Neurophysiological Correlates of Self-monitoring Differentially Moderate the Relation Between Maternal Depression and Children’s Behaviour Problems

by

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

Department of Human Development and Applied Psychology at the University of Toronto

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Department of Human Development and Applied Psychology at the University of Toronto
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Abstract

Research in developmental psychopathology has advanced our understanding of emotional dysregulation underlying children’s behavioural problems and their association with maternal depression. Neurophysiological measures may further clarify these relations. The ERN (error-related negativity) is an event-related potential commonly used to assess self-monitoring, with large amplitudes reflecting internalizing and small amplitudes reflecting externalizing symptoms. The present study investigated the effect of ERN amplitude on the relation between maternal depression and internalizing and externalizing problems in a sample of children referred for aggressive behaviour. ERN amplitudes were recorded while 50 children (8-10 years of age) engaged in a go/no-go emotion induction task. Results indicated an association between high ERN amplitudes and internalizing behaviour and between small ERN amplitudes and externalizing behaviours for children with depressed mothers. These findings suggest that the degree of self-monitoring (indexed by ERN amplitude) moderates the relation between maternal depression and psychopathological outcomes in children.
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<th>Description</th>
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<tbody>
<tr>
<td>ACC</td>
<td>Anterior cingulate cortex</td>
</tr>
<tr>
<td>AMG</td>
<td>Amygdala</td>
</tr>
<tr>
<td>BDI</td>
<td>Beck Depression Inventory</td>
</tr>
<tr>
<td>BI</td>
<td>Behavioural inhibition</td>
</tr>
<tr>
<td>CBCL</td>
<td>Child Behaviour Checklist</td>
</tr>
<tr>
<td>CBF</td>
<td>Cerebral blood flow</td>
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<tr>
<td>CBT</td>
<td>Cognitive behavioural therapy</td>
</tr>
<tr>
<td>dACC</td>
<td>Dorsal anterior cingulate cortex</td>
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<tr>
<td>dlPFC</td>
<td>Dorsolateral prefrontal cortex</td>
</tr>
<tr>
<td>EEG</td>
<td>Electroencephalography</td>
</tr>
<tr>
<td>EF</td>
<td>Executive function</td>
</tr>
<tr>
<td>ERN</td>
<td>Error-related negativity</td>
</tr>
<tr>
<td>ERP</td>
<td>Event-related potential</td>
</tr>
<tr>
<td>fMRI</td>
<td>Functional magnetic resonance imaging</td>
</tr>
<tr>
<td>GAD</td>
<td>Generalised anxiety disorder</td>
</tr>
<tr>
<td>MDD</td>
<td>Major depressive disorder</td>
</tr>
<tr>
<td>mPFC</td>
<td>Medial prefrontal cortex</td>
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<tr>
<td>NA</td>
<td>Negative affect</td>
</tr>
<tr>
<td>OCD</td>
<td>Obsessive-compulsive disorder</td>
</tr>
<tr>
<td>OFC</td>
<td>Orbitalfrontal cortex</td>
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<tr>
<td>PFC</td>
<td>Prefrontal cortex</td>
</tr>
<tr>
<td>PTSD</td>
<td>Post-traumatic stress disorder</td>
</tr>
<tr>
<td>RSA</td>
<td>Respiratory sinus arrhythmia</td>
</tr>
<tr>
<td>sMRI</td>
<td>Structural magnetic resonance imaging</td>
</tr>
<tr>
<td>vACC</td>
<td>Ventral anterior cingulate cortex</td>
</tr>
<tr>
<td>vlPFC</td>
<td>Ventrolateral prefrontal cortex</td>
</tr>
<tr>
<td>vmPFC</td>
<td>Ventromedial prefrontal cortex</td>
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Chapter 1
Introduction

1 Introduction
1.1 Overview

The ability to appropriately regulate emotions is a critical component of children’s socioemotional development. However, children who display aggressive and antisocial behaviours, termed externalizing problems, display a difficulty in monitoring their feelings of anger and frustration. These disturbances in socioemotional development are commonly encountered and make up approximately half of all referrals for behavioural problems to children’s mental health agencies (Patterson et al., 1993). However, a vast majority of these children also experience high levels of co-occurring internalizing problems, characterized by social withdrawal, anxiety, and fearfulness (e.g., Flament et al., 1990). Both externalizing and internalizing problems may be characterized by a fundamental difficulty in self-monitoring, which impedes the performance of effective emotional regulation (Lewis et al., 2008; Stieben et al., 2007).

One environmental influence believed to be associated with the genesis of these psychopathological symptoms and their underlying self-regulatory deficits is maternal depression. Although extensive research has been conducted on the effects of maternal depression on children’s internalizing and externalizing behaviour problems, it is not clear what factors predispose children toward either of the behavioural outcomes. Furthermore, there is only so much that behavioural studies can tell us about the mechanisms of cognitive and emotional processing specific to these relations. Neurobiological differences in self-monitoring may help
elucidate trait-like characteristics that increase a child’s vulnerability for experiencing a diverse range of socioemotional impairments linked to maternal depression. The present study investigated the neural correlates of self-monitoring in a comorbid sample of children referred for aggressive behaviour problems to assess their influence on the relation between maternal depression and both internalizing and externalizing symptoms.

1.2 Impact of maternal depression on children’s behaviour problems

With a multitude of environmental influences acting on children from birth, distinguishing the critical risk factors that predict developmental impairment is a multifaceted task. Previous studies have identified socio-economic status (SES) (Lovejoy et al., 2000), peer and teacher interrelationships (Calkins et al., 1999), and factors related to family dynamics such as birth order and mother-child attachment (Goodman & Gotlib, 1999) as influences on children’s development. However, a key focus in this area of research has been on the early familial environment and, more specifically, the role of maternal depression.

Whereas predictions from maternal depression to various child outcomes have been reported by researchers in developmental psychopathology, no clinical diagnosis is generally identified. Rather, maternal depression is normally characterized as depressed mood, symptoms of which are tapped by standardized instruments such as the Beck Depression Inventory (BDI). These symptoms include social withdrawal, self-dissatisfaction, and irritability among many others (Beck et al., 1988). It is important to emphasize that depression in this study, as in most research in developmental psychopathology, is not determined by clinical diagnosis as would be evaluated by a psychiatric assessment.
Characteristic profiles of depressed mothers have been well studied. Mothers suffering from depression have a tendency to reveal more negative cognitive thought patterns, behaviours and affect (Goodman & Gotlib, 1999). Findings from self-reports as well as observational studies reveal that depressed mothers have elevated negativity in self-perceptions, enhanced memory for negative events as well as more internal, stable, and global attributions of negative events compared to healthy controls (Rogosch et al., 2004; Goodman & Gotlib, 1999). Observational studies suggest that, not only do depressed mothers show more negative behaviours towards their children (Foster et al., 2008), but they also display less maternal positivity compared to non-depressed mothers (Jacob & Johnson, 1997). When interacting with their offspring, depressed mothers demonstrate excessive intrusiveness, criticism, irritability, and use of coercive, conflict-causing, disciplinary techniques (Rogosch et al., 2004; Weissman et al., 1987; Lovejoy et al., 2000). These are associated with less emotional engagement, more withdrawal, and reduced overall warmth during mother-child interactions (e.g., Wan et al., 2009; Goodman & Gotlib, 1999). The negative behaviours and affect to which children of depressed mothers are exposed has led to the belief that maternal depression may be associated with poor child functioning in a range of developmental domains (Goodman & Gotlib, 1999).

Therefore, it is of no surprise to researchers that exposure to maternal depression poses an increased risk for child psychopathology (e.g., Goodman & Gotlib, 1999). A large body of work has shown associations between maternal depression and both internalizing (e.g., Silk et al., 2006; Warner et al., 1999; Weissman et al., 1997) and externalizing problems in children (e.g., Ashman et al., 2008; Gross et al., 2008; Kim-Cohen et al., 2005). Internalizing behavioural problems have been characterised by symptoms of social withdrawal, loneliness, anxiety, inhibition, depression, shyness, and fearfulness (Flament et al., 1990). It has been estimated that children of depressed mothers are six times more likely to suffer from depression and anxiety-
related symptoms than children of non-depressed mothers (Downey & Coyne, 1990), with a 3 - 4 fold risk for developing depression prior to adulthood (Goodman & Golib, 1999; Weissman, et al., 1997). Ormel et al. (2005) assessed over 2000 children (10- and 11-years of age) at two time points (2-3 years apart) for socioemotional problems in relation to a history of parental (95.6% mothers) psychopathological symptoms. The researchers found that a history of internalizing disorders in parents predicted internalizing but not externalizing symptoms in children, and vice versa (Ormel et al., 2005). An analysis of a community sample of 15-year old offspring and their depressed and non-depressed mothers also revealed interesting results (Phillips et al., 2005); having a depressed mother within the first 5 years of life was shown to be significantly related to depressive disorders and particularly to anxiety disorders at the age of 15 years (Phillips et al., 2005). Another longitudinal study revealed that maternal depression and greater fearfulness/shyness in children predicted internalizing behaviour over the course of 12 years (Leve et al., 2005). These studies reveal strong associations of maternal depression with children’s and adolescents’ internalizing problems.

Although the impact of maternal depression has been associated with internalizing problems in offspring, the literature also emphasizes its influence on externalizing problems including conduct disorder (Hammen et al., 1990) and substance abuse (Weissman et al., 1997). Parents and teachers most commonly characterize children with externalizing behaviours as impulsive, disruptive, hyperactive, aggressive, and delinquent (Connor et al., 2004; Bank et al., 1993). In a longitudinal study with 24-month and 6-year old children from a community sample (n=1,364), symptoms of maternal depression and parenting behaviours were found to be strongly associated with externalizing behaviours in the younger cohort of boys (Blatt-Eisengart, et al., 2009). Whereas this relationship attenuated with time in boys, girls’ externalizing symptoms were positively associated with increased levels of maternal depression from 24 months to 6 years of
age (Blatt-Eisengart, et al., 2009). A longitudinal study assessing internalizing and externalizing trajectories in children from age 5 through to adolescence (17 years of age) also revealed sex differences (Leve et al., 2005). Study results showed that among boys who expressed low impulsivity, maternal depression predicted externalizing problems later on; however, in girls, externalizing outcomes were only predicted when impulsivity was initially high (or when fear/shyness was low) (Leve, et al., 2005). In summary, these findings suggest that maternal depression in early childhood is associated with the development of externalizing problems in offspring.

Most research focuses on internalizing and externalizing symptoms in isolation, however many children suffer from comorbid socioemotional problems (e.g., Lewis et al., 2008; Stieben et al., 2007). Unfortunately, not only do we know very little about such heterogeneous populations, but even less research has considered the effects of maternal depression on their development. In a cross-sectional study that attempted to identify maternal predictors of children’s (aged 2-6 years old) behaviour problems, chronic stressors and the mother’s depressive symptoms accounted for 27% of the variability in internalizing behaviour and 21% of the variability in externalizing behaviour (Hall et al., 2008). Since the great majority of referred children show both internalizing and externalizing behaviour problems, a better understanding of the behavioural profiles of the individuals in this population could benefit from an examination of the underlying difficulties that make them differently susceptible to the impact of maternal depression. However, what makes some children more susceptible to the impact of maternal depression than others, and what accounts for the different outcomes attributable to maternal depression? The factors that increase or decrease children’s vulnerability to the negative influence of maternal psychopathology, as well as the kind of symptoms resulting from that vulnerability, may depend on children’s capacity to regulate their emotions.
1.3 Emotion regulation and children’s behaviour problems

A fundamental skill underlying healthy socioemotional development is the ability to effectively regulate negative emotions and the impulses that accompany them. Although scientists continue to disagree over its main constituents, emotion regulation, also termed emotional self-regulation, can be understood as cognitive operations that modify the appraisals, feelings, and/or behaviours that accompany emotions (Gross, 2002; Lewis et al., 2007; Lewis, in press). Emotions are multidimensional in nature, encompassing experiential, behavioural, and physiological components (Amstadter, 2008), and they give rise to a diverse set of individually specific control processes aimed at regulating them. Ultimately, children regulate emotions to maximize the experience or expression of positive emotions and to minimize the experience or expression of negative ones (Quirk & Beer, 2006). However, successful engagement in social interactions often requires children to learn how to regulate emotional expression on the basis of contextual cues (Eippert et al., 2007; Gross, 2002), even if they are not fully consistent with their internal feelings or goals.

Self-regulation falls under the umbrella of cognitive activities known as executive functions (EFs) (Lewis, in press). Commonly studied cognitive processes in the EF literature include focused or sustained attention, inhibitory control, switching, decision making between two optimal options, and action- and self-monitoring (e.g., conflict detection and error monitoring) (Zelazo & Mueller, 2002; Séguin & Zelazo, 2005). Emotional self-regulatory strategies, such as avoidance and the ability (or inability) to distract oneself from distressing stimuli, like the negative affect and behaviours that children of depressed mothers are exposed to, have been studied in developmental psychology. Appropriate emotional self-regulation reflected in the refocusing of attention onto a non-distressing stimulus is an adaptive means of reducing distress overall (e.g.,
Calkins & Johnson, 1998). On the other hand, the inability to shift attention away from such distress can leave children overwhelmed with negative affect (Derryberry & Rothbart, 1988). Consequently, attention shifting has been associated with fewer behaviour problems in children (Eisenberg et al., 2000; Gilliom et al., 2002; Valiente et al., 2003). However, only a limited amount of literature discusses how the ability to perform self-regulation develops into late childhood or how the inability to regulate their own emotions with respect to environmental influences underlies children’s susceptibility to the effects of maternal depression on their socioemotional development.

Given that effective emotional self-regulation is a critical aspect of children’s personality and social development, abnormalities in emotional processing may reflect poor developmental outcomes, including behaviour problems and difficulties interacting with peers (Calkins et al., 1999; Eisenberg et al., 2001). Failing to effectively self-regulate may be a critical factor underlying the onset and progression of externalizing and internalizing behaviour problems in children and adolescents (eg: Eisenberg et al., 2007; Eisenberg, et al., 2001; Lewis et al., 2008; Stieben et al., 2007). Such socioemotional and behavioural issues seem to be associated with difficulty in controlling negative emotions. More specifically, aggressive and antisocial acts of children are a reflection of underregulation (undercontrol) of feelings of frustration and anger, whereas the behavioural inhibition, fear and sadness commonly characterizing internalizers are a reflection of emotional overregulation (Stieben et al., 2007; Lewis et al., 2006; Eisenberg et al., 2001). Underregulation of children with externalizing behaviour has been associated with lower effortful control (Ormel et al., 2005) and a lack of awareness of responsibility for one’s actions (e.g., Derryberry & Reed, 1994; Posner & Rothbart, 2000). It has also been proposed that children with purely externalizing symptoms, who have been characterised with high impulsivity and emotional reactivity, may lack self-reflection and evaluation over their impulsive behaviour
that differentiates them from children who experience comorbid internalizing problems (Oland & Shaw, 2005). These children are thus often disliked by their peers (Calkins et al., 1999) and have difficulties interacting with teachers and other adult figures in their lives (Connor et al., 2004; Hinshaw et al., 1995). Hence, the primary cause for aggressive behaviours in children with externalizing behaviour problems may be the triggering of maladaptive self-regulatory techniques by emotionally challenging situations, which foster increased and underregulated negative emotions, including frustration and anger.

Conversely, overregulated emotional control in clinically anxious individuals is regarded as an over-engagement and rigidity in the use of maladaptive self-regulatory strategies (Eisenberg et al., 2001; Lewis et al., 2008). Internalizing psychopathology is thus typically associated with the inability to regulate excess negative affect, usually anxiety or fear (Ormel et al., 2005), which contributes to impaired cognitive and emotional functioning (Amstadter, 2008). Studies looking at regulatory styles of children with internalizing behaviour problems reveal that they have an attentional bias to emotional (especially threatening) stimuli including angry faces (Telzer et al., 2008) and a difficulty shifting their attention elsewhere. Using a visual probe task, Waters et al. (2008) measured attentional bias in 7-12 year old children (n = 23) with generalised anxiety disorder (GAD) and found that severely anxious children showed an attentional bias toward emotional faces (both happy and angry faces), whereas healthy controls and those children with milder GAD levels did not. In fact, anxious individuals even have a tendency to interpret ambiguous social information (especially ambiguous facial expressions) as threatening (Yoon & Zinbarg, 2007). It seems that the distress experienced in response to such minor aversive environmental cues among internalizers is associated with a heightened sense of self-awareness and involuntary inhibition of one’s actions (Eisenberg et al., 2000) related to shyness and feelings of shame (Eisenberg et al., 2001). This results in persistent, anxious rumination and self-blame.
(Garnefski et al., 2005) compared to aggressive and low-socialized individuals (Dikman & Allen, 2000). The increased levels of anxiety and fearfulness experienced by individuals with internalizing problems thus leave little opportunity for recruiting a more flexible and adaptive repertoire of regulatory strategies to control negative emotions (Eisenberg et al., 2001; Lewis, in press).

Although externalizing and internalizing behaviours have often been thought of as occurring separately, prevalence studies reveal an overwhelmingly high rate of comorbidity between these clusters of symptoms (Ingram & Price, 2002). In fact, many children referred for externalizing behaviours have been found to make up a heterogeneous population experiencing both externalizing and internalizing symptomatology (e.g., Garnefski et al., 2005; Lewis et al., 2008; Stieben et al., 2007). As I have previously noted, children’s aggressive and antisocial acts are a reflection of underregulated frustration and anger. However, as described in Panksepp’s (1998) model of motivated aggression, the inability to regulate one’s experience of extreme sadness, or feelings of fear and anxiety, can also provoke the onset of aggressive acts. Thus, children comorbid for internalizing and externalizing symptoms also reveal great difficulty performing effective emotion regulation. Garnefski and colleagues (2005) measured specific cognitive emotional regulation strategies of 271 12- to 18-year-old healthy adolescents with internalizing problems, externalizing problems, or a comorbidity for both internalizing and externalizing problems, compared to a control group. Analyses revealed that scores on self-blame and rumination were significantly greater among adolescents who were pure internalizers or comorbid for externalizing and internalizing, compared to the pure externalizers and controls (Garnefski et al., 2005). Consequently, a comprehensive study of populations comorbid for internalizing and externalizing behaviour problems requires an investigation of the underlying emotional dysregulation associated with two emotional dimensions: anxiety, fear, self-blame, and excess
rumination, on the one hand, and anger, frustration, and impulsivity on the other, which may also reveal individual differences in children’s level of self-monitoring.

Self-monitoring seems to be an important component of emotional self-regulation (Luu, et al., 2000b). The notion of self-monitoring has been more commonly reviewed in social psychology and personality research (e.g., Tice, 1992; Kulik et al., 1986). In the early years of work in this area of research, Snyder and colleagues showed that the notion of self-monitoring was relevant to individual differences in sensitivity to social or situational cues (e.g., Snyder, 1974; Snyder & Gangestad, 1986), which can be observed in the regulation of individuals’ expressive self-presentation (Snyder & Gangestad, 1986). Tice (1992) assessed low and high self-monitoring in 80 university students, and found that high self-monitoring was more strongly associated with internalizing behaviour than low-self-monitoring, and that this effect was strongest in interpersonal settings compared to private settings. These findings may be associated with distinct self-regulatory styles and behavioural problems that I had previously discussed; the inability to regulate negative emotions in low self-monitors may reflect an underregulated style of emotional control, whereas a stimulus-bound style of emotional overregulation may be more closely linked with enhanced self-monitoring. On the other hand, children with high levels of comorbid internalizing and externalizing problems may display individual variability in levels of self-monitoring, which may originate from the inability to regulate the experience of sadness, fear, and anxiety, as well as anger and frustration (Panskepp, 1998). Failing to effectively self-monitor may thus not only be an important factor underlying the onset and progression of externalizing and internalizing behaviour problems, but also be critical in the genesis of comorbidity for these psychopathological symptoms among children and adolescents.

Because emotion regulation is critical to children’s early social and emotional development, and
caregivers are thought to play an important role in fostering its maturation (Ashman et al., 2008; Blandon et al., 2008), many researchers suggest that maternal depression has an effect upon children’s self-regulation that underlies the behavioural outcomes I have discussed (e.g., Goodman & Gotlib, 1999; Silk et al., 2006). Styles of emotion regulation can also be viewed as stable trait-like variables that modulate children’s responsiveness to emotionally challenging situations. For children who display high levels of self-monitoring, the negative feelings associated with exposure to maternal depression, including rejection and emotional disengagement, may increase children’s rumination and self-blaming, reflecting internalizing symptoms (Nolen-Hoeksema & Girgus, 1994; Silk et al. 2006). In contrast, children with low self-monitoring characteristic of emotionally underregulated individuals may be more readily angered, frustrated, and display externalizing behaviours in response to maternal depression. Such maladaptive self-regulatory skills of children with depressed mothers may underlie children’s predispositions for different psychopathological outcomes.

It is becoming a widespread concern of researchers to understand the role of emotional self-regulation in development, and how impairment of emotion regulation may influence a child’s susceptibility to negative environmental influences. However, few have moved beyond behavioural measures. Current methodologies offer many other avenues for assessing emotion and its regulation, including psychophysiological and neurophysiological techniques. Furthermore, as the field of developmental neuroscience continues to expand, it is becoming evident that a comprehensive model of the emotional dysfunctions underlying psychopathological disorders will require a greater understanding of their neurobiological underpinnings (e.g., Lewis et al., 2008; Wager, et al., 2008). For now, relatively little attention has been given to the study of neural correlates of emotion regulation in children and how they may increase their risk for experiencing an array of psychopathological outcomes in the context of maternal depression. The
study of the neural mechanisms underlying distinct styles of over- and underregulation would contribute to our understanding of these distinct developmental pathways.

1.4 Emotion regulation and the brain

1.4.1 Cortical regions involved in emotion regulation

Research in developmental neuroscience within the past decade has significantly advanced our understanding of the cognitive (or executive) mechanisms that are involved in regulating emotion-related responses (Lewis et al., 2008; Stieben et al., 2007). Since the associations between emotion regulation and developmental psychopathology, as well as the impact of maternal depression on problematic behavioural outcomes, have been investigated, further neuroscientific research may help elucidate the relations among these variables of interest. In this section, I briefly review this literature and discuss its relevance for the goals of the present study.

Emotion regulation is thought to be mediated by neural circuitry involving several cortical regions. These incorporate the prefrontal cortex (PFC), including the ventral and lateral orbitofrontal cortex (OFC), and dorsolateral PFC (dLPFC), as well as the adjacent anterior cingulate cortex (ACC) (Beauregard et al., 2006; Davidson et al., 2002). The PFC has been shown to exert inhibitory control over the amygdala (AMG), another major area of focus in research on emotional processing (Davidson et al., 2000), and to be involved in attention, decision-making, self- and conflict-monitoring, as well as other cognitive activities (e.g., Kim & Hamann, 2007; Ladouceur et al., 2007). In the study of appraisal, inhibitory control, self-monitoring, characteristic structures and functional activation patterns have led to the distinction of the dorsal from the ventral prefrontal system (e.g., Bush et al., 2000; Drevets & Raichle, 1998; Luu et al., 2003). The ventral prefrontal system (e.g., ventral ACC and OFC) seems to underlie attention and inhibition of behavioural impulses in anticipation of threat, whereas the dorsal...
prefrontal system (e.g., dorsal ACC and dlPFC) seems to be involved in more deliberate switching of attention and behaviour (Lewis et al., 2008; Philips et al., 2003).

The ACC is a key structure in the regulation of emotion and cognition (e.g., Ladouceur et al., 2007; Luu et al., 2003). It is thought to be the brain’s “error detection and correction device” (Carter et al., 1998) involved in self-monitoring and behavioural regulation (e.g., Bush et al., 2000). The ACC’s two functionally distinct subdivisions make it a critical component of the circuitry regulating both cognitive and emotional processing (Bush et al., 2000). The affective ACC component is predominantly involved in detecting emotional and motivational information, as well as regulating emotional responses; as part of the ventral prefrontal system, the ventral ACC (vACC) has elaborate connections with the OFC, periaqueductal grey, AMG, and other limbic structures (Bush et al., 2000; Devinsky et al., 1995). A rostral ACC (rACC) area commonly associated with the affective division of the vACC (Devinsky et al., 1995) was also identified by Yamasaki et al. (2002) and thought to act as a bridging point between the dorsal and ventral subdivisions. On the other hand, strong reciprocal connections with the lateral PFC, parietal cortex, and premotor and supplementary motor areas (Bush et al., 2000) make the dorsal cognitive division of the ACC (dACC) an integral part of processes involving attention and executive function, error detection, working memory, motivation, and anticipation (Bush et al., 2000). Evidence for these predictions has come from work on the differential activation patterns of the ventral versus the dorsal subdivisions monitored when participants engage in the Emotional Counting Stroop task (Whalen et al., 1998) versus the Counting and interference Stroop tasks, respectively (Counting Stroop: Bush et al., 1998; interference Stroop: Drevets & Raichle, 1998). Furthermore, reciprocal suppression among these areas has been observed in regional cerebral blood flow of participants switching from engaging in a neutral or emotional task (greater ventral
activity) to performing tasks requiring higher cognitive processes (greater dorsal activity) (Drevets & Raichle, 1998).

Regional activation within the ventral and dorsal prefrontal systems, including the neighboring ACC, has been systematically associated with internalizing and externalizing psychopathology, mainly in adults and adolescents. Cortical patterns reflecting abnormal emotional styles of over- and under-regulation have been elucidated and associated with these psychopathological tendencies. More specifically, the underactivation of both dorsal and ventral prefrontal systems are believed to characterize externalizing problems and reduced self-monitoring (Davidson et al., 2000; Lewis, in press). More recently, Eshel and colleagues (2007) found that adolescents who engage in risky behaviour show a reduction in activity levels of both the dorsal and ventral prefrontal systems. Several studies point toward reduced activation particularly in the OF- and AC- cortices as neural correlates of underregulated behaviour and deficits in self-monitoring (Davidson et al., 2000). In a most recent study using functional magnetic resonance imaging (fMRI) and structural magnetic resonance imaging (sMRI), 14 – 17 year old adolescents were asked to imagine aggressive and neutral (non-aggressive) interactions with a peer (Strenziok et al., 2009). The findings suggest that even imagining an aggressive interaction is associated with a reduction in ventral-medial prefrontal cortex (vmPFC) activation (Strenziok et al., 2009).

Conversely, numerous studies report that overactivation of the ventral system characterizes anxiety and other internalizing problems (e.g., Drevets & Raichle, 1998; Monk et al., 2006). More specifically, hyperactivation of the OFC and vACC are commonly reported in neuroimaging studies investigating anxious and depressed subjects (Drevets, 2000b; Drevets & Raichle, 1998). It has been shown that inducing anxious states in individuals with various anxiety disorders including obsessive-compulsive disorder (OCD), specific phobias, and post traumatic
stress disorder (PTSD) results in an increase in cerebral blood flow (CBF) within the left ventrolateral PFC (vlPFC) and other ventral systems; inducing sadness and anxiety in controls (healthy individuals) has provided similar results (see review by Drevets & Raichle, 1998). Many researchers, including Drevets et al (1992, 1995), have shown that individuals suffering from major depressive disorder (MDD) also show increased CBF and metabolism in these areas, with the vlPFC and the right posterior orbital cortices in particular (reviewed by Drevets, 2000a). In a study assessing attention to threat in non-clinically anxious youth, trait anxiety was not only positively associated with a bias toward angry faces, but also with right vlPFC activation in response to all emotional faces (Telzer et al., 2008). Monk et al. (2006) obtained similar results in a sample of 18 adolescents with GAD in comparison with healthy controls. Reduced internalizing symptomatology, on the other hand, has been associated with decreased vACC activation in the case of anxiety (Davidson et al., 2002) and with successful treatment for depression (Drevets, 2000b). Furthermore, Kalisch et al. (2006) showed that while inducing anticipatory anxiety, participants were able to attenuate dorsal medial prefrontal/rostral ACC activity under high, compared to low, cognitive load. This evidence suggests that ventral activation in anxious and depressed subjects may also initiate deactivation of dorsal prefrontal systems.

Concurrent symptoms of aggression and anxiety in comorbid samples make it difficult to predict patterns of prefrontal activation and deactivation associated with emotional dysregulation. In a sample of children comorbid for internalizing and externalizing problems, Lewis and colleagues (2008) found that attenuation of ventral activity was associated with behavioural improvement from pre- to post-treatment. However, children who improved with cognitive behavioural therapy (CBT) also maintained high levels of dorsal activation while their ventral activity had subsided (Lewis et al., 2008). The authors speculated that behavioural improvement was associated with a shift in the emotion regulation habits of children with behaviour problems mediated by the
ventral prefrontal cortex (Lewis et al., 2008). Furthermore, work in this understudied area of research has also recently produced other interesting findings. Using a provocation manipulation paradigm previously shown to anger participants (Pedersen et al., 2000), Denson and colleagues (2009) induced angry rumination in 20 undergraduates ($M_{age} = 18.68$) after insulting them. Although rumination is generally associated with internalizing symptoms, work in aggression has proposed that angry rumination is also observable and similarly contains components of negative affect, self-reflection and emotional regulation that control the degree to which anger is expressed (Denson et al., 2009). This seems to reflect an internalizing component associated with aggression. The primary findings revealed that individual differences on an aggression scale as well as self-reported feelings of anger were positively associated with dACC activation, whereas individual differences in displaced aggression and self-reported rumination were related to activation in the medial PFC (Denson et al., 2009). This work may suggest that heterogeneity (anger as well as increased rumination) in normal populations can be associated with individual differences in self-regulation and may also help illuminate the mechanisms underlying impaired emotional self-regulation in children with clinical levels of internalizing as well as externalizing behaviours. Although our understanding is still in its infancy, the neural correlates of multiple regulatory systems may thus be involved in performing partial control over internalizing and externalizing symptoms (Davidson et al., 2002; Lewis et al., 2008; Stieben et al., 2007).

1.4.2 ERP correlates of emotion regulation: the ERN

Neuroscientific approaches to studying these phenomena use lesion studies and neuroimaging tools including functional magnetic resonance imaging (fMRI), electroencephalography (EEG), positron emission tomography (PET) and magnetoencephalography (MEG), to investigate cortical activation profiles in regions of interest. EEG is a particularly useful neuroimaging instrument for the study of neural correlates of emotion regulation in children. Unlike many of
the other commonly used techniques, EEG does not require metabolic measures of change. Instead it is a non-invasive measure of the electrical activity of cortical neurons (Ladouceur et al., 2006). EEG data are often processed to provide event-related potentials (ERPs), or averaged EEG waveforms time-locked to a stimulus or response. Relative to other neuroimaging techniques, EEG has millisecond-level temporal resolution, thus providing researchers with the neural correlates of specific cognitive processes as they arise. Especially when evaluating self-monitoring in clinical populations, such temporal acuity is critical in establishing links with observable, behavioural outcomes.

Electrophysiological research suggests that a reliable index of self-monitoring processes is a sharp negative deflection in the ERP known as the error-related negativity (ERN) (Dehaene, et al., 1994; Falkenstein et al., 2000; Gehring, et al., 1993; Ladouceur et al., 2006). The ERN is a response-locked ERP component occurring approximately 50-150 ms after response to a stimulus (Dehaene et al., 1994; Gehring et al., 1993; Hajcak et al., 2003). It is most distinct after commission of an error on speeded performance tasks (Falkenstein et al., 2000; Gehring et al., 1993).

Several theories have been proposed to elucidate the precise role of the ERN. The theory of error-detection postulates that the ERN reflects the recognition of a mismatch between the representation of the intended and actual action (Falkenstein et al., 2000). Although awareness of error may be necessary for an ERN to occur (Dehaene et al., 1994), other researchers postulate that the ERN amplitude does not reflect the mere detection of an error but rather the monitoring of response conflict that arises between the intended and actual response selection (Botvinick et al., 2004; Yu, & Zhou, 2009). Error trials would thus be expected to induce a high level of response conflict compared to correct trials, resulting in larger ERN amplitudes (Ladouceur et al.,
It is generally agreed that the ERN’s role in the error-detection system is not to correct errors, but to signal the need for higher-order cognitive control processes to adjust performance (Botvinick et al., 2004; van Veen & Carter, 2002; Yeung et al., 2004), the result of which may be a slowing of responses on trials following errors (Gehring et al., 1993). Thus overall, the ERN seems to serve an on-going self-monitoring function rather than checking for incorrect responses (Luu et al., 2000b).

Additionally, the generation of the ERN is believed to be affected by error salience and motivation (Bernstein, 1995; Bush et al., 2000) as well as the extent to which participants believe that they have erred (Coles et al., 2001); ERN amplitudes are larger when the need for accuracy on a task is emphasized over speed of performance (Gehring et al., 1993). Finally, based on the assumption that increasingly late responses elicit more attentional self-monitoring in order to avoid the error of responding past the deadline, Luu and colleagues (2000b) examined ERN amplitude activity associated with responses that were slightly, moderately, or very late. As predicted, they found that increasingly late response were associated with increasingly large ERN amplitudes. The authors speculate that previously studied conflicting responses engage processes of self-monitoring; however, self-monitoring itself (as reflected in the ERN) does not require conflict in order for it to occur (Luu et al., 2000b). Rather, the ERN seems to tap processes of action monitoring and self-monitoring in situations where performance matters.

Substantial evidence utilizing EEG and fMRI source-localization methods points towards a primary medial frontal generator of the ERN commonly identified as the ACC (Dehaene et al., 1994; Hajcak et al., 2003; Hajcak & Olvet, 2008; van Veen & Carter, 2002). In support of a common role in error detection and more general response monitoring, fMRI findings point to ACC activation during the ERN on both correct and incorrect trials that elicit conflict (Carter et
al., 1998; van Veen & Carter, 2002); thus, similarly to the ERN, the ACC seems to come on-line at times of action-monitor rather than mere error detection (Carter et al., 1998). Also, the ACC is believed to be involved in cognitive and emotional processing, which supports the finding of common peak activation patterns of the ERN and ACC that reflect affective responses to errors (e.g., Bush et al., 2000; Luu et al., 2000a).

Furthermore, electrophysiological data provide support for a late maturation of the ACC, which may partially explain reduced ERN signals commonly observed in normal young children under the age of 12 years (Ladouceur et al., 2004; Santesso et al., 2006). It may also be the reason for a subsequent increase in ERN amplitude throughout adolescence (Davies et al., 2004; Kim et al., 2007) as the ACC develops into adulthood. Ladouceur et al. (2007) used a flanker paradigm (arrow version) to detect the development of the action monitoring in early adolescence ($M_{\text{age}} = 12$ years), late adolescence ($M_{\text{age}} = 16$ years), and adults ($M_{\text{age}} = 29$ years). The authors source localized the ERN component to the ACC and proposed that the maturation of the ACC in the development of action monitoring played a key role in the increase in ERN amplitude from early adolescence to late adolescence and adulthood (Ladouceur et al., 2007). For this reason, measures of ERN amplitude and associated ACC activity measured in children just before they reach adolescence may be particularly revealing in determining early signs of individual differences in action monitoring and self-monitoring. Thus, although more research is needed to confidently source the ERN, the current assumptions proposing its association to the ACC coincide with aforementioned beliefs about the functional and structural roles of this medial frontal region.

1.4.3 The ERN in relation to adult and child psychopathology

Individual differences in ERN amplitudes have consistently been associated with behavioural problems that may reflect individual differences in self-monitoring. Neurophysiological data have
shown that smaller ERN amplitudes reflect reduced self-monitoring in individuals with externalizing behaviour problems (Hall et al., 2007). Enhanced impulsiveness (Pailing et al., 2002) or low conscientiousness, in response to errors where no reward was expected (Pailing & Segalowitz, 2004), and low socialization characterised by high levels of rebelliousness and aggression (Dikman & Allen, 2000) have all been associated with reduced ERN amplitudes. These findings are particularly useful in associating impulsive/aggressive behaviours with neural correlates of reduced self-monitoring.

In contrast, larger ERN amplitudes have been associated with an array of internalizing symptoms, including anxiety and depression (Ladouceur et al., 2007; Olvet & Hajcak, 2008). Undergraduate students with excessive worry and general anxiety were shown to have increased ERN amplitudes following correct and incorrect responses compared to both non-worriers and phobic controls (Hajcak et al., 2003). Similarly, individuals who score high on trait negative affect (NA) reveal greater ERN amplitudes (Hajcak et al., 2004; Luu et al., 2000). Investigation of ERPs has been performed extensively in individuals with obsessive-compulsive disorder (OCD), as well as in samples of participants exhibiting signs of the characteristic OCD symptoms (Debener et al., 2005; Dehaene et al., 1994). Findings consistently point toward a significantly greater ERN amplitude in OCD patients compared to healthy adults on Flanker tasks (Grundler et al., 2009) and Stroop tasks (Hajcak & Simons, 2002). Santesso and colleagues (2006) also found that greater ERN and Pe amplitudes predicted parental reports of their 10-year old (normal) children’s enhanced OCD behaviours. Previous investigations have also demonstrated that ERN amplitudes increase with OCD symptom severity (Gehring et al., 2000). Finally, McDermott et al. (2009) assessed risk for anxiety disorders in adolescents using ERPs from a Flanker Task and childhood behavioural inhibition (BI). Results showed that the interaction of early BI and large ERNs in
adolescence (enhanced self-monitoring) was significantly associated with clinical anxiety (McDermott et al., 2009). Taken together, these studies indicate a consistent link between anxiety or anxiety-related disorders and enhanced ERN amplitudes, particularly in adults.

Understanding the variations in neural circuitry that are related to dysfunctional styles of emotional self-regulation may help researchers elucidate the underlying mechanisms of socioemotional and behavioural problems in children as well. As reviewed earlier, maternal depression can also predict symptoms of anxiety and aggression and pose an increased risk for the development of psychopathology in children. I suggest that these factors may be considered in tandem to construct stronger models for predicting behavioural outcomes in children. Specifically, neurophysiological research has indexed fluctuations in ERN amplitude associated with different levels of self-monitoring. Hence, variations in children’s ERN amplitude may influence the associated between maternal depression and internalizing and externalizing behaviour problems in children.

1.5 Modeling the ERN as a moderator

In the present study, a moderation model was proposed to investigate the effect of children’s self-monitoring on the association between maternal depression and children’s psychopathological outcomes. A moderator is a variable that affects the direction and/or strength of the relation between the predictor (independent variable) and the dependent variable (Baron & Kenny, 1986). In the current study, the predictor was maternal depression, the moderator was child self-monitoring, and the dependent variables were internalizing and externalizing behaviour. As stated by Coan & Allen (2004, p.8), “moderators are essentially third variables that represent conditions under which some independent variable becomes maximally potent or effective”. This framework can efficiently test the effect of high and low ERN amplitudes in children (reflecting high and
low self-monitoring) as conditions under which maternal depression becomes most potent in predicting behavioural outcomes in children. Specifically, I predicted that the effect of maternal depression on child internalizing behaviours would be maximal when children displayed enhanced self-monitoring (larger ERN amplitudes), whereas the effect of maternal depression on externalizing behaviours would be maximal for children with low self-monitoring (smaller ERN amplitudes). The moderator model is sketched in Figure 1.

*Figure 1.* Conceptual representation of the moderation model. ERN amplitude moderates the relation between maternal depression (predictor) and children's internalizing and externalizing behaviour problems (dependent variables).
As can be seen in Figure 2, there are three pathways that need to be tested. The first path represents the causal influence of the predictor (maternal depression) on the behavioural outcome (internalizing and externalizing behaviour, tested separately). Path 2 shows the independent influence of the ERN on behavioural outcomes. Finally, the third pathway demonstrates the influence of the interaction effect between the predictor and the moderator on the outcome variable. Only the effect of the interaction term (path 3) needs to be significant to support the moderator hypothesis (Coan & Allen, 2004). If there is an effect on the outcome for the predictor or the moderator (path 1 and 2), there is potential for mediation effects and particular consideration has to be taken when interpreting the results. This is because moderator variables and predictors always function as independent variables. Hence, although a correlation between maternal depression and ERN amplitude or between ERN amplitude and internalizing or
externalizing problems would not automatically exclude the existence of a moderator effect, this interpretation would have to be weighed against other possible interpretations.

1.6 Present study

The present study set forth the following goals:

1) To assess the relation between maternal depression and school-aged children’s (8-10 years of age) scores on internalizing and externalizing behaviour problems.

2) To investigate the effect of a neural correlate of self-monitoring in children, indexed by the ERN, on the relation between maternal depression and children’s internalizing and externalizing symptoms.

Before presenting specific hypotheses about cortical activation patterns in children referred for aggressive behaviour problems, it is important to revisit the literature on children’s socioemotional problems as a consequence of maternal depression. Abnormal behaviour of children with depressed relative to healthy mothers can be observed from infancy onward and has been documented throughout the literature. Although few studies have examined child-mother interactions in older children, there is evidence to show that maternal depression predicts externalizing and internalizing behaviour problems. One possible mechanism explaining this association is children’s ability to regulate their emotions. Children with behavioural problems may differ in their ability to regulate negative emotions and, more specifically, in their level of self-monitoring. Emotional overregulation underlying increased anxiety, fear, and rumination characteristic of internalizing problems, may reflect enhanced self-monitoring. Conversely, children with externalizing behaviour problems display underregulated, impulsive behaviour, and
disregard for the consequences of their actions, that presumably result from reduced self-monitoring. In comorbid populations of children, both of these constellations may be present. Given that characteristic styles of emotion regulation underlie socioemotional problems, and that the neural correlates of self-monitoring have been specified in previous research, we can make predictions with regard to the neural signatures of children at risk for behavioural problems. Larger amplitudes of the ERN component in children (reflecting increased self-monitoring) as well as maternal depression have both been associated with children’s internalizing behaviours. Although maternal depression has also been associated with externalizing problems in children, these populations reveal small ERN amplitudes (reflecting low self-monitoring). The model I have proposed suggests that aggressive children comorbid for externalizing and internalizing problems who also show high levels of self-monitoring (greater ERN amplitude) are at an increased risk for experiencing internalizing symptoms under the impact of maternal depression, whereas maternal depression is more likely to be associated with externalizing behaviour in children who are characterized by low self-monitoring (small ERN amplitude). A comorbid population of referred children, with large variability in both scales, may thus be the ideal sample to test the relation between maternal depression and two different symptom types, externalizing and internalizing.

1.7 Design

Clinically referred children were seen with their parents for a single assessment session. Measures of children’s socioemotional problems as well as ratings of maternal depression were obtained from parental reports.

To test for differences in ERN amplitude in an emotionally challenging situation, the testing paradigm used a three-block design with a rigged algorithm: children gained points in block A,
lost points in block B to elicit negative emotion, and regained the lost points in block C. Previous studies confirm that negative emotions such as anger, anxiety, and sadness are induced in block B when children lose many points (Stieben et al., 2007). With this design, the negative emotions experienced by the end of block B were expected to carry over into block C. In order to stay motivated throughout the game, children were told about the rewards of gaining many points. During the go/no-go game, electrical activity in the cortex was recorded at the scalp, and the ERN, an ERP component associated with self-monitoring in both children and adults, was extracted for further processing.

Regression analyses were used to assess discrepancies in the effect of the ERN on the relation between ratings of maternal depression and children’s behaviour problems.

1.8 Hypotheses

The literature suggests that maternal depression has an impact on the development of both internalizing and externalizing symptoms in children. Although both subtypes of behavioural problems are consistently reported, children with depressed mothers have more often shown depression and anxiety.

1) Scores on both internalizing and externalizing behaviours in children will be correlated with self-reported scores of maternal depression.

Because maternal depression can lead to increased internalizing or externalizing, high vs. low ERN amplitudes (reflecting high vs. low self-monitoring) could be the factor that determines which outcome is most likely for any given child. Maternal depression is thus expected to lead to the development of internalizing behaviours among children who exhibit high levels of self-
monitoring, and to increase children’s susceptibility for developing externalizing problems when they exhibit low levels of self-monitoring.

2) **Children’s ERN amplitudes moderate the relation between maternal depression and internalizing vs. externalizing child behaviour outcomes, whereby large ERN amplitudes predict internalizing and small ERN amplitudes predict externalizing behaviours.**

If ERN differences indeed tap individual styles of emotion regulation, then these differences would be most predictive when measured during a negative emotional experience. The current go/no-go paradigm uses a three-block design. Negative emotions are induced in block B (Stieben et al., 2007) when children lose their points, and the negative emotions continue to be experienced as children struggle to regain points in block C (Lewis et al., 2008).

3) **ERN amplitudes in block C (after emotion induction) and not in block A (prior to emotion induction), will predict child behavioural outcomes.**
Chapter 2
Methods

2.1 Participants

Participants were recruited from two outpatient group treatment programs for aggressive children before commencement of treatment. Both programs targeted the same population and were based on the same treatment approach. Referral was made by mental health professionals, teachers, and/or parents over the course of 2 years. Participants were excluded if they had a considerable developmental delay, or if they lived outside the Greater Toronto Area, where the study was conducted. The study included 50 children, 8- to 10-years of age ($M_{\text{age}} = 9.10, \text{SD} = .78$; 41 boys) to perform the analyses. Parental ratings of children’s behaviour on the externalizing subscale of the CBCL ranged from 54 to 83 ($M = 72.12; \text{SD} = 6.84$) and from 45 to 83 ($M = 63.02; \text{SD} = 8.62$) on the internalizing subscale. The participants’ mothers also completed the BDI; scores on the BDI ranged from 0 to 36 ($M = 13.08; \text{SD} = 8.26$), based on items endorsed as descriptive of one’s mood over the past two weeks. For a more detailed description of the sample in this study, child characteristics are in Table 1. A breakdown of the children’s family demographics is provided in Table 2.
Table 1.
Age, Internalizing Scores, Externalizing Scores, and Ratings on Maternal Depression.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Boys (N=41)</th>
<th>Girls (N=9)</th>
<th>All (N=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age *</td>
<td>9.10 (.83)</td>
<td>9.11 (.60)</td>
<td>9.10 (.78)</td>
</tr>
<tr>
<td>CBCL Internalizing score *</td>
<td>62.83 (9.00)</td>
<td>63.89 (6.99)</td>
<td>63.02 (8.62)</td>
</tr>
<tr>
<td>CBCL Externalizing score *</td>
<td>71.83 (6.94)</td>
<td>73.44 (6.58)</td>
<td>72.12 (6.84)</td>
</tr>
<tr>
<td>BDI score *</td>
<td>12.66 (8.19)</td>
<td>15.00 (8.84)</td>
<td>13.08 (8.26)</td>
</tr>
</tbody>
</table>

*Analyses revealed no significant differences between boys and girls on age ($p = .96$), internalizing ($p = .74$) or externalizing ($p = .57$), as well as in their mothers’ ratings of depression on the BDI ($p = .45$).
Table 2.
Children’s Family Demographics (N=50)

<table>
<thead>
<tr>
<th>Family demographics</th>
<th>Number of children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Living arrangement</td>
<td></td>
</tr>
<tr>
<td>Both parents</td>
<td>18</td>
</tr>
<tr>
<td>With mother and step-parent</td>
<td>4</td>
</tr>
<tr>
<td>With father and step-parent</td>
<td>1</td>
</tr>
<tr>
<td>Mother only</td>
<td>20</td>
</tr>
<tr>
<td>Adoptive</td>
<td>3</td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
</tr>
<tr>
<td>European</td>
<td>38</td>
</tr>
<tr>
<td>Asian</td>
<td>1</td>
</tr>
<tr>
<td>African/ Caribbean</td>
<td>7</td>
</tr>
<tr>
<td>Latin</td>
<td>0</td>
</tr>
<tr>
<td>Other</td>
<td>3</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
</tr>
<tr>
<td>Mother's Education (highest level completed)</td>
<td></td>
</tr>
<tr>
<td>Grade 8 or less</td>
<td>3</td>
</tr>
<tr>
<td>Did not graduate high school</td>
<td>4</td>
</tr>
<tr>
<td>High school</td>
<td>11</td>
</tr>
<tr>
<td>Community college</td>
<td>20</td>
</tr>
<tr>
<td>University</td>
<td>7</td>
</tr>
<tr>
<td>Postgraduate/ professional degree</td>
<td>2</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
</tr>
<tr>
<td>Father's education (highest level completed)</td>
<td></td>
</tr>
<tr>
<td>Grade 8 or less</td>
<td>3</td>
</tr>
<tr>
<td>Did not graduate high school</td>
<td>7</td>
</tr>
<tr>
<td>High school</td>
<td>11</td>
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<tr>
<td>Community college</td>
<td>11</td>
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<tr>
<td>University</td>
<td>4</td>
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<tr>
<td>Postgraduate/ professional degree</td>
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</tr>
<tr>
<td>Other</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>1</td>
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<tr>
<td>Family Income ($)</td>
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</tr>
<tr>
<td>0 - 29,000</td>
<td>13</td>
</tr>
<tr>
<td>30,000 - 49,000</td>
<td>15</td>
</tr>
<tr>
<td>50,000 and above</td>
<td>20</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
</tr>
</tbody>
</table>
2.2 Procedure

Each child attended the testing session with his/her mother. After becoming more familiar with the study setting, the planned procedure, and the EEG sensor nets, children gave their assent to participate in the study. Parents filled out consent forms and completed questionnaires (BDI and CBCL) in a nearby room. In the meantime, children were shown two bins full of toys and told that they could win a prize for completing the EEG computer game. One of the bins contained small, undesirable toys such as small plastic cars, whereas the other bin contained larger, more desirable toys including action figures, large stuffed animals, puzzles, activities, and $10.00 gift certificates to local toy stores. It was then emphasized to the children that successful performance (accumulation of points) in the game would be rewarded with a desirable toy of their choice, whereas fewer points by the end of the game would limit their choice of a prize to toys from the less desirable bin.

The EEG testing took place in a control room where children were seated in front of a computer monitor with one experimenter present. Another experimenter sat in the adjacent room and operated the EEG recording system. A chin rest was used to control the child’s distance and alignment to the monitor. The electrode sensor net was applied and children were instructed to respond by clicking a button on the response pad with the index finger of their dominant hand. Each participant completed a practice block (or two if necessary) of 30 trials prior to game commencement in order to ensure that they understood the task instructions.

2.3 Measures and tasks

2.3.1 Measures

*Child Behaviour Checklist (CBCL; Achenbach, 1991).* The CBCL is a standardized, highly reliable, and valid measure of children’s emotional and behavioural problems. Parents are asked
to report on their child’s behavioural problems. The measure yields standardized T-scores for total behaviour problems, as well as internalizing and externalizing subtypes. For the purpose of this study, only standardized T-scores for internalizing and externalizing problems were used. Specifically, the internalizing subscale incorporates scores on social withdrawal, somatic complaints, and anxiety/depression, whereas the combination of scores on delinquent behaviour and aggressive behaviour makes up the externalizing subscale. Symptom severity is determined as follows: 0 to 66 (normal), 67 to 70 (borderline clinical), and above 70 (clinical).

*Beck Depression Inventory (BDI; Beck, A.T., Steer, R.A., Garbin, M.G., 1988).* The BDI is a reliable and valid self-reported measure of depressive symptomatology shown to correlate strongly with clinicians’ evaluations of depression (Modell et al., 2001). The level of depression, based on scores from 0 to 63, can be categorized into one of four main diagnostic categories: (1) <10 (no or minimal depression) (2) 11 to 18 (mild depression), (3) 19 to 29 (moderate to severe depression), (4) 30 to 63 (severe depression).

**2.3.2. ERP Task**

The emotion induction go/no-go task used in the present study was partly adapted from a task developed by Garavan, Ross, and Stein (1999), and presented using E-Prime software (Psychological Software Tools, Pittsburgh, PA). The paradigm normally consists of go and no-go conditions. Participants’ success depends on their ability to press a button as fast as possible when they see stimuli to which a response is expected (the “go” condition) and to inhibit the impulse to respond to prepotent stimuli (the “no-go” condition). In the present version of the task, each letter presented on the monitor was a “go” trial, except for letters appearing a second time in succession; the second viewing of the same letter was a “no-go” trial. For example, if the child saw an “x”, followed by a “y” and another “y”, the child would be expected to respond (“go”
trial) to both the “x” and the first “y”, and to inhibit themselves from responding (“no-go” trial) to the second “y” in this sequence.

Although participants were unaware of the task design, the task was composed of three distinct blocks: blocks A, B, and C. Blocks A and C were identical in trial count (each consisting of 200 trials), and in the number of trials per condition (66 no-go trials). Go and no-go trials appeared in pseudorandom sequence. Block B was intended to induce negative emotions, such as anxiety, sadness, anger, and frustration, and was shorter in length to reduce the duration of possible distress (150 trials; 40 no-go conditions). The negative emotion induction was based on changing the point-adjustment algorithm: children gained points in block A, due to a generous reward formula, but lost all or the majority of their points in block B, when that formula was more challenging. They regained their points in block C with a return to the previous formula. This allowed all children to be successful in the end and win their desired prize.

Children were reminded at the beginning of the task, and at the onset of each block, that a high number of points was required to win the “big prize.” Points were added for correct no-go responses and deducted for response errors on both go and no-go trials. Every 5 to 25 trials, the computer displayed the number of points accumulated throughout the whole game. The points lost were shown in red and accompanied by an unpleasant “buzzer” sound to emphasize poor performance. Conversely, good performance was marked by a gain of points displayed in green and a pleasant “tinkling” sound. Throughout the game, error feedback was provided by a red bar in the middle of the screen following incorrect responses, omitted responses, and late responses.

Additionally, different pairs of letters were used as stimuli in each block (block A: x, y; block B: o, p; block C: u, d). Dynamic adjustment of stimulus duration was applied to maintain the no-go error rate for the task at 50% ± 10%. Stimulus duration was increased with each erroneous
response made on no-go trials. However, decreases in stimulus time only occurred when the no-go trial followed a correct go trial. This constraint was incorporated to prevent stimulus time adjustments that may have been made on the basis of chronic non-responding. The use of dynamic adjustment was intended to provide the same level of challenge for all participants at all ages, and to obtain a sufficient number of correct no-go trials for ERP averaging.

### 2.4 Analyses

#### 2.4.1 EEG data collection and analysis

EEG was recorded using a 128-channel Geodesic Sensor Net and sampled at 250 Hz, using EGI software (Electrical Geodesic, Inc., Eugene, OR). Data acquisition was started after impedances for all EEG channels were below 50 kΩ. All channels were referenced to Cz (channel vRef) during the recording, and later re-referenced against an average reference. Eye blink and eye movement artifacts (70 µV threshold), signals exceeding 200 µV, and fast transients exceeding 100 µV were removed during the averaging process. Data were filtered using a FIR bandpass filter with a lowpass frequency of 30 Hz and a highpass frequency of 1 Hz.

Incorrect no-go data were segmented into epochs from 400 ms before to 800 ms after the response. The ERN was identified as the most negative deflection between 0 ms and 150 ms post response onset from three main channels: 6 (FCz), 11 (Fz), and vRef (Cz). Incorrect no-go trials that did not have a correct go trial preceding and following them were removed (they most likely reflected attentional lapses or chronic non-responding). After segmentation, the data were put through an automatic artifact detection tool, and 10% were checked manually. EEG values for the 0-150 ms window were then averaged for all non-artifact trials.

Measures of ERN amplitude recorded at each of the three electrode sites for blocks A and C, as well as their means across sites within each of these blocks, were computed and considered in this
study. Finally, 8 variables, each reflecting a measure of ERN amplitude, were included in the regression analyses.

Table 3.
Measures of ERN Amplitude in Blocks A and C

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (μV)</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERN amplitude at FCz</td>
<td>-2.74</td>
<td>3.77</td>
</tr>
<tr>
<td>ERN amplitude at Fz</td>
<td>-3.42</td>
<td>3.52</td>
</tr>
<tr>
<td>ERN amplitude at Cz</td>
<td>-1.05</td>
<td>4.99</td>
</tr>
<tr>
<td>Mean ERN amplitude across all channels (FCz, Fz, Cz)</td>
<td>-2.40</td>
<td>3.51</td>
</tr>
<tr>
<td>Block C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERN amplitude at FCz</td>
<td>-3.74</td>
<td>4.12</td>
</tr>
<tr>
<td>ERN amplitude at Fz</td>
<td>-3.97</td>
<td>4.34</td>
</tr>
<tr>
<td>ERN amplitude at Cz</td>
<td>-1.76</td>
<td>3.85</td>
</tr>
<tr>
<td>Mean ERN amplitude across all channels (FCz, Fz, Cz)</td>
<td>-3.15</td>
<td>3.66</td>
</tr>
</tbody>
</table>

2.4.2 Statistical analysis

All statistical analyses were carried out using the Statistical Package for Social Sciences, Version 15. Descriptive statistics were computed, including means and standard deviations, for each variable considered in the analysis. All of the initial variables were continuous. Bivariate analyses were then performed between the behavioural and neurophysiological measures of interest. The assumptions of the moderator model had to be affirmed in order to perform the main regression analyses in this study. A multiple linear regression model was chosen to test for the moderating effect of ERN amplitude on the relation between maternal depression and children’s behavioural outcomes. Z-scores of all continuous variables were computed before being included in the
analysis. As a final step, the file was split to clearly distinguish the predictive value of maternal depression on internalizing and externalizing behaviours under separate conditions of large and small ERN amplitudes.
Chapter 3

Results

3.1 Preliminary analyses

Children’s internalizing and externalizing scores were obtained from the respective subscales on the CBCL. It is relevant to note that, consistent with other clinical studies, this sample was clinically impaired in terms of internalizing and externalizing problems. The sample mean was above the borderline-clinical cutoff (T = 67) on externalizing (M = 72.12; SD = 6.84), and below this borderline-clinical cutoff for internalizing (M = 63.02; SD = 8.62).

The self-reported BDI measure of maternal depression (M = 13.08; SD = 8.27) revealed that 34% of the sample experienced no or minimal depression (score of 0 – 9), 46% of the sample was mildly to moderately depressed (score of 10 – 18), 6% of mothers showed signs of moderate to severe depression (score of 19 – 29), and a considerable 14% were found to suffer from severe levels of depression (score of 30 – 63).

An outlier analysis was conducted on both the standardized behavioural and neurophysiological measures. Outliers were recoded to values of 2 standard deviations from the mean.

Relations between Maternal depression, children’s self-monitoring (ERN amplitude), and behavioural outcomes

To support the moderation hypothesis, only the effect of the interaction term (predictor x moderator) on the outcome needs to be significant. Although particular consideration must be taken in interpreting the results, both the predictor and moderator may reveal independent associations with the outcome variable without violating the assumptions of the moderator model.
Before the regressions were performed, bivariate correlations were analysed between all of the variables of interest. Bivariate analyses revealed results that were consistent with the primary assumption of the moderator model: both internalizing ($r = .37, p < .01$) and externalizing ($r = .29, p < .05$) were significantly correlated with maternal depression. There was no significant correlation between the outcome measures and the moderator for any of the 8 measures of ERN amplitude included in the regression analyses. A significant correlation was found between maternal depression and only one of the moderator variables of interest ($r = -.29, p < .05$).
Table 4
Bivariate Correlations Between All Behavioural Measures and Neurophysiological Measures of ERN Amplitude

<table>
<thead>
<tr>
<th>Variables</th>
<th>Maternal Depression</th>
<th>Internalizing Scores</th>
<th>Externalizing Scores</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Behavioural Measures</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Depression</td>
<td>1</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Internalizing scores</td>
<td>.37**</td>
<td>1</td>
<td>–</td>
</tr>
<tr>
<td>Externalizing scores</td>
<td>.29*</td>
<td>.48**</td>
<td>1</td>
</tr>
<tr>
<td><strong>Neurophysiological Measures</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Block A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERN amp at FCz</td>
<td>-.19</td>
<td>-.06</td>
<td>.02</td>
</tr>
<tr>
<td>ERN amp at Fz</td>
<td>.01</td>
<td>-.08</td>
<td>.06</td>
</tr>
<tr>
<td>ERN amp at Cz</td>
<td>-.10</td>
<td>-.01</td>
<td>-.01</td>
</tr>
<tr>
<td>Mean ERN amp (FCz, Fz, Cz)</td>
<td>-.10</td>
<td>-.05</td>
<td>.02</td>
</tr>
<tr>
<td>Block C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERN amp at FCz</td>
<td>-.22</td>
<td>-.00</td>
<td>-.03</td>
</tr>
<tr>
<td>ERN amp at Fz</td>
<td>-.29*</td>
<td>-.04</td>
<td>-.01</td>
</tr>
<tr>
<td>ERN amp at Cz</td>
<td>-.03</td>
<td>.06</td>
<td>.09</td>
</tr>
<tr>
<td>Mean ERN amp (FCz, Fz, Cz)</td>
<td>-.21</td>
<td>.00</td>
<td>.02</td>
</tr>
</tbody>
</table>

**Correlation is significant at the 0.01 level (2-tailed)
*Correlation is significant at the 0.05 level (2-tailed)

3.2 Regression analyses

3.2.1 Hierarchical regression analyses

A series of multiple linear regressions was performed to test for the moderating effect of ERN amplitude on the relation between maternal depression and children’s behavioural outcomes. Z-scores of all continuous variables were computed before being included in the analysis. The regression was run using a hierarchical method in a two-step fashion. The first step included maternal depression (predictor) and ERN amplitude (moderator) as the predictors. The interaction
term (maternal depression x ERN amplitude) was entered in the second step. This procedure was repeated for each measure of ERN amplitude in order to test for the predictive value of maternal depression on internalizing (dependent variable) and externalizing (dependent variable) symptoms in children.

The ERN as a moderator of the relation between maternal depression and children’s behaviour problems

After performing a series of multiple hierarchical regression analyses, ERN amplitude in block C, but not in block A, was found to have a significant moderating effect on internalizing behaviours. In block A, none of the four interaction terms (maternal depression x ERN amplitude) were found to have any significant value in predicting internalizing behaviour. The final regression equations were: (1) $F(3, 46) = 2.72, ns$, for mean ERN amplitude across channels, (2) $F(3, 46) = 2.45, ns$, for ERN amplitude at FCz, (3) $F(3, 44) = 3.56, p < .02$ for ERN amplitude at Fz, and (4) $F(3, 46) = 2.48, ns$, for ERN amplitude at Cz. Although the final regression equation was significant when ERN amplitude at Fz was entered into the equation, the contribution of the interaction term was not significant ($R^2$ change = .001, $p = .78$), suggesting that maternal depression accounted for the majority of variance in internalizing behaviour ($R^2 = .138, p < .05$), with no moderator effect.

However, two ERN measures had a significant moderating effect on internalizing in block C. When mean ERN amplitude in block C was tested as a moderator, both maternal depression ($R^2 = .14, p = .03$) and the interaction term (maternal depression x ERN) ($R^2$ change = .07, $p = .04$) significantly predicted internalizing behaviour, $F(3, 46) = 4.27, p = .01$ (Table 5). In block C, ERN amplitude recorded at Fz also yielded significant moderation effects. Maternal depression ($R^2 = .14, p = .03$) and the interaction term (maternal depression x ERN) ($R^2$ change = .24, $p = .02$) significantly predicted internalizing behaviour, $F(3, 46) = 4.95, p < .01$ (Table 6).
Nonsignificant results in block C were found for the moderation models based on (1) ERN amplitude at FCz, \( F(3, 46) = 3.15, p = .03 \) (no significant contribution of interaction term, \( R^2 \) change = .02, \( ns \)) and (2) ERN amplitude at Cz, \( F(3, 46) = ns \). Children’s self-monitoring, defined by two measures of ERN amplitude in block C, thus showed significant moderation of the relation between maternal depression and children’s internalizing behaviour problems.

At this initial stage of analysis, results did not reveal any significant moderation effects on externalizing behaviour in block A or C. Similar to the findings for internalizing, the interaction of ERN amplitude and maternal depression was not found to predict externalizing after any of the moderator variables from block A were entered into the regression: (1) \( F(3, 46) = 1.43, ns \), for mean ERN amplitude across channels, (2) \( F(3, 46) = 1.63, ns \), for ERN amplitude at FCz (3) \( F(3, 44) = .605, ns \), for ERN amplitude at Fz, and (4) \( F(3, 46) = 1.02, ns \), for ERN amplitude at Cz. This was consistent with hypotheses for block A. However, contrary to the hypotheses, both the interaction terms previously found to be significant in predicting internalizing had no value in predicting externalizing behaviour in block C: interaction terms based on ERN amplitude across channels in block C, (1) \( F(3, 46) = 1.92, ns \), and with (2) ERN amplitude at Fz in block C, \( F(3, 46) = 1.04, ns \), revealed no moderating effects on the relation between maternal depression and externalizing problems. Furthermore, interaction terms based on (3) ERN amplitude in block C at FCz, \( F(3, 46) = 1.43, ns \), and (4) at Cz, \( F(3, 46) = 1.89, ns \), also yielded nonsignificant results.
Table 5
Summary of Hierarchical Regression for ERN Amplitude at Fz in Block C on the Relation Between Maternal Depression and Children’s Internalizing Behaviour (N=50).

<table>
<thead>
<tr>
<th>ERN Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Depression</td>
<td>.40</td>
<td>.14</td>
<td>.40**</td>
</tr>
<tr>
<td>ERN amp at Fz</td>
<td>.08</td>
<td>.16</td>
<td>.07</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Depression</td>
<td>.42</td>
<td>.14</td>
<td>.42**</td>
</tr>
<tr>
<td>ERN amp at Fz</td>
<td>.04</td>
<td>.15</td>
<td>.03</td>
</tr>
<tr>
<td>Maternal Depression x ERN amp</td>
<td>-.45</td>
<td>.18</td>
<td>-.32*</td>
</tr>
</tbody>
</table>

*p < .05; **p < .01.

Table 6
Summary of Hierarchical Regression for mean ERN Amplitude in Block C on the Relation Between Maternal Depression and Children’s Internalizing Behaviour (N=50).

<table>
<thead>
<tr>
<th>ERN Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Depression</td>
<td>.40</td>
<td>.14</td>
<td>.40**</td>
</tr>
<tr>
<td>Mean ERN amplitude</td>
<td>.09</td>
<td>.14</td>
<td>.09</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Depression</td>
<td>.38</td>
<td>.14</td>
<td>.38**</td>
</tr>
<tr>
<td>Mean ERN amplitude</td>
<td>.02</td>
<td>.14</td>
<td>.02</td>
</tr>
<tr>
<td>Maternal Depression x mean ERN amp</td>
<td>-.38</td>
<td>.18</td>
<td>-.28*</td>
</tr>
</tbody>
</table>

*p < .05; **p < .01.
3.2.2 Post-hoc analyses: dichotomizing the moderator variable

A moderating relationship is based on the assumption that low and high values of a variable will predict different values of an outcome variable. One way to test this association would be to split the file. Further regression analyses were thus performed by splitting the file and specifically testing the hypothesis that maternal depression predicts internalizing behaviours in children with high self-monitoring (large ERN amplitudes), and externalizing behaviour in children who display low self-monitoring (small ERN amplitudes). Only the mean ERN amplitude across sites in block C (which showed significant results in the initial statistical regressions) was tested at this next stage of analysis. The standardized measure of mean ERN amplitude was recoded into a dichotomous variable. It was dichotomized based on z-scores: (1) ERN z-scores from -2 to 0 represented large ERN amplitudes, and (2) ERN z-scores from 0 to 2 represented small ERN amplitudes. The remaining variables (maternal depression, internalizing, and externalizing) were left unaltered. The file was then split by the dichotomous variable. A one-step regression tested the value of maternal depression alone in predicting internalizing and externalizing behaviours independently for children with small versus large ERNs. Analyses revealed an effect of maternal depression ($R^2 = .37, p < .01$) on internalizing for children with large mean values of ERN amplitude in block C, $F (1, 20) = 11.69, p < .01$) (Table 7, Figure 3). Maternal depression showed no association with internalizing behaviour for smaller mean ERN values.
Table 7
Summary of One-Step Regression Analyses with Maternal Depression as a Predictor of Internalizing Behaviour for Large and Small Values of Mean ERN Amplitude in Block C

<table>
<thead>
<tr>
<th>ERN Variable</th>
<th>R</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean of FCz, Fz, and Cz</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large ERN amplitude (N=22)</td>
<td>.61</td>
<td>.37**</td>
</tr>
<tr>
<td>Small ERN amplitude (N=28)</td>
<td>.13</td>
<td>.02</td>
</tr>
</tbody>
</table>

**p < .01

*Figure 3.* One-step regression of internalizing behaviour in children on maternal depression, by (1) large ERN amplitudes and (2) small ERN amplitudes, as defined by mean ERN amplitude in block C. All values are in z-scores.
Although regression analyses with continuous values of ERN amplitude did not yield any significant results for externalizing, the same procedure, based on a dichotomous recoding of ERN amplitudes, revealed findings consistent with the hypothesis. Among children who showed smaller mean values of ERN amplitude in block C, maternal depression ($R^2 = .22, p = .01$) significantly predicted externalizing behaviours, $F(1, 26) = 7.24, p = .01$ (Table 8, Figure 4), whereas no significant prediction was found when ERN values were large.

Table 8
Summary of One-Step Regression Analyses with Maternal Depression as a Predictor of Externalizing Behaviour for Large and Small Values of Mean ERN Amplitude in Block C

<table>
<thead>
<tr>
<th>ERN Variable</th>
<th>R</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean of FCz, Fz, and Cz</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large ERN amplitude (N=22)</td>
<td>.03</td>
<td>.001</td>
</tr>
<tr>
<td>Small ERN amplitude (N=28)</td>
<td>.47</td>
<td>.22*</td>
</tr>
</tbody>
</table>

* $p < .05$
Figure 4. One-step regression of externalizing behaviour in children on maternal depression, by (1) large ERN amplitudes and (2) small ERN amplitudes, as defined by the mean ERN amplitude in block C. All values are in z-scores.
Discussion

The present study investigated the cortical activity underlying emotional self-regulation in relation to children referred for aggressive behaviour problems who have depressed mothers. Because our sample of clinically referred children was characterized by comorbidity in internalizing and externalizing scores, children’s difficulties in emotion regulation were thought to be characterised by both overregulation and underregulation of negative emotions. Specifically, I predicted that individual differences in neurophysiological measures of self-monitoring would be associated with behavioural outcomes correlated with maternal depression. Variation in the ERN has been linked with individual differences in self-monitoring (e.g., Gehring et al., 2000; Hall et al., 2007; Ladouceur et al., 2007; McDermott et al., 2009; Olvet & Hajcak, 2008), and this ERP component was therefore tested as a moderator of the relation between maternal depression and child behaviour outcomes. Based on previous work associating both maternal depression and ERN amplitudes with internalizing and externalizing symptoms (Downey & Coyne, 1990; Goodman & Gotlib, 1999; Ormel et al., 2005; Silk et al., 2006; Weissman et al., 1997), it was predicted that ERN amplitudes in children would differentially moderate the relation between maternal depression and children’s socioemotional problems.

The results supported the hypothesis that the ERN moderated the relation between maternal depression and internalizing behaviours in children (in block C only), on the basis of regression analyses with continuous ERN variables. However, the continuous ERN measures in interaction with maternal depression did not predict *externalizing* behaviour. Therefore, a categorical variable for ERN amplitude had to be created to capture the relation between the low values of the moderator and high scores on externalizing. It was only after splitting the file by large vs. small ERN amplitudes, that maternal depression was found to predict high internalizing for high
levels of ERN, and high externalizing for low levels of ERN. Moreover, this association was remarkably strong in block C but nonexistent in block A.

Characteristic negative cognitive thought patterns, behaviours and affect of depressed mothers (Goodman & Gotlib, 1999) have been associated with the genesis of both children’s internalizing (e.g., Hall et al., 2008; Silk et al., 2006; Weissman et al., 1997) and externalizing problems (e.g., Blatt-Eisengart, et al., 2009; Hall et al., 2008; Hammen et al., 1990). Additionally, individual variation in ERN amplitude has also been differentially related to these dimensions of psychopathology (for a reviewed see Olvet & Hajcak, 2008). Considering these consistent associations in the literature, it was unexpected to find moderating effects of a continuous measure of ERN amplitude on internalizing, but not externalizing, behaviour. This may suggest that incremental differences in ERN magnitude are more sensitive to differences in internalizing than externalizing symptoms. Furthermore, the relations among these variables appear to be non-linear and may include threshold effects.

However, the effect of maternal depression on externalizing behaviour was revealed under the condition of small ERNs after the file had been split. It is possible that there was a large degree of variability in the ERN and externalizing data as a result of elevated levels of comorbidity in the sample, allowing for only extreme values on both measures to be associated with each other. Hence, it may be that in this sample, only global differences in values of ERN predispose children to experiencing externalizing symptoms when exposed to maternal depression. Nonetheless, splitting the file by large and small ERN values revealed a large amount of variance in both internalizing and externalizing scores, respectively, that was accounted for by maternal depression. It may thus be suggested that children who display higher levels of self-monitoring and have depressed mothers are at risk for developing anxious and/or depressive symptoms.
Conversely, only children who display abnormally low levels of self-monitoring seem to be at a particularly high risk for developing aggressive and antisocial behaviours.

To exemplify these associations, let us imagine a child living with a depressed mother. This child may lack emotional engagement, warmth, positive interactions, and be concurrently exposed to his or her mother’s consistent criticism and coercive parenting (Goodman & Gotlib, 1999; Lovejoy, 2000; Rogosch et al., 2004). As I had previously discussed, a child’s reaction to such an emotionally challenging situation may be driven by his or her tendency to engage in low or high self-monitoring; children who tend to underregulate, as expressed by low self-monitoring, would have difficulty identifying and inhibiting angry feelings, increasing the likelihood of acting out in an impulsive/aggressive manner. Alternatively, for children who display high levels of self-monitoring, the feeling of rejection and emotional disengagement may increase their tendency to ruminate, engage in self-blaming, and make negative self-attributions in response to the negativity expressed by their mothers. As a result, these self-monitoring styles, exacerbated by the high levels of negative emotions experienced in response to maternal depression, may be the underlying mechanisms that increase children’s likelihood of developing internalizing and externalizing problems. Although mostly neurophysiological research has investigated the mechanisms of action-monitoring and self-monitoring, previous work has found that the inability to refocus attention away from distress in children and adolescents is related to rumination, overregulation, and sad mood, all shown to be characteristic of internalizing symptoms (Nolen-Hoeksema, 1994; Silk et al. 2006). In line with the results of the current study, children with small ERN amplitudes who display low levels of self-monitoring are more likely to experience anger, show disregard for their behaviour and develop externalizing problems, whereas children with large ERN amplitudes who show enhanced self-monitoring may experience anxiety, attribute blame for their mother’s negative behaviour to themselves, and develop internalizing
symptoms. As such, maladaptive self-regulatory skills may raise children’s vulnerability for developing socioemotional problems when exposed to maternal depression.

4.1 The moderator model

The moderator model, chosen to test the theoretical framework for this study, has thus been effective in establishing a differential relation between maternal depression and children’s behavioural outcomes. It depicts different conditions, defined by levels of children’s self-monitoring, that allow for maternal depression to be most potent in predicting internalizing and externalizing problems. In this sense, small ERN amplitude triggers one effect of the predictor, whereas large ERN amplitude triggers a completely different effect. In contrast, a test of the ERN’s mediating effects on the relation between maternal depression and behavioural outcomes would not only have required the variables in the model to all be associated, but would also have characterized the ERN as state-related (Baron & Kenny, 1988). In other words, in a significant model of the ERN’s mediating effects, it would have had to be assumed that maternal depression was dependent on the ERN in order to be associated with child behaviour: the stronger the association between maternal depression and the ERN (as well as between the ERN and behavioural outcomes), the weaker the direct association between maternal depression and child behaviour, since the causal effect works through the ERN (Coan & Allen, 2004). Instead, as the moderator model proposes, high levels of trait-like self-monitoring (large ERN amplitude) in the context of maternal depression predispose children for internalizing problems, whereas low levels of self-monitoring predispose children for externalizing problems.
4.2 Block effects and emotion induction

The study was designed to compare ERP results for blocks A and C (two structurally identical blocks) before and after emotion induction. Block A was designed to be relatively easy so that children could earn many points at the beginning of the game, whereas block C began right after children had learned that they lost most, if not all, of their points in block B. It was thought that children’s levels of anxiety would be particularly high at the beginning of block C when points were still low, and that increased vigilance and monitoring of children’s own actions would occur as points were being regained until the end of block C. As expected, significant moderating effects of ERN amplitude were only found in block C (at Fz and as a mean across all sites). Using the same paradigm, most recent (unpublished) work from our lab looked at changes in cardiovascular measures from blocks A to C. Respiratory sinus arrhythmia (RSA), reflecting parasympathetic activity, was found to decrease from blocks A to C in a linear fashion, in normally developing children (Chapman et al., under review). It was believed that an increase in arousal (increase in negative emotionality from block A to C) would be associated with reduced RSA levels. Although it was expected that RSA levels would return to normal by the end of block C when children regained their points, analyses from this work suggest that RSA levels remained low. The authors suspect that along with incomplete recovery of reaction times in block C, the low RSA levels by the end of block C reflect persistent experience of negative emotionality (Chapman et al., under review). Given these results, findings from the current study suggest that ERN amplitude effectively taps processes of emotion regulation since individual differences are only seen once negative emotions have been induced and maintained. Considering that self-monitoring may be particularly enhanced among children high on internalizing, the mechanisms underlying self-monitoring among comorbid populations may come on-line much more readily when the individual is distressed and experiencing an increase in negative emotions (block C). On
the other hand, increased frustration associated with losing points may have also enhanced engagement in the task (greater vigilance and action-monitoring) among children high on externalizing who wanted to regain points and win their desired prize. This may explain to a large extent, why individual differences in self-monitoring were much more potent in block C than in block A.

This study investigated the conjoint effects of children’s individual styles of self-monitoring with the environmental influence of maternal depression on children’s socioemotional problems. However, despite previous studies with adults and children that have associated large and small ERN amplitudes with internalizing (e.g., Gehring et al., 2000; Grundler et al., 2009; Ladouceur et al., 2007; Olvet & Hajcak, 2008) and externalizing (e.g., Dikman & Allen, 2000; Hall et al., 2007; Pailing et al., 2002), respectively, this association was not found in the current study. A high level of comorbidity between internalizing and externalizing symptoms may have partially accounted for the lack of any direct associations between these symptoms and the ERN per se. Since this neurophysiological measure represents self-monitoring specific to each participant, independent of scores on internalizing and externalizing, there may have been a high level of variability in the ERN data among the majority of children. This suggests that ERN amplitude alone may not have been sufficiently sensitive to the tendencies within each of the subscales. However, since the interaction of ERN and maternal depression was found to differentially affect behavioural outcomes, the results suggest that ERN amplitudes tap a self-regulatory mechanism that affects the relation between maternal depression and children’s behavioural problems.

### 4.3 Limitations

The current study has several limitations that need to be addressed. Firstly, it is important to keep in mind that scores on the internalizing and externalizing subscales of the CBCL were based on
Parental reports of children’s emotional and behavioural problems. Because these reports may have been affected by maternal subjectivity, they must be considered with caution. More importantly, many of the mothers in this study experienced moderate to severe levels of depression, which may have additionally contributed to the level of bias with which they reported on their children’s socioemotional functioning (Modell et al., 2001). Furthermore, the history of depression among mothers was not investigated in the present study. It is possible that for children who were exposed to a depressed caregiver from birth, compared to those with only more recent exposure, maternal depression differentially impacted their behavioural outcomes (e.g., Garber et al., 2008; Goodman & Gotlib, 1999; Silk et al., 2006). However, an analysis of this type would have required information regarding the course of depression, which was not gathered for the purposes of this study.

Furthermore, although it has been acknowledged that children’s behaviour can elicit specific parental behaviours (e.g., Feng et al., 2007; Gross et al., 2008), this was not investigated. Higher levels of externalizing and internalizing behaviours may contribute to increased depression in mothers, who then provide less rewarding interactions, warmth, and positive feedback (Blatt-Eisengart et al., 2004). However, the cross-sectional and prospective nature of the study did not allow for such analyses to be conducted or for conclusions to be made with regards to transactional influences between the mother and child over an extended period of time.

Another limitation of this study is the small number of segments comprising the incorrect no-gos for ERN extraction. Averaged waveforms based on a low number of trials are more subject to noise and artifacts, leading to distorted outcomes. Although statistically removing this effect is not a perfect solution, block-specific segment counts can be added into the regression, because they potentially contribute to the amplitude variation. However, in order to minimize the amount
of variables added into the regression, this step was not taken. Finally, independent physiological measures of negative emotion were not recorded for the current study in order to lessen the amount of distress and potential distraction that additional wiring would cause for these aggressive children. Although the physiological data from Chapman et al.’s (under review) work supported the current interpretation of emotion regulation during block C, it is difficult to confidently relate the present findings to their results because they did not look at ERN amplitudes specifically as a measure of self-monitoring. Also, the RSA data collected by that group of researchers were from a sample of normally developing children, whereas clinically referred (and especially comorbid) children may reveal different fluctuations in negative emotionality throughout the task.

4.4 Conclusion

Maternal depression was found to significantly predict internalizing symptoms in children who displayed high self-monitoring, reflected in large ERN amplitudes, and to predict externalizing behaviour problems among children with low levels of self-monitoring, reflected in small ERN amplitudes. This work confirms previous findings that have revealed a relation between maternal depression and children’s psychopathological symptoms, but also goes further to investigate the mechanisms of emotional self-regulation that seem to underlie a differential susceptibility to maternal depression. To my knowledge, this is the first study to assess the neural correlates of self-monitoring in clinically referred children as they moderate the association between maternal depression and children’s psychopathological symptoms.

As an important component of emotion regulation, individual differences in self-monitoring may be relevant to determining tendencies for psychopathological outcomes among clinical populations of children. Early intervention programs teaching effective coping and self-regulatory
strategies among children who seem to exhibit difficulties regulating their emotions may be important to prevent the genesis of psychopathology. Certainly, clinical intervention would need to be tailored to the particular needs of each child in either enhancing or lowering self-monitoring. Finally, future research will benefit from a more in-depth analysis of the neural correlates of self-monitoring as means of detecting early signs of risk for internalizing and/or externalizing problems among children.
References


