal controls were assessed in the same way as the ADHD cases with the exception of the parent and teacher interviews (PICS-IV and TTI-IV) due to time constraints. In lieu of interviews, control children’s parents and teachers were given several diagnostic and behavioural questionnaires that were also given to the ADHD group prior to clinic admission. These included the Conners Parent Rating Scales Revised Long Version, (CPRS-R:L; Conners, 1997), Conners Teacher Rating Scales Revised Long Version (CTRS-R:L; Conners, 1997) and the Ontario Child Health Study Scales (OCHS; Boyle, Offord, Racine, & Fleming, 1993). All diagnostic and behavioural questionnaires were screened prior to any research testing in order to ensure that children met inclusionary criteria for the control group.

All children received an extensive assessment of cognitive, psychoeducational, language, and social-emotional functioning as part of a larger research battery. A psychological associate, supervised by a registered clinical psychologist, assessed intellectual ability using the Wechsler Intelligence Scale for Children 4th ed. (WISC-IV; Wechsler, 2003). Academic achievement was assessed in reading and arithmetic through a comprehensive
battery of achievement tests (Woodcock Reading Mastery Test Revised Word Identification and Word Attack subtests, WRMT-R; Woodcock, 1987; Wide Range Achievement Test 3rd ed., WRAT-III; Wilkinson, 1993). Children also completed self-report measures of anxiety symptoms (Multidimensional Anxiety Scale for Children, MASC; March, Parker, Sullivan, Stallings, & Connors, 1997) and depression (Children’s Depression Inventory, CDI; Kovacs, 1985). A global measure of impairment was obtained by having parents and teachers rate each participant on the Ontario Child Health Survey Scales (OCHS; Boyle et al., 1993). Impairment scores were standardized by age and gender using general population norms. Due to time and resource constraints, only ADHD cases received a hearing (pure tone audiometric screening), vision (screening of visual acuity), and language assessment by a registered speech pathologist using the Clinical Evaluation of Language Fundamentals 3rd. ed. (CELF-III; Semel, Wiig, & Secord, 1995).

Inclusion criteria

To be classified as ADHD for research purposes, children had to meet DSM-IV-TR criteria for ADHD (American Psychiatric Association, 2000) defined as at least 6 of 9 inattentive, 6 of 9 hyperactive–impulsive symptoms, or both. To ensure that children were at least moderately impaired in two settings as per DSM-IV-TR, it was required that they met criteria for ADHD from the parent or the teacher interview and questionnaires, and a minimum of 4 ADHD symptoms were reported according to the second informant, and they had at least a “moderately impaired” rating on both the parent and teacher
impairment scales. Participants were categorized into ADHD subtypes (Inattentive, Hyperactive–Impulsive, or Combined) based on all information and using DSM-IV-TR criteria (see Appendix A for DSM-IV-TR criteria for ADHD and ADHD subtypes).

ADHD children were also categorized into those with and without oppositional defiant disorder (ODD) and conduct disorder (CD). ODD was diagnosed if there were four or more DSM-IV-TR symptoms as reported by the teacher and parent together. Conduct disorder (CD) was diagnosed if the number of DSM-IV-TR CD symptoms in the parent and teacher interviews totaled three or more. CD and ODD were combined into a single entity for analysis (ODD/CD) if a participant met criteria for ODD, CD or both. Anxiety disorders were diagnosed if identified on the parent interview or if the participant scored above threshold on the MASC. Quantitative scores for ADHD symptoms were calculated by summing appropriate PICS-IV and TTI-IV items.

Exclusion criteria

Participants were excluded if they fulfilled any of the following criteria: (a) IQ below 80 or above 130 on the full scale IQ of the WISC-IV, (b) presence of a pervasive developmental disorder (including Asperger’s and Autism), non-verbal learning disability, psychosis, obsessive compulsive disorder (OCD), Tourettes syndrome or tics, serious medical condition, substance abuse, or a history of traumatic brain injury, (c) concurrent treatment with medication other than a stimulant, (d) specific language impairment (CELF total language score was below 85) as it may interfere with
assessment and cognitive testing, or (e) hearing or visual impairment. Control participants were excluded from this study if they met criteria for ADHD or if other behavioural, psychiatric or learning problems were endorsed on the screening questionnaires or self-report measures.

Measures

A modified flanker task (Eriksen & Eriksen, 1974) was used in this study to present participants with a task where they are likely to make numerous errors. This task was chosen to examine post-error slowing as it has been widely used in the investigation of performance monitoring and research has consistently revealed that the typical behavioural adjustment to errors on this task is the slowing of one’s responses (Dikman & Allen, 2000; Falkenstein et al., 1991). Two variations (conditions) of the task were presented using identical stimuli and instruction sets (see Figure 1). Both versions of the task were administered on a computer using Presentation software (Neurobehavioural Systems Inc.) to control the presentation and timing of all stimuli. Participants were required to make a discriminative response to a target stimulus. Each version of the task consisted of 25 trials presented in 5 blocks for a total of 125 trials. Within each task, a practice block was run prior to the test being administered. A fixation symbol, *, was displayed at the beginning of each trial for 500 ms. Following the presentation of the fixate, a stimulus array was presented on screen for 150 ms, which was then followed by a mask consisting of a grey bar covering the same dimensions of the array. The mask was presented for 350 ms to eliminate any visual carry-over effects that may have
occurred after the stimulus array disappeared from the screen. A blank screen then appeared for 1500 ms, allotting time for a response to be made before the next trial began, for a total trial time of 2500 ms. The amount of time between trials was selected to ensure that a response to each trial was completed prior to the beginning of the following trial. These timing parameters ensured that any refractory effects would not be carried over from trial to trial, and therefore reduced the likelihood that such refractory effects would account for any differences in reaction times. Task parameters were also established to produce a task where participants made numerous errors.

Stimuli consisted of an array of five white letters presented on a black background in the centre of the computer screen, where the middle letter was always the target. To increase task difficulty (thereby increasing error rate), four different letters were used as targets. Targets M and N were mapped to the left index finger and targets S and H were mapped to the right index finger. There were eight sequences of stimuli that were presented randomly within each block (see Appendix B). On every trial each target was flanked by distracter stimuli that were associated with the opposite or conflicting response as the target. In other words, all trials had distracters that were incongruent with the target (e.g. HHHHH, SSNSS, NNHNN, HHHHH). Research has shown that typically in the incongruent condition reaction times are longer and error rates are greater than in the congruent condition. This is likely the result of cognitive and motor conflict during response selection between the target and the distracters (Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988). There were 63 trials with S or H as a target and 62 trials with M or N as a target. There were
two task versions used: a neutral condition, where participants were never told how they were performing, and a feedback condition, where participants were provided with knowledge of their performance on each trial throughout the entire task. Participants in both groups received identical instructions for each version of the task and were instructed to be as accurate as possible while performing the task as quickly as possible. A detailed description of each condition follows.

Neutral condition

This task was designed to explore the processes involved in performance monitoring, particularly post-error slowing when participants are required to monitor for and detect errors in their performance themselves. In the neutral condition, participants were presented with neutral auditory tones immediately following each response. Participants were instructed that the tones did not represent information regarding performance; rather the tones were just the computer acknowledging that they had provided a response. This task was not initiated until participants understood instructions clearly and demonstrated this understanding during the practice block. The neutral condition provides task reaction times without any feedback about performance.

Feedback condition

A second condition of the modified flanker task was used to explore the effect of knowledge of performance on post-error slowing, thereby removing the need for
participants to detect errors themselves by making error detection explicit. In the feedback condition participants received valid auditory feedback immediately following a response. A high pitched tone emitted from the computer speakers indicated a correct response and low pitched tone indicated an incorrect response (or no response, which is still incorrect). These tones provided participants with explicit knowledge of their performance. The meaning of the feedback tones was explained to participants and understanding of the instructions and feedback tones was evaluated during the practice block before beginning this task. The feedback condition provides task reaction times when explicit feedback about performance is given.

**Procedure**

All procedures were explicitly outlined in our information and consent forms and were approved by the research ethics board at The Hospital for Sick Children. Informed consent and assent was obtained prior to participation in the research session. The parent, teacher, and child assessments were conducted without knowledge of the screening diagnosis or the results of other portions of the assessment including the results of the modified flanker tasks. All children were free of medication for at least 24 hours on the day of assessment. A drug free trial of at least 3 days was arranged before teacher ratings and interviews when the child’s teacher had not observed the child without medication within the preceding 6 months.
The same research assistants administered the tasks to both groups and the physical set up of the computer was also the same across groups. Research assistants ensured task understanding and valid performance of the practice block before testing began. During testing, task validity was also evaluated by monitoring participants responding and ensuring that there were no intentional periods of not responding. Possible practice effects were eliminated by randomizing the order in which participants completed the tasks. As the tasks were administered to both the ADHD and the control samples within the clinic as part of a larger research battery, if a different task was presented between the two modified flanker conditions, the type of that task was also randomized. This ensured that no particular type of task (e.g. pencil and paper, computerized, working memory, or inhibitory control task) could influence performance on the modified flanker tasks.

**Analysis**

Post-Error slowing and group differences were examined in several ways. First, post-error slowing was compared in the ADHD and controls groups using a two-group (ADHD vs. Control) by two-condition (neutral vs. feedback) Analysis of Variance (ANOVA). This analysis was conducted in order to determine whether all participants differed with respect to slowing between conditions and to determine whether groups differed in slowing across conditions. Second, a comparison of the proportion of the ADHD group and the control group that slowed to any extent across conditions was conducted using a chi-squared test. Third, each condition was examined independently with a one-way ANOVA comparing post-error slowing and a chi-squared test comparing
the proportion of slowing between groups. Fourth, also within each condition, the
association between post-error slowing and various other measures was examined in the
ADHD and control groups. Fourth, a series of t-tests comparing post-error slowing
between conditions in each group independently was also examined.
Chapter 3

Results

Post-error slowing was calculated by subtracting the reaction time for all correct trials following correct trials from the reaction time of all correct trials following error trials (i.e. post-error slowing = post-error reaction time – post-correct reaction time).

Individual participant data was not included in any of the analyses if accuracy was 100% or was below 40%. All outliers, defined as scores + or – 2 standard deviations from the mean of the dependent measure of post-error slowing in either condition were also excluded from all analyses (3 control participants and 8 ADHD participants were excluded due to outlying values). For all analyses, whenever the homogeneity of variance assumption for an ANOVA or ANCOVA was violated, a Welch $F$ was calculated and used for interpretation. ADHD participants ranged in age from 6 to 16 years old with a mean age of 9.3 years and control participants ranged in age from 7 to 16 years old with a mean age of 10.7 years (see Table 1 for a summary of demographic information). The ADHD sample was 76% male and 24% female and the control sample was 32% male and 68% female. Children in the control group had a mean full scale IQ of 112.6, and children in the ADHD group had a mean full scale IQ of 101.5. Results of a one-way analysis of variance (ANOVA) revealed a significant difference in the age, gender and full scale IQ of the two groups (age: Welch $F(1, 60) = 5.83$, $p < .02$; gender: $F(1,112) = 24.21$, $p < .0005$; full scale IQ: $F(1, 107) = 24.92$, $p < .0005$).
The ADHD sample was composed of a heterogeneous group of ADHD children, with the goal of providing more ecological validity to the present study. Within the ADHD group 39% of participants had the Inattentive Subtype, 18% had the Hyperactive-Impulsive Subtype, and 43% had the Combined Subtype as defined by the DSM-IV-TR (American Psychiatric Association, 2000). This distribution is typical of clinic samples of ADHD. Also within the ADHD sample, 37% met criteria for a comorbid diagnosis of oppositional defiant disorder (ODD), only 1 participant met criteria for a comorbid diagnosis of Conduct Disorder (CD), 18% met criteria for a comorbid diagnosis of Generalized Anxiety Disorder or Separation Anxiety, 11% met criteria for a comorbid diagnosis of Major Depression, 12% met criteria for a Reading Disorder and 8% met criteria for a Math Disability.

**Main and interaction effects**

In order to evaluate the effects of group membership and the two conditions on post-error slowing a 2 X 2 ANOVA was conducted. The ANOVA indicated no significant interaction between group membership and condition, $F(1, 218) = 0.94, n.s.$, or for a main effect of condition, $F(1, 218) = 2.57, n.s.$ (see Table 2 and Figure 3). However, a significant main effect for group membership was found, $F(1, 218) = 8.69, p < 0.005$. Another 2 X 2 ANOVA was conducted and a similar result was found with respect to percent correct (accuracy) with no significant interaction, $F(1, 218) = 0.16, n.s.$, or main effect of condition, $F(1, 218) = 2.06, n.s.$, but a significant main effect of group $F(1, 218) = 14.1, p < 0.0005$ (see Table 2 and Figure 4).
Further 2 X 2 ANOVAs were performed on the mean correct reaction time and the mean error reaction time. Results indicate no significant interaction or main effect of group or condition on either of these measures (Mean correct reaction time: Group $F(1, 218) = 0.96$, n.s.; Condition $F(1, 218) = 0.80$, n.s.; Interaction $F(1, 218) = 0.008$, n.s.; Mean error reaction time: Group $F(1, 218) = 1.50$, n.s.; Condition $F(1, 218) = 0.47$, n.s.; Interaction $F(1, 218) = 0.01$, n.s.). However, in contrast to mean reaction times, mean variability in reaction times showed a significant main effect of group, but no main effect of condition or interaction (Mean correct reaction time variability: Group $F(1, 218) = 7.37$, $p < .008$; Condition $F(1, 218) = 0.74$, n.s.; Interaction $F(1, 218) = 0.27$, n.s.; Mean error reaction time variability: Group $F(1, 218) = 9.00$, $p < .004$; Condition $F(1, 218) = 1.21$, n.s.; Interaction $F(1, 218) = 0.70$, n.s.).

**Proportion of slowing between groups**

A chi-squared test compared the proportion of children who slowed to any extent following errors across conditions in the ADHD and control groups. It was found that 68% of the control group exhibited any post-error slowing across conditions compared to only 49% of the ADHD group. This secondary analysis supports the findings of the main effect of group, in that significantly more control children exhibited slowing behaviour following errors compared to children with ADHD, $\chi^2 (1) = 7.02$, $p < .009$. 
Within condition analyses

Neutral condition

Results of a one-way analysis of variance (ANOVA) revealed a significant difference in the mean number of errors made by children across groups on this task, with control children making significantly less errors and therefore having greater accuracy, Welch $F(1, 97) = 6.15, p < .02$ (see Table 3). The results of another one-way ANOVA revealed that control children and ADHD children did not differ on correct or incorrect reaction times of this task (mean correct reaction time: $F(1, 111) = 0.549, n.s.$; mean error reaction time: $F(1, 111) = 0.633, n.s.$). However, another one-way ANOVA revealed that ADHD children had significantly more variability in their reaction times on this task than controls (mean correct reaction time variability: $F(1, 111) = 5.94, p < .02$; mean error reaction time variability, $F(1, 111) = 5.20, p < .025$).

A one-way ANOVA determined that there was no significant difference between groups on post-error slowing in the neutral task, Welch $F(1, 56) = 1.61, n.s.$ There was also no significant relationship between age or full scale IQ and post-error slowing in this condition (age: $r(113) = .15, p < .13$; full scale IQ: $r(108) = .03, p < .76$) therefore eliminating these potential confounds. However, a significant relationship between gender and post-error slowing was found, $r(113) = .20, p < .04$. Therefore, as gender was significantly associated with post-error slowing, a one-way analysis of covariance (ANCOVA) with gender entered as a covariate was conducted in order to determine if,
when gender was controlled for, the difference between groups remained non significant. The ANCOVA was not significant \( F(1,110)=0.388, \text{n.s.} \) indicating that the ADHD and control groups did not differ significantly in their amount of post-error slowing in the neutral condition even after controlling for the effects of gender. An examination of the means (see Table 2) reveals that although children in the ADHD group exhibited no post-error slowing on average, it was not significantly less than the post-error slowing displayed by the control group.

In order to determine if the proportion of individuals in the control group that exhibited post-error slowing was greater than the proportion that exhibited it in the ADHD group, a chi-squared test was conducted. It was found that 63% of the control group slowed in the neutral task and only 45% of the ADHD group slowed. However, a chi-squared test of the relationship between group membership and slowing was not significant, \( \chi^2 (1) = 3.09, \text{n.s.} \).

For both groups, a significant relationship was found between accuracy and post-error slowing (ADHD: \( r(73) = .39, p = .001 \); Control: \( r(40)= .37, p < .02 \)). Given that increased task accuracy was significantly related to increased post-error slowing across groups, further analyses into if post-error slowing is related to accuracy in academic achievement was warranted. Scores on academic measures of reading and arithmetic were available for both ADHD and control participants and were entered into this analysis. Results revealed no significant relationship between post-error slowing and performance on single word reading (WRMT-R Word Identification and Work Attack
Results indicated no significant difference among the three ADHD subtypes on post-error slowing, $F(2, 70) = 1.61, n.s.$ Symptom severity, defined as the total number of ADHD symptoms reported by parents and teachers independently, also yielded a non significant relationship with post-error slowing (parent: $r(71) = -.05, p < .71$; teacher: $r(71) = .06, p < .65$). Parent and teacher reported measures of impairment obtained from the OCHS Scales (Boyle et al., 1993) and clinician rated measures of impairment on the Children’s Global Assessment Scale (Shaffer, Gould, Bird, et al., 2000), were also found to have no significant relationship with post-error slowing (parent: $r(68) = .08, p < .54$; teacher: $r(66) = .20, p < .12$; clinician: $r(51) = .02, p < .92$). The effect of comorbidity was also examined. Results indicated no significant difference in post-error slowing among
ADHD without ODD/CD and ADHD with CD/ODD children, $F(1,71) = 0.149$, n.s.

Furthermore, there was no significant relationship between post-error slowing and parent or teacher reported symptoms of ODD/CD (parent: $r(71) = .02$, $p < .85$; teacher: $r(71) = .21$, $p < .08$).

**Feedback condition**

Results of a one-way analysis of variance (ANOVA) revealed a significant difference in the mean number of errors made by children across groups on this task, with control children making significantly less errors and therefore having greater accuracy, *Welch* $F(1, 105) = 12.51$, $p = .001$ (see Table 3). The results of another one-way ANOVA revealed that control children and ADHD children did not differ on correct or incorrect reaction times of this task (mean correct reaction time: $F(1, 107) = 0.416$, n.s.; mean error reaction time, $F(1, 107) = 0.879$, n.s.). Similarly, and in contrast to the neutral condition, another one-way ANOVA revealed that ADHD children were not significantly more variable in their reaction times on this task (mean correct reaction time variability: $F(1, 107) = 2.13$, n.s.; mean error reaction time variability, $F(1, 107) = 3.85$, n.s.).

Again unlike the neutral condition, a one-way ANOVA determined that there was a significant difference between groups on post-error slowing, $F(1, 107) = 7.13$, $p < .01$. There was no significant relationship between age, gender or full scale IQ and post-error slowing in this condition (age: $r(109) = .16$, $p < .10$; gender: $r(109) = .16$, $p < .10$; full scale IQ: $r(104) = .09$, $p < .39$) eliminating these potential confounds. In addition,
despite both groups not differing significantly in their mean correct and incorrect task reaction times or variability, there is still a significant difference between groups on post-error slowing.

In order to determine if the proportion of individuals in the control group that exhibited post-error slowing was greater than the proportion that exhibited it in the ADHD group, a chi-squared test was conducted. It was found that 73% of the control group slowed in the feedback condition and only 53% of the ADHD group slowed. A chi-squared test of the relationship between group membership and slowing was significant, $\chi^2 (1) = 4.14, p < .045$. In the feedback condition, the proportion of control participants who displayed post-error slowing was significantly greater than in the ADHD group.

For both groups, a significant relationship was found between accuracy and post-error slowing (ADHD: $r(73) = .40, p = .001$; Control: $r(37)= .49, p = .002$). Again, given that increased task accuracy was significantly related to increased post-error slowing across groups, further analyses into if post-error slowing is related to accuracy in academic achievement was warranted. Scores on academic measures of reading and arithmetics were available for both ADHD and control participants and were entered into this analysis. Results revealed no significant relationship between post-error slowing and performance on single word reading (WRMT-R Word Identification and Work Attack Subtests; Woodcock, 1987) and reading comprehension (WRAT-III Global Reading Score; Wilkinson, 1993) in either group (ADHD: $r(67) = -.18, p < .14$, $r(67) = -.05, p < .68$, $r(45) = -.18, p < .25$ respectively, Control: $r(36) = -.17, p < .32$, $r(36) = -.14, p < .41$,
Arithmetic and mathematical reasoning performance scores (WRAT-III Arithmetic and Calculation Subtests; Wilkinson, 1993) were also not significantly related to behavioural adjustment following errors (ADHD: $r(67) = -.19, p < .13$ and $r(23) = -.13, p < .57$ respectively; Control: $r(36) = .07, p < .69$ and $r(14) = -.16, p < .58$ respectively). Furthermore, measures of working memory (digit span forwards and backwards standard scores on the WISC-IV; Wechsler, 2003) were not significantly related to post-error slowing in either group (ADHD: $r(64) = .14, p < .26$ and $r(64) = .07, p < .61$ respectively; Control: $r(26) = .16, p < .43$ and $r(26) = .09, p < .67$ respectively).

Results indicated no significant difference among the three ADHD subtypes on post-error slowing, $F(2, 69) = 0.70, n.s.$ Symptom severity, defined as the total number of ADHD symptoms reported was reported by parents and teachers independently. The total parent reported symptoms of ADHD was significantly related to post-error slowing in this condition ($r(70) = -.28, p < .02$), where as the total teacher reported ADHD symptoms was not related to post-error slowing ($r(70) = .04, p < .74$). The relationship between parent reported ADHD symptoms and post-error slowing indicates that as total number of ADHD symptoms increase, the amount of post-error slowing observed in the feedback condition decreases.

Parent and teacher reported measures of impairment obtained from the OCHS Scales (Boyle et al., 1993) and clinician rated measures of impairment on the Children’s Global Assessment Scale (Shaffer et al., 2000), were found to have no significant relationship with post-error slowing (parent: $r(69) = .08, p < .52$; teacher: $r(66) = .23, p < .07$;
clinician: \( r(50) = .09, p < .56 \). The effect of comorbidity was also examined. Results indicated no significant difference in post-error slowing among ADHD without ODD/CD and ADHD with CD/ODD children, \( Welch \, F(1, 40) = 0.555, n.s. \) Furthermore, there was no significant relationship between post-error slowing and parent or teacher reported symptoms of ODD/CD (parent: \( r(70) = .09, p < .47 \); teacher: \( r(70) = .18, p < .14 \)).

**Within group analyses**

In order to investigate how post-error slowing changed across the two task conditions within each group, several t-tests were conducted for those in each group that had valid data for both conditions. Mean comparisons across conditions indicated a significant effect of condition in both post-error slowing and percent correct for the control sample (\( t(36) = -1.78, p < .045 \) and \( t(36) = -2.44, p = .01 \) respectively). Both post-error slowing and percent correct were significantly different across conditions for the control group with participants slowing significantly more and being significantly more accurate in the feedback condition. In contrast, the ADHD group did not show a significant difference in the amount of post-error slowing or in the percent correct across conditions (\( t(70) = -.66, p < .26 \) and \( t(70) = -1.47, p < .08 \) respectively; see Table 4, Figure 5 and Figure 6).

In both groups, there was no significant difference across conditions with respect to mean correct reaction time, mean correct reaction time variability, mean error reaction time, mean error reaction time variability, or in the proportion of individuals that displayed
post-error slowing (ADHD: $t(70) = 1.88, p < .07$, $t(70) = 1.81, p < .08$, $t(70) = 1.56, p < .13$, $t(70) = -1.42, p < .17$, $t(70) = -1.00, p < .33$ respectively; Control: $t(36) = .39, p < .70$, $t(36) = .38, p < .71$, $t(36) = -.10, p < .92$, $t(36) = -1.03, p < .32$, $t(36) = -1.28, p < .22$ respectively).

Even though the ADHD group did not significantly slow more in the feedback condition versus the neutral condition, overall as a group they did slow on average 12.6 ms more in the feedback condition. In comparison, the control group slowed on average 73.7 ms more in the feedback condition. Despite this large difference in means, the difference in the amount of observed slowing between the conditions was not significant between groups, Welch $F(1, 52)= 1.78, n.s.$

In the ADHD group, post-error slowing in the neutral condition was significantly correlated with the feedback condition, $r(71) = .40, p < .0005$. However, in the control group, post-error slowing across conditions was not significantly related, $r(37) = .20, p < .24$. The percent correct, or accuracy, was significantly related in each group across conditions (ADHD: $r(71) = .80, p < .0005$; Control: $r(37) = .39, p < .02$).
Chapter 4

Discussion

The goal of the present study was to specify the deficit in performance monitoring within ADHD by examining the sub-process of post-error behavioural adjustment with and without requiring error detection. Previous research investigating performance monitoring in children with ADHD has reported a deficit within this population; however, whether these children possess a deficit in their ability to detect their errors or whether they have difficulty implementing effective post-error strategies has remained unclear. In order to examine this a task paradigm (the modified flanker task; Eriksen & Eriksen, 1974) was used that elicited many errors, required performance monitoring, was amenable to feedback manipulation, and had task accuracy benefits when post-error slowing was employed.

Post-error adjustment: Specifying the performance monitoring deficit in ADHD

Results indicate a main effect of group on post-error slowing with controls slowing significantly more than children with ADHD across conditions. This indicates that children with ADHD possess a deficit in post-error slowing and is consistent with the predictions of the current study and with previous reports of such a deficit in children with ADHD (Jonkman et al., 2007; Wiersema, et al., 2005; Sergeant & van der Meere, 1988; Schachar et al., 2004; Krusch et al., 1996). Furthermore, this significant main
effect of group is also supported by the finding that the proportion of control children that slowed to any extent following errors was also significantly more than children with ADHD across conditions.

Whether children with ADHD have an error detection deficit, the first sub-process associated with performance monitoring, is important to address as an error detection deficit would not enable appropriate behavioural adjustment following errors. The impact of error detection was mediated in this study by providing participants with immediate and overt error detection in the feedback condition. This was done to remove the reliance on self-detection of errors and to observe and isolate the post-error slowing sub-process. As predicted, results indicated that there was no significant main effect of condition (i.e. experimentally providing participants with error detection or not) on post-error slowing across groups. Therefore, as was hypothesized, this finding specifies the performance monitoring deficit within children with ADHD as one of post-error behavioural adjustment and not error detection. As such, it is posited that children with ADHD do not have an error detection deficit as results indicate that children with ADHD and normal controls differ significantly with respect to their post-error slowing across conditions, and providing immediate and overt error detection did not normalize post-error slowing in children with ADHD.

This is a highly relevant finding as past studies have not clearly delineated the performance monitoring deficit in children with ADHD. In part, this lack of clarity has resulted from electrophysiological (ERP) studies examining children with ADHD as they
commit errors on cognitive tasks. Given the results of the current study, it is posited that the discrepancy in the findings on an error detection deficit in ADHD may reflect the notion that the error-related negativity (ERN) does not separate error detection and post-error slowing components with clear precision and therefore, aspects of post-error slowing may be being captured within the ERN. An example of this may be studies that have shown a reduced ERN in children with ADHD. For example, Liotti et al. (2005) showed that children with ADHD displayed reduced ERN amplitude on error trials compared to controls, and Groen et al. (2008) showed both a decreased ERN and error-positivity (Pe) in children with ADHD in a feedback-based learning task. It is possible that the results reported by these studies actually reflect aspects of deficient post-error slowing in the ADHD sample.

However, it is perhaps more plausible that the ERN is more sensitive and specific to error detection and therefore, studies finding no deficit in ERN in children with ADHD are only measuring one component of performance monitoring, error detection. This notion and the results of the current study are consistent with a study that combined ERP techniques and behavioural data in children with ADHD. Wieserma et al., 2005 found that children with ADHD failed to adjust their speed of responding after making an error, but revealed that the ERN was the same for ADHD and control children, but that children with ADHD showed diminished Pe. Based on these findings, the authors posited that children with ADHD are normal in error detection processes, but show abnormal post-error strategy/adjustments and are therefore deviant in performance monitoring processes associated with the conscious evaluation of errors (Wieserma et al., 2005). Another ERP
study on children with ADHD also found that ERN amplitude was unaffected and Pe amplitude was reduced in the ADHD group (Jonkman et al., 2007). As both of these studies have found no significant differences between their ADHD and control group for the amplitude and latency of the ERN, it is suggested by both authors that this is reflective of the absence of an ADHD deficit in error detection (Wieserma et al., 2005; Jonkman et al., 2007). In addition, one study even found that although ADHD status did not affect performance on the task, the ADHD sample exceeded controls in ERN amplitude (Burgio-Murphy et al., 2007). The authors interpreted this as an attempt to compensate for a deficit in post-error behavioural adjustment. Given that not every child with ADHD will exhibit a performance monitoring deficit, as not all children with ADHD exhibit deficits in inhibitory control, working memory, etc., it is possible that the mixed results of the ERN in children with ADHD is also the result of different ADHD samples with heterogeneous cognitive deficits.

In summary, despite the discrepancy in ERP research concerning the nature of the ERN in children with ADHD, the results of the present study indicate that in children with ADHD the performance monitoring deficit is specific to a post-error adjustment deficit and not an error detection deficit. In addition, the post-error slowing deficit was most significantly observed in the feedback condition when self-detecting of errors was not required. Therefore, being provided with explicit feedback about errors did not ameliorate the post-error adjustment deficit in the ADHD group. Furthermore, regardless of having to self-detect errors or experimentally being provided with explicit error-
detection, children with ADHD did not significantly change their post-error slowing across conditions.

This demonstrates that the post-error slowing deficit in ADHD children is not mitigated by providing external feedback of error detection, which removes the reliance on self-detection of errors. Rather, this result supports the notion that children with ADHD are deficient in using the detection of errors to their optimal advantage while performing tasks. Further, this suggests that post-error slowing is not merely a function of error detection per se but perhaps is an additional and distinct process. Therefore, this mechanism may be distinct in part because of several other factors that are also involved in reinforcement learning, including knowledge of behavioural strategies, or a deficiency in the underlying cognitive and/or neural mechanisms supporting post-error adjustment. As such, these factors involved in post-error behavioural adjustments and their relation to ADHD are discussed in the sections below.

**The post-error adjustment deficit in ADHD: How does it fit into the cognitive model of performance monitoring?**

Within the current study, results indicate that the deficit in performance monitoring in children with ADHD has been localized to the application of behavioural adjustment strategies. This pattern of results can be understood within the framework of the widely accepted cognitive model of performance monitoring presented in Figure 1 (Holroyd et al., 2005; a full description of the model is presented above in Chapter 1: Introduction).
The current results indicate that children with ADHD are able to self-detect their errors, thereby enabling the monitor module to send TD error signals to the task module for adjustment or modification of the desired response representation. Within the flanker task, the model predicts that the size (or strength) of the TD error signal is associated with the amount of post-error slowing, in that the stronger the TD signal, the greater the increase in post-error slowing. The TD error signals serve to improve performance by adjusting the representation of the desired response ascribed to the task module. This adjustment is the proposed process at work during the observed post-error slowing. Therefore, it is proposed that children with ADHD have difficulty with their monitor module, specifically the TD unit layer and TD error signal generation. As such, ADHD children’s TD unit generates a weaker signal compared to controls (given that control participants are believed to show normal modulation of the mesolimbic pathway), resulting in ADHD children’s smaller behavioural adjustments following a weaker signal. These weaker TD error signals could be the result of hypofunctioning of dopamine within striatal regions in individuals with ADHD (i.e., the mesolimbic system is not functioning on par with that of controls in that reinforcement learning is altered). The results obtained from the ADHD sample across tasks are consistent with such a hypothesis. Therefore, the decreased slowing displayed by children with ADHD compared to controls may correspond to poorly generated, weaker TD error signals from the monitor module. Therefore, it is likely that the significantly less accuracy found across conditions in children with ADHD is the result of deficit TD error signal generation and therefore, less post-error slowing and less correct responses.
To date, behavioral and neurophysiological evidence has suggested that ADHD is indeed characterized by the impact of abnormal reward prediction error signals carried by the midbrain dopamine system to frontal brain areas that implement cognitive control. As such, another model has linked the proposed cognitive architecture of performance monitoring with neurological pathways that have been activated during the sub-processes of performance monitoring as observed through fMRI. In particular, Chevrier and Schachar (2009) have proposed that the actor-critic architecture of the dopamine system can guide our predictions about which distinct neurological circuits might accomplish error detection and error signal magnitude processing. Dopamine structures in the striatum are thought to be divided into a ventral ‘adaptive-critic’ that can learn stimulus-response relationships based on performance errors (akin to the monitor module that modulates the representation of the desired response), and a dorsal ‘instrumental-actor’ (akin to the task module) that implements these new mappings in behaviour. This model proposes that the actor is affected by the critic and that without the critic, although the actor can still perform the task, no reinforcement learning can occur. This actor-critic architecture is an important model in the study of performance monitoring as it maps the cognitive sub-processes involved to specific brain circuitry.

**Possible influence of feedback on post-error slowing**

Although not significant, there was an indication of a possible interaction between group and condition on post-error slowing. Results indicate that the control group “benefited” from the feedback condition significantly more than the ADHD group. In addition,
controls exhibited significantly more slowing and more accuracy in the feedback condition than they did in the neutral condition. Given these results an examination of the possible influence that feedback of performance had on post-error slowing is warranted. Therefore, we must first consider the manipulation that was actually present in the feedback condition.

In the feedback condition, it is obvious that error detection was manipulated, but to say that it significantly reduced the need for self-detection of errors by participants is hypothesized. Given the resulting increase in the controls post-error slowing in the feedback condition compared to the neutral condition, it is hypothesized that their slowing increased as a result of detecting each and every error. Therefore, for controls, perhaps the feedback condition did eliminate the need to self-detect errors; however, for ADHD children, it did not. In addition, past research on cognitive tasks of performance monitoring have shown that external feedback elicits ERNs in individuals similar to those generated when depending on the self-detection of errors (Ullsperger, & von Cramon, 2003; Miltner, Braun, & Coles, 1997; Luu et al., 2003).

Alternatively, ADHD children may have reacted differentially than controls to their errors being pointed out to them. Controls may have seen this as productive feedback allowing them to shape their behaviour to better their performance; where as children with ADHD may have seen this as negative, punitive, or perhaps even frustrating especially given that they had significantly more errors than the control group.
As such, there is a growing body of literature that has begun to illuminate the potential influences (both contextual and intrinsic) that can be at work in performance monitoring. Although the majority of these studies were conducted using ERP techniques, the findings reported could apply to behavioural phenomena as well due to the aforementioned link between ERP measures and behavioural indices of performance monitoring. In electrophysiological research, both the ERN and the Pe component (thought to be associated with error detection and post-error adjustments respectively) may be sensitive to intrinsic (e.g., personality) and contextual (e.g., reward/instruction) manipulations. It is important to note that the magnitude of the ERN has been found to be associated with the magnitude of the resulting post-error slowing (Gehring et al., 1993; Scheffers et al., 1996) and as such, possible influences on the ERN likely also influence post-error slowing.

In particular, it has been proposed that the ERN may be mediated by reinforcement and/or affective states (Gehring et al., 1993; Pailing & Segalowitz, 2004a; Gehring & Fencsik, 2001; Hajcak, Moser, Yeung, & Simons, 2005; Holroyd & Coles, 2002; Nieuwenhuis, Holroyd, Mol, & Coles, 2004). In addition, this relationship is likely bidirectional in that detection of, and adjustment to errors has the potential to influence reinforcement quality or quantity and/or emotional/affective states, and in turn these may influence the relevance or salience of errors. As such, studies do reveal that ERN amplitude is also affected by error salience in that the higher the error salience for a participant, the greater the ERN wave generated (Gehring et al., 1993; Dikman & Allen, 2000; Holroyd et al., 2004; Pailing & Segalowitz, 2004b). Research also reports
enhanced ERNs when emotionally arousing stimuli were used in a modified Eriksen flanker task (Larson, Perlstein, Stigge-Kaufman, Kelly, & Dotson, 2006), and in affectively distressed participants, such as those with obsessive-compulsive disorder (OCD; Gehring, Himle, & Nisenson, 2000). In addition, Luu et al. (2000) examined the relationship between ERN, personality, and affective dimensions of distress and discovered that individuals with high negative-emotionality and affect had larger ERN amplitudes than other participants. In contrast, ERN amplitude is reduced in individuals with schizophrenia who often display flat affect (Mathalon et al., 2002). Similarly, a study by Dikman and Allen (2000) reported that individuals with low socialization produced smaller ERNs compared with highly-socialized individuals. It has also been suggested that the ERN itself demonstrates sensitivity to established goal states and reflects, in part, the negative affective response to the realization that one has committed an error (Luu et al., 2003).

Based on these findings, electrophysiological research indicates the important role of various contextual and intrinsic influences on performance monitoring indices such as the ERN. Furthermore, it is important to reiterate, and is of most relevance to the performance monitoring deficit in children with ADHD, that the magnitude of the ERN has been found to be associated with the magnitude of the resulting post-error slowing (Gehring et al., 1993; Scheffers et al., 1996). In addition to these various findings about possible contextual and intrinsic influences, there are several other factors that may also influence performance monitoring, and more specifically influence post-error slowing. As such, these possible influences are discussed below.
Other factors that may influence post-error slowing

Several other factors were investigated in order to determine whether the findings regarding post-error slowing are specifically attributable to ADHD and not to differences in other between-group factors. As such, the relationship of variables such as age, gender, comorbidity, ADHD subtype, full scale IQ, and academic performance with post-error slowing were all examined.

The present study did not find age to be a significant factor in post-error slowing in either condition. However, previous research investigating the effect of age on performance monitoring has been stimulated by a developmental model of the ERN that posits that the functioning of the ACC (considered to be the source generator of the ERN) does not reach adult levels until late adolescence (Davies, Segalowitz, & Gavin, 2004). Therefore, this suggests that the ability to detect one’s errors would improve as one develops and ages. Although, the current study did not find age to be significantly related to post-error slowing, it is likely that the ERN is reflective of error detection and so perhaps age differences with respect to error detection mechanisms would be more likely. In fact, a recent study comparing older and younger age groups found that the groups did not differ in their ability to adjust response strategies after making an error (i.e., the Pe amplitude did not change with age), but that the ERN amplitude increased with age (Wiersema, van der Meere, & Roeyers, 2007). In comparison, gender was a significant factor in post-error slowing on the neutral task, with males displaying post-error speeding compared with post-error slowing shown by females. However, the difference in post-error slowing
between the two groups remained non significant for the neutral condition when gender was entered into the analyses as a covariate. Gender was not a significant factor in post-error slowing in the feedback condition.

Two disorders found to be commonly comorbid with ADHD, Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD), and their relationship with post-error slowing was also investigated in this study. Two subgroups consisting of participants with ADHD and no ODD/CD, and with ADHD and ODD/CD were formed in order to examine the effect of comorbidity on post-error slowing. Results indicated no significant differences between the subgroups on post-error slowing, indicating that comorbidity did not affect post-error behavioural adjustments. This finding is consistent with results generated by other studies investigating executive function deficits in ADHD. For example, studies have found that deficits in executive functioning of children with ADHD remained independent of the presence of comorbid ODD and CD (Oosterlaan, Scheres, & Sergeant, 2005; Schachar et al., 2000).

In addition, the three ADHD subtypes (inattentive subtype, hyperactive/impulsive subtype, and combined subtype) were examined for their potential relationship with post-error slowing. Results indicated no significant differences among any of the subtypes on post-error slowing, suggesting that individuals with ADHD exhibited similar performance regardless of subtype classification. The finding that ADHD subtype classification was not related to post-error slowing is consistent with results from
previous studies revealing no differences in performance among ADHD subtypes in other executive function tasks (Oosterlaan et al., 2005; Oosterlaan, Logan, & Sergeant, 1998).

Furthermore, no significant relationship was found between full scale IQ and post-error slowing, eliminating the potential confound of IQ. Performance on academic measures of reading (single word reading and reading comprehension) and mathematics (arithmetic and mathematical reasoning) were also examined for their relationship with post-error slowing. Results revealed no significant relationship between reading ability or mathematical performance and post-error slowing. Therefore, the relationship between the performance monitoring deficit and ADHD appears to be specific in that no association was found with age, gender, full scale IQ, ODD/CD, or with measures of academic functioning.

Notably, results of the present study indicate that the greater number of ADHD symptoms reported by a child’s parents, the less post-error slowing displayed in the feedback condition by that child. This finding suggests the predictive validity of a performance monitoring deficit in children with ADHD as less slowing after errors was correlated with a greater number of ADHD symptoms as reported by parents. This relationship was also found in previous research on performance monitoring in children with ADHD examining post-error slowing after failures of inhibition (Schachar et al., 2004).
Alternative accounts of post-error slowing: Ruling out other cognitive processes

Task reaction times can be influenced by several cognitive processes that may also affect post-error slowing. Therefore, alternative explanations for post-error slowing must be considered. In order to examine this, both the speed and variability of responding for the two groups was examined within each condition of this study to ensure that any differences in post-error slowing was not accounted for by the overall speed of responding. Results revealed that the mean reaction time on correct and incorrect trials was not significantly different across groups or conditions. As such, the possibility that the post-error slowing observed in the control group was a function of slower reaction times overall is ruled out. Another analysis was conducted to examine whether the ADHD and control groups differed on reaction time variability (i.e. standard deviation of reaction times) in responding on correct and error trials for both conditions. It was found that there was a main effect of group on reaction time variability, with children with ADHD showing greater variability across conditions. The finding that the control group displayed less variability in responding is consistent with past research that has demonstrated that children with ADHD are more variable in cognitive task performance than control children (Castellanos et al., 2005). The observed variability has been attributed, in part, to the ADHD symptoms of inattention and hyperactive impulsivity. However, the significant variability in responding between the two groups may have affected the resulting measure of post-error slowing in the neutral task, but statistical analyses were employed that took this variability into account when comparing groups.
(i.e. a Welch F was used for interpretation). Therefore, the non-significant difference between groups on post-error slowing in the neutral task is considered to be a valid result.

In addition, the types of trials presented within a task should also be considered when examining reaction time performance. The term “switch costs” has been used to reflect the notion that participants display slower reaction times on switch trials compared with repeated trials (Rogers & Monsell, 1995; Meiran, 1996; Rubinstein, Meyer, & Evans, 2001). Within the modified flanker tasks used in this study, a repeat trial constitutes two successive trials that have a target mapped to the same response button (e.g., M as a target, then an N as the target on the following trial, both of which correspond to a left index finger button press). A switch would be from target to a distractor, as when participants are presented for example with a M as a target on one trial, and then a M occurring as a distractor on the successive trial. These factors were considered when designing the tasks in the event that they may have an effect on the variables of interest in this study. Research has indicated that on switch trials, participants display a slight increase in the amount of response time in order to alter their task set configuration (Wylie, Javitt, & Foxe, 2001; Wylie & Allport, 2000). To overcome this, each set of eight stimulus arrays (see Appendix B) were presented an equal number of times within each block. The notion that one group could have been more affected then the other by switching costs was considered; however, given that there was an equal number of trials where switching and non-switching occurred in each condition, the effect of switching is believed to be minimal.
Educational and clinical implications for ADHD

Reinforcement learning is the basis of educational teaching and clinical work with children, especially ADHD children. The association of reinforcement learning with performance monitoring and the deficit found in the current study suggests that these are areas of significant difficulty for children with ADHD. This likely impacts their daily functioning and in particular, impairs their functioning within an educational context. Of course, results obtained in a controlled laboratory setting are not always generalizable to a typical classroom setting. In addition, error detection and behavioural adjustment processes are two of several factors that can affect academic performance. However, specifying the deficit in performance monitoring in children with ADHD is educationally and clinically informative as it allows for more precise targeting of behavioural interventions for children with ADHD. The finding that children with ADHD are not able to adjust their performance following errors, but that they are likely aware that they have made an error, is an important step in beginning to understand the learning and behavioural profiles demonstrated by a significant number of these children. In fact, one study on children with ADHD found that they have a diminished capacity to adjust their behaviour when they are learning by performance feedback, and this capacity worsened as length of the task increased (Groen et al., 2008). Given that a majority of academic learning and behavioural and cognitive-behavioural intervention require reinforcement learning and learning through performance feedback, if children with ADHD cannot adjust their behaviour following errors, they will most certainly have difficulty learning in many contexts.
Given that the specificity of the deficit is in post-error adjustments, it is relevant to question if children with ADHD have knowledge of the strategies required to adjust their behaviour appropriately after making an error. That is, do they know and understand what to do after making an error? Perhaps children with ADHD are aware of what strategies to employ but are unable to adjust their behaviour due to biological underpinnings. Indeed, this is an area that future research should focus on to further our understanding of this behavioral deficit.

Notably, results of this study also found a significant main effect of group on the percent correct (accuracy) across conditions, indicating that control children had significantly more accuracy than ADHD children on this task. It was also found that accuracy was positively correlated with post-error slowing for both groups in both conditions, indicating that when children slowed more after errors their task accuracy also increased. This relationship points to the advantage of slowing after errors on this task and also points to the task validity of the modified flanker task (Eriksen & Eriksen, 1974) for the measurement of post-error slowing. Although post-error slowing on this task was not correlated with performance on academic measures, it is likely that post-error slowing is task specific, in that post-error slowing in one task may not correlate with increased accuracy in another task. As such, it is plausible for future studies to examine the relationship between post-error slowing on basic academic tasks that have external feedback about performance and the same academic task accuracy of children with ADHD. It is likely that children with ADHD are not able to take the appropriate steps
necessary, or perhaps do not know what steps to take, to make the behavioural adjustments that are required to avoid errors.

From a clinical perspective, studies investigating cognitive-behavioural and behavioural therapy, two approaches that draw on reinforcement learning principles and self-monitoring techniques, have indicated that these approaches can be effective in producing and maintaining improvement in behaviour of children with ADHD. These programs tend to focus on providing alternative ways of cognitive problem-solving and on direct reinforcement of certain behaviour. However, behaviour therapy programs for example, do not always provide direct instruction to children on how to actively monitor their behaviour for errors and most importantly, on how to apply appropriate strategies following the commission of errors. Reinforcing a behaviour increases the chances that the behaviour will reoccur; however, it does not necessarily provide a child with strategies for success once they have committed an error. Introducing such strategies along with reinforcement could be especially beneficial for children with ADHD.

**Limitations of the current study and possible directions for future research in performance monitoring and ADHD**

A possible limitation of the current study, and an issue to be considered in future research, is that the ability to accurately self-detect errors may vary as a function of specific task parameters. The task used in the current study may not have allowed for appropriate reflection and self-monitoring of performance because it was too speeded of a
task for children and therefore, perhaps reduced the accuracy with which participants were able to self-detect errors. Therefore, future research should explore the error detection sub-process within a variety of tasks in order to fully comprehend how the process is impacted, if at all, by task/situational parameters. Following this line of research, studies investigating the effect of reinforcement on error detection would provide further insight into this sub-process of performance monitoring. Given reports in the literature of the influence of contextual and intrinsic factors on the amplitude of the ERN (which is believed to reflect the process of error detection), it is vital to ascertain whether similar effects can be found with behavioural measures of error detection.

Behavioural adjustment after errors in the form of post-error slowing was the appropriate strategy to implement given the type of task used in this study. Extending the investigation of behavioural adjustment following errors in ADHD to include tasks where a different form of behavioural adjustment, such as decreasing one’s reaction time, would be beneficial. This would further specify the performance monitoring deficit evident in ADHD as one of post-error adjustment through replication of the deficit using a post-error adjustment other than slowing. In addition, it would be ideal to explore both sub-processes of performance monitoring independently, but within the same task thus allowing future research to examine the two sub-processes concurrently without the confounds of task parameters and timing that two or more experimental conditions present. The results of the current study indicate that individuals possess adequate abilities in one sub-process of performance monitoring while displaying a deficit in the other sub-process. Therefore, studies that fuse these two processes together may not be reflecting the true nature of any dysfunction in performance monitoring. The ability to
measure both error detection and behavioural adjustment independently, but within the same task would also permit the direct investigation of the relationship between the two sub-processes.

It would be interesting to investigate the impact of reinforcement and reward on performance monitoring ability in ADHD, further expanding current theoretical models of ADHD, and informing therapeutic strategies with this population. Careful consideration to reinforcement schedules and response-reinforcement delay should be given to such studies as research has demonstrated that children with ADHD prefer immediate versus delayed rewards (Sonuga-Barke, 2003), exhibit deterioration of performance under conditions of partial compared to continuous reward (Douglas & Parry, 1994), and have less effective reinforcement learning as the response-reinforcer delay is increased (Lattal & Gleeson, 1990; Grice, 1948). It would also be interesting to explore the manner in which including reinforcement schedules with extinction phases would alter behavioural adjustment performance in individuals with ADHD. These variables would be of interest as research has found that a learned behaviour takes much longer to extinguish in individuals with ADHD than controls (Johansen, Aase, Meyer, & Sagvolden, 2002). Despite the need for future research to investigate the effects of reward on performance monitoring, research using reward will always present unique challenges in terms of reinforcement schedules and contingencies, quantity and quality of reinforcement, if a manipulation was actually reinforcing as was intended and if it was similarly reinforcing for all participants.
The literature on cognitive processes in ADHD consistently reports improvement in task performance for this group while on stimulant medication (Pelham, Milich, & Walker, 1986; Berman, Douglas, & Barr, 1999). In addition, behavioural research on children with ADHD has shown that stimulant medication increased post-error slowing on a Sternberg task, while also increasing speed and accuracy (Krusch, et al., 1996). Furthermore, ERP studies investigated the effects of methylphenidate in children with ADHD children found reduced errors in both low and high-conflict conditions and normalized Pe amplitudes (Jonkman et al., 2007; Groen et al., 2008). However, the research into the effects of stimulant medication on post-error behavioural adjustment with ADHD is still very limited. Such research is extremely relevant to ADHD as stimulant medications are the most commonly prescribed treatment for individuals with the disorder. It would be of considerable theoretical and clinical interest to further explore whether stimulant medication and its resultant impact on dopamine functioning, alters performance monitoring ability in children with ADHD. Furthermore, identifying which sub-process, error detection or post-error adjustments, displays the improvement with stimulant medication would be extremely informative.

Research into performance monitoring in ADHD could also be extended to include familial and hereditary components of the process. Sibling and twin studies would provide insight into whether the ability to monitor one’s performance for errors and engage in subsequent adjustment of behaviour is predicted by familial ability. Research examining the heritability of cognitive processes in ADHD has recently expanded to include performance on flanker tasks. One such study has reported that performance on a
flanker task was heritable within a sample of ADHD boys and their siblings (Albrecht et al., 2008). If hereditary of performance monitoring ability on the flanker task can be discovered within ADHD families, it will also support the notion that this may be a core cognitive deficit of ADHD. Much like inhibitory control deficits, a core cognitive deficit found in many children with ADHD, performance monitoring deficits also fall under the umbrella of executive functioning and self-regulation difficulties. Therefore, a finding of heritability in performance monitoring would support it becoming a candidate endophenotype in ADHD (for a review see Crosbie, Perusse, Barr, & Schachar, 2008 and Anokhin, Golosheykin, & Heath, 2008).

In summary, the results generated from the current study identify the performance monitoring deficit found within children with ADHD to be specific to behavioural adjustments and/or strategies following the commission of errors. Furthermore, the results indicate that children with ADHD do not have an error-detection deficit. These findings provide a clearer understanding of the specific nature of the performance monitoring deficit in ADHD and will advance future research toward gaining a more comprehensive understanding of the post-error adjustment deficit in ADHD.
References


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cortex: a source analysis of the magnetic equivalent of the error-related negativity. *Biological Psychology, 64*, 157-166.


Table 1

*Summary of Demographic Information for Samples*

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<th>Age</th>
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Table 2

*Main and Interaction Effects*

**Main Effect of Group**

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<td>Mean SE</td>
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<td></td>
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<tr>
<td>Post-error slowing (ms)</td>
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<td>8.68</td>
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<td>Percent correct (accuracy)</td>
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**Main Effect of Condition**

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<td>Mean SE</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-error slowing (ms)</td>
<td>23.0 16.7</td>
<td>61.4 17.2</td>
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<tr>
<td>Percent correct (accuracy)</td>
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**Group X Condition Interaction**

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<tr>
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<td>Mean SE</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Post-error slowing (ms)</td>
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<td>108.2 27.9</td>
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<tr>
<td></td>
<td>-0.615 19.9</td>
<td>14.5 20.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent correct (accuracy)</td>
<td>81.0 2.29</td>
<td>84.8 2.38</td>
<td>0.16</td>
<td>.687</td>
</tr>
<tr>
<td></td>
<td>74.2 1.69</td>
<td>76.3 1.70</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. *indicates significance at the 0.05 level, **indicates significance at the 0.005 level, *** indicates significance at the 0.0005 level.
Table 3

*Within Condition Performance*

<table>
<thead>
<tr>
<th>Measure</th>
<th>ADHD Mean</th>
<th>ADHD SD</th>
<th>Control Mean</th>
<th>Control SD</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-error slowing (ms)</td>
<td>-0.62</td>
<td>132.1</td>
<td>46.7</td>
<td>214.5</td>
<td>1.61</td>
<td>.210</td>
</tr>
<tr>
<td>Percent correct (accuracy)</td>
<td>74.2</td>
<td>16.0</td>
<td>81.0</td>
<td>12.7</td>
<td>6.15</td>
<td>.015*</td>
</tr>
</tbody>
</table>

*Feedback Condition Performance*

<table>
<thead>
<tr>
<th>Measure</th>
<th>ADHD Mean</th>
<th>ADHD SD</th>
<th>Control Mean</th>
<th>Control SD</th>
<th>F-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-error slowing (ms)</td>
<td>14.5</td>
<td>162.0</td>
<td>108.2</td>
<td>194.3</td>
<td>7.133</td>
<td>.009**</td>
</tr>
<tr>
<td>Percent correct (accuracy)</td>
<td>76.3</td>
<td>15.8</td>
<td>84.8</td>
<td>9.2</td>
<td>12.51</td>
<td>.001**</td>
</tr>
</tbody>
</table>

*Note.* *indicates significance at the 0.05 level, **indicates significance at the 0.005 level*
Table 4

*Performance Across Conditions*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Neutral Mean</th>
<th>Neutral SD</th>
<th>Feedback Mean</th>
<th>Feedback SD</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-error Slowing (ms)</td>
<td>3.7</td>
<td>130.7</td>
<td>16.3</td>
<td>162.4</td>
<td>-.656</td>
<td>.257</td>
</tr>
<tr>
<td>ADHD</td>
<td>34.5</td>
<td>204.8</td>
<td>108.2</td>
<td>194.3</td>
<td>-1.775</td>
<td>.042*</td>
</tr>
<tr>
<td>Control</td>
<td>74.3</td>
<td>16.0</td>
<td>76.1</td>
<td>15.8</td>
<td>-1.467</td>
<td>.074</td>
</tr>
<tr>
<td>Percent Accuracy</td>
<td>79.9</td>
<td>12.5</td>
<td>84.8</td>
<td>9.2</td>
<td>-2.438</td>
<td>.010*</td>
</tr>
</tbody>
</table>

*Note.* *indicates significance at the 0.05 level
Figure 1. Cognitive model of performance monitoring
Figure 2. Representation of a trial from the modified flanker task
Figure 3. Non-significant interaction of group and condition on post-error slowing in milliseconds (ms)
Figure 4. Non-significant interaction of group and condition on percent correct (accuracy)
Figure 5. Mean post-error slowing in milliseconds (ms) across conditions
Figure 6. Mean percent correct (accuracy) across conditions
Appendix A. DSM-IV-TR criteria for Attention Deficit Hyperactivity Disorder

A. Either (1) or (2):

(1) six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

**Inattention**

a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities  
b) often has difficulty sustaining attention in tasks or play activities  
c) often does not seem to listen when spoken to directly  
d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)  
e) often has difficulty organizing tasks and activities  
f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)  
g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)  
h) is often easily distracted by extraneous stimuli  
i) is often forgetful in daily activities

(2) six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

**Hyperactivity**

a) often fidgets with hands or feet or squirms in seat  
b) often leaves seat in classroom or in other situations in which remaining seated is expected  
c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)  
d) often has difficulty playing or engaging in leisure activities quietly  
e) is often “on the go” or often acts as if “driven by a motor”  
f) often talks excessively

**Impulsivity**

 g) often blurts out answers before questions have been completed  
h) often has difficulty awaiting turn  
i) often interrupts or intrudes on others (e.g., butts into conversations or games)

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.
C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

_Code_ based on type:

**314.01 Attention Deficit/Hyperactivity Disorder, Combined Type:**
if both Criteria A1 and A2 are met for the past 6 months

**314.00 Attention Deficit/Hyperactivity Disorder, Predominantly Inattentive Type:**
if Criterion A1 is met but Criterion A2 is not met for the past 6 months

**314.01 Attention Deficit/Hyperactivity Disorder, Predominantly Hyperactive Impulsive Type:**
if Criterion A2 is met but Criterion A1 is not met for the past 6 months

_Coding note:_ For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "In Partial Remission" should be specified.
Appendix B. Eight stimulus array configurations presented in the modified flanker task

NNHNN
NNSNN
MMSMM
MMHMM
SSMSS
SSNSS
HHMHH
HHNHH