Using EEG to examine inhibitory abilities in children: The effects of time pressure, physical activity, and emotion

by

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Abstract

The ability to inhibit inappropriate actions and irrelevant information is crucial for adaptive functioning. Inhibitory skills emerge early in life, with significant growth occurring during the period of early and middle childhood. The objective of this dissertation was to utilize electroencephalography (EEG) to investigate the neural correlates underlying inhibitory abilities in children. In the three different studies that comprise this dissertation, I investigated how inhibitory performance and the neural correlates underlying inhibitory performance were modulated by three different factors.

In the first study, I examined the effect of inducing time pressure on inhibitory performance in children and how this was reflected in the neural correlates. Children's response execution performance was impaired under time pressure, suggesting that the time pressure manipulation may have caused children to adopt a cautious response strategy. Further, the topography of the event-related potentials (ERPs) underlying inhibitory performance, the N2 and the P3, were different in children from that reported in the adult literature.

In the second study, I investigated the association between physical activity and response inhibition in early childhood. Physical activity was not associated with behavioural performance on the GNG task. However, the P3 on No-go trials occurred earlier in children who had greater parent-reported participation in non-organized activities, indicating faster and more efficient evaluation and activation of inhibitory processes. The findings from this study also highlight the importance of considering the types of physical activities that children engage in when examining the effects of physical activity in early childhood.

In the third study, I examined how emotions affect cognitive performance in early and middle childhood, and whether this effect varied as a function of the valence of the emotion and the cognitive demands of the task. The effect of emotions on cognitive performance was

contingent on the valence of the emotion, but this effect did not vary across cognitive control demands in children. Stimuli with negatively valenced, angry emotion elicited a longer N2 latency in children relative to positively valenced, happy emotion and neutral stimuli. The effect of negatively valenced emotion lasted longer in younger children who showed a more pronounced late frontal negativity ERP component.

The findings from all three studies broaden our understanding on how inhibitory performance and the underlying neural correlates are modulated by these three different factors. Across the three studies, the results indicate differences in the neural correlates underlying inhibitory abilities in children. The findings from these studies may potentially be applied in both clinical and educational settings to improve behavioural and learning outcomes in children.

Preface

This dissertation is original work by Siti Aishah Abdul Rahman. Ethics approval was obtained for all three studies included in this dissertation. Ethics approval for the study in Chapter Two was obtained from University of Nebraska-Lincoln Institutional Review Board. Ethics approval for the research in Chapter Three *(Title: Dose-Response Relationships between Physical Activity, Sedentary Behaviour, and Neurocognitive Development among Children of the Early Years, ID: MS6_Pro00045567)* and Chapter Four *(Title: Brain and Cognitive Development in the Transition to School, ID: MS2_Pro00023362)* were obtained from the University of Alberta Research Ethics Board.

Chapter Two was a project done in collaboration with Drs. Daniel Carroll (University of Sheffield) and Kimberly Andrews Espy (University of Nebraska-Lincoln and University of Arizona) under the supervision of Dr. Sandra Wiebe (University of Alberta). Data was collected at the University of Nebraska-Lincoln by Dr. Sandra Wiebe, Dr. Daniel Carroll, Sari Raber, Megan Herrington, and Scott McLean. I processed and analyzed the data, and drafted the manuscript. This chapter has been published in *Developmental Neuropsychology*, 42:5, 336 – 350.

Chapter Three used data from the baseline phase of a longitudinal study, *Physical Activity and Cognition in Early Childhood*, done in collaboration with Dr. Valerie Carson (University of Alberta). Danielle Pertschy, Stephanie Constantin, and I collected the data at the University of Alberta. Luciano Hood, Alice Yan, and Nicholas Kuzik helped with data processing. I analyzed the data and drafted the manuscript.

Chapter Four was a study carried out at the University of Alberta. Jonah Elke, Tyler Harrison, Katherine Maki, and I collected the data. I processed and analyzed the data, and drafted the manuscript. My supervisor, Dr. Sandra Wiebe, and I were involved in the concept formation and manuscript composition of all the chapters included in this dissertation.

Dedication

I would like to dedicate this dissertation to my Mother, Father, Sister, and Brothers.

Thank you for the love, encouragement, and prayers.

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Numerous individuals and organizations helped in making this dissertation a reality. My supervisor, Dr. Sandra Wiebe, played a crucial role at every stage of the project, and I am truly indebted to her for her patience, guidance, and support. My committee members, Drs. Anthony Singhal and Esther Fujiwara were extremely helpful, and I am grateful for the valuable insights and suggestions that they provided. I would also like to thank Drs. Esther Kim and Andrew Simpson for taking the time out of their busy schedules to be a part of my examining committee, as well as Dr. Clayton Dickson for charing the exam. Thanks also to Dr. Valerie Carson and Nicholas Kuzik who assisted with the Physical Activity and Cognition in Early Childhood project. Members of the Alberta Brain and Cognitive Development Lab played an important role in the various processes involved in conducting the research addressed in this dissertation and I am thankful for their assistance. The studies were conducted with generous funding from various organizations. I am grateful to the Neuroscience and Mental Health Institute for the scholarship support they provided me throughout my graduate program. I would also like to thank the Women and Chldren's Health Research Institute, National Sciences and Engineering Research Council, and Policy Wise for Children and Families for generously funding the research studies included in this dissertation.

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CHAPTER 1

Using EEG to examine inhibitory abilities in children: The effects of time pressure, physical activity, and emotion

Goal-oriented behaviour often requires overcoming automatic cognitive processes or well-learned behavioural tendencies. The deliberate adjustment of behaviour and mental processes to meet present goals and task demands is known as cognitive control (Crone & Steinbeis, 2017; Nigg, 2016). One key component of cognitive control is inhibition (Miyake, Friedman, Emerson, Witzki, & Howerten, 2000). Inhibition refers to the ability to withhold responses or suppress irrelevant information (Dempster, 1993). It is a control process that is crucial for overcoming interferences and regulating behaviour. Inhibition that involves overcoming an automatic but inappropriate behaviour is known as response inhibition. Inhibition of interfering, irrelevant information is known as interference suppression.

Both types of inhibitory processes response inhibition and interference suppression are bound together by some commonalities. They are both control processes that involve an intentional overriding of interferences in an attempt to attain a desired outcome. Both processes also employ a prefrontally-mediated neural circuitry (Dempster, 1992; Diamond, 2013), and are impaired in patients with prefrontal lesions (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Stuss, Floden, Alexander, Levine, & Katz, 2003). There are also several differences between the two inhibitory processes. Besides the most obvious difference regarding the nature of the interference presented, interference suppression and response inhibition also differ in the level at which inhibition is observed. Interference suppression involves the control of mental, attentional processes, whereas response inhibition is involves the control of motor behaviour.

Inhibitory abilities follow a protracted trajectory of development, and are still developing in children. Nonetheless, they are crucial in these early developing years and have been associated with intelligence (Dempster, 1991; Duan, Wei, Wang, & Shi, 2010), conscience (Kochanska, Murray, & Coy, 1997), and self-regulatory behaviour (Shoda, Mischel, & Peake, 1990; Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996). The critical function served by inhibitory abilities in enabling adaptive behaviour is perhaps most evident in clinical disorders where inhibitory impairments are a prominent feature. For instance, those with attention deficit hyperactive disorder (ADHD) have been reported to have greater social problems as well as difficulties in education and employment (Barkley, Fischer, Smallish, & Fletcher, 2006).

My objective in this dissertation is to utilize electroencephalography (EEG) to examine how response inhibition and interference suppression performance in children are influenced by three different factors: time pressure, emotion, and physical activity. In this introductory chapter, I will provide a brief overview of (1) the development of response inhibition and interference suppression, (2) the neural networks that underlie these abilities and their development, and (3) the use of electrophysiological methods in examining them in children. I will end by providing a brief summary of the three studies included in this dissertation.

The development of inhibitory processes

Inhibitory abilities emerge early in infancy and improve gradually. At four months of age, infants are able to inhibit automatic saccades to irrelevant information (Johnson, 1995). At about seven months of age, they are able to inhibit reflex grasp reactions (Diamond, 1990). Towards the end of the first year of life, infants are able to inhibit a prepotent tendency to reach straight for an object and instead, reach around the opening at the side (Diamond, 1990). Despite these developments, inhibitory abilities at infancy and toddlerhood are rudimentary, and children are unable to perform well on more complex measures of inhibition. For instance, two-year-olds were unable to inhibit perceptual interference (Gerardi-Caulton, 2000) or performed only at chance level (Rothbart. Ellis, Rueda, & Posner, 2003). The preschool period between the ages of three to five has been consistently identified as a time window during which children experience a steep growth in their inhibitory abilities

(Garon et al., 2008; Wiebe, Sheffield, & Espy, 2012). It is during this period that children begin to perform well on more complex inhibition tasks similar to those that are used with adults (Gerstadt, Hong, & Diamond, 1994; Perner, Lang, & Kloo, 2002; Wiebe et al., 2012). However, the development of inhibition is far from being complete at this stage and continues to develop into adolescence (Luna, 2009; Luna, Garver, Urban, Lazar, & Sweeney, 2004). In laboratory settings, numerous measures have been used to assess inhibitory abilities in children. As response inhibition and interference suppression involve interference being presented at the motor and perceptual levels, they have typically been assessed using different measures. The Go/No-go and Flanker tasks are two measures that have been commonly employed to assess these functions, and they have informed us on the development of response inhibition and interference suppression abilities, respectively. In the following section, I briefly describe the two paradigms, and outline what each has taught us about developmental improvements in inhibition in early childhood.

Go/No-go (GNG) Task: The GNG task is a well-validated measure of response inhibition (Simpson & Riggs, 2006). It has been used across different ages, and has also been adapted into preschool versions in order to make the task engaging for young children (Cragg & Nation, 2008; Simpson & Riggs, 2006; Wiebe et al., 2012). In a typical GNG task, children are required to make a button press response to a frequently occurring stimulus (Go). This dominant response tendency has to be inhibited to a different, infrequently occurring stimulus (No-go). Several studies have reported that children begin to perform well on this task only towards the end of the third year of life (Dowsett & Livesey, 2000; Livesey & Morgan, 1991; Wiebe et al., 2012). Younger three-year-olds, despite being able to verbalize the rules of the task, perform poorly and struggle to inhibit their responses on No-go trials. As they grow older, children perform better on the GNG task, with improvements seen throughout early and middle childhood (Cragg & Nation, 2008; Wiebe et al., 2012). These improvements have been taken to reflect an enhanced capacity to inhibit inappropriate responses, and some have argued that this stems from a more efficient inhibitory process whereby children are able to inhibit their responses at an earlier stage (Cragg & Nation, 2008). Although there is converging evidence to suggest improvements in performance with age, there is less consensus on the age at which performance reaches peak. Some have reported that performance reaches a plateau in adolescence (Levin et al., 2001; Luna et al., 2004), whereas other have found performance to improve into early adulthood (Rubia et al., 2006). The inconsistency in findings could be due to differences in the design of the GNG tasks used in the studies. In addition, peak performance on the GNG task may not necessarily indicate the complete development of response inhibition. It is possible that with more complex measures of response inhibition, performance improvements may be detectable into adulthood (Livesey, 1991; Posner, Rothbart, Sheese, & Voelker, 2014).

Flanker Task: The flanker task has been used extensively to study several cognitive processes, including interference suppression (Eriksen & Eriksen, 1974; Ridderinkhof & van der Molen, 1995; Ridderinkhof, van der Molen, Band, & Bashore, 1997). The task consists of a central target stimulus (e.g. >) presented alongside distractor flankers that are congruent or incongruent to the information carried by the target (e.g. >>> or <><). Responses are based on the central target, and the interference presented by the flankers has to be inhibited. This is especially important on the incongruent trials where the flankers signal an inaccurate response. The greater interference presented on incongruent trials is evinced by the longer response time (RT) that is typically observed on incongruent trials, relative to congruent trials. Like the GNG task, the flanker task has been adapted to child-friendly versions (Rueda et al., 2004). This preschool version of the task has been used successfully with children aged four and above, although difficulties have been noted with three-year-olds (Rueda et al., 2004). With age, children respond faster and are better able to suppress interference from the

flankers. The developmental improvements are suggested to arise from both children's ability to more efficiently suppress interference from the flankers (Cragg, 2016; Pasto & Burack, 1997), and rapidly resolve the competing responses primed by the flankers (Erb, Moher, Song, & Sobel, 2016; Ridderinkhof & van der Molen, 1997). Using a flanker task with arrows, Ridderinkhof and van der Molen (1995, 1997) reported that adult level performance was reached at 10 - 12 years of age. Others, using a preschool version of the task, have reported performance to reach a plateau at the age of seven (Rueda et al., 2004).

Neural network underlying inhibitory processes

Response inhibition and interference suppression engage a distributed neural network comprising multiple brain regions (Figure 1-1). They include the prefrontal cortex (PFC), anterior cingulate cortex (ACC), pre supplementary motor area (preSMA), superior parietal gyrus, and basal ganglia (Aron, 2007; Blasi et al., 2006; Bunge et al, 2002; Casey et al., 2000). Successful inhibition of both motor and perceptual interference involves several discrete stages, each supported by the different brain regions outlined above. For instance, the dorsolateral and inferior frontal cortices within the PFC play a pivotal role in biasing brain activity in a goal-directed manner (Aron, 2007; Casey et al., 1997). The ACC serves an equally critical role by monitoring and detecting competition during task performance. Parietal regions aid in attentional selection and in maintaining stimulus-response associations (Bunge, Hazeltine, Scanlon, Rosen, & Gabrieli, 2002; Casey et al., 2000; Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000). As all of these operations are crucial for both response inhibition and interference suppression, it is not surprising that both inhibitory processes engage a largely overlapping neural network. However, the extent to which the brain regions are recruited for each process has been found to differ. Comparing the level of activity observed in these brain regions during response inhibition and interference suppression, Blasi and colleagues (2006) reported greater activity in the prefrontal and

parietal areas for response inhibition, and greater activity in the ACC for the interference suppression (Blasi et al., 2006). Moreover, while the basal ganglia has been found to be involved in both processes, different basal ganglia nuclei have been implicated in response inhibition and interference suppression. The subthalamic nucleus has been suggested to be vital in applying a global 'brake' for inhibiting responses — as required for response inhibition, whereas the striatum has been suggested to be involved in selectively inhibiting, or delaying a response till a decision is made — as required for interference suppression (Aron, 2011).

The development of response inhibition and interference suppression in children is tied to the maturation of the neural networks underlying them. In children, brain development is ongoing, and the neural network outlined above is still developing. Although the size of the brain changes little after the age of five, the brain continues to display structural and functional changes as it matures (Durston et al., 2006; Reiss, Abrams, Singer, Ross, & Denckla, 1996). The timeline for these changes differs by region, with sensory and motor regions maturing earlier than association regions like the prefrontal and parietal cortices (Gogtay et al., 2004; Sowell et al., 2003), both of which have been implicated in inhibitory processes. The PFC follows a protracted rate of development, and is one of the last brain regions to mature. This pattern is observed in the rate of synaptogenesis (Huttenlocher & Dabholkar, 1997), as well as in changes in both gray and white matter volumes (Giedd et al., 1999; Gogtay et al., 2004). Gray matter volume in the PFC increases steadily in the first few years, peaking in early adolescence and then rapidly declining. In contrast, white matter volume in the PFC increases progressively in early and middle childhood, the pattern continuing till the age of 40 - 50 years before starting to show a decline (Bartzokis et al., 2001; Sowell et al, 2003). White matter volume reflects myelination of axonal tracts that are crucial for rapid communication between distant brain regions. As inhibitory processes rely

on a distributed neural network, the development of these white matter tracts is important for the maturation of inhibitory processes. This is also supported by findings showing both increased white matter volume and increased connectivity between frontal, parietal and striatal regions to be associated with better inhibitory performance (Liston et al., 2006; Silveri et al., 2006).

Notwithstanding the immature inhibitory network, by the age of four, children are able to perform well on inhibitory tasks similar to those used with adults, and which have been adapted for use with children. Electrophysiological and brain imaging findings show that children compensate for the immature inhibitory neural network by recruiting different and more diffuse brain regions (Brown et al. 2005; Bunge et al., 2002; Durston et al., 2006; Lamm, Zelazo, & Lewis, 2006). Casey and colleagues (1997) reported that while both adults and seven- to twelve-year-olds recruited the DLPFC during response inhibition, relative to adults, children displayed a larger volume of activation within this region. In another study, eight- to twelve-year-olds recruited more posterior parietal regions during response inhibition, and greater activation in this region was associated with better response inhibition performance. In contrast, it was activity in the prefrontal regions that was associated with better response inhibition performance in adults (Bunge et al., 2002). These patterns of findings indicate that the parietal regions of the inhibitory network mature earlier than the anterior, frontal regions, and that children rely more heavily on posterior brain regions to support response inhibition. This is also consistent with observations of a posterior to anterior pattern of brain development (Reiss et al., 1996; Shaw et al., 2008).

Similar developmental differences in the engagement of brain regions are also noted during interference suppression. In one study, children displayed a left-lateralized pattern of brain activation to support interference suppression, whereas adults recruited regions that were mostly right-lateralized (Bunge et al., 2002). In another study, both seven- to elevenyear-olds and adolescents displayed reduced activation in prefrontal regions relative to adults during interference suppression. However, only seven- to eleven-year-olds, but not adolescents, displayed reduced activity in the parietal regions relative to adults (Adleman et al., 2002). This parallels the findings seen with response inhibition, and indicates that parietal regions underlying interference suppression mature earlier than prefrontal regions.

Using electrophysiological method to study inhibitory processes

Brain imaging studies have helped to elucidate how children recruit neural networks that are different and more diffuse than the mature, focal pattern of brain activation observed in adults (Adleman et al., 2002; Brown et al. 2005; Bunge et al., 2002; Durston et al., 2006). Electrophysiological studies complement these finding, providing additional insights into the development of the neural networks underlying inhibitory processes. EEG is a tool that allows us to measure tiny fluctuations in electrical current that is primarily generated by a population of pyramidal neurons in the cerebral cortex (Nunez & Srinivasan, 2006). The scalp-recorded voltage reflects a summation of synchronized excitatory and inhibitory postsynaptic potentials. EEG provides a window to non-invasively examine the neural correlates underlying cognitive processes like inhibition. Relative to the other methods that are also utilized in the study of cognitive neuroscience, EEG offers several advantages. For instance, EEG offers superior temporal resolution on a time scale of milliseconds. As cognitive processes occur rapidly, this property of EEG allows for the capturing of the various stages of a cognitive process with little loss of information. In addition, EEG places less restriction on movements relative to brain imaging methods. This characteristic is especially relevant when the study population concerns children who are often not as compliant with instructions and are more prone to making movements during data acquisition.

The EEG data collected while children complete the cognitive tasks must undergo several stages of processing to remove 'noise' or signals that do not reflect brain activity. The processed EEG data can be analyzed in several ways, and one common method is to analyze event-related potentials (ERPs). ERPs are the peaks and troughs that are observed on an averaged EEG waveform, and they occur in relation to the occurrence of an event, such as the onset of a stimulus or response (Luck & Kappenman, 2011). ERPs inform us about the neural mechanisms involved in a cognitive processes in several ways. First, the amplitude of the ERP reflects the amount of synchronized neuronal activity observed in relation to an event. Larger amplitudes indicate greater synchronization and recruitment of neural resources. Second, the latency or the time where a peak/trough is seen informs us about the speed at which the cognitive process associated with the ERP occurs. Lastly, the topography of the ERP can provide some information about the cortical generators of the ERP, although caution has to be exercised with this inference due to the volume conduction property of ERPs. ERPs are typically described in terms of their polarity, latency (or ordinal position), topography, and functional significance (Männel, 2008). For example, the term, 'inhibition-related N2' signifies that it is a second negative peak observed on a waveform in relation to an event that elicits inhibitory processes.

There are two ERP components that have been consistently associated with inhibitory processes. The N2 is a negative peak that occurs about 200 - 300 ms after stimulus onset and is observed in the frontocentral regions of the brain (Jodo & Kayama, 1992, Eimer, 1993; Fallgatter & Strik, 1999). The P3 is a positive peak that is seen about 300 – 600 ms after stimulus onset in the frontal or parietal regions of the brain (Eimer, 1993; Kopp et al., 1996). Under conditions requiring inhibition, the amplitude of the N2 and P3 are enhanced, and this is taken to reflect the greater cognitive resources required for successful inhibition.

Numerous studies have examined the inhibition-related N2 and P3 in adults under varying contexts. Fewer have examined them in children (Ciesielski, Harris, & Cofer, 2004; Jonkman et al., 2006; Johnstone et al., 2005), especially in early childhood (Buss, Dennis, Brooker, Sippel, 2011; Lahat, Todd, Mahy, Lau, & Zelazo, 2010). In the few studies that have, as in adults, the N2 and P3 amplitude are found to be enhanced under conditions requiring inhibition (Buss et al., 2011; Johnstone et al., 2005; Lewis et al., 2007; Rueda et al., 2004). However, the N2 and P3 that are observed in children are found to differ from those in adults in several other ways. The amplitude of both ERPs are greater in children than in adults (Ciesielski et al., 2004; Jonkman et al., 2006). Also, the latencies of the ERPs are longer in children than in adults (Johnstone et al., 2005; Lamm et al., 2006). Over the course of development, both the amplitude and latency decrease (Hoyniak, 2017), signalling increased efficiency in the recruitment of neural resources to carry out inhibition. These changes in the amplitude and latency of the N2 and the P3 are also accompanied by improvements in inhibitory performance. The developmental progressions seen in both the ERPs and cognitive performance have been attributed to cellular changes such as synaptic pruning and axonal myelination of neurons that occur with age (Huttenlocher, 1990). Synaptic pruning involves the elimination of redundant neuronal connections and the strengthening of frequently used connections. The fine-tuning of neuronal connections allows for more efficient recruitment of neural resources during cognitive processing. Myelination of axonal tracts enables neuronal signals to be transmitted faster, which in turn allows for rapid information and cognitive processing (Tasaki, 1939).

In addition to changes in the amplitude and latency, changes have also been noted in the topography of the N2 and P3 observed in children. Specifically, in adults, the enhancement of the N2 and P3 amplitude that occurs under conditions requiring inhibition are seen in the frontal electrode regions (Jodo & Kayama, 1992; Eimer, 1993; Kopp et al., 1996). In contrast, several studies in children have reported this N2/P3 enhancement in more posterior electrode regions (Ciesielski et al., 2004; Johnstone et al., 2007; Jonkman et al., 2007). Laterality differences have also been reported for the N2 component (Chevalier, Kelsey, Wiebe, & Espy, 2014; Lamm et al., 2006). In these studies, children displayed a leftlateralized negativity under inhibitory requirements, a pattern that is different from that observed in adults where the N2 is frequently seen in midline electrodes (Jodo & Kayama, 1992; Eimer, 1993; Kopp et al., 1996).

The differences in the characteristics of these two ERPs in children — especially in amplitude and topography — parallel brain imaging findings where children are seen to recruit slightly different and more diffuse brain regions to support inhibitory performance relative to adults (Brown et al. 2005; Bunge et al., 2002; Durston et al., 2006; Lamm, Zelazo, & Lewis, 2006). More specifically, the greater ERP amplitudes recorded in children indexing greater synchronized neuronal activation are consistent with the diffuse brain activation patterns seen in imaging studies. Similarly, the topographical differences observed in the N2 and P3 in children align well with the posterior brain activation patterns reported in imaging studies. The lack of correspondence between ERP latency findings and findings from brain imaging studies is likely due to the limited temporal resolution of brain imaging methods that are dependent of haemodynamic responses. Nonetheless, these differences observed in the neural correlates underlying inhibitory processes in children are in accordance with the observations that their behavioural performance on inhibitory tasks undergoes continued development in childhood (Garon et al., 2008; Wiebe, et al., 2012). Taken together, the existing literature informs us that although children are able to perform well on inhibitory tasks and that their neural system is able to support performance, these are not yet at the optimal levels. It is only with development that they attain the level of efficiency seen in adults.

Scope of this Dissertation

In this introductory chapter, I have outlined the development of response inhibition and interference suppression, and the development of the neural correlates supporting these processes. As reviewed above, early childhood is an important milestone for the development of inhibitory processes, and children's performance on tasks assessing these abilities improves with age. Because inhibitory processes are immature and developing rapidly at this stage, it is important to understand the factors that can influence their performance. The studies in this dissertation address how three different factors influence inhibitory performance, and how they modulate the ERPs underlying inhibitory performance in children. In the three different studies that comprise my dissertation, I explored how factors on three different levels — (1) time pressure: an attribute that is intrinsic to inhibition, (2) physical activity: a participant-related attribute, (3) emotion: as a stimulus-related attribute modulated inhibitory performance and the underlying neural correlates. Below, I provide a brief overview of the issues investigated in each study.

Study One: The neural correlates underlying response inhibition in children: Evidence from a Go/No-go task

In Study One, I examined how inducing time pressure influenced response inhibition performance in young children and the underlying neural correlates. Two groups (Fast, Slow) of five-year-olds completed a GNG task while scalp EEG was recorded. Children from the Fast group were given less time to respond (750 ms) relative to the children from the Slow group (1500 ms). The motivation for examining time pressure as a factor was based off a prominent model of response inhibition, the horse race model, which depicts the process of response inhibition as a race against response execution (Logan & Cowan, 1984). In this conceptualization of response inhibition, time/speed is a crucial factor in determining the success of response inhibition. By inducing time pressure, the objective was to speed up responding in the 'fast' group, making responding a prepotent behaviour, and to examine how the neural correlates underlying inhibition were modulated to enable successful inhibition under this condition. Previous findings in children have shown that inducing time pressure increases inhibitory demands of the task as indicated by reduced performance accuracy (Cragg & Nation, 2008; Simpson & Riggs, 2006). However, these studies did not examine how the neural correlates were modulated by time pressure. In adults, a similar time pressure manipulation was found to increase the amplitude of the N2 ERP component, suggesting that greater neural resources were required to support performance under the increased inhibitory demands (Jodo & Kayama, 1992). Thus, the goal in this study was to understand how behavioural and neural indices of response inhibition were modulated under time pressure in early childhood.

Study Two: The association between physical activity and response inhibition in early childhood

In Study Two, I examined how individual differences in physical activity levels in early childhood were associated with response inhibition performance and the neural correlates. Physical activity has been identified as a lifestyle factor that enhances cognitive performance in both adulthood (Kramer & Erickson, 2007; Hillman, Belopolsky, Snook, Kramer, McAuley, 2004) and middle childhood (Chaddock-Heyman et al., 2014; Hillman et al., 2014). Importantly, these findings also show that the enhancement seen in behavioural performance is accompanied by changes to the indices of neurocognitive functioning, including ERPs (Hillman et al., 2004; 2014) and blood oxygenation level-dependent responses (Chaddock-Heyman et al., 2014; Voss et al., 2011). As discussed previously, early childhood is a critical period for the development of inhibitory abilities (Garon et al., 2008; Wiebe, et al., 2012). Moreover, it is also a time when the brain is developing rapidly (Lenroot & Giedd, 2006). A better understanding of how physical activity in this crucial time period is associated with inhibitory performance can potentially be utilized as a tool for early intervention. This research question was addressed as part of larger cohort study investigating the effects of physical on cognitive functioning in early childhood. In this dissertation, to fit with the overall topic on inhibitory processes, the discussion is limited to examining the association between physical activity and response inhibition. Two- to four-year-olds completed the GNG task while their scalp EEG was recorded. Objective measures of physical activity were obtained using accelerometry, and subjective parent-report measures were obtained through a questionnaire. The objective was to investigate if physical activity levels predicted response inhibition performance and the underlying neural correlates in early childhood.

Study Three: Valence matters: An electrophysiological study on how emotions influence cognitive performance in children

Study Three was an investigation of how positive (happy) and negative (angry) emotions influenced cognitive performance in children under varying cognitive control demands. In this study, I used interference suppression as a means of varying the cognitive control demands of the task. Findings in adults show that the effect that emotions have on cognitive performance may be attenuated by increasing the cognitive control demands of the task (Lim, Padmala, & Pessoa, 2008; Cohen, Henik, & Mor, 2011). In children, while there is some evidence to indicate that the valence of emotional stimuli may determine the effect that it exerts on cognitive performance (Augusti, Torheim, & Melinder, 2012; Lewis et al., 2007), there is a paucity of research examining how cognitive control demands might modulate this. A group of four- to eight-year-olds completed a colour flanker task while their scalp EEG was recorded. The task required children to indicate the colour of the border (red or blue) around a central target face displaying a neutral, happy, or angry expression. The target face was flanked by two neutral distractor faces. On incongruent trials, the colour of the border around the flankers differed from that around the target. The interference presented by the incongruent flankers was intended to increase the cognitive control demands required on the incongruent trials. The objective of this study was three-folds: First, to examine if the effect of emotions on children's cognitive performance differed by valence; second, to examine if this effect varied with the cognitive control demands of the task; and lastly, to examine if these effects differed across early and middle childhood.

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Figure 1-1: Schematic representation of the neural network underlying inhibitory processes.

DLPFC: Dorsolateral prefrontal cortex; ACC: Anterior cingulate cortex; IFC: Inferior frontal cortex; BG: Basal ganglia; STN: Subthalamic nucleus; PreSMA: Pre supplementary motor area; M1: Primary motor cortex.

Figure is based on information sourced from: Aron, 2011; Blasi et al., 2000; Bunge et al., 2002; Casey et al., 2000; Hazeltine et al., 2003

CHAPTER 2

Neural correlates of response inhibition in early childhood: Evidence from a Go/No-go task

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Imagine that you have just stepped out of your house and see a child running after a ball that had gone to the road. Right then, you notice a car coming, and you instinctively call out to the child to stop. Will the child be able to comply with your instructions? This scenario is just one example of the adaptive importance of response inhibition, or the process of stopping an action that has been initiated. This ability emerges and undergoes rapid growth in early childhood, particularly between three to six years (Carver, Livesy, & Charles, 2001; Garon, Bryson, & Smith, 2008; Wiebe, Sheffield, & Espy, 2012), and continues to mature into early adulthood (Band, van der Molen, Overtoom, & Verbaten, 2000). There is a parallel, protracted developmental trajectory of the prefrontal brain regions implicated in higher order cognitive functions (Fuster, 2002). Within the prefrontal cortex, the anterior cingulate, dorsolateral, ventrolateral, inferior frontal and medial prefrontal cortices have been identified as a part of the neural network underlying response inhibition (Casey et al., 1997; Rubia et al., 2001; Tamm, Menon, & Reiss, 2002). The immaturity of these regions in children may underlie the difficulties they face in inhibiting responses. The goal of the present study was to examine response inhibition and its neural correlates in young children, while manipulating task demands expected to affect inhibitory load. Numerous factors have been found to increase inhibitory load - for example, working memory load (Wijeakumar et al., 2015), level of interference (Ciesielski, Harris, & Cofer, 2004), and preceding context (Durston, Thomas, Yang, Ulug, Zimmerman, & Casey, 2002). In this study, we choose to focus on one factor, time pressure (Cragg & Nation, 2008; Jodo & Kayama, 1992; Simpson & Riggs, 2006).

Response inhibition and Time pressure

One prominent perspective on response inhibition is the horse race model (Logan & Cowan, 1984), which conceptualizes it as a race between a 'go' process, initiating the response, and a 'stop' process, inhibiting it. Successful inhibition requires that the 'stop' process be completed in time to interrupt the ongoing 'go' process. Failed inhibitions occur when the 'stop' process is too slow and the 'go' process is completed first. Thus, the timing of these two processes is crucial in determining whether inhibition is successful. Increasing the speed of the 'go' process requires a corresponding increase in the speed of the stop process, and should therefore increase inhibitory load; several previous studies support this suggestion (Cragg & Nation, 2008; Simpson & Riggs, 2006). However, while faster response initiation can be directly observed in response times, faster stopping cannot be directly observed. Consequently, behavioural studies of response inhibition have relied primarily on failed inhibitions to inform us about inhibitory processing. This limitation can be surmounted by neuroimaging methods such as event-related potentials (ERPs), as brain activity is ongoing and can be measured in the absence of observable behaviour. Jodo and Kayama (1992) demonstrated the utility of this approach in adults, showing that when the time window allowed for a response was decreased, response initiation was speeded up, and the increased demands were reflected in ERP characteristics.

Two studies (Cragg & Nation, 2008; Simpson & Riggs, 2006) have examined the effects of time pressure on response inhibition in children behaviourally. Both studies used variants of the Go/No-go (GNG) paradigm, commonly used to assess response inhibition in children (e.g., Durston et al., 2002; Wiebe et al., 2012). In a typical GNG task, participants respond to a frequently occurring stimulus type (Go trials), and withhold responding to a less frequently occurring stimulus type (No-go trials). Because the majority of trials require a response, the task induces a prepotent tendency to respond, and consequently, No-go trials require inhibitory control. The extent to which individuals are able to inhibit responding on No-go trials serves as a measure of their inhibitory abilities. Cragg and Nation (2008) found that when a shorter time window was allowed for a response, task accuracy decreased in both 5 - 7-year-old and 9 - 11-year-old children, suggesting that the response inhibition demands of the task increased with time pressure. Similarly, in a study of 3-year-olds, Simpson and Riggs (2006) found time pressure increased inhibitory demands of the task. However, in their study, this was true only to a certain extent. When the time window was too short for children to respond, Go accuracy plummeted and No-go accuracy no longer correlated with another inhibition measure. Findings from both of these studies suggest that manipulating time pressure should affect response initiation and/or inhibitory demands in children, as it does in adults.

The neural correlates underlying response inhibition

Although numerous studies have documented young children's behavioural performance on measures of inhibition (Cragg & Nation, 2008; Simpson & Riggs, 2006; Wiebe et al., 2012), only a few studies have examined the neural correlates underlying their performance. Cognitive processes like response inhibition occur on a millisecond time scale, and electroencephalography (EEG) is one of the few neuroimaging methods with the necessary temporal resolution to investigate the neural underpinnings of these processes. Two ERP components, the N2 and P3, have been consistently identified as markers of inhibitory processes: The N2 is a negative peak observed at frontal electrode sites between 200 and 500 ms after stimulus onset (Carter & Van Veen, 2007; Falkenstein, Hoormann, & Hohnsbein, 1999; Jonkman, Sniedt, & Kremner, 2007); the P3 is a positive peak observed at frontocentral electrode sites between 300 and 600 ms after stimulus onset (Bokura, Yamaguchi, & Kobayashi, 2001; Eimer, 1993; Kopp, Mattler, Goertz, & Rist, 1996). In GNG paradigms, the amplitude and latency of these two ERP components differ between No-go trials, where inhibition is required, and Go trials, where it is not (Bokura et al., 2001; Falkenstein et al., 1999; Fallgatter & Strik, 1999).

A few studies with young children have utilized EEG with the GNG task. However, none of these studies have addressed how manipulating time pressure would modulate the neural correlates of response inhibition. In one study that did examine the role of timing, the investigation was limited to how the timing of action decision and response initiation influenced response inhibition in 5- year-old children (Chevalier, Kelsey, Wiebe, & Espy, 2014). In that study, Chevalier and colleagues used a modified GNG task that allowed children to fully or partially inhibit their responses, and they found that relative to partial inhibitions, successful inhibitions were marked by an earlier onset of a late negative slow wave thought to represent action decision. Another study examined cross-cultural differences between European- and Chinese-Canadian 5-year-olds. They found no behavioural differences, but Chinese-Canadian children showed a more pronounced N2 component (Lahat, Todd, Mahy, Lau, & Zelazo, 2010). Davis, Bruce, Snyder, and Nelson (2003) found that while adults displayed the enhanced P3 on No-go trials, 6-year-old children did not. Instead, a late positive peak at frontal electrodes distinguished No-go trials from Go trials. Two studies have examined response inhibition in GNG tasks that also required emotion regulation. In a study with both children and adolescents, the N2 and P3 on No-go trials were greater during conditions of negative emotion induction. (Lewis, Lamm, Stieben, Segalowitz, & Zelazo, 2006). Adopting a similar paradigm, another study with 5-6 year-olds reported that better response inhibition performance during negative emotional induction was accompanied by greater EEG power in the theta frequency range,

though no differences in the N2 amplitude were seen (Farbiash & Berger, 2015). Several other studies have also incorporated EEG with the GNG paradigm to examine brain activity related to error detection and monitoring in 5 to 7-year-old children (Torpey, Hajcak, Kim, Kujawa, & Klein, 2012; Torpey, Hajcak, & Klein, 2009). These studies, however, did not look at the neural correlates associated with response inhibition in early childhood.

More research using ERP to study response inhibition has been conducted in middle childhood. Most of these studies have found that ERPs modulated by response inhibition demands were more evident at posterior electrode sites (Brydges, Anderson, Reid, & Fox, 2013; Ciesielski et al., 2004; Durston et al., 2002; Jonkman et al., 2007). Jonkman, Lansbergen, and Stuader (2003) directly compared 9-year-old children and adults, and found that the No-go P3 was maximal at posterior electrode sites in children, but frontally maximal in adults. Furthermore, several studies have found laterality differences in brain activity during response inhibition between children and adults. While response inhibition in adults is typically associated with greater activity in the right hemisphere (Aron, Robbins, & Poldrack, 2004), several studies have found that children display greater activity in the left hemisphere (Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Johnstone, Pleffer, Barry, Clarke, & Smith, 2005; Lamm, Zelazo, & Davis, 2006). In addition to the studies highlighted above, Hoyniak (2017) recently performed a meta-analysis of 65 studies that have utilized the GNG task to assess inhibition in children aged 2 to 12 years old. This meta-analysis found that the N2 was larger on No-go trials than on Go trials, bolstering its position as a neural marker of response inhibition. Furthermore, the N2 decreased in both amplitude and latency with age. However, of these studies, only five involved children in early childhood, and none examined how time pressure influenced response inhibition performance or its neural correlates.

Study goals and hypotheses

In the present study, our main goal was to examine the neural correlates of response inhibition in early childhood, and to do so under varying inhibitory demands. We used the preschool GNG paradigm, because several previous studies have manipulated the inhibitory demands of this task by inducing time pressure (Cragg & Nation, 2008; Jodo & Kayama, 1992; Simpson & Riggs, 2006). In the GNG task, response inhibition is indexed by children's ability to withhold responding on No-go trials. We chose to focus on 5-year-olds because many studies have found that children at this age are capable of performing well on GNG tasks (Chevalier et al., 2014; Wiebe et al, 2012), and in ERP studies, it is important that children complete sufficient correct trials to generate stable ERP averages. Additionally, this age captures an important phase transition to formal schooling where there is an increased need for children to inhibit inappropriate responses (Blair & Razza, 2007; Lahat et al., 2010). It is, therefore, important for us to gain insights into factors that may influence response inhibition performance in children at this age. Because of the number of trials required in ERP studies, young children's limited attention span, and to minimize the influence of practice effects and fatigue on children's performance, we chose to implement the time pressure manipulation in a between-subjects design. Children were randomly assigned to the Fast or Slow condition, and completed the GNG task with shorter or longer time windows in which they could respond. We hypothesized that children in the Fast condition would experience increased inhibitory demands induced by time pressure and that this would be reflected in their performance and their brain activity, particularly the N2 and P3 components.

Method

Participants

The sample included 31 children (15 boys, 16 girls) between 5;0 and 5;11 years (M = 5;8 years, SD = 3 months). Data from eleven additional children were excluded due to poor task performance (n = 6) and/or excessive EEG movement or eye-blink artifact (n = 5). Children were randomly assigned to either the Fast or Slow GNG conditions. There were 15 children (7 boys, 8 girls) between 5:0 and 5:11 years (M = 5:8 years, SD = 3 months) in the Slow condition and 16 children (8 boys, 8 girls) between 5;3 and 5;11 years (M = 5;9 years, SD = 2 months) in the Fast condition. Participants were recruited from a small city in the Midwestern United States through local businesses that served families with young children (e.g., preschools, health offices, pediatricians) and by word of mouth. To be included in the study, children had to be born fullterm and have no history of neurological or behavioral disorders. The ethnic composition of the children in the two conditions was similar: the Slow condition included 11 European-American, 1 African-American and 3 mixed ethnicity children. The Fast condition included 12 European-American, 1 African-American, 1 Asian-American and 2 mixed ethnicity children. Parentreported health insurance status was used as a proxy for socioeconomic status (SES). Most participants (71%; 11 in each condition) were from middle or upper-middle SES, with private health insurance. Fewer participants (29%; 4 in the Slow condition, 5 in the Fast condition) were from low SES and eligible for public health insurance.

Procedure

The study was carried out at a child development laboratory at a university in the Midwestern United States. Parents accompanied their children to the lab and were briefed about the study before providing informed consent. After EEG net application, children completed two tasks, with the GNG task administered second. The first task was a measure of set-shifting that took approximately 10 minutes. It was unrelated to the present investigation and was the same for all children. Parents remained in the testing room throughout the session.

Response Inhibition Task. The Preschool GNG task (adapted from Wiebe et al., 2012) was a computerized fishing game (Figure 2-1), presented using E-Prime 2.0 Professional (Psychology Software Tools, Sharpsburg, PA). Children were instructed to respond by pressing a single button on a button box whenever a fish appeared (Go trials) and withhold responding when a shark appeared (No-go trials)¹. Each trial began with the onset of a stimulus that remained on the screen for a maximum of 750 ms (Fast) or 1500 ms (Slow), and disappeared when the child responded. On correct Go trials, a net appeared over the fish with a 'bubbling' sound to indicate that the fish had been caught. On incorrect No-go trials, a picture of a broken fishing net appeared over the shark with a 'buzzer' sound to indicate the shark had broken through the net. The feedback lasted for 1000 ms. No feedback was given on trials when the child did not press the button. Each trial was followed by an inter-stimulus interval of 1000 ms.

At the beginning of the task, children were shown a picture of all the stimuli and asked to point out the fish and sharks to ensure they could distinguish between Go and No-go stimuli.

¹ To make the rules of the task easy for children to understand, two different categories of stimuli (fish, sharks) were used on Go and No-go trials. However, this may have introduced differences between the Go and No-go trials, for example in stimulus salience. To investigate whether the fish and shark stimuli differed in salience, we administered a target detection task to an adult sample (n = 6). This study identified two stimuli as outliers: one fish was .53 standard deviations above the mean in salience and one shark was .52 standard deviations below the mean. We reanalyzed the behavioral data using repeated measures ANOVA excluding these two stimuli. For accuracy, there was a main effect of speed condition (F(1, 29) = 12.33, p < .01, $\eta^2 = .298$) and an interaction between speed condition and trial type (F(1, 29) = 15.22, p < .01, $\eta^2 = .344$). For RT, there were main effects of speed condition (F(1, 29) = 68.0, p < .001, $\eta^2 = .701$) and trial type (F(1, 29) = 1381.48, p < .001, $\eta^2 = .979$). As the pattern of findings did not differ from those including the complete stimulus set, the latter are reported in the Results section.

Next, children completed a block of 13 practice trials and then proceeded with the test trials. There were a total of 200 (Fast) or 160 (Slow) test trials² with 75% Go trials (Fast: 150 trials, Slow: 120 trials) and 25% No-go trials (Fast: 50 trials, Slow: 40 trials). Following the prototypical design of the GNG task (Cragg & Nation, 2008; Durston et al., 2002; Lahat et al., 2010), Go trials were probabilistically dominant to increase children's bias to respond, thereby maximizing response inhibition requirements on No-go trials. Children in the Fast condition completed a greater number of trials to ensure there was sufficient EEG data on correct trials to permit ERP analyses, as we expected children to make more errors in the speeded condition.

All stimuli measured 10 by 13 cm and were presented at the centre of the screen on a 20inch DELL desktop monitor. In total, there were 10 different Go stimuli and 3 different No-go stimuli and each stimulus was presented with roughly equal frequency across the task (10 -12%). As there were more Go trials (75%) than No-go trials (25%), it was necessary to have a greater variety of Go stimuli to ensure each stimulus was presented at equal frequency across the task.

Electrophysiological Recording and Data Processing. EEG was recorded using a 128channel Hydrocel Geodesic Sensor Net (Electrical Geodesics Inc., Eugene, OR). First, children's head circumference was measured to allow selection of the appropriate net, and the vertex was

² Because we expected children in the Fast condition to make more errors, we had them complete a greater number of trials. To test whether this difference in procedure affected study findings, we analyzed the behavioural data using repeated measures ANOVA excluding the last block of trials from children in the Fast condition so that both conditions contributed an equal number of trials. For accuracy, there was a main effect of speed condition (F(1,29) = 17.25, p < .001, η^2 = .373) and an interaction between speed condition and trial type (F(1, 29) = 13.53, p < .01, η^2 = .318). For RT, there were main effects of speed condition (F(1, 29) = 17.94, p < .001, η^2 = .382) and trial type (F(1, 29) = 33.59, p < .001, $\eta^2 = .537$). As the pattern of findings did not differ from those including the complete stimulus set, the latter are reported in the Results section.

marked to ensure correct net placement. The net was soaked in an electrolyte solution prior to application. Electrode impedances were measured and maintained below 50 k Ω . If needed, additional electrolyte solution was applied to electrodes with high impedances. During task performance, EEG data was recorded continuously at a sampling rate of 250 Hz, with a 0.1 – 100Hz bandpass filter, referenced to the vertex.

ERP analysis was conducted with Net Station software (Version 4.3.1, Electrical Geodesics Inc., Eugene, OR). A 0.3-30 Hz bandpass filter was used to filter data offline. EEG data was segmented into 1300 ms epochs beginning 100 ms before stimulus onset and ending 1200 ms post-stimulus. Artifact detection was performed on segmented files and all electrodes where signal fluctuations exceeded 200 µV were marked bad. Segments with more than 12 bad electrodes were rejected. Spline interpolation was used to replace bad electrodes in otherwise acceptable segments. EOG correction was conducted using Gratton's algorithm (Gratton, Coles, & Donchin, 1983). In addition, the first author visually inspected all segments for EOG or movement artifacts, and corrected segment and channel markups if needed. As recommended by the Net Station data processing manual, following EOG correction, channels were reassessed for artifacts with previously marked information overwritten if necessary, and then, bad electrodes were replaced using spline interpolation. All usable segments were averaged, separately for Go and No-go trial types. EEG data was re-referenced to an average reference using the polar average reference effect correction (Junghöfer, Elbert, Tucker, & Braun, 1999) and then, baseline-corrected using the data 100 ms prior to stimulus onset. Incorrect trials and trials with a RT less than 200 ms were excluded from analysis as they do not reflect deliberate behaviour (Haith, Hazan, & Goodman, 1988). All ERP data included in the analysis had a minimum of 10 artifact-free trials in each trial type (Go, No-go). In the Slow condition, the number of Go and

No-go trials included in data analysis ranged from 27 - 62 (Total = 120 trials) and 15 - 37 (Total = 40 trials) respectively. In the Fast condition, the number of Go and No-go trials included ranged from 33 - 87 (Total = 200 trials) and 14 - 42 (Total = 50 trials) respectively.

Then, mean amplitude and peak latency measures were calculated for the inhibitionrelated ERP components, N2 and P3. Because visual inspection of the waveforms suggested that the P1 component at parietal electrodes differed between the Fast and Slow conditions, mean amplitude and peak latency of the P1 were also calculated. Mean amplitude was defined as the average amplitude of the waveform within the time window selected for each ERP component. Peak latency was defined as the time taken from the onset of the stimulus to the maximum peak of the ERP component within the time window selected. N2 was examined at the frontal, frontocentral, and central electrode sites and was defined as the negative deflection in the time window between 260 and 560 ms. P1 and P3 were examined at parietal electrode sites. P1 was defined as a positive deflection between 60 and 150 ms, and P3 was defined as the positive deflection between 310 and 610 ms. The time windows were determined after considering previous studies with similar age groups (Ciesielski et al., 2004; Davis et al., 2003; Lahat et al., 2010; Jonkman et al., 2003) and visual examination of the grand averaged and individual waveforms. Peak latency and mean amplitude measures were averaged across electrodes within clusters selected to be compatible with the 10-20 electrode placement system. The electrodes included in each cluster are presented in Figure 2-2. Because visual inspection of waveforms showed that the N2 component was more pronounced laterally than in midline regions, electrode clusters were defined within Left, Midline and Right locations.

Data Analysis

All analyses were conducted using SPSS Version 21.0 (IBM Corp., Armonk, NY). Behavioral and ERP measures were analyzed using general linear models analysis of variance (ANOVA), to account for the unequal sample sizes in the Fast and Slow conditions. Greenhouse-Geisser correction was applied in certain cases where sphericity assumptions were violated. Means are reported as least-squares means, and partial eta-squared (η^2) is reported as a measure of effect size for all statistically significant findings.

Analyses of behavioural measures, accuracy and reaction time (RT) were conducted for speed condition (Fast, Slow) and trial type (Go, No-go). Accuracy was calculated as the number of correct trials as a proportion of the total number of Go or No-go trials. RT was measured as the time from stimulus onset to when a button press was recorded.

Analyses of ERP measures, N2 and P3 mean amplitude and latency were conducted for speed condition (Fast, Slow), trial type (Go, No-go), region (Frontal, Frontocentral, Central and Parietal), and laterality (Left, Midline and Right).

Results

Descriptive statistics for accuracy and response time (RT) are presented in Table 2-1, broken down by speed condition and trial type. Those for ERP amplitude and latency measures are presented in Table 2-2, broken down by speed condition, trial type, region, and laterality. Grand-averaged ERP waveforms are shown in Figure 2-3, separately for each electrode cluster.

Behavioral Performance

Accuracy and RT were analyzed using speed condition (Fast, Slow) x trial type (Go, Nogo) mixed ANOVAs with repeated measures on trial type. For accuracy, there was a main effect of speed condition (F(1,29) = 21.2, p < .001, $\eta^2 = .420$) and a statistically significant interaction between speed condition and trial type (F(1, 29) = 9.15, p < .01, $\eta^2 = .240$). Overall, children in the Fast condition responded less accurately (M = .87, SD = .01) than children in the Slow condition (M = .94, SD = .01). Follow-up analysis of the interaction effect (F(1, 29) = 12.08, p < .01, $\eta^2 = .294$) showed that only in the Slow condition, children had greater accuracy on Go trials (M = .98, SD = .01) than on No-go trials (M = .91, SD = .02). Examined another way, the speed condition effect was significant only for Go trials (F(1, 29) = 32.92, p < .01, $\eta^2 = .532$): children in the Fast condition performed less accurately (M = .87, SD = .01) than children in the Slow condition (M = .98, SD = .01).

Analyses of RT revealed main effects of speed condition ($F(1, 32) = 14.3, p < .005, \eta^2 = .331$) and trial type ($F(1, 32) = 33.5, p < .001, \eta^2 = .536$). Across both trial types, children in the Fast condition responded more quickly (M = 493.9 ms, SD = 19.64) than children in the Slow condition (M = 600.8 ms, SD = 20.28), as expected given the time pressure manipulation. Errors of commission on No-go trials (M = 487.3 ms, SD = 20.61) were characterized by shorter RTs than correct Go trials (M = 607.4 ms, SD = 13.75). This finding, characteristic of inhibitory failures, follows the predictions of the horse race model, indicating the earlier completion of the Go process in No-go trials as the underlying reason for inhibitory failures. The interaction between trial type and speed condition was not significant (p > .05; see Appendix A for details).

ERP Amplitude and Latency

P1 amplitude and latency were analyzed using speed condition (Fast, Slow) x trial type (Go, No-go) x laterality (Left, Midline, Right) mixed ANOVAs with repeated measures on trial type and laterality. For P1 amplitude, there were main effects of speed condition ($F(1, 29) = 4.26, p < .05, \eta^2 = .128$) and laterality ($F(2, 58) = 42.23, p < .001, \eta^2 = .593$). P1 amplitude was greater for children in the Slow condition ($M = 7.3 \mu V, SD = .86$) than children in the Fast

condition ($M = 4.9 \ \mu\text{V}$, SD = .84). It was also greater at the midline electrodes ($M = 8.5 \ \mu\text{V}$, SD = .73) than at the right ($M = 6.8 \ \mu\text{V}$, SD = .81) or left ($M = 3.0 \ \mu\text{V}$, SD = .53) electrodes and greater at the right ($M = 6.8 \ \mu\text{V}$, SD = .81) than left ($M = 3.0 \ \mu\text{V}$, SD = .53) electrodes. No other main effects or interactions were significant.

For P1 latency, there was a main effect of laterality (F(2, 58) = 42.23, p < .001, $\eta^2 = .593$). P1 latency was earlier in the left electrodes (M = 118.8 ms, SD = 3.9) than at the right electrodes (M = 128.6 ms, SD = 1.8). P1 latency at midline electrodes (M = 125.7 ms, SD = 1.8) did not differ from right or left electrodes. No other main effects or interactions were significant.

N2 amplitude and latency were analyzed using speed condition (Fast, Slow) x trial type (Go, No-go) x region (Frontal, Frontocentral, Central) x laterality (Left, Midline, Right) mixed ANOVAs with repeated measures on trial type, region, and laterality. For amplitude, there was a main effect of region (F(2, 58) = 34.5, p < .001, $\eta^2 = .544$), an interaction between trial type and laterality $(F(2, 58) = 5.3, p < .01, \eta^2 = .154)$ and a three-way interaction between trial type, laterality and region ($F(4, 116) = 4.2., p < .01, \eta^2 = .127$). N2 amplitude was significantly greater at frontal electrodes ($M = -5.3 \mu V$, SD = .68) than at frontocentral ($M = -4.6 \mu V$, SD = .63) or central electrodes ($M = -1.6 \mu V$, SD = .65) and it was significantly greater at frontocentral (M = -4.6 μ V, SD = .63) than at central electrodes ($M = -1.6 \mu$ V, SD = .65). At left electrodes only $(F(1, 29) = 6.7., p < .05, \eta^2 = .188)$, N2 amplitude was greater on No-go trials ($M = -5.3 \mu V$, SD = .88) than Go trials ($M = -4.2 \mu V$, SD = .68), whereas at midline and right electrodes, amplitude did not differ by trial type (p > .05). Finally, follow-up tests of the three-way interaction between trial type, laterality, and region showed that the increased No-go amplitude at left electrodes was only significant at frontal ($F(1, 29) = 5.9., p < .05, \eta^2 = .169$) (Go: $M = -5.4 \mu V$, SD = .90; Nogo: $M = -6.7 \,\mu\text{V}$, SD = 1.0) and frontocentral regions ($F(1, 29) = 7.7., p < .01, \eta^2 = .211$) (Go: M

= -4.6 μ V, *SD* = .71; No-go: *M* = -6.0 μ V, *SD* = .97). N2 amplitude in left electrodes at the central region did not differ between Go and No-go trials (*p* > .05). There were no significant effects involving speed condition (*p* > .05; see Appendix A for details) and no other main effects or interactions were significant.

For N2 latency, there were main effects of trial type ($F(1, 29) = 15.0, p < .01, \eta^2 = .341$), region $(F(1.4, 33.4) = 23.8, p < .001, \eta^2 = .451)$ and laterality $(F(1.5, 44.2) = 7.4, p < .01, \eta^2)$ =.203). N2 peaked later on No-go trials (M = 387.4 ms, SD = 5.6) than on Go trials (M = 369.0ms, SD = 5.1) and at frontal (M = 398.4 ms, SD = 7.2) than at frontocentral (M = 376.8 ms, SD = 7.2) 6.2) or central (M = 359.3 ms, SD = 3.4) regions. N2 latency was also later at left (M = 389.9 ms, SD = 7.6) and midline (M = 382.0 ms, SD = 6.2) electrodes than at right (M = 362.7 ms, SD = 6.2) 5.2) electrodes. These main effects were qualified by interactions between trial type and region $(F(2, 58) = 6.8, p < .01, \eta^2 = .189)$ and between laterality and region $(F(3, 71.8) = 4.8, p < .01, \eta^2)$ = .141). The N2 peaked later on No-go trials than Go trials in the frontal $(F(1, 29) = 17.2, p < 10^{-1})$.01, $\eta^2 = .372$) (Go: (M = 383.8 ms, SD = 8.2); No-go: (M = 412.9 ms, SD = 7.9)) and frontocentral electrodes ($F(1, 29) = 10.0., p < .01, \eta^2 = .256$) (Go: (M = 366 ms, SD = 6.4); Nogo: (M = 387.7 ms, SD = 7.7)), but not at central electrodes (p > .05). Furthermore, N2 peaked later at left and midline leads relative to right leads in the frontal $(F(2, 28) = 8.9, p < .01, \eta^2 =$.389) (left: (M = 413.1 ms, SD = 10.1); midline: (M = 409.9 ms, SD = 10.4); right: (M = 372.1 ms, SD = 10.4)ms, SD = 6.7) and frontocentral regions ($F(2, 28) = 4.3., p < .05, \eta^2 = .234$) (left: (M = 377.3) ms, SD = 7.7); midline: (M = 393.7, SD = 10.4); right: (M = 359.6 ms, SD = 5.7)). In the central region, N2 latency did not significantly differ between left, right and midline leads (p > .05). Again, there were no significant effects involving speed condition (p > .05; see Appendix A for details) and no other main effects or interactions were significant.

P3 amplitude and latency were analyzed using speed condition (Fast, Slow) x trial type (Go, No-go) x laterality (Left, Midline, Right) mixed ANOVAs with repeated measures on trial type and laterality. Analyses of P3 amplitude revealed main effects of trial type (F(1, 29) = 13.0, p < .01, $\eta^2 = .310$) and laterality (F(1.6, 45.8) = 7.0, p < .01, $\eta^2 = .195$). P3 amplitude was greater on No-go trials ($M = 12.3 \mu V$, SD = .92) than Go trials ($M = 9.6 \mu V$, SD = 1.1). Topographically, P3 amplitude was greater in midline ($M = 11.8 \mu V$, SD = 1.2) and right electrodes ($M = 12.6 \mu V$, SD = 1.2) than in left electrodes ($M = 8.5 \mu V$, SD = .96).

For P3 latency, there was a main effect of laterality ($F(2,58) = 12.1, p < .01, \eta^2 = .195$). The P3 latency was significantly earlier in the midline electrodes (M = 369.6 ms, SD = 11.2) than in the right (M = 398.6 ms, SD = 13.1) or the left electrodes (M = 429.4 ms, SD = 12.8) and significantly earlier in the right electrodes (M = 398.6 ms, SD = 13.1) than in the left electrodes (M = 429.4 ms, SD = 12.8). There were no significant effects involving speed condition for both P3 amplitude and latency (p > .05; see Appendix A for details). No other main effects or interactions were significant.

Discussion

We sought to examine the neural correlates of successful response inhibition in early childhood, and how response inhibition is modulated under conditions of time pressure. Children completed a GNG task where they had to inhibit responding on the less frequent No-go trials. The time pressure manipulation affected children's task performance: children in the Fast condition responded faster but less accurately than children in the Slow condition. In addition, children in the Slow condition displayed greater accuracy on Go trials than on No-go trials, whereas children in the Fast condition performed equivalently on Go and No-go trials. The time pressure manipulation also affected early ERP activity, as the P1 was greater for children in the Slow condition than the Fast condition. However, ERP markers of response inhibition did not differ between the Fast and Slow conditions. Across both conditions, relative to Go trials, No-go trials elicited a left-lateralized enhanced N2 and an enhanced P3 at midline electrode sites.

The N2 and the P3

The enhanced N2 observed on No-go trials as compared to Go trials is consistent with the literature, and typically thought to reflect neural activity underlying response inhibition. Alongside amplitude effects, we found parallel differences in N2 latency, with longer N2 latencies for No-go trials and at left frontal electrodes. However, in adults, N2 differences are typically observed at midline (Bekker, Kenemans, & Verbaten, 2005; Jonkman et al., 2007) or right (Bokura et al., 2001) electrode sites, whereas in our study the effect was left-lateralized. Interestingly, in a previous study with 7- to 16-year-olds, both younger participants and participants who performed poorly exhibited greater left lateralization of the No-go N2 (Lamm et al., 2006). This suggests that there may be reorganization of networks underlying response inhibition, with the shift from the left-lateralized to midline and right-lateralized activity indicating a more mature neural network. Another alternative is that the left-lateralized N2 indicates that children employ a different strategy to perform the task. There is evidence to indicate that children frequently employ verbal strategies to perform cognitive tasks that are inherently non-verbal (Berk, 1992; Winsler & Naglieri, 2003), which would presumably lead to greater reliance on the left hemisphere. For example, in the present study children may have used verbal labeling or self-talk as a strategy to withhold a button press on No-go trials. In a neuroimaging study, 9- to 12-year-old children performing a cognitive control task displayed greater activity in the left hemisphere, and verbal ability was correlated with performance (Bunge et al., 2002). Future research should examine how children's use of different strategies

on cognitive control tasks influences their performance as well as the neural resources recruited to perform them.

We also found an enhanced No-go P3 — that is, the P3 was more pronounced on No-go trials than on Go trials (Bokura et al., 2001), although there were no P3 latency differences by trial type. However, as with the N2, the topography of the enhanced No-go P3 observed in our study differed markedly from studies of adults. In our study of early childhood, the No-go P3 was observed at posterior midline electrode sites, whereas in adults, the enhanced No-go P3 is typically seen at frontal midline electrode sites, a phenomenon referred to as the 'No-go anteriorization' (Fallgatter et al., 1999). At posterior electrode sites, adults show a more pronounced Go P3 than No-go P3 reflecting attention to targets (Bruin, Wijers, & Van Staveren, 2001). A posterior P3 is also seen in adults in the oddball task indicating the processing of infrequent targets (Friedman, Cycowicz, & Gaeta, 2001; Gaeta, Friedman, & Hunt, 2003). One might question whether the P3 effect in the present study is an oddball effect; however, we believe it is unlikely to be a result of infrequent target probability, as individual Go and No-go stimuli were presented with equal frequency. We argue that the difference in the topography of the No-go P3 found in our study could indicate children's reliance on additional posterior brain regions to support response inhibition. Similar findings have been observed in middle childhood with regard to the N2 (Jonkman et al., 2007). Using source localization methods, Jonkman and colleagues found that the neural activity underlying response inhibition in adults was adequately explained by frontal sources, but in children, contributions from additional posterior sources were required. Supporting evidence also comes from studies utilizing brain imaging techniques that have shown that as children develop, the neural networks controlling inhibitory processes

shift from a more posterior, distributed pattern to a more frontal, localized one (Bunge et al., 2002; Casey, Thomas, Davidson, Kunz, & Franzen, 2002; Durston et al., 2006).

Considered together, our ERP findings suggest that the neural correlates underlying response inhibition in early childhood differ in important ways from those seen in adulthood. Consistent with the adult literature, both the N2 and the P3 were more pronounced on No-go trials than on Go trials in 5-year old children. However, the topography of these No-go effects differed markedly, suggesting that the brain regions supporting response inhibition in early childhood differ from those in adults.

It is sometimes argued that the enhanced N2 and P3 amplitudes seen on No-go trials are a result of motor-related neural activity rather than a reflection of inhibition. This argument is based on the grounds that unlike Go trials, No-go trials do not require a motor response and that this disparity could explain the amplitude differences observed in both ERP components. However, given that motor preparation is typically associated with a negative-going response (Shibasaki, Barrett, Halliday, & Halliday, 1980), we should have observed an *increased* N2 amplitude on Go trials. Our findings, in contrast, show increased N2 amplitude on No-go trials making its association with inhibitory processes a more tenable explanation. Similarly, attributing the No-go P3 to motor related activity can be ruled out based on the findings of studies where an enhanced No-go P3 was found despite eliminating motor demands from the task (Smith, Johnstone, & Barry, 2008). However, it should be noted that these studies have typically looked at adults, so future studies should examine motoric contributions to No-go ERP effects in children, particularly given topographic differences between the No-go P3 in children and adults.

We experimentally manipulated time pressure by giving children in the Fast condition a shorter time window in which they could make a response. We expected this manipulation to increase the prepotency of responding, leading to greater inhibitory demands on No-go trials. However, examination of our accuracy findings showed that despite their faster response times, children in the Fast condition did not appear to display prepotent responding, in that their No-go performance was equivalent to children in the Slow condition. Rather, differences between the conditions emerged only on Go trials, where children in the Fast condition made more errors. One possible explanation for the differences in accuracy between the Fast and Slow conditions is that the time pressure manipulation resulted in differences in attentional engagement on the task. This suggestion is consistent with the finding that the parietal P1 component was more pronounced in the Slow condition than in the Fast condition. Greater P1 amplitude is typically associated with heightened selective attention or higher levels of vigilance (Key, Dove, & Maguire, 2005). Hence, it is possible that the lower Go accuracy observed in the Fast condition was due to the time pressure manipulation interfering with children's ability to recruit or sustain attentional resources. However, another possibility is that this difference is due to children's inability to respond quickly enough. To test this possibility, we examined the Go reaction time distribution of children in the Fast condition to evaluate whether they were truncated. All children responded between 400 - 500 ms on a majority of Go trials and, with one exception, RTs greater than 650 ms comprised less than 20 percent of each child's RT distribution. This suggests that children successfully adjusted their speed of responding to the time pressure manipulation, and the behavioral differences between the conditions were not simply a result of the children in the Fast condition having insufficient time to respond. Behavioral differences could also indicate that children in the Fast condition adopted a cautious strategy, placing a

higher priority on not catching the sharks on No-go trials, at the expense of missing more fish on Go trials.

One might ask whether the P1 difference between the Fast and Slow condition affected the other ERP findings. Notably, this difference involved only the speed condition factor, with no hint of a main effect or interaction involving trial type. The converse was true for the N2 and P3, which differed between Go and No-go trials but were not modulated by the speed condition manipulation. Therefore, although there were indications that early attentional processes were affected by the speed condition manipulation, it seems unlikely that these differences contributed to later inhibitory processes.

Limitations

An important limitation of this study is its between-subject design. This was necessary to prevent training effects and exposure to the task. Furthermore, the short attention span of young children would have made it hard for them to participate in both versions of the task, given the high number of trials required in each condition for ERP studies. Children were randomly assigned to the two conditions to minimize the possible confounding effects of unrelated third variables; however, sampling error may have resulted in differences in the makeup up of the children in the two conditions that may have contributed to the findings. Unfortunately, measures of IQ and processing speed were not administered, and information on parental education levels was not collected to allow us to assess and control for possible confounding differences.

In order to make the task appropriate for young children, it was designed with fish as the Go stimuli and sharks as the No-go stimuli. However, because stimuli were drawn from two distinct categories, any differences between the stimulus sets could have contributed to observed differences between Go and No-go trials (for example, salience). A supplementary analysis (see

footnote 1) indicated that behavioral findings from our study remained unchanged after taking into consideration differences in stimulus salience. Furthermore, as the Go stimuli were more colorful, differences in salience should have resulted in a more pronounced N2 for the Go trials, whereas our findings showed the reversed pattern, a more pronounced N2 for No-go trials.

Another limitation of this study is our inability to examine the error-related negativity (ERN) — the neural correlate associated with error detection and monitoring. While several other studies utilizing similar task designs have examined ERN (Torpey et al., 2009; 2012), we were unable to do so in this study because the children made few errors. However, analyzing ERN could potentially give us additional insights into the development of response inhibition abilities in early childhood, and future studies should undertake such an investigation.

Conclusion

We ventured to understand the neural correlates underlying response inhibition in early childhood, a period critical for the development of the neural networks underlying higher order cognitive processes like response inhibition. Few studies have examined the neural correlates underlying response inhibition in early childhood. We found both similarities and differences in the pattern of brain activity underlying response inhibition in early childhood compared with that seen in adults. Response inhibition was associated with an enhanced left-lateralized frontal N2 and a midline posterior P3, differing topographically from patterns in adults. These differences may suggest that the immaturity of inhibitory brain networks may result in children's recruitment of additional, different brain regions to perform response inhibition tasks.

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Table 2-1. Means and standard deviations (in parentheses) for accuracy (proportion correct) and

 reaction time (milliseconds) by trial type and speed condition.

	Go Trials		No-go Trials	
	Slow	Fast	Slow	Fast
Accuracy	.98 (.03)	.87 (.07)	.91 (.04)	.88 (.08)
Reaction Time	664 (100.6)	550 (43.3)	537 (151.4)	437 (63.6)

Note: Reaction times represent correct go trials and incorrect no-go trials.

Table 2-2. Means and standard deviations (in parentheses) for ERP waveform characteristics

(amplitude and latency), by trial type and speed condition.

Component Region Laterality	Go		No-go	
	Slow	Fast	Slow	Fast
N2 Amplitude (µV)				
Frontal				
Midline	-5.07 (4.67)	-5.71 (4.19)	-4.53 (5.57)	-4.91 (5.00)
Right	-5.18 (4.94)	-4.78 (2.82)	-4.52 (6.23)	-4.62 (3.39)
Left	-5.01 (5.47)	-5.72 (4.49)	-5.74 (7.02)	-7.74 (4.44)
Frontocentral				
Midline	-5.21 (4.99)	-4.81 (3.30)	-4.44 (6.26)	-4.63 (4.58)
Right	-4.13 (4.44)	-4.22 (3.33)	-2.85 (7.28)	-3.30 (3.66)
Left	-4.28 (4.33)	-4.89 (3.55)	-5.09 (6.34)	-6.93 (4.38)
Central				
Midline	97 (5.34)	-1.86 (3.49)	-1.99 (3.32)	68 (6.71)
Right	81 (3.80)	-2.07 (4.10)	39 (6.85)	31 (3.60)
Left	-1.67 (3.72)	-3.43 (3.43)	-1.90 (5.51)	-4.39 (3.49)
N2 Latency (ms)				
Frontal				
Midline	400 (67.1)	384 (71.2)	427 (63.1)	429 (51.6)
Right	378 (48.6)	347 (34.9)	408 (58.5)	355 (33.9)
Left	402 (68.9)	393 (75.8)	436 (66.7)	422 (58.2)
Frontocentral				
Midline	365 (44.1)	365 (44.9)	383 (53.2)	396 (58.9)
Right	361 (34.5)	348 (36.3)	378 (51.2)	351 (40.0)
Left	378 (63.1)	379 (66.7)	415 (67.8)	403 (62.5)
Central	· · ·			
Midline	356 (46.7)	357 (25.6)	359 (48.6)	362 (29.6)
Right	363 (37.3)	347 (30.4)	370 (61.0)	346 (31.4)
Left	367 (45.5)	353 (34.4)	365 (30.3)	366 (51.3)
P3 Amplitude (μV) Parietal				
Midline	11.30 (8.83)	9.84 (6.16)	14.12 (7.73)	11.88 (5.06)
Right	12.71 (9.72)	9.38 (5.73)	15.14 (8.46)	12.96 (3.96)
Left	8.32 (4.41)	6.27 (5.91)	11.52 (6.99)	7.86 (5.78)
P3 Latency (ms) Parietal				
Midline	344 (45.5)	382 (84.3)	372 (75.1)	380 (65.8)
Right	398 (88.3)	401 (84.4)	383 (64.3)	411 (75.3)
Left	401 (63.6)	449 (101.9)	406 (57.4)	461 (84.6)



Figure 2-1. Outline of the Go/No-go task used in the study. Top-left: Correct Go trial (Hit), Topright: Correct No-go trial (Correct miss), Bottom-left: Incorrect Go trial (Miss), Bottom-right: Incorrect No-go trial (False alarm). In the Slow condition, stimulus was displayed for up to 1500 ms and in the Fast condition, stimulus was displayed for up to 750 ms.



Figure 2-2. Layout illustrating the electrodes that were included in the ERP analyses at frontal (F), frontocentral (FC), central (C) and parietal (P) regions.



Figure 2-3. Grand-averaged ERP waveforms separated by region (Frontal, Frontocentral, Central, and Parietal) and laterality (Left, Midline, and Right). Time windows selected for the analyses of the N2, P1 and P3 are displayed in boxes of different shades of gray. N2 (light gray, top three panels) was analyzed between 260-560 ms after stimulus onset at frontal, frontocentral and central electrodes. P1 (light gray, bottom panel) was analyzed between 60-150 ms, and P3, (dark gray, bottom panel) was analyzed between 310-610 ms after stimulus onset at parietal electrodes.

Chapter 3

Physical activity and response inhibition in early childhood: An ERP study using objective and subjective measures of physical activity

In recent years, physical activity has gained recognition as a factor that enhances brain health and cognitive performance (Erickson, Voss, & Prakash, 2011; Hillman, Erickson, & Hatfield, 2017). It has been reported to aid processes vital to supporting brain health, including neurogenesis, synaptogenesis, and angiogenesis (Lista & Sorrentino, 2010; Vivar, Potter, & van Praag, 2013). The benefits of physical activity have been especially noted in cognitive control functions that are essential for goal-directed behaviour (Colcombe & Kramer, 2003; Davis et al., 2011). One aspect that has been most commonly studied in relation to the effects of physical activity is inhibitory control (Ludyga, Gerber, Brand, Holsboer-Traschler, & Pühse, 2016) — the ability to suppress inappropriate responses (response inhibition) or irrelevant information (interference suppression) (Barkley, 1997; Dempster, 1993). Response inhibition emerges very early in infancy (Diamond, 1990; Johnson, 1994), and undergoes a protracted rate of development, with particularly marked development occurring in early childhood, between the ages of three to six (Clark et al., 2013; Wiebe, Sheffield & Espy, 2012). Response inhibition is associated with both academic and social competence in children (Blair & Razza, 2007; Carlson & Moses, 2001), and it has also been linked to social functioning later in life (Rinsky & Hinshaw, 2011). As early childhood is an important period for the development of response inhibition, it is vital to understand how factors such as physical activity may influence its development. Few studies have explored the relationship between physical activity and response inhibition in early childhood (Campbell, Eaton, McKeen, 2002; Carson, Abdul Rahman, & Wiebe, 2017; Palmer, Miller, & Robinson, 2013), and none have examined the association between physical activity and the neural correlates underlying response inhibition. The goal of this study is to address this gap in literature, and examine how physical activity in early childhood is associated with response inhibition performance and the underlying neural correlates.

The mechanisms underlying the effects of physical activity on cognitive functioning

Physical activity refers to bodily movements that are generated by skeletal muscles, which results in energy expenditure (Caspersen, 1989). In both adults and children, physical activity is positively associated with cognitive performance (Ludyga et al., 2016; Sibley & Etnier, 2003). Several pathways have been proposed to explain the salutary effects of physical activity on cognitive functioning. One direct pathway concerns the physiological changes that are brought about by physical activity. For instance, physical activity increases cerebral blood flow (Guiney, Lucas, Cotter, & Machado, 2015; van Praag, 2009), and neurochemicals, such as brain derived neurotrophic factor (Tang, Chu, Hui, Helmeste, & Law, 2008; Williams, & Shen, 2007) and monoamines (Dishman et al., 2006). Physical activity has also been associated with structural changes in the brain. Increases in both white and gray matter volumes, as well as in white matter integrity have been observed with physical activity participation (Colcombe et al., 2006; Erickson et al., 2011; Oberlin et al., 2016; Weinstein et al., 2012). Such increases are most prominently observed in the prefrontal and temporal/hippocampal brain regions (Colcombe et al., 2006; Erickson et al., 2011; Voss et al., 2013), and importantly, these structural changes in the brain have been found to mediate the association between physical activity and the observed enhancement in cognitive performance (Oberlin et al., 2016; Weinstein et al., 2012). Similar findings have also been noted in children. Specifically, participation in physical activity and increased physical fitness were associated with increases in the volumes of both cortical (Frontal and Temporal cortices) and subcortical (Hippocampus, Caudate Nucleus) regions of the brain (Chaddock et al., 2010a; Chaddock et al., 2010b; Esteban-Cornejo et al., 2017).

Other indirect pathways have also been proposed to explain the beneficial effects of physical activity on cognitive functioning, and these address the interrelations that are present between physical activity and cognitive processes. The cerebellum, a brain region more

commonly recognized for its role in motor functions, has been suggested to play an equally important role in cognitive processes (Buckner, 2013; Diamond, 2000; Stoodley, 2012). A close coupling in the activation of the cerebellum and the dorsolateral prefrontal cortex has been noted during cognitive tasks (Bellebaum & Daum, 2007; Raichle et al., 1994). The prefrontal cortex has also been implicated in the learning and acquisition of complex motor skills (Serrien, Ivry & Swinnen, 2007). Hence, the overlap of key brain regions involved in cognitive performance. Another indirect mechanism that has been proposed addresses the cognitive demands that are inherent in physical activities. Physical activities that involve group participation require planning and strategizing in order to adapt to the rapidly changing demands of the activity. The 'cognitive training' that is obtained from engaging in such physical activities has been suggested to transfer to and aid performance on cognitive tasks (Best, 2010; Sibley & Etnier, 2003; Tomporowski & McCullick, 2009).

Physical activity and response inhibition

To date, only three published studies have assessed the association between physical activity and response inhibition in early childhood. Of these, one reported a positive association between physical activity and response inhibition performance (Campbell et al., 2002), whereas the other two reported a lack of association (Carson et al., 2017; Palmer et al., 2013). All three studies assessed physical activity objectively using activity trackers. However, Campbell and colleagues (2002) used a composite score from five different tasks to assess response inhibition, whereas in the other two studies (Carson et al., 2017; Palmer et al., 2013), a single response inhibition task was used. Hence, the discrepancies in the findings between the studies could be due to the different methods that were used to assess response inhibition. A recent study also

examined the association between physical activity and inhibitory abilities (response inhibition and interference suppression) in preschool children. In this study, inhibitory abilities in children were negatively associated with moderate- to vigorous-intensity physical activity (Willoughby, Wylie, & Catellier, 2018). All four of these studies, however, did not examine the neural correlates underlying response inhibition to investigate how they are associated with physical activity in children.

A few studies in adolescence and adults have examined the association between physical activity and the neural correlates of response inhibition. In these studies, event-related potentials (ERPs) underlying inhibitory abilities (P3 and N2) were examined, and their findings provide some indication on how the neural correlates underlying response inhibition may be modulated by physical activity. In a study with adolescence, both behavioural performance and the P3 ERP component were not influenced by participation in physical activity (Stroth et al., 2009). In contrast to this finding, two studies in adults reported a positive association between physical activity and the P3 (Bae, Ogawa, Yamazaki, 2012; Chu, Alderman, Wei, & Chang, 2015). In one study, adults who participated in physical activity displayed better response inhibition performance than those from the control condition. This enhanced performance was accompanied by increased amplitude and prolonged latency of a P3 ERP component. This finding was interpreted as physical activity aiding in more successful recruitment of neural resources to support response inhibition, but also delaying processing that was necessary to overcome prepotent responding (Chu et al., 2015). Similarly, in another study, physical activity was associated with increased P3 amplitude during response inhibition. However, no differences in behavioural performance or P3 latency were observed in this study (Bae et al., 2012).

A greater number of studies have examined the association between physical activity and the neural correlates underlying interference suppression in school-aged children. In these

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studies, physical activity participation and greater physical fitness were associated with a larger P3 amplitude (Drollette et al., 2014; Hillman et al., 2009; Hillman, Buck, Themanson, Pontifex, & Castelli, 2009; Hillman, Castelli, & Buck, 2005; Pontifex et al., 2011) and a shorter P3 latency (Drollette et al., 2014; Hillman et al., 2005; Pontifex et al., 2011). Importantly, findings from two of these studies provide additional insights on an earlier occurring ERP component that has also been associated with inhibition, the N2. Specifically, the two studies reported that physical activity participation and greater physical fitness in children were associated with a decrease in N2 amplitude (Drollette et al., 2014; Pontifex et al., 2011). The decrease in amplitude was interpreted as physical activity and fitness allowing for more efficient recruitment of neural resources to support performance. In addition, a shorter N2 latency was also observed in children with higher physical fitness levels indicating faster information processing and response selection (Pontifex et al., 2011).

These ERP findings with adults and older children highlight the importance of examining how the neural correlates underlying response inhibition performance are modulated by physical activity. Examining this association in early childhood is especially pertinent as this period is marked by rapid development of the brain. Moreover, there is evidence that environmental influences play a key role in shaping its development (Blair, & Raver, 2012; Fox, Levitt, & Nelson, 2010; Liu, Diorio, Day, Francis, & Meaney, 2000). Therefore, it is important to test if lifestyle factors like physical activity affect the neural correlates supporting response inhibition in the early years.

Response inhibition and the underlying neural correlates in children

Response inhibition in children is commonly assessed using the Go/No-go task (Simpson & Riggs, 2006; Wiebe et al., 2012). In this task, children are required to respond to a frequently

occurring Go stimulus, but have to withhold responding to a less frequently occurring No-go stimulus. The greater frequency of the Go stimulus makes responding the more automatic response, and the extent to which children are able to withhold this prepotent responding on the No-go trials is taken to index their response inhibition abilities. The GNG task has been validated in children as young three years old, and is found to correlate with other measures of response inhibition (Simpson & Riggs, 2006).

As mentioned in the review of previous findings above, electrophysiological recordings obtained during response inhibition performance have consistently identified the two ERP components, the N2 and P3, to underlie inhibitory performance. These two ERPs show enhanced amplitude under conditions requiring inhibition, as in the No-go trials of the GNG task. The enhanced amplitude is thought to reflect the greater engagement of neural resources that is required to support the inhibitory demands on No-go trials. The N2 is observed at frontocentral electrode regions, and is identified as a negative deflection occurring about 200 – 500 ms after stimulus onset (Carter & Van Veen, 2007; Falkenstein, Hoormann & Hohnsbein, 1999; Jonkman, Sniedt, Kremner, 2007). The P3 is typically observed in more posterior electrode regions in children, and is identified as a positive deflection occurring about 300 – 600 ms after stimulus onset (Johnstone et al., 2007; Spronk, Jonkman, & Kemner, 2008).

The present study

In the present study, children aged two- and a-half to four- years-old completed the GNG task while their scalp EEG was recorded. Objective and subjective measures of physical activity were obtained to assess the relationship between physical activity, response inhibition, and the underlying neural correlates. While two previous studies with preschool children used accelerometer to objectively assess physical activity, subjective reports were not assessed

(Campbell et al, 2002; Palmer et al., 2014). In preschool children, subjective measures may index more purposeful behaviour (Carson et al., 2017), and examining both objective and subjective measures would provide us with a better understanding of physical activity in children. In addition, in the ERP studies with adults and older children, physical activity was experimentally manipulated by assignment of participants into acute or chronic exercise and control groups. However, in early childhood, a similar design may not be feasible due to logistical reasons and participant compliance. Moreover, assessment of physical activity using accelerometry and parental reports would measure naturally occurring differences in physical activity levels in children and as such, may have greater ecological validity.

Based on previous findings associating physical activity with superior cognitive performance (Campbell et al., 2002; Chu, Alderman, Wei, & Chang, 2015; Hillman et al., 2009), we hypothesized that higher levels of physical activity in children would be associated with better response inhibition. Following previous ERP findings, we hypothesized that higher levels of physical activity will be associated with reduced N2 amplitude and enhanced P3 amplitude, and that it will be associated with earlier latencies of both the N2 and P3.

Method

Participants

Children included in this study were part of a larger cohort of 100 children (53 girls, 47 boys) involved in the Physical Activity and Cognition in Early Childhood (PACE) study (Carson et al., 2017). The present sample included 58 children (36 girls, 22 boys) aged between 2; 5 years and 5; 0 years (M = 3; 8 years, SD = 9 months). An additional 42 children visited the lab but were not included in the analysis because they did not understand the task rules (n = 4), or did not want to complete the task (n = 15), did not provide sufficient ERP data (n = 14) or did not

provide sufficient accelerometer data (n = 9). To test whether missing data was systematically related to child characteristics, a logistic regression analysis was carried out regressing missingness on child age, gender, and maternal education (as a proxy for socioeconomic status). Only age was a significant predictor with older children being less likely to have missing data (χ^2 = 11.20, p < .05, Odds Ratio = 0.94). Participants were recruited through flyers sent to preschools, playgroups, daycares, and elementary schools. Parents reported that all children were born full-term (\geq 37 weeks), had no history of neurological or behavioural disorders, and English was the primary language spoken in the home.

Procedures

The study was carried out at a child development laboratory at a university in a mid-sized Western Canadian city. Parents accompanied their children to the lab and were briefed about the study before providing informed consent. After EEG net application, children first completed the GNG task and later another task that is not related to the present study. Children also completed the Peabody Picture Vocabulary Test (PPVT), a measure of receptive vocabulary, and the score was used as a proxy for IQ. Parents remained in the testing room throughout the session and completed several questionnaires. At the end of the visit, parents were given an Actigraph accelerometer monitor for children to wear for seven days and were provided with instructions on how to use it. They were also given a postage paid envelope for them to mail back the accelerometers to the lab. All children received a toy and parents received a gift card in appreciation of their participation in the study.

Physical Activity Measures. Objective measures of physical activity were collected using an Actigraph accelerometer (Model: wGT3X-BT). Children wore the accelerometer (attached to an elastic waistband) around their hips for a period of seven days (including weekdays and weekends), and removed it during nap and bed times, and during bathing and

swimming. The accelerometer collected movement counts in 15-second intervals. Based on the number of counts, each 15-second interval was classified as light-intensity physical activity (LPA; 26 to 419 counts), or moderate- to vigorous-intensity activity (MVPA; \geq 420 counts). These two values were summed to derive total objective physical activity level. The cut-off points were age-appropriate and based on previous findings with children (Janssen et al., 2013). Instances with 80 or more consecutive 15-second intervals with zero counts were classified as non-wear time. To be included in the analyses, children were required to have worn the accelerometer for at least four valid days. A valid day required at least 1440 15-second intervals of wear time each day (equivalent to at least six hours each day; Hinkley et al., 2012). To calculate the number of minutes per day of LPA and MVPA, the total number of 15-second intervals was divided by four, and then divided by the number of valid days. These values were then standardized to adjust for wear time. Standardization was done using the residuals obtained from regressing each variable on total wear time (Willett & Stampfer, 1986).

Subjective measures of physical activity were also obtained from parental-reports. In the demographic questionnaires that parents filled out during the lab visit, they reported on the number of hours their child spent in organized (e.g., swimming, skating, gymnastics) and non-organized physical activities (e.g., going for a walk, playing at a splash pad, bike ride, playing at the park/yard). The response options were: (1) never, (2) less than 2 hours per week, (3) 2-3 hours per week, (4) 4-6 hours per week, and (5) 7+ hours per week. Mid-point of the response options (i.e., 0, 1, 2.5, 5, 7) was used as a measure of time spent in organized and non-organized activities. The sum of these two components (organized and non-organized) was used as a measure of total subjective physical activity.

Response Inhibition Task. The GNG task has been used to assess response inhibition in children as young as three years old (Simpson & Riggs, 2006; Wiebe et al., 2012) and its utility

as a measure of response inhibition has been validated by demonstrating high correlations with other measures of response inhibition (Simpson & Riggs, 2006). The Preschool GNG task (adapted from Wiebe et al., 2012) was a computerized fishing game (Figure 3-1), presented using E-Prime 2.0 Professional (Psychology Software Tools, Sharpsburg, PA). Children were required to respond to a frequently occurring Go stimulus (fish) by pressing a single button on a button box, but had to withhold responding to a less frequently occurring No-go stimulus (sharks). The greater frequency of the Go stimulus makes responding the more automatic response, and the extent to which children are able to withhold this automatic responding on the No-go trials is taken as a measure of their response inhibition abilities.

Each trial in the task began with the onset of a stimulus (fish or shark). The stimulus remained on the screen for a maximum duration (1500 ms, 2000 ms, or 2500 ms) and disappeared when the child responded. The maximum duration in which the stimulus was displayed was adjusted according to the child's age so as to ensure that the task difficulty was age-appropriate. Previous findings have shown that when administering the GNG task with children, it is important to ensure that the time window allowed for responding is sufficient to allow children an opportunity to respond, but at the same time, not so long that the inhibitory demands of the task are removed (Simpson & Riggs, 2006). For children younger than 3.25 years, the maximum response duration was 2500 ms. For children aged 4.0 years or older, the maximum response duration was 1500 ms. These time windows were selected based on previous studies with similar age groups (Simpson & Riggs, 2006; Wiebe et al., 2012). The three task versions of the task were identical, and only differed in the time window allowed for a response.

When the child responded correctly on Go trials, a net appeared over the fish with a 'bubbling' sound to indicate that the fish had been caught. When the child responded incorrectly

on No-go trials, a picture of a broken fishing net appeared over the shark with a 'buzzer' sound to indicate the shark had broken through the net. Both feedbacks lasted for 1000 ms. No feedback was given on trials when the child did not press the button. Each trial was followed by an inter-stimulus interval of 1000 ms.

Following the prototypical design of the GNG task (Cragg & Nation, 2008; Wiebe et al., 2012), Go trials were probabilistically dominant (75%) to increase children's bias to respond, thereby maximizing response inhibition requirements on No-go trials. The stimuli (six fish and two sharks) were presented at the centre of the screen on a 20-inch Dell desktop monitor and measured 10 cm by 13 cm. Each stimulus was presented with equal frequency across the test trials (12.5%). At the beginning of the task, children were shown a picture of all the stimuli and asked to point out the fish and sharks to ensure they could distinguish between Go and No-go stimuli. In addition, to ensure that children understood the rules of the task, they completed a block of 24 practice trials, similar to test trials and consisting of 50% fish and 50% shark stimuli. Once it was established that children understood the task and were responding appropriately, they proceeded to the test trials. There were a total of 176 test trials including 132 Go trials (75%) and 44 No-go trials (25%). Test trials were divided into four blocks of 44 trials.

Electrophysiological Recording and Data Processing. EEG was recorded using a 128channel Hydrocel Geodesic Sensor Net (Electrical Geodesics Inc., Eugene, OR). First, children's head circumference was measured to allow selection of the appropriate net, and the vertex was marked to ensure correct net placement. The net was soaked in an electrolyte solution prior to application. Electrode impedances were measured and maintained below 50 k Ω . If needed, additional electrolyte solution was applied to electrodes with high impedances. During task performance, EEG data was recorded continuously at a sampling rate of 250 Hz, with a 0.1 – 100 Hz bandpass filter, referenced to the vertex.

ERP analysis was conducted with Net Station software (Version 4.3.1, Electrical Geodesics Inc., Eugene, OR). A 0.3-30 Hz bandpass filter was used to filter data offline. EEG data was segmented into 1300 ms epochs beginning 100 ms before stimulus onset and ending 1200 ms post-stimulus. Artifact detection was performed on segmented files and all electrodes where signal fluctuations exceeded 200 μ V were marked bad. Segments with more than 12 bad electrodes (10% of channels) were rejected. Spline interpolation was used to replace bad electrodes in otherwise acceptable segments. EOG correction was conducted using Gratton's algorithm (Gratton, Coles & Donchin, 1983). In addition, the first author visually inspected all segments for EOG or movement artifacts, and corrected segment and channel markups if needed. Following EOG correction, channels were reassessed for artifacts with previously marked information overwritten if necessary and then bad electrodes replaced using spline interpolation. All usable segments were averaged, separately for Go and No-go trial types. EEG data was rereferenced to an average reference using the polar average reference effect correction (Junghöfer, Elbert, Tucker, & Braun, 1999), and then baseline-corrected using the data 100 ms prior to stimulus onset. Incorrect trials and trials with a reaction time (RT) less than 200 ms were excluded from analysis as they do not reflect deliberate behaviour (Haith, Hazan & Goodman, 1988). All participants included in the analysis had a minimum of 10 artifact-free trials in each trial type.

Then, adaptive mean amplitude and peak latency measures were calculated for the inhibition-related ERP components, N2 and P3. Adaptive mean amplitude was defined as the average amplitude centred around the peak, including 40 ms before and after the peak. This selection was based on visual examination of grand-averaged and individual waveforms. Peak latency was defined as the time taken from the stimulus onset to the maximum amplitude within the time window selected. The N2 was examined at frontal electrode sites and was defined as the

peak negative deflection occurring in the time window between 260 and 560 ms. The P3 was examined at parietal electrode sites, and was defined as a peak positive deflection occurring between 310 and 610 ms. The electrode selection was based on previous studies examining these components in children (Abdul Rahman et al., 2017; Jonkman, Lansbergen, & Stuader, 2003), confirmed by visual inspection of the waveforms showing N2 to be maximal at frontal electrodes and P3 to be maximal at parietal electrodes. The time windows were determined based on previous studies with similar age groups (Ciesielski et al., 2004; Lahat et al., 2010) and visual examination of the grand averaged and individual waveforms. Peak latency and adaptive mean amplitude measures were averaged across electrodes within clusters selected to be compatible with the 10-20 electrode placement system. The electrodes included in each cluster are presented in Figure 3-2. Based on visual inspection of the waveforms and previous findings reporting the N2 to be left-lateralized in children (Abdul Rahman, Carroll, Espy, & Wiebe, 2017; Lamm, Zelazo, & Davis, 2003), electrode clusters for N2 were defined within left, midline and right locations. As visual inspection showed P3 to be maximal at midline electrodes, only midline electrode clusters were analyzed for P3.

Data Analysis

Preliminary analyses: Behavioural and ERP measures of response inhibition

All analyses were conducted using SPSS Version 21.0 (IBM Corp., Armonk, NY). Descriptive statistics for demographic information are presented in Table 3-1. Descriptive statistics for behavioural and ERP measures of response inhibition are presented in Table 3-2. Descriptive statistics for physical activity measures are presented in Table 3-3. All variables were checked for outliers ($\geq \pm 3$ standard deviations), and outliers were trimmed to three standard deviations for three participants. Distribution of all outcome variables were checked to assess normality and no transformations were required. To test the validity of the GNG task, behavioural (Accuracy, RT) and ERP measures (N2 amplitude, N2 latency, P3 amplitude, P3 latency) on the GNG task were analyzed using repeated measures ANCOVA, controlling for age and task version, with trial type (Go, No-go) as a within-subject factor. Only for the analyses of N2 amplitude and latency, laterality (Left, Midline, Right) was included as a within-subject factor. Greenhouse-Geisser correction was applied in cases where sphericity assumptions were violated. Means are reported as least-squares means, and partial eta-squared (η^2) is reported as a measure of effect size for all statistically significant findings. Grand averaged ERP waveforms are presented in Figure 3-3. *Primary analyses: The association between physical activity and response inhibition*

To examine the association between physical activity and response inhibition, first, partial correlations between physical activity and response inhibition measures were assessed, while controlling for age (Table 3-4). To limit the number of variables, only the measures relevant to the investigation (behavioural accuracy and ERP amplitude and latency measures on No-go trials) were included. Further, for the N2 ERP measures, only the electrodes showing maximal activity (Frontal left electrode cluster, as assessed from preliminary ERP analyses) were included.

Next, we performed hierarchical regression analysis to follow-up on any significant associations that were observed between physical activity and response inhibition measures, as revealed by the correlations analyses. The regression model included age, task version, and maternal education as covariates. Maternal education was included as a proxy for socio economic status. As with the correlations, analyses were limited to No-go measures and electrode clusters showing maximal activity. Statistical significance was determined using p < .05 as the criteria.

Results

Preliminary analyses

Behavioural and ERP measures of response inhibition

Repeated measures ANCOVA, controlling for age and task version, with trial type (Go, No-go) as a within-subject factor was carried out to assess children's response inhibition performance and the ERP components, N2 and P3. Descriptive statistics are presented in Table 3-2.

There was a main effect of trial type for both accuracy (F(1, 54) = 26.92, p < .01, $\eta^2 = ..333$) and RT (F(1, 45) = 6.16, p < .05, $\eta^2 = .120$). Unexpectedly, children displayed greater accuracy on No-go trials (M = .92, SD = .07) relative to Go trials (M = .85, SD = .08). As is typical of inhibitory failures, children were faster on No-go trials (M = 826.88 ms , SD = 355.65) than on Go trials (M = 928.45 ms, SD = 195.02). There were no significant effects of age or task version for accuracy or RT.

For N2 amplitude, there were main effects of trial type (F(1, 54) = 17.73, p < .01, $\eta^2 = .247$), and laterality (F(2, 108) = 116.57, p < .01, $\eta^2 = .683$). N2 amplitude was greater on No-go trials ($M = -9.30 \mu$ V, SD = .62) than on Go trials ($M = -7.35 \mu$ V, SD = .49), indicating greater engagement of neural resources on No-go trials. N2 amplitude was also greater at the left electrodes ($M = -14.27 \mu$ V, SD = .77) than at the midline ($M = -5.94 \mu$ V, SD = .62) or right electrodes ($M = -4.77 \mu$ V, SD = .50).

For N2 latency, there was a main effect of laterality (F(2, 108) = 25.18, p < .01, $\eta^2 = ...318$). N2 latency was longer at the left electrodes (M = 398.33 ms, SD = 5.54) than at the midline (M = 358.26 ms, SD = 6.83), or right electrodes (M = 345.23 ms, SD = 6.71), which did not differ (p > .05). There was also an interaction between trial type and laterality (F(2, 108) = 4.76, p < .05, $\eta^2 = .081$). On both Go and No-go trials, N2 peaked later at the left electrodes (Go:

M = 402.31 ms, SD = 6.09; No-go: M = 394.35 ms, SD = 6.88) than at the midline (Go: M = 347.69 ms, SD = 7.58; No-go: M = 368.84 ms, SD = 7.96), or right electrodes (Go: M = 338.37 ms, SD = 7.92; No-go: M = 352.09 ms, SD = 7.90). N2 latency did not differ between midline or right electrodes on both Go and No-go trials (p > .05).

For P3 amplitude, there was a marginally significant main effect of trial type (F(1, 54) = $3.45, p = .07, \eta^2 = .060$), with a trend of greater P3 amplitude on No-go trials ($M = 27.45 \mu$ V, SD = 1.47) than on Go trials ($M = 26.02 \mu$ V, SD = 1.37). For P3 latency, no main effects or interactions were significant.

Age and task version were not significant for N2 or P3 amplitude or latency.

Correlations between behavioural and ERP measures of response inhibition

Partial correlations controlling for age are presented in Table 3-4. There were no significant associations between behavioural measures and N2 amplitude/latency measures of response inhibition.

Correlations between objective and subjective measures of physical activity

Partial correlations controlling for age are presented in Table 3-4. Total objective physical activity was significantly correlated with total subjective physical activity (r = 0.26, p < .05). After controlling for age, the correlation between total objective physical activity and total subjective physical activity was marginally significant (r = 0.24, p = .073).

Primary analyses

Correlations between measures of physical activity and response inhibition

Partial correlations controlling for age are presented in Table 3-4. No-go accuracy was not correlated with any physical activity measures.

For ERP measures, only the association between P3 latency on No-go trials and nonorganized activities was significant (r = -0.29, p < .01). Children with who spent more time on non-organized activities had a longer P3 latency on No-go trials.

Hierarchical regression analyses

To examine if physical activity predicted response inhibition measures, a series of hierarchical regression analyses were performed and the results are presented in Table 3-5. To limit the number of analyses performed, findings from the correlational analyses were used to select the predictors and outcome variables tested in the regression models. Parent-reported participation in non-organized activities was correlated with No-go P3 latency, so we tested if participation in non-organized activities predicted No-go P3 latency. At the first step in the hierarchical regression analyses, age, task version, and maternal education were entered into the model to control for potential confounds. At the second step, non-organized physical activity participation was added to test if it predicted No-go P3 latency after controlling for the covariates.

Non-organized activity significantly predicted No-go P3 latency ($\beta = -.319$, t(52) = -2.43, p < .05). However, the regression model did not reach significance ($R^2 = .141$, F(5, 52) = 1.71, p = .15).

Discussion

Our objective in this study was to examine the association between physical activity and response inhibition in early childhood. We obtained both objective and subjective measures of physical activity to assess if individual differences in physical activity predicted inhibitory performance on a GNG task and the ERP components associated with response inhibition. We found that subjectively assessed participation in non-organized activities predicted No-go P3

latency. However, contrary to our prediction, we did not find children who were more physically active to display better response inhibition performance, as indexed by No-go accuracy. In addition, physical activity was not associated with N2 or P3 amplitudes. These findings are further discussed below. We also address some limitations present in our study that may have contributed to the discrepancy in the findings between this present study and previous findings in adults and school-aged children.

Associations between physical activity and response inhibition

Our findings revealed that only one measure of response inhibition, No-go P3 latency, was significantly association with physical activity in children. Children with higher levels of non-organized activity had a shorter P3 latency on No-go trials. Considering the role of the P3 in inhibitory processes, this suggests that the activation of the response inhibition process occurred at a faster pace in children with greater participation in non-organized activities. The P3 has also been associated with post-perceptual evaluative processes such as the updating of the occurrence of task-relevant events (Donchin, 1981). Interpreting our P3 findings in light of this alternate account, it appears that children with greater participation in non-organized activities were faster in evaluating and updating the occurrence of No-go trials. This pattern of finding-that higher physical activity levels is associated with faster, more efficient inhibitory or evaluative processing in children—is consistent with findings in adults where physical activity has been associated with faster information processing (Desjardins-Crépeau et al., 2016; Hillman et al., 2006). Physical activity has been associated with greater white matter integrity (Chaddock-Heyman et al., 2014; Gons et al., 2018) and myelination (Feter et al., 2018). The structural integrity of the white matter in the brain is important for efficient communication between different brain regions (Magistro et al., 2015), and this could be one mechanism contributing to

the earlier P3 observed on the No-go trials in children who had higher participation in nonorganized physical activities. However, the association between physical activities and white matter integrity has thus far only been investigated in school-aged children (Chaddock-Heyman et al., 2014) and adults (Gons et al., 2018). This is a potential avenue to be explored in future investigations and would expand our understanding on the effects of physical activity in early childhood.

A vital point to note in this observation is that the association was observed with nonorganized activities that were subjectively assessed through parental reports. Subjective parental reports are likely to have captured physical activities that are functional and intentional due to the definition of physical activity included in the questionnaire that parents completed (i.e., that makes him/her out of breath or warmer than usual). On the other hand, as the actigraph accelerometers were worn by children during the vast majority of their waking hours, it is likely to have captured activities that are both functional and non-functional. Hence, differences in the nature of activities captured by objective and subjective measures of physical activity could explain the lack of association with objectively assessed measures of physical activity. This also raises the importance of considering the nature and type of physical activities when considering the effects of physical activity in early childhood.

In our study, No-go accuracy, as well as N2 and P3 amplitude measures of response inhibition were not associated with physical activity in children. While this finding is unexpected given the vast majority of studies reporting a positive association (Campbell et al., 2002; Chu et al., 2015; Hillman et al, 2009; 2014), similar findings were also reported in one study with preschoolers. In this study, Palmers and colleagues (2013) reported a lack of association between objectively measured physical activity and response inhibition performance in four-year-olds. The authors acknowledged that the response inhibition task that they utilized may not have been sufficiently sensitive, and that this could have contributed to the lack of an association. When examining children's performance on the GNG task in our study, we find that children's accuracy on No-go trials was significantly greater than their accuracy on Go trials. While this may prompt questions on the inhibitory requirements of the task, our ERP findings provide evidence that the task was tapping on inhibitory abilities. Specifically, we found that the N2 amplitude was more pronounced on No-go trials than on Go trials. P3 amplitude showed a similar pattern, although this effect was only marginally significant. Together, our ERP findings provide evidence of inhibitory demands being present of the GNG task. There are, however, several other limitations of our study that may have contributed to these findings, and these are discussed below.

Study Strengths and Limitations

One of the main strengths of this study was the inclusion of both objective and subjective measures of physical activity. While the two previous studies examining the association between physical activity and response inhibition in early childhood had used accelerometry to measure physical activity (Campbell et al., 2002; Palmer et al., 2013), this study is the first to employ both accelerometry and subjective reports of physical activity. Objectively measuring physical activity using accelerometry is considered to be a more sensitive method (Adamo, Prince, Tricco, Connor-Gorber, & Tremblay, 2009), and the higher values captured by the accelerometer relative to parental subjective reports attests to this. However, it also suggests that subjective measures may be capturing more purposeful physical activity rather than incidental physical activity. This is likely due to the definition of physical activity provided in the questionnaire that parents completed (i.e., that makes him/her out of breath or warmer than usual).

In interpreting the study findings, several limitations of this study must be kept in mind. Although we argued that the high No-go accuracy does not indicate a lack of inhibitory demands in our task, it could have contributed to the lack of association with physical activity measures in other ways. Specifically, the ceiling level performance indicates that the task was not too challenging for children. As previous findings have shown that the beneficial effects of physical activity are most pronounced when the cognitive task is sufficiently challenging (Ludyga et al., 2016; Sibley & Beilock, 2007), and in low performers (Drollete et al., 2013), the lack of finding an association between physical activity and response inhibition in our study may have been due to the task not being sufficiently difficult for children.

Our study examined individual differences in physical activity levels in children. While this is a strength of the study in that we examined naturally occurring differences in physical activity as opposed to experimentally created conditions, this methodology differs from those adopted in many of the previous findings where positive associations were reported (Campbell et al., 2002; Chu et al., 2015; Hillman et al, 2009; 2014). This also means that the context under which children engaged in physical activity, as well as the nature of the physical activity were not controlled in this study. Such information may be vital in order to achieve a greater understanding of the relationship between physical activity and response inhibition, especially since one of the mechanisms that have been proposed to explain the beneficial effects of physical activity on cognitive functioning concerns the complexity of movements and the extent to which the physical activity is cognitively engaging. As such, future studies investigating this relationship should attempt to capture this information.

This study is the first attempt to investigate the association between physical activity and the neural correlates underlying response inhibition in early childhood. Although the association between physical activity and some of the ERP indices of response inhibition (N2/P3 amplitude)

did not reach significance, these null findings must be interpreted with caution. Given the modest sample size of this study, we had sufficient power to detect only effects in the medium to large range. The effect sizes for the regression analyses of the ERP indices were small ($R^2 = 0.02 - 0.10$), and it is possible that these effects may have attained significance with a larger sample size. This is an issue that needs to be investigated in future studies with a larger sample size.

Conclusion

This study is a crucial first step towards the examining the association between physical activity and the neural correlates underlying response inhibition in early childhood. This is an important avenue of investigation given our understanding of the role that environmental factors play in guiding brain development in these early years. Our findings showed that physical activity predicted the latency of the No-go P3 ERP component in children. Physical activity was not associated with any other indices of response inhibition.

Physical activity has been found to be an effective intervention tool in improving cognitive functioning in the ageing literature (Lautenschlager et al., 2008; Nascimento et al., 2015), as well as in children who are overweight (Davis et al., 2011), or who have been diagnosed with ADHD (Hoza et al., 2015; Smith et al, 2013). In order to determine the potential for its application in early childhood, there is a need for continued investigation of this relationship, while addressing the limitations of this study that were highlighted.

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Variables	Mean (SD)	Range	Skewness	Kurtosis
Age (months)	45.47 (9)	30 - 60	-0.18	-1.20
Male (<i>n</i> = 22)	44.82 (9)	31 - 60	-0.02	-1.12
Female $(n = 36)$	45.86 (9)	30 - 59	-0.31	-1.20
IQ (PPVT)	117.57 (11)	97 - 142	0.21	-0.58
Maternal Education (years)	16.91 (2)	12 - 20	0.10	-1.03

Table 3-1. Descriptive Statistics for Demographic Variables

Variables	Mean (SD)		Ra	nge	Skewness		Kurtosis	
	Go	No-go	Go	No-go	Go	No-go	Go	No-go
Accuracy (%)	.85 (.08)	.92 (.07)	.6598	.70 – 1.0	-0.51	-0.95	-0.56	0.86
RT (ms)	946.2 (190)	826.8 (356)	607.4 - 1495.2	339 - 1980.3	0.47	1.51	-0.03	2.59
N2 Amplitude (µV)								
Left	-13.39 (6.1)	-15.14 (6.5)	-30.34 - 0.92	-30.721.35	-0.36	-0.09	0.10	-0.41
Midline	-4.77 (4.9)	-7.20 (6.0)	-23.64 - 5.51	-26.71 - 5.43	0.77	-0.75	2.59	0.94
Right	-3.97 (3.3)	-5.58 (5.7)	-13.52 - 5.83	-19.1 - 7.79	-0.40	-0.25	1.67	0.13
N2 Latency (ms)								
Left	402.3 (47)	394.3 (53)	286.7 - 464.0	246.7 - 464.0	-0.53	-0.60	-0.70	-0.20
Midline	347.7 (59)	368.8 (60)	185.3 - 461.0	212.0 - 464.0	-0.39	-0.74	-0.05	0.24
Right	338.4 (59)	352.1 (62)	205.3 - 450.7	192.0 - 456.0	-0.41	-0.44	-0.21	-0.19
P3 Amplitude (µV)								
Midline	26.08 (10.4)	27.45 (11.0)	7.94 - 56.35	4.33 - 54.56	0.72	0.71	1.43	0.32
P3 Latency (ms)								
Midline	453.5 (88.0)	465.1 (91)	316.0 - 616.0	316.0 - 616.0	0.09	-0.07	-0.93	-1.25

 Table 3-2. Descriptive Statistics for Behavioural and ERP Measures of Response Inhibition

Variables	Mean (SD)	Range	Skewness	Kurtosis		
LPA	263.55 (28.7)	186.59 - 322.30	-0.39	0.36		
MVPA	87.17 (27.9)	27.22 - 148.54	0.30	-0.25		
Total Objective PA	350.72 (43.1)	265.52 - 438.37	-0.19	-0.80		
Organized Activities	15.22 (11.97)	0 - 60	1.76	5.62		
Non-organized Activities	43.37 (14.76)	12.86 - 60	-0.49	-0.81		
Total Subjective PA	58.60 (15.76)	21.43 - 102.86	0.01	0.26		

 Table 3-3. Descriptive Statistics for Objective and Subjective Measures of Physical Activity (mins/day).

LPA = Light intensity physical activity; MVPA = Moderate-to-vigorous-intensity physical activity; PA = Physical activity

	1	2	3	4	5	6	7	8	9	10	11	12
1. LPA	-											
2. MVPA	0.19	-										
3. Total Objective PA	0.79**	0.75**	-									
4. Organized PA	0.10	-0.10	0.00	-								
5. Non-organized PA	0.19	0.20	0.25+	-0.34*	-							
6. Total Subjective PA	0.25+	0.11	0.24+	0.42**	0.71**	-						
7. No-go Accuracy	-0.13	-0.12	-0.16	-0.06	0.04	-0.00	-					
8. No-go RT	0.23	0.37*	0.39**	-0.16	0.05	-0.08	-0.30*	-				
9. N2 NG Amplitude	0.00	-0.05	-0.03	-0.04	-0.09	-0.11	-0.05	0.02	-			
10. P3 NG Amplitude	-0.19	0.09	-0.07	-0.09	-0.12	-0.18	0.03	-0.14	-0.48**	-		
11. N2 NG Latency	0.19	-0.06	0.10	0.01	0.01	0.01	-0.05	0.11	-0.52**	0.37**	-	
12. P3 NG Latency	0.16	0.05	0.14	0.16	-0.29*	-0.16	-0.02	-0.29*	0.16	-0.03	0.02	-

Table 3-4. Partial Correlations between Measures of Physical Activity and Response Inhibition (Behavioural and ERP),

Controlling for Age

*******p* < 0.01; **p* < 0.05; *p* < 0.10. *LPA* = *light-intensity physical activity; MVPA* = *Moderate- to vigorous-intensity physical activity; PA* = *Physical activity; NG* = *No-go; RT* = *Reaction Time*

No-go P3 Latency ΔR^2 Predictor *b (SE)* β Model 3 .044 Step 1 Intercept 465.48 (12.11) 2.90 (4.32) Age -.296 Task Version 1 25.21 (20.21) -.205 Task Version 2 -35.29 (40.86) -.404 Maternal Education 4.33 (5.01) .116 .098* Step 2 Intercept 465.69 (11.58) Age 2.94 (4.13) .300 Task Version 1 24.32 (19.34) .198 -35.41 (39.09) Task Version 2 -.406

Table 3-5. Hierarchical Regression Analyses for No-go P3 Latency, Reporting Unstandardized

and Standardized Beta Coefficients, and ΔR^2

Maternal Education

Non-organized Activities

* p < 0.01, ** p < 0.05; MVPA = Moderate- to vigorous-intensity physical activity; $PA = Physical \ activity$

6.67 (4.89)

-1.96 (0.81) *

.179

-.319*



Figure 3-1. Schematic diagram outlining task flow. Stimulus duration of Go and No-go trials were adjusted according to the child's age and varied between 1500 – 2500 ms.



Figure 3-2. An Illustration of the electrode clusters used in the analyses of the N2 and P3 ERP components. N2 was calculated at Frontal (left, midline and right) electrode clusters, and P3 was analyzed at mid-parietal electrode cluster.



Figure 3-3. Grand-averaged waveforms of the N2 (left) and the P3 (right). Time windows selected for the analyses of the N2 and P3 are indicated by the rectangular box in the figure above. The N2 was analyzed between 260 - 560 ms and was maximal at the frontal left electrode cluster (presented above). TheP3 was analyzed between 310 - 610 ms after stimulus onset and was maximal at mid parietal electrode cluster.

CHAPTER 4

Valence matters: An electrophysiological study on how emotions influence cognitive

performance in children

Emotions are ubiquitous. We experience and encounter them on a daily basis, especially in the form of facial expressions. Emotions inform us about situations, as well as actions and mental states of others (Cole, Martin, & Dennis, 2006; Lemerise & Arsenio, 2000). Importantly, emotions influence our behaviour and cognitive performance even when present 'in the background' and not directly task-relevant (Anderson, 2005; Brown & Kulik, 1977; Gray, 2004). Existing evidence indicates that emotions both facilitate (Kanske & Kotz, 2010) and impair cognitive performance (Cohen-Gilbert & Thomas, 2013), and the direction of this influence may depend on the valence of the emotion (Augusti, Torheim, & Melinder, 2012; Lewis, Todd, & Honsberger, 2007).

Although a large corpus of research has addressed how emotions influence cognitive performance in adults, fewer studies have examined this relationship in children. A greater understanding of this relationship is crucial as childhood is marked by rapid growth in emotion processing and cognitive abilities (Gross & Ballif, 1991; Wiebe, Sheffield, & Espy, 2012). Moreover, studies in adults show that emotional influence on cognitive performance is not static. In particular, when the cognitive control demands of a task are increased, emotional influence is attenuated (Cohen, Henik, & Mor, 2011; Lim, Padmala, & Pessoa, 2008). In children, we know much less about how emotional influence on cognitive performance is affected by the cognitive control demands of a task. This is an important question to address as both cognitive control abilities, and the underlying brain regions are still developing in children (Gogtay et al., 2004; Sowell, Thompson, & Toga, 2004). Our goals in the present study were to examine how emotions influence cognitive performance in children, and to address whether this influence is modulated by emotional valence and cognitive control demands. To understand how these

interactions are reflected in the neural correlates underlying cognitive performance, we examined electrophysiological measures alongside cognitive performance.

The effect of emotions on cognitive performance in children

The Dual Competition Model (DCM; Pessoa, 2009) is a framework explaining emotional influence on cognitive performance. It posits that emotions receive preferential processing and enhanced representation due to their salience. However, emotions' effect on cognitive performance is contingent on whether they lead to a competition for neural resources. Emotions that are threatening or task-irrelevant would recruit neural resources away from the task, resulting in impaired cognitive performance. Emotions that are task-irrelevant and low on threat threatening would also impair cognitive performance, but to a lesser extent. On the other hand, emotions that are task-relevant and low on threat would facilitate cognitive performance due to the enhanced perceptual representation that they receive. While the DCM has garnered support from findings in adults (Pessoa, Padmala, Kenzer, & Bauer, 2011), it has been less frequently studied in children (Augusti et al., 2012; Urben, Van der Linden, & Barisnikov, 2012).

Children make significant progress in processing and identifying emotions in the early years. Within the first year of life, infants are able to discriminate emotional expressions and respond to them appropriately (Sorce, Emde, Campos, & Klinnert, 1985). By the age of three years, children can use emotional words and label emotional expressions accurately (Ridgeway, Waters, & Kuczaj, 1985). Children get better in processing emotions as they grow older (Kolb, Wilson, & Taylor, 1992), and this developmental trajectory differs across emotions. In particular, early on, children show an advantage in recognizing happy emotions, with slower growth in proficiency for the other emotions (Gao & Maurer, 2010; Montirosso et al., 2008; Todd, Evans, Morris, Lewis, & Taylor, 2011). The advantage for happy emotions extends beyond recognition. Several studies have found that children display a 'positivity bias' on cognitive tasks with emotional stimuli, with better cognitive performance observed in the presence of happy relative to other emotional stimuli (Augusti et al., 2012; Bahn, Vesker, García Alanis, Schwarzer, & Kauschke, 2017; Lewis et al., 2007; Tottenham, Hare, & Casey, 2011). At the same time, children's cognitive performance is hampered in the presence of negative emotions (Augusti et al, 2012; Lewis et al., 2007). However, other studies have found that the inclusion of emotions, including happy emotions, renders a cognitive task more challenging for children (Lagatutta, Sayfan, & Monsour, 2011). Overall, despite some inconsistencies, studies investigating how emotions influence cognitive performance in children generally indicate that the direction of this influence may be dependent on its valence. Importantly, the bias for positive emotion to facilitate cognitive performance in children has been found both when it was relevant and irrelevant to the task, counter to the predictions made by the DCM.

While there is evidence that emotion processing and recognition develops over early and middle childhood (Montirosso et al., 2008; Todd et al., 2011), it is unclear if the effect that emotions exert on cognitive performance also changes with development. As children become more adept in processing emotions and make strides in their cognitive abilities, the manner in which emotions influence their cognitive performance may change. For example, in one study, the extent to which negative emotions impaired cognitive performance decreased between a younger group of nine- to ten-year-olds and an older group of eleven- to twelve-year-olds (Augusti et al., 2012). In contrast, another study found that emotional influence on cognitive performance did not differ between middle childhood and adolescence (Urben et al., 2012).

These studies, however, did not examine if the effect that emotions had on cognitive performance changed between early and middle childhood.

The effect of cognitive control demands on emotional influence

Even as there is converging evidence indicating that emotions influence cognitive performance, there is also some indication that this influence is modulated by cognitive control. Cognitive control refers to top-down processes that support goal-directed behaviour (Miller, 2000). In adults, when the need for cognitive control is increased, emotional influence on cognitive performance is attenuated (Cohen et al., 2011; Lim et al., 2008). These findings have been interpreted in terms of a limited resources model: as increased cognitive control demands require greater resources, top-down control mechanisms limit the processing of emotions and their influence on cognitive performance. This interpretation is also consistent with brain imaging findings showing increased activity in the dorsolateral prefrontal cortex and the anterior cingulate cortex, coupled with decreased activity in the amygdala under conditions of increased cognitive control demands (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Kanske & Kotz, 2011a; Mohanty et al., 2007).

These control processes and their underlying brain regions are immature in children and continue to develop into early adulthood (Crone, Peters, & Steinbeis, 2018; Gogtay et al., 2004; Sowell et al., 2004). To date, only a few studies have investigated how varying cognitive control demands modulates emotional influence on cognitive performance in children (Lewis et al., 2007; Tottenham et al., 2011; Urben, et al., 2012). These studies have used tasks assessing response inhibition, namely the Go/No-go task (GNG) and the Stop-signal task (SST), and cognitive control demands were manipulated by varying inhibitory load. Both tasks require children to respond to a frequently occurring stimulus (Go) while inhibiting response to a less

frequently occurring stimulus (No-go/Stop). Because responding is required more frequently, greater cognitive control is needed to overcome this dominant response behaviour on trials requiring inhibition. These studies all found that when cognitive control demands were low, positive emotions facilitated cognitive performance (Lewis et al., 2007; Tottenham et al., 2011; Urben et al., 2012). On trials with greater cognitive control demands, both positive and negative emotions impaired performance (Tottenham et al., 2011; Urben et al., 2012). Although these studies with children indicate that emotional influence on cognitive performance varies with cognitive control demands, the nature of the relationship differed from that seen in adults:

These developmental studies give some indication of how cognitive control demands modulate emotional influence on children's cognitive performance. However, a drawback of the tasks used in these studies is that response requirements between trials requiring high and low cognitive control also differed. Go trials (lower cognitive control demands) required a response whereas No-go or Stop trials (greater cognitive control demands) did not. These differences in response requirements could be important because in adults positive emotions have been shown to elicit approach, whereas negative emotions elicit avoidance (Roelofs, Minelli, Mars, van Peer & Toni, 2009). Hence, the compatibility between the behavior primed by the emotion and the response requirements of the task may have contributed to the findings (i.e., facilitation observed for positive emotions on trials with lower cognitive demands may have arisen because approach behavior elicited by positive emotion is compatible with the Go response). This problem can be circumvented by utilizing tasks that require inhibition, but without the confounding response requirement differences. The flanker task (Eriksen & Eriksen, 1974) is commonly used to assess inhibition of irrelevant information, a process also referred to as interference suppression. In the flanker task, a central target stimulus (e.g., >) is flanked by congruent (e.g., >>>) or incongruent (e.g., <><) distractors, and participants are required to respond to the central target. Cognitive control demands are greater on incongruent trials, as the conflicting information provided by the distractors has to be inhibited. This is typically reflected in the longer reaction times (RT) observed on incongruent trials. Children are able to perform well on flanker tasks by the age of four (Rueda et al., 2004). Hence, the task provides a platform to examine how emotions influence cognitive performance, and to assess the impact of cognitive control demands on this influence using a single paradigm with identical response requirements across cognitive control conditions.

Neural correlates of emotion-cognition interactions

Electrophysiological methods can provide a window into the time course of emotional influence on cognitive performance, due to their excellent temporal resolution. For example, studies using event-related potentials (ERPs) have found emotional effects on the N2, an ERP predominantly seen at frontocentral electrodes about 200 – 500 ms after stimulus onset (Carter & Van Veen, 2007; Jonkman, Sniedt, & Kremner, 2007). The N2 has been associated with inhibitory processes, displaying a more pronounced amplitude under conditions requiring inhibition, as in the incongruent trials of a flanker task (Bartholow et al., 2005; van Veen & Carter, 2002). In inhibitory tasks assessing emotional influence, N2 amplitude is typically enhanced on emotional trials relative to neutral trials, and this pattern has been observed in both adults (Kanske & Kotz, 2010; 2011b) and children (Lamm, White, Martin, & Fox, 2012; Lewis et al., 2007). In one study of children, N2 latency was also modulated by emotions, with the N2 occurring earlier for negative emotions (Lewis et al., 2007). Furthermore, the effect of emotions on N2 amplitude has been found to vary with cognitive control demands. In a study of nine-year-

old children, the N2 amplitude was enhanced by emotional stimuli only under high cognitive control demands (Lamm et al., 2012). In contrast, a study of four- to six-year-olds reported that the N2 amplitude was enhanced by emotional stimuli only under low cognitive control demands (Lewis et al., 2007). These contrasting findings may be explained by the age differences between samples, as children have more mature cognitive and emotion processing abilities in middle than early childhood. However, both these studies used a GNG task, introducing concerns about the role of differing response requirements between trial types.

Study goals and hypotheses

In this study, our goal was to understand how emotions influence cognitive performance in children. Two groups of children representing early and middle childhood completed a flanker task with task-irrelevant, emotional facial stimuli (happy, angry, and neutral faces). The decision to use happy and angry emotions was based on previous findings reporting valence effects in children, and also to ensure that the emotions differed in valence but not arousal. Children were instructed to respond based on the color of the border around the central, target face, regardless of facial emotion. This meant that emotion was embedded in the target stimuli but was irrelevant to the task that children were performing. The two flankers at the sides of the target were neutral faces on all trials, but the color of the border around the flankers was manipulated to be congruent or incongruent to that around the target. This task design allowed us to examine whether emotional influence on children's cognitive performance is modulated by (1) the valence (happy vs angry) and (2) the cognitive control demands of the task (congruent vs incongruent). In addition, we wanted to examine (3) how emotions influenced the neural correlates underlying cognitive performance, the amplitude and latency of the N2. Finally, as children experience rapid growth in their emotion processing and cognitive abilities in the

transition from early to middle childhood, we examined (4) if the influence of emotion on cognitive performance differed between age groups representing these developmental periods.

Based on previous findings with children (Lewis et al., 2007), we hypothesized that the manner in which emotions influence cognitive performance in children would be contingent on the valence of the emotion. Specifically, we hypothesized that children would perform better on trials with happy targets than on trials with neutral or angry targets, displaying the 'positivity' bias' found in previous studies (Augusti et al., 2012; Bahn et al., 2017; Lewis et al., 2007). Following previous findings showing cognitive performance in children to be hampered by negative stimuli (Augusti et al., 2012; Lewis et al., 2007), as well as DCM's prediction that threatening emotions would divert resources away from the cognitive task, we also hypothesized that children would perform worse on trials with angry targets than on trials with happy or neutral targets. Because cognitive control capacities improve with age, we hypothesized that the influence of emotions on cognitive performance would be stronger in younger children than in older children. In addition, following previous ERP findings, we predicted that the influence of emotions on the N2 would be modulated by the valence of the emotion and that its amplitude will be greater and latency earlier for trials with angry faces than on trials with happy or neutral faces (Lewis et al., 2007).

Methods

Participants

The study was conducted in accordance with the American Psychological Association ethical guidelines, and with the approval of the university Research Ethics Board. The final sample included a total of 51 children (25 girls, 26 boys) in two age groups. The younger group included 28 children (14 girls, 14 boys) between 4;5 and 6;0 years (M = 5;3 years, SD = 5 months), and the older group included 23 children (11 girls, 12 boys) between 7;0 and 8;5 years (M = 7;5 years, SD = 6 months). An additional 9 children visited the lab but did not provide usable data due to equipment failure (n = 2) or refusal to complete the task (n = 7). Data from 14 children were excluded from ERP analyses due to insufficient artifact-free trials. The ERP subsample included 37 children (15 girls, 22 boys; 20 younger children; 17 older children) The sample's average IQ was 111, measured using the Peabody Picture Vocabulary Test (PPVT-4; Dunn & Dunn, 2007). Children excluded from ERP analyses did not differ from children included in the ERP analyses in IQ or age. The median level of maternal education was 16 years. Participants were recruited through online advertising, as well as flyers distributed at preschools, playgroups, daycares, and elementary schools. Parents described their children as European-Canadian (n = 29), African-Canadian (n = 3), Asian-Canadian (n = 6), mixed ethnicity (n = 10), or others (n = 3). Parents reported that all children were born full-term (≥ 37 weeks), had no history of neurological or behavioral disorders, and that English was the primary language spoken in the home.

Procedures

The study was carried out at a child development laboratory at a university in a mid-sized Western Canadian city. Parents accompanied their children to the lab and were briefed about the study before providing informed consent. After EEG net application, children completed two tasks, with the Face Flanker task administered second. The first task was unrelated to the present investigation and was the same for all children. Parents remained in the testing room throughout the session and completed several questionnaires on family demographics, child temperament, and behavior. At the end of the visit, all children received a small toy, and parents received a gift card in appreciation.

The Flanker Task. A computerized flanker task (Figure 4-1) was framed as a soccer game to make it engaging for children. Children were told that they would see pictures of soccer players and they would be helping to group them into two teams — a Red and a Blue team. They were instructed to do so by pressing either a red or blue button on a button box that matched the color of the border around the central target face. Children were told to ignore the distractor faces on both sides of the target face as they sometimes gave incorrect information. They were also told that sometimes the players might look angry or happy but that they should ignore the facial expression.

Each trial began with a black fixation cross presented centrally, lasting for 1000 ms. This was followed by the presentation of three identical faces — a target face presented centrally, flanked horizontally by two distractor faces. Each face had a red or blue border around it. On congruent trials, the color of the border around both the target and distractors were the same. On incongruent trials, the color of the border around the target differed from those around the distractors. The target face displayed either a neutral or emotional (happy or angry) expression while the distractor faces always displayed neutral expressions. On each trial, the target and distractor faces remained on the screen for a maximum of 2000 ms and ended when the child responded. This was followed by a feedback display that lasted for 1500 ms. On correct trials, the feedback was a smiling thumbs-up sign with the sound of an applause. On incorrect trials, the feedback was a frowning thumbs-down sign with the sound of a buzzer.

The task took 15 - 20 minutes to administer, and comprised 144 test trials divided into 6 blocks. Before the test trials, children were given 44 practice trials to familiarize them with the task and to ensure that they understood task instructions. The task included an equal number of

congruent and incongruent trials. Within these categories, there were an equal number of targets with red or blue border. Target gender (male, female) and target emotion (neutral, happy, angry) were also presented at equal numbers across the categories. Stimuli were presented in a fixed, pseudo-randomized order for all participants. The same face (model) was not presented in any two consecutive trials and the same target emotion was not presented in more than three consecutive trials. The positions of the red and blue buttons on the button box were counterbalanced across participants.

The task was presented on a 20-inch Dell monitor using E-Prime 2.0 Professional (Psychology Software Tools, Sharpsburg, PA) at a viewing distance of 30 cm. Responses were collected using a button box (Electrical Geodesics, Model C-PKG-RSP-1000-001).

Task stimuli were selected from the NimStim Set of Facial Expressions (Tottenham et al., 2009). Faces of 12 Caucasian models (6 male, 6 female) displaying calm, happy, and angry expressions were selected. Calm exemplars were selected to serve as the neutral expression in our study as previous studies have reported that children perceive the more prototypical neutral expressions to be ambiguous or negative (Gross & Baliff, 1991; Thomas et al., 2001; Tottenham et al., 2009). The selected stimuli had high reliability and validity as exemplars of the emotions (Tottenham et al., 2009). A total of 36 face stimuli (12 models x 3 expressions were used in the task. An additional 18 face stimuli were used for training and practice trials. All stimuli measured 3 cm by 3 cm and were gray-scaled images of equal luminance. The horizontal distance between the target and each distractor face was 1.2 cm.

Electrophysiological Recording and Data Processing. EEG was recorded using a 128channel Hydrocel Geodesic Sensor Net (Electrical Geodesics Inc., Eugene, OR). First, children's head circumference was measured to allow selection of the appropriate net, and the vertex was marked to ensure correct net placement. The net was soaked in an electrolyte solution prior to application. Electrode impedances were measured and maintained below 50 k Ω . If needed, additional electrolyte solution was applied to electrodes with high impedances. During task performance, EEG data was recorded continuously at a sampling rate of 250 Hz with the vertex used as the reference.

ERP analysis was conducted with Net Station software (Version 4.3.1, Electrical Geodesics Inc., Eugene, OR). A 0.3-30 Hz bandpass filter was used to filter data offline. EEG data was segmented into 1100 ms epochs beginning 100 ms before stimulus onset and ending 1000 ms post-stimulus. Artifact detection was performed on segmented files and all electrodes where signal fluctuations exceeded 200 µV were marked bad. Segments with more than 12 bad electrodes were rejected. Spline interpolation was used to replace bad electrodes in otherwise acceptable segments. EOG correction was conducted using Gratton's algorithm (Gratton, Coles & Donchin, 1983). In addition, the first author visually inspected all segments for EOG or movement artifacts, and corrected segment and channel markups if needed. Following EOG correction, channels were reassessed for artifacts with previously marked information overwritten if necessary (some channels that were previously marked bad may be acceptable after EOG correction) and then bad electrodes replaced using spline interpolation. All usable segments were averaged separately for congruency (congruent, incongruent), emotion (neutral, happy, angry), and age group (young, old). EEG data was re-referenced to an average reference using the polar average reference effect correction (Junghöfer, Elbert, Tucker, & Braun, 1999), and then baseline-corrected using the data 100 ms prior to stimulus onset. Incorrect trials and trials with a RT less than 200 ms were excluded from analysis as they do not reflect deliberate

behaviour. ERP data of all participants included in the analysis had a minimum to 10 artifact-free trials in each condition.

Adaptive mean and peak latency measures were calculated for the N2 ERP component. As visual inspection of the waveforms showed that a sustained, late frontal negativity (LFN) differed across emotions, the LFN was also examined; mean amplitude was calculated because the LFN did not have a distinct peak. In addition, as the task included emotional face stimuli, emotion and face-related ERPs, P1 and N170 were calculated to ensure that any effects observed on the N2 were not due to early processing differences. The N2 and LFN were examined at the frontal electrode sites. The N2 was defined as a negative deflection in the time window between 200 and 450 ms and the LFN was defined as a slow negative wave occurring within the time window between 450 and 700 ms. P1 and N170 were examined at temporal-occipital electrode sites. P1 was defined as the first positive peak occurring within 80 - 120 ms and N170 was defined as the first negative peak occurring 130 - 240 ms. The time windows were determined after considering previous studies with similar age groups (Batty & Taylor, 2006; Buss, Dennis, Brooker, & Sippel, 2011; Rueda, Posner, Rothbart, & Davis-Stober, 2004), and visual examination of the grand averaged and individual waveforms. Topographic maps were visually inspected to identify electrode sites displaying maximal activity during the defined time window. The electrode sites chosen for the analysis through visual inspection were also consistent with previous studies (Lewis et al., 2007; Rueda et al., 2004). Electrode sites included lateral (left, right) and midline electrode clusters as the topographic maps showed the N2 to be maximal in lateral electrodes. Amplitude and peak latency measures were derived by averaging values across electrodes within clusters selected to be compatible with the 10-20 electrode placement system (Figure 4-2).

Data Analysis

All analyses were conducted using SPSS Version 21.0 (IBM Corp., Armonk, NY). Behavioral and ERP measures were analyzed using general linear models analysis of variance (ANOVA) applying the Greenhouse-Geisser correction when sphericity assumptions were violated. Means are reported as least-squares means and partial eta-squared (η^2) is reported as a measure of effect size for all statistically significant findings. We performed sensitivity analyses in G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) to determine the effect sizes that we would have statistical power to detect given our sample size. The analyses showed that we would have 80% power to detect medium to large effects for behavioural measures ($\eta^2 = .053 - .148$) and large effects for ERP measures ($\eta^2 = .074 - .200$).

Results

Behavioral Measures

Accuracy was analyzed using mixed ANOVAs with target emotion (neutral, happy, angry) and flanker congruency (congruent, incongruent) as within-subject factors and age group (young, old) as a between-subject factor. Descriptive statistics for behavioral performance are presented in Table 4-1.

For accuracy, there were main effects of target emotion ($F(2, 98) = 7.64, p < .01, \eta^2 =$.135) and age group ($F(1, 49) = 5.88, p < .05, \eta^2 = .107$). Children responded more accurately on trials with happy targets (M = .92, SD = .01) than on trials with angry (M = .90, SD = .01) or neutral targets (M = .89, SD = .01), which did not differ (p > .05). Older children (M = .93, SD = .01) responded more accurately than younger children (M = .88, SD = .01). Emotional influence on accuracy was not modulated by cognitive control demands, as the interaction between target

emotion and flanker congruency was not significant (F(2, 98) = 1.86, p = .17, $\eta^2 = .037$). No other main effects or interactions were significant.

RT analyses revealed significant main effects of target emotion (F(2, 98) = 7.42, p < .01, $\eta^2 = .132$), flanker congruency ($F(1, 49) = 23.50 p < .001, \eta^2 = .324$) and age group ($F(1, 49) = 14.61, p < .01, \eta^2 = .230$). Children were slower to respond on trials with angry targets (M = 961.3 ms, SD = 21.88) than on trials with happy (M = 933.3, SD = 20.78) or neutral targets (M = 932.7 ms, SD = 20.40), which did not differ (p > .05). Children were also slower to respond on incongruent trials (M = 961.6 ms, SD = 21.32) than on congruent trials (M = 923.2 ms, SD = 20.33), confirming that cognitive control demands were greater on incongruent trials. Finally, younger children (M = 1020.6 ms, SD = 27.47) responded slower than older children (M = 864.3 ms, SD = 30.30). Emotional influence on response time was not modulated by cognitive control demands as the interaction between target emotion and flanker congruency was not significant ($F(2, 98) = 0.60, p = .55, \eta^2 = .012$). No other main effects or interactions were significant.

ERP Measures

Amplitude and latency measures for N2 and LFN were analyzed using mixed ANOVAs with target emotion (neutral, happy, angry), flanker congruency (congruent, incongruent), and laterality (left, midline, right) as within-subject factors, and age group (young, old) as a between-subject factor. ERP waveforms by emotion and congruency conditions for each age group are presented in Figure 4-3.

For N2 amplitude, there were main effects of flanker congruency ($F(1, 35) = 16.79, p < .001, \eta^2 = .324$), age group ($F(1, 35) = 5.62, p < .05, \eta^2 = .138$), and laterality ($F(2, 70) = 6.98, p < .01, \eta^2 = .166$). Contrary to the pattern commonly observed in adults, N2 amplitude was greater on congruent ($M = -7.57 \mu V, SD = .50$) than on incongruent trials ($M = -6.34 \mu V, SD = .50$)

.52). The N2 was greater in younger children ($M = -8.12 \ \mu V$, SD = .67) than in older children ($M = -5.79 \ \mu V$, SD = .72). It was also greater at the left electrodes ($M = -7.89 \ \mu V$, SD = .56) than at midline ($M = -7.08 \ \mu V$, SD = .62) or right electrodes ($M = -5.91 \ \mu V$, SD = .55), and greater at the midline than at the right electrodes. Emotional influence on N2 amplitude was not modulated by cognitive control demands, as the interaction between target emotion and flanker congruency was not significant (F(2, 70) = 0.95, p = .39, $\eta^2 = .026$).

For N2 latency, there were main effects of target emotion ($F(2, 70) = 5.08, p < .01, \eta^2 = .127$) and age group ($F(1, 35) = 5.67, p < .05, \eta^2 = .139$). N2 latency was longer for angry (M = 344.20 ms, SD = 4.63) targets than for neutral (M = 333.20 ms, SD = 4.87) targets. N2 latency for happy targets (M = 338.50 ms, SD = 4.69) did not differ from neutral or angry targets (p > .05). N2 latency was longer in younger children (M = 348.85 ms, SD = 5.82) than in older children (M = 328.42 ms, SD = 6.31). Emotional influence on N2 latency was not modulated by cognitive control demands, as the interaction between target emotion and flanker congruency was not significant ($F(2, 70) = 1.27, p = .29, \eta^2 = .035$).

For LFN amplitude, there was a main effect of congruency ($F(1, 35) = 5.29, p < .05, \eta^2 = .131$), age group ($F(1, 35) = 9.50, p < .01, \eta^2 = .213$), and laterality ($F(2, 70) = 14.91, p < .001, \eta^2 = .299$). LFN amplitude was more pronounced on congruent trials ($M = -4.19 \mu V, SD = .46$) than on incongruent trials ($M = -3.38 \mu V, SD = .45$), and in younger children ($M = -5.10 \mu V, SD = .57$) than in older children ($M = -2.47 \mu V, SD = .62$). It was also more pronounced at the left electrode sites ($M = -5.27 \mu V, SD = .53$) than at the midline ($M = -3.90 \mu V, SD = .56$), or right electrode sites ($M = -2.19 \mu V, SD = .50$), and at the midline electrode sites than at the right electrode sites. There was also a significant interaction between target emotion and age group ($F(2, 70) = 5.33, p < .01, \eta^2 = .132$). Only in younger children, LFN amplitude was greater for

angry targets ($M = -6.26 \ \mu\text{V}$, SD = .63) than for neutral ($M = -5.18 \ \mu\text{V}$, SD = .62) or happy targets ($M = -4.58 \ \mu\text{V}$, SD = .77), which did not differ (p > .05). In older children, LFN amplitude did not differ between neutral, happy or angry targets (p > .05).

To ensure that any effects observed on the N2 and LFN were not due to differences associated with early sensory processing of emotions or faces, ERPs related to face and emotion processing, P1 and N170, were analyzed using mixed ANOVAs with target emotion (neutral, happy, angry), flanker congruency (congruent, incongruent), and laterality (left, midline, right) as within-subject factors, and age group (young, old) as a between-subject factor. The amplitudes and latencies of the P1 and N170 did not differ by target emotion or flanker congruency conditions (p > .05).

Discussion

Our goal in this study was to investigate how emotions influenced cognitive performance in early and middle childhood. We specifically examined if this influence differed by emotional valence and cognitive control demands. We used a modified flanker task with emotional face stimuli where cognitive control demands were greater on the incongruent trials and lower on the congruent trials. Our findings revealed that happy and angry emotions differed in the manner in which they influenced cognitive performance in children. Emotional influence on both behavioural performance and the N2 was not modulated by cognitive control demands, and these effects were similar across early and middle childhood. We discuss below our behavioural and ERP findings, beginning with findings related to emotional valence, followed by the effect of varying cognitive control demands of the task, and finally, findings pertaining to developmental differences across early and middle childhood.

The effect of emotional valence on cognitive performance

As predicted, we found that emotional influence on children's behavioural performance was modulated by valence: happy target emotions facilitated cognitive performance while angry target emotions impaired cognitive performance. Across both age groups, children responded more accurately on trials with happy targets, and responded more slowly on trials with angry targets. Parallel to our RT findings, N2 latency was longer for angry targets. In addition, children in the younger group showed a larger late frontal negativity (LFN) for angry targets than for neutral or happy targets.

Our finding that children responded more accurately to happy targets parallels previous reports of a 'positivity bias' displayed by children on different cognitive tasks (Augusti et al., 2012; Bahn et al., 2017; Lewis et al., 2007). One possible explanation for the facilitation observed with happy emotions is that children are able to process them more efficiently (Todd et al., 2011). This suggestion is also consistent with emotion recognition studies that have reported that children attain adult-level performance in recognizing happy emotions before the other emotions (Durand, Gallay, Seigneuric, Robichon, & Baudouin 2007; Gao & Maurer, 2010). The finding of a 'positivity bias', however, contradicts predictions made by the DCM. According to the DCM, emotions that are low in threat — such as the happy targets in our study — do not require as much processing, leaving cognitive resources intact for task-related processing. However, it explicitly predicts a behavioural facilitation only when the emotion is task-relevant. In our study, it is possible that the enhanced representation received by the task-irrelevant happy targets was extended to the task-relevant borders, due to the close proximity between them. Future studies should examine if task-irrelevant happy emotions facilitate cognitive performance
even when not presented in such close proximity to task-relevant stimuli on which responses are based on.

Our findings showing that children responded more slowly and had a longer N2 latency on trials with angry targets are in line with the predictions made by the DCM. The DCM postulates that emotions that are threatening or task-irrelevant divert cognitive resources away from the task, thereby impairing task performance. In the present study, if the angry targets were perceived as threatening, they would be expected to divert cognitive resources away from taskrelated processing (color judgement of the target border), resulting in both the longer RTs and N2 latency. This account also explains the larger LFN observed for angry targets, possibly indicating that they recruited greater attentional resources.

While the longer N2 latency found for angry targets is in accordance with the prediction made by the DCM, it contrasts with previous findings by Lewis and colleagues (2007). In that study, the N2 was found to occur earlier for angry faces than for neutral or happy faces. As this finding was observed in a condition that involved passive viewing of the stimuli, the difference in study design could account for the discrepancy in findings across studies. In the present study, children responded to the target border color, and emotions were irrelevant to task performance. To the extent that the N2 reflects task-related processing, the longer N2 latency could indicate that task-related processing was delayed on trials with angry targets.

The effect of cognitive control demands on emotional influence

In addition to valence, we also sought to examine whether emotional influence on cognitive performance was modulated by cognitive control demands. We found no indication that varying cognitive control demands modulated emotional influence on children's cognitive performance. This finding differs from that reported in adults where increasing the cognitive control demands of a task has been reported to attenuate emotional influence on cognitive performance. This modulatory effect is attributed to top-down control mechanisms, namely a network consisting of the lateral prefrontal regions and the ACC (Etkin et al., 2006; Kanske & Kotz, 2011a; Mohanty et al., 2007). Under increased cognitive control requirements, activity in this network is found to increase in adults, coupled with decreased activity in the amygdala. The discrepancy between these findings in adults and ours with children could be due to the immaturity of these top-down control mechanisms in children. This interpretation is consistent with evidence showing that the regions implicated in this top-down control are still developing in children (Gogtay et al., 2004; Sowell et al., 2004). One might suggest that the lack of finding emotional influence to vary with cognitive control demands is due to the congruent and incongruent trials not differing in cognitive control demands. We consider this explanation unlikely as our behavioural findings showed that children were slower to respond on incongruent trials than on congruent trials, attesting the greater cognitive control demanded by the incongruent trials. This study is the first to directly address if varying the cognitive control demands of a task modulates emotional influence on children's cognitive performance. In the few other studies where cognitive control requirements were varied, the response requirements between the low and high cognitive control trials also differed, making it less clear if the observed emotional effects were due to the increased cognitive control demands. These findings contribute to a greater understanding of emotion-cognition interaction in the study of development.

In interpreting this null finding, it must also be recognized that because our sample size was modest, we had sufficient power to detect only effects that ranged from medium to large. Observed effect sizes for the interaction between emotion and congruency were all in the small to medium range ($\eta^2 = .012 - .037$), and with a larger sample size they may have reached statistical significance.

Age differences

As expected, older children performed better on the task than younger children, displaying greater accuracy and faster responding. Developmental improvements were also observed in our ERP findings. Older children had smaller N2 amplitude and earlier N2 latency than younger children. These findings are in accordance with our understanding of the gains observed in children's cognitive abilities and processing speed with age, and they parallel previous results in children (Johnstone, Pleffer, Barry, Clarke, & Smith 2005; Johnstone et al., 2007; Rueda et al., 2004), linked to neurodevelopmental changes such as synaptic pruning and myelination occurring during early and middle childhood (Gogtay et al., 2004; Lenroot & Giedd, 2006).

Based on our understanding that children get better in their cognitive and emotion processing abilities with age, we predicted that cognitive performance in the older children would be less affected by emotional influence. Contrary to our prediction, emotional influence on cognitive performance did not differ between the young and old groups. Previous studies that have examined age-related changes in emotional influence on cognitive performance have reported mixed findings (Augusti et al.,2012; Urben et al., 2012). Part of this discrepancy could be due to the different emotions examined in these studies. In the study where emotional influence varied with age, the effects were observed specifically for fearful emotions (Augusti et al., 2012), which was not examined in the other study (Urben et al., 2012), or ours. As that study also involved children aged between nine- to twelve-years, future studies should examine if similar changes are observed from early to middle childhood. Another possible reason for the lack of finding age-related changes in emotional influence on cognitive performance could be the late maturation of the fronto-amygdala connections that underlie emotion-cognition interactions. In a study examining the development of fronto-amygdala connections from early childhood to adulthood, changes in connectivity patterns were found to emerge only at the age of ten (Gee et al., 2013). As the children in our study were between the ages of four and eight, they may not have started to display such changes. Future studies including older children and adolescents are needed to bridge the gap with the adult literature, to better understand the dynamics of emotion-cognition interactions.

While behavioural findings showed that angry targets had a similar effect in both young and old groups of children, we found that a late frontal negativity (LFN) was larger for angry targets than for neutral or happy targets only in the younger group. The morphology of this negativity resembles a sustained anterior negativity reported in studies of selective attention (Eimer, 1997; Karayanidis & Michie, 1996). In these studies, selectively attending to a stimulus attribute or location produced a sharp anterior negativity (similar to the N2 in our study) that was followed by an extended negative potential (similar to the LFN in our study). The larger LFN observed for angry targets could be an indication of greater attention being allocated towards them. This is consistent with the notion that threatening emotions take up cognitive resources, and implies that the younger children had greater difficulty from directing attention away from the task-irrelevant angry targets. However, angry targets impaired behavioural performance in both young and old groups of children. The discrepancy between behavioural and LFN findings can be reconciled by taking into consideration that this effect was observed at a late occurring ERP component and not in the earlier components. This suggests that older children may have been better able to disengage and direct their attentional resources away from the task-irrelevant angry targets.

Aside from an age-emotion interaction effect, we also found that the LFN was more pronounced on congruent trials than on incongruent trials. A similar pattern was observed with the N2 that preceded the LFN. Typically, the N2 amplitude is larger on incongruent trials than on congruent trials, and this is attributed to the greater recruitment of neural resources that is required to support the increased cognitive control demands on the incongruent trials (Buss et al., 2011; Forster, Carter, Cohen, & Cho, 2011; van Veen & Carter, 2002). However, this pattern is more frequently reported in adults, and several studies have failed to replicate this 'incongruent N2-effect' in children (Brydges et al., 2013; Rueda et al., 2004; Vuilier et al., 2016). As children in these studies performed well on the incongruent trials, the lack of finding the 'incongruent N2effect' was attributed to children employing a different, more diffuse neural network to meet the increased cognitive control demands on the incongruent trials. It is possible that children's immature cognitive abilities may have contributed to the absence of the 'incongruent N2-effect' in our study. Besides not finding the N2 to be enhanced on the incongruent trials, we found the N2 amplitude to be more enhanced on the congruent trials. Taking into consideration the morphological similarity of the N2 and LFN observed in our study to the frontocentral negativities seen in studies of selective attention, one possibility is that children were better able to selectively attend to the targets on congruent trials. The compatibility between the target and flankers results in less interference on congruent trials relative to incongruent trials. It is possible that this reduced interference allowed children to selectively attend to the targets more successfully on the congruent trials, and that the larger N2 and LFN observed on congruent trials reflects this successful engagement of selective attention.

Conclusion

In this study, we ventured to understand how emotional influence on cognitive performance is modulated by valence and cognitive control demands in early and middle childhood. We found the valence of the emotion to be a crucial factor in determining how emotions influenced cognitive performance in early and middle childhood. Children displayed greater accuracy for happy targets but were slower to respond to angry targets. N2 latency was longer for angry targets, and in younger children only, the LFN was greater for angry targets. In contrast to findings with adults, we found that in early and middle childhood, emotional influence on cognitive performance was not modulated by the cognitive control demands of the task. This finding suggests that children may not yet have developed the capacity to overcome emotional influence, and is consistent with our understanding that cognitive control capacities are still developing in children. Our study is the first to have directly assessed if emotioncognition interactions are dynamic in children and contributes to a greater understanding of emotion-cognition interactions in childhood. Children are frequently exposed to emotions — at home as well as in academic and social settings. As early and middle childhood is also a period where children are making rapid gains in their cognitive abilities, the findings from our study could have potential implications on how the emotional climate at home and in school can impact children's learning.

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Table 4-1. Means and Standard Deviations (in parentheses) for Accuracy (proportion correct) andReaction Time (milliseconds) by Age Group, Congruency, and Target Emotion.

	Young		Old	
	Congruent	Incongruent	Congruent	Incongruent
Accuracy (%)				
Neutral	.85 (.11)	.87 (.10)	.94 (.06)	.92 (.10)
Нарру	.90 (.09)	.91 (.07)	.94 (.05)	.94 (.07)
Angry	.90 (.08)	.86 (.10)	.92 (.08)	.91 (.08)
Reaction Time (ms)				
Neutral	984 (146.2)	1037 (150.3)	845 (150.6)	865 (165.5)
Нарру	998 (137.0)	1022 (165.4)	841 (167.0)	873 (153.6)
Angry	1018 (161.5)	1065 (158.7)	854 (163.9)	908 (172.8)



Figure 4-1. Task Outline - Trial began with the onset of a fixation cross lasting for 1000ms. This was followed by the onset of the stimulus. Stimulus display terminated with response or after 2000ms. A feedback then appeared indicating if response made was correct (thumbs up) or incorrect (thumbs down) and lasted for 1500ms.



Figure 4-2. Montage illustrating the electrodes used in the analysis of the N2 and the Late Frontal Negativity (LFN) at the Left, Midline, and Right electrode clusters.

—— Neutral Congruent	······ Neutral Incongruent
—— Happy Congruent	······ Happy Incongruent
—— Angry Congruent	······ Angry Incongruent



Younger Children

Older Children



Figure 4-3. ERP waveforms of young (top panel) and old (bottom panel) groups of children at the frontal electrode clusters (left, midline, and right). Time windows of the N2 and the Late Frontal Negativity are shown in the dark gray and light gray boxes, respectively.

Chapter 5

General Discussion

The objective of this dissertation is to investigate how the behavioural and ERP indices of inhibitory abilities in children are influenced by three different factors, namely, time pressure, physical activity, and emotion. I examined two inhibitory processes: response inhibition and interference suppression. The findings from all three studies provided unique insights into how the respective factors addressed in each study modulated inhibitory performance and the underlying neural correlates. In Study One, I found that time pressure impaired performance on Go trials but that it did not affect response inhibition performance. I also found that the topography of the neural correlates underlying response inhibition was left-lateralized and had a more posterior distribution in children than has been found in adults. In Study Two, I found that physical activity in children was associated with their P3 latency on No-go trials, indicating that children who are more active took a longer time to activate inhibitory process or evaluate the stimulus on No-go trials. In Study Three, I found that emotions had a global effect on children's cognitive performance, which differed by valence, but that emotional influences did not interact with interference suppression demands.

In this chapter, my goal is to briefly highlight the contributions made by this dissertation towards a better understanding of inhibitory abilities in children. I will begin by providing a review of the findings from each of the three studies. Then, I will discuss the commonalities and differences observed in the findings between the studies, and how they contribute towards a greater understanding of the neural correlates underlying inhibitory processes in children. I will also address some of the limitations of the studies and suggest future directions for research on this subject. I will conclude by highlighting the significance of the findings and some avenues for their potential application.

Study One: The neural correlates underlying response inhibition in children: Evidence from a Go/No-go task

Often, the need for inhibition arises when the response is prepotent or automatic, but inappropriate in the given context. As captured by the horse race model, successful response inhibition requires the inhibitory process to complete before the execution process does. By inducing time pressure, we sought to speed up the execution process to understand how this affects response inhibition performance in children, and how the neural correlates are modulated to allow for inhibition. Contrary to our expectation, time pressure did not influence the behavioural and ERP measures of response inhibition in children. However, I found that that when subjected to time pressure, children strategized to optimize their performance in both inhibition (No-go trials) and execution (Go trials). As expected, children in the Fast condition, who were given less time (750 ms) to execute the response and catch the fish, responded faster than the children in the Slow condition, who were given more time to respond (1500 ms). But children in the Fast condition were also less accurate on the Go trials than children in the Slow condition. This finding suggests that children in the Fast condition may have adopted a more cautious strategy towards responding, rather than engaging in a prepotent response strategy. One possible explanation for this is that they may have weighed the consequences of inhibitory failures on No-go trials more heavily than that of failing to respond on Go trials. Specifically, inhibitory failures (i.e., catching a shark) resulted in a broken fishing net, whereas omission errors (i.e., not catching a fish) resulted in the fish swimming away. Children may have felt the consequences of the former more strongly, and thus, may have been more motivated to avoid it. Although the engagement of a cautious strategy to help with inhibition was unexpected, it aligns well with other findings in children. For instance, in a longitudinal study, Wiebe and colleagues (2012) reported that improvements in response inhibition in preschool children were accompanied by slowing down of responses. Moreover, another study also reported that children's response inhibition performance can be improved by imposing a delay after the signal to respond (Diamond, Kirkham, & Amso, 2002). While the findings from this study is interesting in its own right, future studies should examine how response inhibition performance and the neutral correlates are influenced when responding is prepotent. There were also some interesting ERP findings from this study. These findings are discussed below in relation to the findings from the Studies Two and Three.

Study Two: Physical activity and response inhibition in early childhood: An ERP study using objective and subjective measures of physical activity

Numerous studies in adults (Bherer, Erickson, Liu-Ambrose, 2013; Ludyga, Gerber, Brand, Holsboer-Traschler, & Pühse, 2016) and school-aged children (Chaddock, Pontifex, Hillman, & Kramer, 2011; Sibley & Etier, 2003) have demonstrated the beneficial effects of physical activity on cognitive functioning. Inhibitory abilities have been the outcome of interest in a large number of these studies (Ludyga, et al., 2016), and there is some indication that of the various cognitive control processes, inhibitory abilities may preferentially benefit from physical activities (Barenberg, Burse, & Dutke, 2011; Nascimento et al., 2015). To address the paucity of research addressing this relationship in early childhood, in my second study, I examined if physical activity levels were associated with response inhibition performance and the neural correlates underlying performance. Of the response inhibition measures assessed, only No-go P3 latency was associated with physical activity. Specifically, children who were more active displayed a longer P3 latency on No-go trials. This suggests that children with higher levels of physical activity may have been slower in activating inhibitory processes or in evaluating the stimulus on No-go trials. This pattern is opposite to the increased processing speed that is typically observed with physical activity intervention in older adults (Desjardins-Crépeau et al., 2016; Hillman et al., 2006). The differences in findings could be due to the manner in which physical activity was defined and measured. While the previous studies with adults involved physical activities that were structured and purposeful (e.g., sports, exercise activities), this may not have been the case with the non-organized activity measure captured in our study. One possibility is that participation in non-organized activities may have involved non-functional or excessive movement, and thus the lack of behavioural control required in such activities may have given rise to a less efficient activation of inhibitory process or evaluation of the No-go stimulus by children with higher levels parent-reported participation in organized activities.

Alternatively, the longer P3 latency on No-go trials may be an indication of children being more careful in evaluating the stimulus in order to minimize omission and commission errors. As findings in early childhood suggest that improved response inhibition performance is characterized by an initial decrease in the speed of responding (Wiebe, et al., 2012), the longer No-go P3 latency in children with higher levels of physical activity could be a reflection of physical activity participation guiding children in adopting strategies that help with optimizing task performance.

Neither No-go accuracy nor the amplitude of the N2 and the P3 were associated with physical activity. Similar behavioural finding has been noted in a previous study with preschool children (Palmer, Miller, & Robinson, 2013). However, as these findings with preschool children diverge from those reported in adults (Bae, Ogawa, Yamazaki , 2012; Chu, Alderman, ,Wei, &

Chang, 2015), some limitations present in this study may have contributed to the observed null associations (These are discussed in detail in Chapter 2).

Study Three: Valence matters: An electrophysiological study on how emotions influence cognitive performance in children

In my third study, I examined if the effect of emotions on cognitive performance differed by valence and cognitive demands. Within a flanker paradigm, cognitive demand was manipulated by the varying the need for interference suppression (i.e., congruent versus incongruent trials), and emotion was manipulated by varying the expression displayed by the target face (neutral, happy, or angry). Previous findings with children have reported a 'positivity bias' where superior cognitive performance is observed on trials with positive emotions (Augusti, Torheim, & Melinder, 2012; Bahn, Vesker, García Alanis, Schwarzer, & Kauschke, 2017; Lewis, Todd, & Honsberger, 2007). However, as findings in adults show that the influence that emotions exert on cognitive performance can be attenuated by cognitive control demands (Lim, Padmala, & Pessoa, 2008; Cohen, Henik, & Mor, 2011), the goal in the second study was to examine if a similar phenomenon existed in children.

The findings showed that emotional information had a global effect on children's cognitive performance. Specifically, emotional influence on children's cognitive performance depended on emotional valence. Children's accuracy was greater on positive, happy trials, but their reaction time was delayed on negative, angry trials. These findings are consistent with previous findings in children (Augusti et al., 2012; Bahn et al., 2017; Lewis et al., 2007), but are only partially consistent with the dual competition model (DCM). The DCM is a prominent framework outlining emotional influence on cognitive performance (Pessoa, 2009). It predicts that when emotions compete with cognitive processes for neural resources, as happens when

emotions are threatening or irrelevant, they lead to impaired task performance. Accordingly, I found that angry targets, which are threatening, resulted in slower responding in children. However, the DCM also predicts that emotions that are irrelevant would result in impaired cognitive performance regardless of the level of threat posed, as they would divert cognitive resources away from task-related processing. Following this idea, we would expect happy targets to have also impaired task performance as they were irrelevant to the colour judgment that children were required to make in the task. Yet, we found that children's performance was facilitated on trials with a happy target.

Although this pattern seems to suggest that the effect that emotions have on cognitive performance in children is not contingent on the relevancy of the emotion to the task, it is also important to note that in this study, emotions were proximate to the task-relevant borders. Similarly, in previous findings with children where a 'positivity bias' has been noted, emotions were embedded within the task-relevant dimension (Bahn et al., 2017; Lewis et al., 2007). Hence, in our study it is possible that this close proximity between the borders and the targets allowed for the enhanced perceptual representation received by the happy targets to be extended to the target borders.

I also found that the effect of emotions on cognitive performance did not vary with cognitive demands (i.e., interference suppression requirements), in that there was no interaction between emotion and flanker congruency condition. The contrast between our findings and those observed in adults could be driven by the immaturity of the brain regions that are involved in cognitive control and in the integration of emotional-cognitive processes. These regions, including the prefrontal cortex and the anterior cingulate cortex have been found to reach maturity only in adulthood (Kelly et al., 2009; Gogtay et al., 2004; Sowell, Thompson, & Toga,

2004). Because of the immaturity of these regions in children, children may not yet have the capacity to control the influence of emotions with increased task demands. As discussed below, the atypical pattern of N2 that was found in children in this study also lends support to this interpretation.

The neural correlates of inhibitory functioning in children

Following previous findings, I predicted that the amplitude of the N2 in all three studies would be enhanced on trials requiring inhibition. The enhancement of the N2 on trials where inhibition is required is thought to index the greater cognitive demands of inhibitory processes, and the greater neural resources that are required to meet these demands (Buss, Dennis, Brooker, & Sippel, 2011; Forster, Carter, Cohen, & Cho, 2011; van Veen & Carter, 2002). In Studies One and Two where response inhibition was assessed, the N2 was greater on trials requiring inhibition. This was in line with both my predictions and previous findings. However, while the N2 showed the expected enhancement on trials requiring inhibition, its topography differed from that typically observed in adults. The N2 was left-lateralized in children, whereas in adults, it is maximal in the midline (Bekker, Kenemans & Verbaten, 2005; Jonkman et al., 2007) or right electrode regions (Bokura et al., 2001). The different topography of the N2 indicates that the neural network underlying inhibitory abilities in children may be different from that recruited by adults. Although the poor spatial resolution of ERPs make this interpretation difficult to verify. other studies that have used source localization methods offer some support for this interpretation (Lamm, Zelazo, & Lewis, 2006). It is also further corroborated by findings from functional magnetic resonance imaging (fMRI) studies that have superior spatial resolution (Bunge et al., 2002). When considering together both the behavioural findings that indicate immature inhibitory abilities in children and the different pattern of brain activity observed

during inhibitory performance, one interpretation of the left-lateralized N2 is that it represents an immature neural network underlying response inhibition in children. To compensate for this immaturity, children may be engaging in strategies that are different from those employed by adults to support inhibitory performance. For instance, children may be engaging in a verbal strategy to help with inhibition. This is consistent with findings that report that children engage in inner speech to aid with performance on tasks that are inherently nonverbal (Winsler & Naglieri, 2003). As language-related processing predominantly involves the left hemisphere, the engagement of a verbal strategy to perform the task could explain the left-lateralized finding in children.

In Study Three, which involved interference suppression, the N2 was not enhanced on incongruent trials requiring inhibition. This finding was in contrast to my prediction and findings in adults reporting the N2 to be enhanced on incongruent trials where the conflicting information provided by the flankers has to be suppressed. However, in children, a number of studies have failed to find this N2 enhancement on incongruent trials (Brydges, Anderson, Reid, & Fox, 2013; Rueda, Posner, Rothbart, & Davis-Stober, 2004; Vuillier, Bryce, Szücs, & Whitebread, 2016). In our study, despite children not displaying an enhanced N2 on incongruent trials, their accuracy on incongruent trials was high. This shows that children were able to overcome the interference presented by the flankers on incongruent trials. But the lack of an enhanced incongruent N2 also suggests that children may not be employing their neural resources in the same manner as adults to support their performance. Brain imaging studies in children provide some clues in understanding this finding. In these studies, it was reported that children displayed a more diffuse pattern of brain activation relative to adults, while performing the same task. (Bunge et al., 2002; Casey, Giedd, & Thomas, 2000). Hence, it is possible that the lack on an N2 enhancement on

incongruent trials was due to children employing a more distributed neural network to support their performance.

There was also an unexpected finding: an enhanced N2 on congruent trials, a pattern opposite to what has been reported in adults. As the anterior N2 has been associated with several other cognitive control functions besides inhibition (Folstein & Van Petten, 2008), this novel pattern of finding was interpreted in light of a selective attention mechanism. Taking into consideration the temporal and morphological similarity of the N2 observed in this study to that observed in studies where participants are required to selectively attend to a target feature (Eimer, 1997, Karavanidis & Michie, 1997), I interpreted the enhanced N2 to index children's greater selective attention to targets on congruent trials. The compatibility of the flankers to the target on congruent trials may have made it easier for children to selectively attend to the target. On incongruent trials, the interference presented by the incompatible flankers may have made it more challenging for children to selectively attend to the target. In order to ensure that this unexpected N2 finding was not due to the design of the task, a pilot study was done with adult participants using the same task. In adults, the N2 showed the expected pattern and was more pronounced on incongruent trials ($M = -2.19 \mu V$, SD = .72) than congruent trials (M = -1.37) μ V, SD = .75). However, this effect only reached marginal significance (F(1, 9) = 4.45, p = .64, $\eta^2 = .330$), likely due to the small sample size (n = 10) of the pilot study.

Considering the N2 findings from the three studies in this dissertation, along with evidence from existing developmental literature (Brydges et al., 2013; Rueda et al., 2004; Vuillier et al., 2016), the N2 ERP component seems to more reliably index response inhibition than interference suppression in children. However, while the findings largely support the conception of the N2 as a marker of response inhibition, it is important to note that other alternatives have also been proposed. One prominent alternative theory is that the N2 reflects the detection of conflicting information (Albert, López-Martín, Hinojosa, & Carretié, 2013; Nieuwenhuis, Yeung, Wildenberg, & Ridderinkhof, 2003). Other have also suggested that it represents a pre-motor inhibition process or a 'red flag' signalling the need to inhibit (Kok, 1986). Based on the current data set, and due to the conflicting response tendency that is inherently present on No-go trials, it is not possible to negate this alternative interpretation. However, it should be noted that even with equiprobable Go and No-go trials (and hence, less conflict on No-go trials), an enhanced No-go N2 has been reported (Eimer, 1993; Pfefferbaum, Ford, Weller, & Kopell, 1985).

The P3 is another inhibition-related ERP component that was examined in this dissertation. Of the three studies, a clear P3 peak was only observed in the two studies assessing response inhibition, and was not observed during interference suppression. By considering the key difference between response inhibition and interference suppression — that the former requires the inhibition of a motor response, whereas the latter involves the inhibition of perceptual information — the difference in the P3 findings can be better appreciated. The P3 has been suggested to index a motor inhibition process (Enriquez-Geppert, Konrad, Pantev, & Huster, 2010; Smith, Johnstone, & Barry, 2008), a suggestion that is supported by the observation that the onset latency of the P3 on No-go trials parallels the time take to respond on Go trials (Wessel & Aron, 2015). The absence of the P3 in the interference suppression task may be due to the fact that interference suppression does not require a complete motor inhibition, but rather, the inhibition of the interfering information provided by the flankers. Concordant with this explanation, in one study also using the Flanker task, P3 amplitude enhancement was only observed on incongruent trials when preparatory motor activation was observed, but not when

such preparatory motor responses were not elicited (Gehring, Gratton, Coles, & Donchin, 1992). In the flanker task used in my study, the stimulus-response mapping was indirect. Specifically, children had to press a left or a right button based on the colour of the border around the target face. Compared to flanker tasks where a left/right-pointing arrow prompts a left/right button press, the stimulus-response mapping in the colour-flanker task is less automatic. As such, a lack of preparatory motor response may explain the absence of the P3. Unfortunately, as motor/response-related ERPS were not analyzed in the study, future work will need to verify this explanation.

Some have questioned the extent to which the ERP components, N2 and P3, index inhibitory functioning, claiming that movement-related artifacts underlie the amplitude differences observed (Salisbury, Griggs, Shenton, & McCarley, 2004). This argument is especially relevant to Studies One and Two where motor requirements differed between Go and No-go trials. Several studies with adults have provided evidence refuting this suggestion, and proving that N2/P3 modulation are present even when motor requirements are eliminated (Pfefferbaum et al., 1985; Smith et al., 2008). This is an avenue of research that is yet to be investigated in developmental population. On a similar note, examining movement-related ERPs, such as lateralized readiness potentials, may help to deepen our understanding of the neural correlates underlying inhibitory functioning in children.

Limitations

A detailed account of the limitations present in each study is provided within the respective chapters. Here, I will summarize some common limitations.

The sample size included in each of the three studies was modest (n = 31 - 58). In studies involving children, obtaining a large enough sample is often a challenge, and the sample size in

most previous developmental research with children is in a similar range (Brydges et al., 2013; Buss et al., 2011; Lewis et al., 2007). However, it should be noted that the small sample size limited our power to detect significant effects. Specifically, given the sample size, there was sufficient power to detect effects that ranged from medium to large in size and hence, it is possible that real but small effects may have gone undetected. As such, the null results present in the studies should be interpreted with caution, and where these null results ran counter to theoretical predictions or the existing literature, attempts to replicate the findings with a larger sample should be undertaken.

The samples included in each study were convenience samples, and were largely comprised of children from middle to high socio-economic backgrounds. The overrepresentation of children from these backgrounds is likely due to factors such as parental motivation to participate in research studies, as well as resource availability (e.g., ability to take time off work) that may have restricted the participation of children from a lower socio-economic background. Given previous findings showing that socioeconomic status can influence inhibitory functioning (Sarsour et al., 2011), and more generally, cognitive performance in children (Farah et al., 2009; Hackman & Farah, 2009), the findings from the three studies may not be generalizable to children from a lower socioeconomic status.

As reviewed above, in all three studies, I found some differences in the neural correlates underlying inhibitory processes in children, as compared to that observed in adults. However, the tasks used in the three studies were also adapted for use with children. While this was done to set the task difficulty at a level that was appropriate for children, as well as to make it engaging for them, it also meant that the design of the tasks were different from those used with adults. Therefore, it is possible that the differences in task design may have contributed to the differences that were observed with the N2 and P3 ERP components in children. This possibility could have been better investigated by including adult participants in the study. While an attempt at such an investigation was made in Study Three (see findings from Pilot Study briefly mentioned above), it was not done so in Studies One and Two.

Significance of Findings

The findings from the studies in this dissertation broaden our understanding of two types of inhibitory processes, response inhibition and interference suppression, in children.

In Study One, I found that time pressure did not result in prepotent responding in children. Instead, their performance was impaired on Go trials and I interpreted this as an indication of children strategizing to help with response inhibition. While this finding was contrary to our expectation, it has a potential for application in clinical settings. For instance, in children with ADHD, impulsive responding is one of the defining characteristics (Barkley, 1997). There is an avenue to investigate if these children can be trained to overcome impulsive responding by slowing down and responding cautiously. A similar initiative is adopted in individuals who stutter: In speech therapy, a key strategy is teaching patients to control the rate of their speech by slowing down (Hayhow, Cray, & Enderby, 2002).

Study Two examined whether physical activity in early childhood is associated with the neural correlates underlying response inhibition. There is insufficient research in this population, and a key strength is our use of both objective and subjective measures to assess physical activity in children. Children who were more active showed a longer No-go P3 latency, suggesting slower activation of the inhibitory process, or perhaps reflecting a more careful evaluation of the stimuli. The findings from this study bring to light certain factors that needs be addressed in

future investigations in this domain, including considering the context in which physical activity occurs, as well including a sufficiently challenging assessment of response inhibition.

Findings from Study Three offer unique contributions to the existing literature on how emotions affect cognitive performance in children under varying cognitive demands. I found that emotional influence on children's cognitive performance was contingent on valence, and that it did not differ by the level of cognitive control demanded by the task. The findings from the study provided some support for the DCM, but also revealed some findings in contrast to its predictions. In line with the DCM's prediction, angry targets impaired task performance in children. However, contrary to its prediction, I found that happy targets facilitated task performance even when they were irrelevant to the task.

Findings from this study may carry potential implications for use in educational settings. For instance, the 'positivity bias' that we found (and which has been reported in previous findings) has a potential to be translated into educational practice to enhance learning outcomes. Incorporating positive emotions in educational materials and fostering a positive emotional climate may benefit learning outcomes and is an area that warrants further investigation.

The findings from all three studies elucidate important differences in the manner in which children may be employing neural resources to support inhibitory performance. Particularly, the findings from this dissertation indicate that children recruit a left-lateralized and more posterior neural network to support response inhibition. Children also did not display the enhanced N2 that has been suggested to support interference suppression in adults — another finding that suggests differences in the neural correlates underlying inhibitory abilities in children.

Conclusion

Findings from this dissertation provided insights into how time pressure, physical activity, and emotion affected inhibitory performance in children. The use of EEG allowed the investigation to venture beyond behavioural outcomes and examine how the neural correlates are modulated by these factors. As inhibitory abilities are crucial for adaptive functioning (Jahromi & Stifter, 2008;Bull & Scerif, 2001; St Claire-Thompson & Gathercole, 2006) and are still undergoing development in children, knowledge of how they are influenced by various factors can potentially be put towards helping children attain optimal development of this function.

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Appendix A

Supplementary Results for Chapter Two

Behavioural Results

RT was analyzed using speed condition (Fast, Slow) x trial type (Go, No-go) mixed ANOVAs with repeated measures on trial type. Analysis of RT revealed that the interaction between trial type and speed condition was not significant (F(1, 29) = 0.12, p = .74, η^2 = .004).

ERP Results

N2 amplitude and latency were analyzed using speed condition (Fast, Slow) x trial type (Go, No-go) x region (Frontal, Frontocentral, Central) x laterality (Left, Midline, Right) mixed ANOVAs with repeated measures on trial type, region, and laterality. Findings revealed that the interaction between trial type and speed condition were not significant for N2 amplitude $(F(1, 29) = 0.27, p = .61, \eta^2 = .009)$ and latency $(F(1, 29) = 0.03, p = .86, \eta^2 = .001)$.

P3 amplitude and latency were analyzed using speed condition (Fast, Slow) x trial type (Go, No-go) x laterality (Left, Midline, Right) mixed ANOVAs with repeated measures on trial type and laterality. The analyses revealed that the interaction between trial type and speed condition was not significant for P3 amplitude (F(1, 29) = 0.08, p = .78, η^2 = .003) and latency (F(1, 29) = 0.00, p = .99, η^2 = .000).

Appendix B

Supplementary Data: Adult Pilot Study for Chapter Four

Method

Participants

Ten adults (6 Males, 4 Females) aged between 20 - 35 years old participated in this pilot study. Participants were Undergraduate and Graduate students at the University of Alberta.

Procedures

The Flanker Task. Participants completed a Colour Flanker task where they responded to indicate the colour of the border around a central target face. The task design and parameters were identical to that used in the study in Chapter Four.

Electrophysiological Recording and Data Processing. EEG was recorded using a 256-channel Hydrocel Geodesic Sensor Net (Electrical Geodesics Inc., Eugene, OR). The data acquisition and processing procedures were identical to that adopted in Chapter Four. Adaptive mean and peak latency measures were calculated for the N2 ERP component. The N2 was examined at the frontal midline electrode sites (Electrodes 15, 16, and 23) where it was maximal and was defined as a negative deflection in the time window between 150 and 250 ms after stimulus onset. The time windows were determined after visual examination of the grand averaged and individual waveforms. Topographic maps were visually inspected to identify electrode sites displaying maximal activity during the defined time window. Amplitude and peak latency measures were derived by averaging values across electrodes within clusters selected to be compatible with the 10-20 electrode placement system.

Results

Behavioral Measures

Accuracy and RT were analyzed using repeated measures ANOVAs with target emotion (neutral, happy, angry) and flanker congruency (congruent, incongruent) as withinsubject factors.

For accuracy, the main effects of target emotion (F(2, 18) = 0.28, p = .76, $\eta^2 = .030$), flanker congruency (F(2, 18) = 2.45, p = .14, $\eta^2 = .213$), and the interaction between target emotion and flanker congruency were not significant (F(2, 18) = 0.65, p = .53, $\eta^2 = .068$).

RT analyses revealed that the main effect of target emotion was not significant (*F*(2, 98) = 1.69, p = .21, $\eta^2 = .158$). However, a significant main effect of flanker congruency was observed (*F*(1, 9) = 6.36 p < .05, $\eta^2 = .414$). Responding was slower on incongruent trials (*M* = 452.6 ms, *SD* = 17.50) than on congruent trials (*M* = 440.4, *SD* = 19.77), confirming that cognitive control demands were greater on incongruent trials. There was also an interaction between target emotion and flanker congruency (*F*(2, 18) = 4.56 p < .05, $\eta^2 = .336$). Only on congruent trials, RT was significantly faster on trials with a neutral target (*M* = 425.4, *SD* = 17.90), than on trials with a happy (*M* = 453.8, *SD* = 23.39), or an angry target (*M* = 441.9, *SD* = 19.53). On incongruent trials, RT on trials with a neutral, happy or angry targets did not differ (p > .05). This pattern of finding conforms to the prediction made by the Dual Competition Model (discussed in greater detail in Chapter Four): On congruent trials with lower cognitive demands, emotion influenced performace, but on incongruent trials with greater cognitive control demands, RT on happy and angry trials did not differ from that on neutral trials.

ERP Measures

Amplitude and latency measures for N2 was analyzed using repeated measures ANOVA with target emotion (neutral, happy, angry) and flanker congruency (congruent, incongruent) as within-subject factors. N2 was analyzed at the midline electrode cluster where it was observed to be maximal. The ERP waveform is included in Appendix C.

For N2 amplitude, the main effect of target emotion was not significant ($F(2, 18) = 1.38, p = .28, \eta^2 = .133$). However, there was a marginally significant main effect of flanker congruency ($F(1, 9) = 4.44, p = .064, \eta^2 = .334$). At a trend level, the N2 amplitude was greater on incongruent ($M = -2.19 \mu$ V, SD = .72) than on congruent trials ($M = -1.37 \mu$ V, SD = .75). This pattern is consistent with previous findings in adults showing the N2 amplitude to be more pronounced on incongruent trials, indicative of the greater neural resources that are required to suppress interference on the incongruent trials. The interaction between target emotion and flanker congruency was not significant ($F(2, 18) = 0.67, p = .53, \eta^2 = .069$).

For N2 latency, the main effect of target emotion (F(2, 18) = 2.34, p = .13, $\eta^2 = .206$), flanker congruency (F(2, 18) = 2.54, p = .15, $\eta^2 = .220$), and the interaction between target emotion and flanker congruency (F(2, 18) = 0.16, p = .86, $\eta^2 = .017$) were all not significant.

Appendix C



Supplementary Figure: Adult Pilot Study for Chapter Four

Supplementary Figure 1: ERP waveform at the Frontal Midline electrode cluster. The N2 (shown above in the boxed region) was measured between 150 - 250 ms.