

**ERP AND BEHAVIORAL INDICATORS OF INHIBITORY CONTROL IN A
HIGH RISK POPULATION**

by
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ABSTRACT

Inhibitory control is an essential skill for multiple domains including academic functioning and peer relationships. The current study assessed the effectiveness of an attachment based parenting intervention (Attachment and Biobehavioral Catch-up), implemented in infancy, on inhibitory control when the children were eight years of age. Inhibitory control was measured among three populations - a high risk intervention group, a high risk control group, and a low risk comparison group. One hundred and four 8-year-old children underwent behavioral and event-related potential (ERP) measures during a forced-choice inhibitory control task - the Stop Signal Reaction Time Task (SSRT). Children in the low risk comparison group performed better than the high risk control group on behavioral indicators of inhibitory control. No significant differences emerged between the high risk intervention group and the low risk comparison group or the high risk control group. Children who did not receive the intervention performed worse on the SSRT task than children in the low risk comparison group. No differences were found in ERP amplitudes between groups.

Chapter 1

INTRODUCTION

Inhibitory control involves inhibiting a prepotent (or reflex-like) response in favor of an alternative response. Inhibitory control is necessary for success in academic and social domains (Champagne, 2010; Kochanska, Murray, & Coy, 1997; Richards & Wadsworth, 2004). Deficits in inhibitory control predict externalizing problems including Attention Deficit Hyperactivity Disorder (ADHD; Campbell, & von Stauffenberg, 2009; Oosterlaan, & Sergeant, 1996; Utendale, & Hastings, 2011). Sensitive parenting plays a significant role in improving the child's development of inhibitory control (Kochanska, Murray, & Harlan, 2000; Rueben, et al. 2016). Children exposed to early adversity are more likely to develop inhibitory control problems than children without similar early experience (McDermott, et al., 2013; Pears, Fisher, Bruce, Kim, and Yoerger, 2010; Rogosch, Dackis, and Cicchetti, 2011; Skowron, Cipriano-Essel, Gatzke-Kopp, Teti, and Ammerman, 2014). Furthermore, children who experience early adversity are less likely to receive sensitive and responsive parenting than children who are not exposed to early adversity (van Ijzendoorn, Schuengel, & Bakermans-Kranenburg, 1999). Due to the combination of early adversity and insensitive parenting, these children are at greater risk for developing externalizing and inhibitory control problems than children who are not exposed to early adversity and receive sensitive parenting (Lewis, Dozier, Ackerman, Sepulveda-Kozakowski, 2007; Teicher et al., 2003; Pears, Fisher, Bruce, Kim, & Yoerger, 2010). It is therefore important to use preventative interventions to target

parenting in populations experiencing early adversity, and in doing so protect these children from developing deficits in inhibitory control. In this study, we aim to analyze the effects of a preventative attachment-based intervention (Attachment and Biobehavioral Catchup; ABC) on later inhibitory control abilities through behavioral and physiological indicators of this regulatory process.

1.1 Inhibitory Control

Inhibitory control is essential for academic functioning and peer relations (Kochanska, Murray, and Coy, 1997). Poor inhibitory control interferes with children's abilities to learn and remain engaged in the classroom. For example, children with inhibitory control deficits are unable to prevent themselves from talking out of turn and disrupting the class. The inability to inhibit aggressive and socially inappropriate behaviors contributes to poor peer interactions (Bagwell, Molina, Pelham, & Hoza, 2001; Ciairano, Visu-Petra, Settanni, 2007; Wheeler, Maedgen, & Carlson, 2000). Huang-Pollock and colleagues (Huang-Pollock, Mikami, Pffner, and McBurnett, 2009) found that children with weaker inhibitory control abilities had increased difficulty detecting verbal cues, used more hostile statements during social interactions, and showed greater difficulty remaining on task than children with stronger inhibitory control abilities. Therefore, deficits in inhibitory control lead to decreased social functioning and difficulties in maintaining peer relationships (Huang-Pollock, Mikami, Pffner, and McBurnett, 2009). Ultimately, poor inhibitory control results in significant problems across multiple areas.

1.2 Early Adversity and Inhibitory Control

Due to the significance of inhibitory control in influencing multiple domains of functioning, it is critical to identify early risk factors for deficits in inhibitory control. The experiences of early adversity are one type of example of these risk factors. Children exposed to early maltreatment and neglect are considered high-risk populations and include children in institutional care, children in foster care, and children who have involvement with child protective services due to maltreatment and neglect. High risk populations are more likely to develop poor inhibitory control than children of low risk populations (McDermott, et al., 2013; Pears et al., 2010; Skowron et al., 2014). These deficits in inhibitory control are evidenced by behavioral and ERP indicators of performance on inhibitory control tasks. Specifically, institutionalized children, children in foster care, and children exposed to early maltreatment show poor inhibitory control through blunted neural reactivity, deficits in processing speed, and increased errors while completing inhibitory control tasks (Cowell, Cicchetti, Rogosch, & Toth, 2015; McDermott, et al., 2013; McDermott, Westerlund, Zeanah, Nelson, & Fox, 2012).

1.3 Parenting and Inhibitory Control

Parenting behaviors play a critical role in the development of regulatory abilities (Bernier, Carlson, & Whipple, 2010). In infancy, a responsive and sensitive parent is able to act as a co-regulator by supporting a young child in healthy regulatory strategies when engaging in emotional, attentional, and behavioral modulation tasks (Kopp, 1989). In particular, parental sensitivity plays a significant role in the co-

regulation process, and, therefore, in the development of self-regulation (Feldman, 2007a). Parental sensitivity represents the ability to be aware and responsive to a child's signals (Cohn & Tronick, 1988; Feldman, 2007a; Feldman & Greenbaum, 1997). Increased parental sensitivity results in stronger self-regulation (Feldman, 2003; Feldman, 2007a; Feldman, 2007b; Feldman & Greenbaum, 1997; Feldman, Greenbaum, & Yirmiya, 1999). For example, Feldman, Greenbaum, & Yirmiya (1999) found that maternal sensitivity measured when children were three and nine months old predicted stronger self-control at two years of age. As children develop, and become increasingly independent through advances in gross motor, cognitive, and language abilities, the parent's continued co-regulation with the child and modeling self-regulatory behaviors, leads the child to taking a more active role in self-regulation (Bridges & Grolnick, 1995; Diener & Mangelsdorf, 1999; Fox & Calkins, 2003). As a result of strong parental sensitivity, children learn that they are able to rely on the caregiver for support (including regulatory support), and develop consistent and adaptive regulatory strategies with the aid of the parent (Calkins & Hill, 2007; Schore, 2001). Due to early experience with a sensitive and supportive parent, these children are able to develop competent independent regulatory strategies.

In contrast, children exposed to inconsistent, frightening, or neglectful parenting do not experience the sensitive interactions necessary for the establishment of independent regulatory capabilities (Juffer, Bakermans-Kraenburg, van IJzendoorn, 2005; Lyons-Ruth, Alpern, & Repacholi, 1993; Perry, Calkins, & Bell, 2016). These children are likely to develop ineffective self-regulation strategies (Heikamp, et al.,

2012). For example, Perry, Calkins, and Bell (2016) found that maternal sensitivity when children were five months old was related to infant vagal withdrawal (an indicator of poor self-regulation) at ten months. The presence of self-regulatory deficits leads to a number of maladaptive outcomes including externalizing behaviors, attention problems, and other executive function deficits (Fearon, et al., 2010; Heikamp, et al., 2013; Kochanska, Philibert, and Barry, 2009).

1.4 The Attachment and Biobehavioral Catchup Intervention

Sensitive parenting acts as a significant buffer against later inhibitory control problems. Therefore, preventative interventions that target parenting can be useful in promoting the development of healthy inhibitory control strategies. The Attachment and Biobehavioral Catchup (ABC) intervention is an attachment-based parenting intervention focused on preventing maladaptive outcomes among populations exposed to early adversity (Dozier et al., 2006). This intervention is conducted in the home of the family and is made up of 10 weekly sessions. The aims of this intervention are to increase parental following the lead and nurturance and decrease frightening behaviors. By targeting parental behavior essential for the child's development of self-regulatory strategies, the intervention enhances the child's ability to self-regulate (Lewis-Morrarty et al., 2012). This intervention promotes sensitive parenting behavior that aides in the child's development of healthy regulatory strategies (Lewis-Morrarty et al., 2012). ABC has been found to improve patterns of diurnal cortisol production, improve the attachment relationship between the parent and child, decrease parental report of children's behavioral problems, enhance children's behavioral regulation

abilities, and increase executive functioning (Bernard et al., 2012; Dozier et al., 2006; Dozier et al., 2008; Lewis-Morrarty et al., 2012).

1.5 Inhibitory Control and Event Related Potentials

Multiple converging measures can be used to better understand inhibitory control; each individual methodology provides distinct information regarding this process. For example, electroencephalogram (EEG) data have been used to assess inhibitory control in both children and adults (Albrecht, Benaschewski, Brandeis, Heinrich, & Rothernberger, 2005; Brandeis et al., 1998; Dimoska, Johnstone, Barry, & Clarke, 2003; Kok, Ramautru, De Ruyter, Band, Ridderkinhof, 2004; Liotti et al., 2007; Lo et al., 2013; Overtom et al., 2002; Pliska, Liotti, & Woldorff, 2000; Schmajuk, Liotti, Busse, & Woldorff, 2006; Solanto et al., 2001). The added value of EEG methodology, beyond behavioral data alone, is providing time-sensitive information regarding neural reactivity to a significant internal or external events. This time-sensitive neural reactivity is represented by event-related potentials (ERPs). As a result, one can break down, and analyze, a larger process (e.g. inhibitory control) into smaller sub-processes (e.g. recognizing the need to inhibit and enacting inhibition). The ability to analyze each step (conflict monitoring and conflict resolution) of the inhibitory control process in a time-sensitive manner allows one to gain specific information regarding temporal reactivity in multiple aspects of the inhibitory control process. Furthermore, ERPs are more sensitive than behavioral data alone in identifying problems with inhibitory control in the conflict monitoring and conflict

resolution processes. ERPs can be used to supplement behavioral data (i.e. reaction time to a stimulus) by providing a temporally sensitive indicator of neural reactivity.

1.6 Inhibitory Control and the Stop Signal Reaction Time Task

The Stop Signal Reaction Time (SSRT) task is regularly used to assess behavioral and ERP indicators of inhibitory control problems. In this forced-choice task, a participant is provided with a “go” stimulus (left or right pointing arrow) prompting a behavioral response of either a left or right button push. This trial type is considered a “go” trial. In about a third of trials the participant is signaled to go, but the “go” stimulus followed shortly by a “stop” signal indicating that the participant should inhibit the prepotent response that had been activated by the “go” stimulus. In this trial, a participant is required to prevent a behavioral response that has already been prompted, and therefore, already in progress. The inhibitory control process in the SSRT task involves a competition between “go” and “stop” responses. If the “go” response is completed, then the participant failed to inhibit the motor response. In contrast, if the stop response is completed, the participant is successful in preventing the button push. These trials are labeled unsuccessful stop trials (USST) and successful stop trials (SST) respectively.

The SSRT task measures inhibitory control abilities, in part, through the identification of the amount of time required for successful inhibition of the button push in response to the “stop” signal. This Stop Signal Reaction time (SSRT) portrays the time necessary for a participant to recognize the “stop” signal *and* inhibit the already commenced behavioral response. For individuals with poor inhibitory control,

more time is required to complete this process (Huang-Pollock, Mikami, Pffiffner, and McBurnett, 2007; Huang-Pollock, Mikami, Pffiffner, and McBurnett, 2009; Solanto et al., 2001). In fact, those with ADHD, a population with inhibitory control deficits, show significantly longer SSRT's in comparison to healthy controls (Albrecht et al., 2005; Brandeis et al., 1998; Dimoska, Johnstone, Barry, & Clarke, 2003; Johnstone, Barry, & Clarke, 2007; Oades, 1998). For example, Oosterlaan and Sergeant (1996) used the SSRT task to compare inhibitory control processes between children diagnosed with ADHD, aggressive children, and anxious children. The authors found that the two former groups presented with poor inhibitory control in comparison to anxious children as evidenced by slower SSRTs indicating increased time required to inhibit a behavioral response.

1.7 The Stop Signal Reaction Time Task and ERPs

The process of inhibitory control is made up of several smaller processes that can be observed through the measurement of ERPs. Kok (1986) describes the ability to recognize the “stop” signal as a “red flag” or indicator of the need to inhibit a behavioral response. Additionally, Rubia and colleagues (Rubia et al., 1999) highlight the process of inhibiting this response as the “brake.” These flag and brake processes can be further elucidated using EEG methodology; specific ERPs have been associated with the individual flag and brake processes. The N2 component is a negative deflection that occurs about 200 ms after the onset of the “stop” signal and represents the ability to recognize the “stop” signal (the “flag”; Kok et al., 2004). This neural reactivity is primarily located in the fronto-central regions of the brain (Kok et al.,

2004; Rubia et al., 1999). In fact, Rubia and colleagues (1999) found decreased activation of the prefrontal cortex among ADHD participants during an inhibitory control task. In addition, the P3 component is a positive deflection that occurs around 300 ms after the “stop” signal and is activated during the “brake” stage of the process when an individual initiates the inhibition of the behavioral response (Kok et al., 2004). This activity occurs primarily in the frontal areas of the brain (Kok et al., 2004). Adults and children with inhibitory control problems show blunted N2 and P3 amplitudes while completing the SSRT task (Albrecht, Benaschewski, Brandeis, Heinrich, & Rothernberger, 2005; Brandeis et al., 1998; Johnstone, Barry, & Clarke, 2007; Liotti et al., 2007; Liotti, Pliszka, Higgins, Perez, Semrud-Clikeman, 2010; Oosterlaan, Logan, & Sergeant, 1998; Overtom et al., 2002; Pliszka, Liotti, & Woldorff, 2000; Solanto et al., 2001). These ERP results suggest that those with inhibitory control deficits show a decrease in neural activity during a task that targets this process. Ultimately, EEG methodology provides additional, temporally sensitive, information concerning specific “flag” and “brake” processes involved in inhibitory control.

1.8 The Present Study

Children who experience early adversity are at greater risk for developing poor inhibitory control than children without exposure to early adversity, as evidenced through both behavioral and physiological measures (Bruce et al., 2013; Lewis et al., 2007; McDermott et al., 2012; Pears et al., 2010). Due to the negative long-term effects of poor inhibitory control it is essential to target this process in a preventative

intervention. Information regarding how individuals with inhibitory control deficits perform on the distinct processes (conflict monitoring and conflict resolution) that make up inhibitory control can be useful in identifying how to target inhibitory control in interventions. ERP methodology is essential in detecting abnormalities within the conflict monitoring and conflict resolution process. The current study aimed to analyze inhibitory control deficits in a high risk population in comparison to a low-risk population. Furthermore, the current study aimed to analyze the effectiveness of a parenting-based attachment intervention intended to improve regulatory processes in children. In the current study, parents in the high-risk group were previously enrolled in either the ABC intervention or a control intervention (Developmental Education for Families; DEF). DEF focused on improving developmental areas and language abilities and was the same length as ABC. Based on previous evidence indicating the efficacy of the ABC intervention in targeting executive functioning and regulatory processes, we hypothesized that children in the DEF group would exhibit poorer inhibitory control than both the ABC group and a low-risk comparison group (Lewis-Morrarty et al., 2012).

Chapter 2

METHOD

2.1 Participants

The study included 152 children (81 males) who ranged in age from 6.92 years to 9.08 years ($M = 8.4657$, $SD = 0.377$). Of these participants, 48 children were excluded for the following reasons: did not meet behavioral criteria for inclusion ($n = 35$), noisy EEG data ($n = 4$), computer or experimenter error ($n = 2$), inability to complete the task ($n = 6$), and too young ($n = 1$). The remaining 104 children (60 males) ranged in age from 8.00 years to 9.08 years ($M = 8.530$, $SD = 0.364$). 57 of these children were members of the high risk group (26 ABC) and the remaining 47 were part of the low risk comparison group. The parents and children in the high risk group were referred by agencies working with Child Protective Services (CPS) in a large mid-Atlantic city. Families were identified on the basis that the children were at risk for maltreatment. These risks include the possible presence of domestic violence, parental substance use, homelessness, and neglect. Dyads were recruited to voluntarily participate in a randomized clinical trial of a parenting intervention and were randomly assigned to the experimental intervention, ABC, or to the control intervention, DEF. The remaining participants were part of a low-risk comparison group made up of children of similar age, race, and ethnicity to the high risk group. These children and their parents were recruited from the area surrounding the University and had no history of involvement with CPS. Analyses were conducted in order to ensure that excluded participants did not differ significantly from included

participants on gender ($\chi^2(1, N = 152) = 2.565, p = .109$) or low vs. high risk group membership ($\chi^2(1, N = 152) = 1.903, p = 0.168$). An independent samples t-test indicated that included participants were significantly older than excluded participants ($t(150) = -3.194, p = 0.002, M_{excluded} = 8.326$ years, $M_{included} = 8.5301$). This difference remained significant even when the participant too young for the study was excluded from analyses ($t(149) = -2.843, p = .005$). Further independent samples t-tests were conducted to identify whether significant differences emerged based on exclusion due to behavioral criteria and inability to complete the task or other reasons, such as, noisy EEG data or experimenter and computer error. These analyses reveal that this difference was significant only for those who were unable to complete the task ($t(143) = 2.811, p = .006$) and not for participants that were excluded due to other reasons ($t(108) = 0.830, p = 0.408$). Therefore, this distinction in age is likely a result of further development attention span in older children.

2.2 Procedure

2.2.1 Participant Recruitment

Parents and children from the high risk group were initially referred to the study after involvement with CPS. Families were originally contacted if children were less than 2 years of age and living with their biological parents. After referral, research staff contacted parents to attain consent. In the case that parents indicated interest in participation in the study, high risk parent-child dyads were randomly assigned to the experimental or control intervention. Children and parents participated in initial pre-intervention visits, intervention visits, and post-intervention visits through 4 years of

age. Some children and parents were contacted when the child was 5 years of age to participate in an additional study. These families were also contacted for an additional laboratory visit at the age of 8 years.

The remaining group of children (low risk comparison group) were recruited by using word of mouth and presentations conducted by research staff at local organizations such as Boys and Girls Clubs, YMCA's, playgrounds, and parks. In order to participate in the study, parents completed a brief screening interview to ensure that there was no presence of a history of involvement with the child welfare system, no history of caregiver hospitalization for psychiatric problems, no history of homelessness, no treatment for substance use, no illegal drug use, and no history of incarceration. These children did not have participate in any studies, specific to this lab, prior to their initial 8-year visit.

2.2.2 Data Collection

For the purpose of the current project all data was collected at the 8-year laboratory visit.

2.3 Measures

2.3.1 Inhibitory Control

In order to measure inhibitory control, children completed the Stop Signal Reaction Time (SSRT) task while wearing an EEG cap. At the beginning of each trial a green arrow was presented in the center of the screen pointing to the left or right. The child was required to press the button associated with the direction of the arrow during “go” trials. In contrast, in “stop” trials a red circle appeared superimposed on

the green arrow at various onset asynchronies (see below). The child was required to inhibit the button press on trials when the red circle appeared if he (or she) can. Children were instructed to engage in the button press as soon as the “go” signal emerged, and avoid waiting for the “stop” signal. For trials during which the child was successful in inhibiting the behavioral response, the trial was labeled a successful stop trial (SST). In contrast, trials in which the child is unsuccessful in inhibiting the behavioral response were categorized as unsuccessful stop trials (USST).

In the Stop Signal task, two types of processes are competing against each other – a ‘go’ process initiated by the green arrow and a ‘stop’ process initiated by the red circle. When the stop process finishes first (‘wins’) the participant is successful in inhibiting the motor response. In contrast, when the go process ‘wins’, the participant is unsuccessful in inhibiting the motor response and proceeds with the button press.

At the beginning of the task, participants completed two blocks of practice trials for a total of 100 practice trials. After completing the practice, participants were provided with four blocks of sixty trials for a total of 240 test trials. Participants were allowed to take a break in between blocks. Each trial lasted for about 1200 ms with an inter-trial interval of 2000 ms. In each “stop” trial there was a delay (Stop Signal Delay; SSD) that varied in duration between the presentation of the “go” stimulus (green arrow) and the “stop” stimulus (red circle). This delay was varied through the use of a tracking procedure (see Logan, 1994). This SSD was purposefully manipulated such that the child was able to inhibit a behavioral response for about half of the “stop” trials. At the commencement of the practice trials, the SSD was set at

200 ms. Every time the participant failed to inhibit a response, 50 ms was subtracted from the SSD in order to shorten the time between the presentation of the “go” signal and the “stop” signal; for example, 200 ms will be changed to 150 ms. Theoretically, the “go” and “stop” processes are independent and race to completion. When the go” signal and “stop” signals are presented closely in succession (small SSD), the “stop” process is initiated quickly after the “go” process. Therefore, the stop process has a sufficient amount of time to be completed and result in successful inhibition. In the case that the participant was successful in inhibiting the behavior response, 50 ms was added to the SSD in order to increase the delay and make inhibiting a behavioral response more difficult; when there is a large SSD, the “go” signal is initiated far sooner than the “stop” signal. As a result, there is not enough time for the “stop” signal to be completed and the participant is not successful in inhibiting the behavioral response. This varying of the SSD continued throughout the task in order to create an average SSD, such that the participant was able to successfully inhibit his behavioral response about half of the time. Throughout the task, about a third of trials were “stop” trials.

SSRT Calculation. During this task a stop signal reaction time (SSRT) is calculated. This reaction time represents the amount of time from the “stop” signal that is needed for the participant to inhibit a motor response. There are several assumptions necessary to calculate the SSRT. Firstly, the “go” and “stop” processes are independent. In addition, the SSRT is assumed to be the same across trials. Lastly, the distribution of “go” processes is assumed to be the same on both “go” and “stop”

trials. As a result of these assumptions, the distribution of “go” trials and the probability that a “go” process will be successfully completed can be used to calculate the SSRT. The “go” trials are rank ordered by increasing reaction time (see Figure 1). In the figure, the dotted line, dissecting the distribution of “go” trials, separates the “go” processes that are likely to be completed (left side) from the “go” processes that are unlikely to be completed (right side). When the “go” process is likely to be completed, inhibition is unsuccessful. Therefore, these trials correspond with USSTs. In order to identify which trial separates the likely to be completed “go” trials from the unlikely to be completed “go” trials (the dissecting line in Figure 1), the number of “go” trials that correspond to USSTs needs to be calculated. This is done by multiplying the percentage of “stop” trials that are USSTs by the total number of “go” trials. The resulting number (n) represents the n th trial in the rank ordered distribution of “go” trials. This n th trial provides the reaction time, from the “go” stimulus that, that separates likely to be completed “go” processes from unlikely to be completed “go” processes. This reaction time is known as the Go RT and is made up of two components (see Figure 1): the delay between the “go” signal and “stop” signal that is necessary for a participant to successfully inhibit (SSD) and the amount of time from the “stop” signal necessary to inhibit a response (SSRT). Therefore, by taking the Go RT and subtracting out the SSD, one is left with the SSRT.

2.4 EEG Recording and Analysis

EEG recording was conducted with a cap of 32 Ag/AgCl embedded electrodes with a forehead ground cite (AFz) and referenced using the right mastoid reference

(M2). The continuous EEG was digitized at 1024 samples per second. Data was processed using Advanced Source Analysis (ASA) software. The data was re-referenced offline using an average mastoid reference. EEG data was corrected for eye blinks and then band-pass filtered from 0.1 to 20 Hz. All trials containing artifacts outside of -75 to 75 μ V were rejected.

Four ERPs were constructed for each participant. In the SSRT task the “go” and “stop” signals are presented closely in succession. As such, the ERP responses produced are separate, but do overlap. Therefore, the “stop” ERPs are contaminated by the “go” ERPs. In order to target the “stop” ERP responses all “go” ERP responses must be subtracted from the “stop” ERP responses. In order to do this, ERPs for both “stop” and “go” trials are identified. First, ERPs that correspond with SSTs and USSTs are identified. Then the average SSD for successful “stop” trials is used to identify the start of the slow “go” ERP and the average SSD for unsuccessful “stop” trials is used to identify the start of the fast “go” ERP. ERPs across trials were baseline corrected using the previous 100 ms and then averaged across trial types. As described above, the “go” ERPs need to be subtracted from the “stop” ERPs as these responses overlap. As SSTs are contaminated by slow “go” responses and USSTs are contaminated by fast “go” responses the following difference waveforms were created: SST – slow “go” and USST – fast “go” (see De Jong, Coles, Logan, and Gratton, 1990). The resulting difference waveforms represent the ERP responses to the “stop” signal for successful inhibition (SST – slow “go”) and unsuccessful inhibition (USST – fast “go”).

Chapter 3

RESULTS

3.1 Behavioral Results

The values for SSRT, Go RT, percent of go trials that were omitted, stop signal delay, successful stop signal delay, and unsuccessful stop signal delay are presented in Table 1 for each group (low risk comparison, high risk intervention, and high risk control). In accordance with the model described to calculate the SSRT, a paired samples t-test confirmed that the stop signal delay for successful trials was shorter than for unsuccessful trials ($t(103) = -14.783, p < .001$).

Given that previous research indicates that SSRT decreases with age, a correlation was conducted between age and SSRT (Williams, Ponsse, Schachar, Logan, & Tannock, 1999). A significant correlation was found between age and SSRT, with SSRT older children demonstrating shorter SSRTs ($r = -.273, p < .01$). Therefore, age was included as a covariate in primary analyses.

Primary analyses using SSRT, a common marker of inhibitory control, included a one-way between subjects ANCOVA that was conducted to examine the effects of group [high risk intervention (ABC), high risk control (DEF), and low risk comparison groups] on reaction time. Results indicate a marginally significant effect of group ($F(2, 100) = 2.948, p = .057$). Post-hoc t-test analyses reveal a significant difference between the high-risk control group ($M = 382.02, SD = 15.11$) and the low-risk comparison group ($M = 327.10, SD = 12.27; t(76) = -2.73, p < .01$) indicating a

significantly longer SSRT for the high-risk control group than the low-risk comparison group. Differences between the high risk intervention group ($M = 351.36$, $SD = 79.93$), the high risk control ($M = 384.99$, $SD = 98.80$) group, and the low risk comparison group ($M = 333.08$, $SD = 69.23$) were not significant ($t(55) = -1.394$, $p > .05$; $t(71) = 1.022$, $p > .05$).

3.2 ERP Analyses

Regions of interest (ROIs) were created for fronto-central (Fz, FC1, FC2, Cz) and central-parietal (Cz, CP1, CP2, Pz) areas based on previous findings (Liotti, et al., 2005). The waveforms at each of the electrodes were averaged to create average waveforms across electrodes for both fronto-central and central-parietal regions. The SSRT task has commonly produced the N2 and P3 components (Kok, et al., 2004), but earlier components (N1 and P2) emerged in this study (see figures 1-3). Based on previous findings (Dimoska, Johnstone, Barry, and Clarke, 2003; Johnstone, et al., 2007; Liotti, et al., 2007; Lo, et al., 2013; Overtom, et al., 2002) and visual inspection of grand-average waveforms, the following time windows were identified for each component: N1 (100-200 ms); P2 (200-275 ms); N2 (275-350 ms); P3 (350-800 ms) and the N1, N2, P2, and P3 components were measured at the fronto-central and central-parietal ROIs. Paired samples t-tests were conducted amplitudes between fronto-central and central-parietal ROIs for SSTs and USSTs. Results revealed no significant difference between ROIs for SSTs and USSTs for the N1 component ($t(101) = -1.122$, $p > 0.05$ and $t(101) = .343$, $p > .05$ respectively). For the P2 component, results revealed significant differences between frontal-central and

central-parietal ROIs for SSTs ($t(101) = 4.851, p < .001$) and USSTs ($t(101) = 4.525, p < .001$) with amplitudes in frontal-central ROI ($M_{\text{frontal-central SST}} = 280.864; M_{\text{frontal-central USST}} = 108.408$) larger than the of central-parietal ROI ($M_{\text{central-parietal SST}} = 74.091; M_{\text{central-parietal USST}} = -117.659$). For the N2 component, no significant differences were found between frontal-central and central-parietal ROIs for SSTs ($t(101) = 1.081, p > .05$) and USSTs ($t(101) = -.401, p > .05$). Lastly for the P3 component, whereas no significant difference was found between frontal-central and central-parietal ROIs for SSTs ($t(101) = -1.590, p > .05$), a significant difference was found between frontal-central and central-parietal ROIs for USST ($t(101) = -5.124, p < .001$) with the frontal-central area ($M_{\text{frontal-central USST}} = 5195.264$) displaying smaller amplitudes than in the central-parietal area ($M_{\text{central-parietal USST}} = 6807.790$). Based on previous findings indicating that these components occur in the frontal-central ROI, the remaining results are analyzed in the frontal-central ROI (Kok, et al. 2004).

3.2.1 N1 Component

A repeated measures ANOVA was conducted to compare effects of group type (intervention group, control group, or comparison group) and trial type (SST and USST) on ERP amplitude for the N1 component. No statistical difference was found based on trial type ($F(2, 99) = .111, p > .05$) or group type ($F(2, 99) = 1.203, p > .05$). No significant interaction was found between group type and trial type ($F(2, 99) = 1.685, p > .05$).

3.2.2 P2 Component

A repeated measures ANOVA was conducted to compare effects of group type (intervention group, control group, or comparison group) and trial type (SST and USST) on ERP amplitude for the P2 component. No significant effect was found for trial type ($F(1, 99) = .688, p > .05$) or group type ($F(2, 99) = 1.411, p > .05$). No significant interaction was found between trial type and group type ($F(2, 99) = 1.969, p > .05$).

3.2.3 N2 Component

A repeated measures ANOVA was conducted to compare effects of group type (intervention group, control group, or comparison group) and trial type (SST and USST) on ERP amplitude for the N2 component. No significant effect was found for trial type ($F(1, 99) = .900, p > .05$) or group type ($F(2, 99) = .576, p > .05$). No significant interaction was found between trial type and group type ($F(2, 99) = 2.107, p > .05$).

3.2.4 P3 Component

A repeated measures ANOVA was conducted to compare effects of group type (intervention group, control group, or comparison group) and trial type (SST and USST) on ERP amplitude for the P3 component. A significant main effect was found for trial type ($F(1, 99) = 21.189, p < .001$) with a larger amplitude for USST ($M = 5062.16$) than for SST ($M = 643.12$). No significant effect was found for group type ($F(2, 99) = .299, p > .05$). No significant interaction was found between trial type and group type ($F(2, 99) = .325, p > .05$).

Table 1 *Means and Standard Deviations for Behavioral Data*

	High Risk Intervention Group (n = 26)		High Risk Control Group (n = 31)		Low Risk Comparison Group (n = 47)	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Stop Signal Reaction Time	351.36	79.93	384.99	98.80	333.08	69.23
Go Reaction Time	690.65	93.31	671.77	119.12	688.94	113.47
% Omissions	9.85	6.99	8.22	6.33	6.50	5.62
Stop Signal Delay	343.14	121.49	293.95	102.56	372.90	122.47
Successful Stop Signal Delay	323.88	111.25	279.02	99.06	348.05	114.38
Unsuccessful Stop Signal Delay	364.61	135.00	309.97	109.70	400.80	132.68

Figure 1

The distribution of rank ordered "go" reaction times across "go" trials

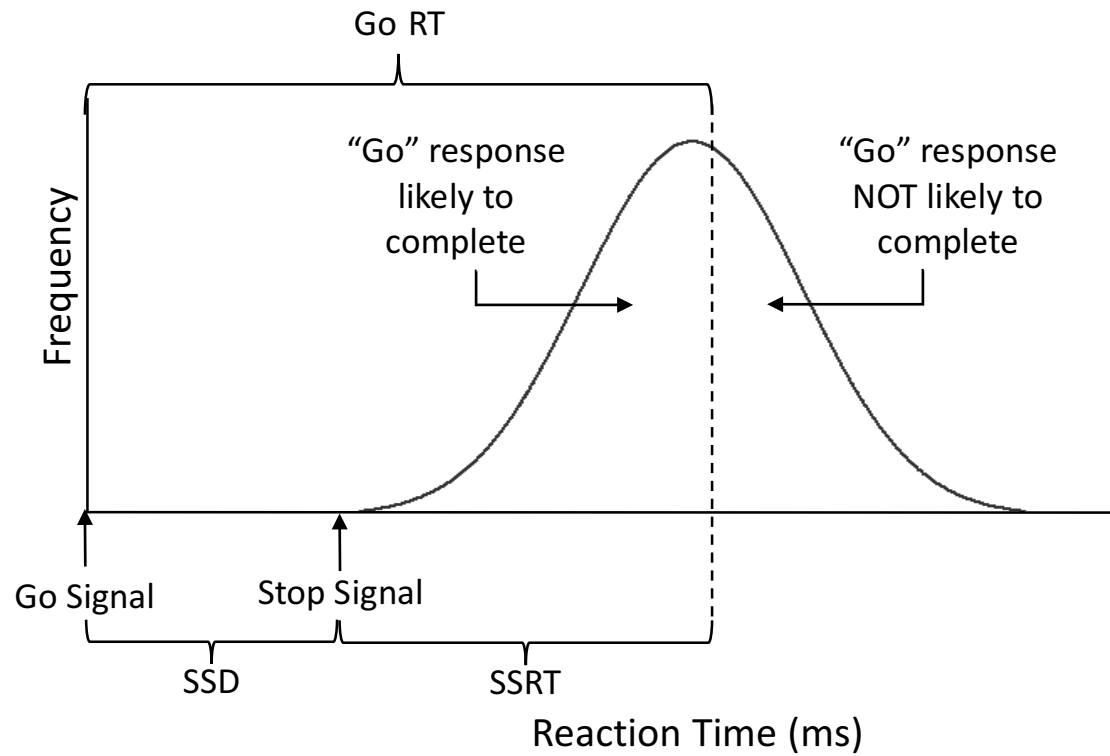


Figure 2

Grand Average Waveforms at the Frontral-central ROI for Successful Stop Trials and Unsuccessful Stop Trials Across Groups

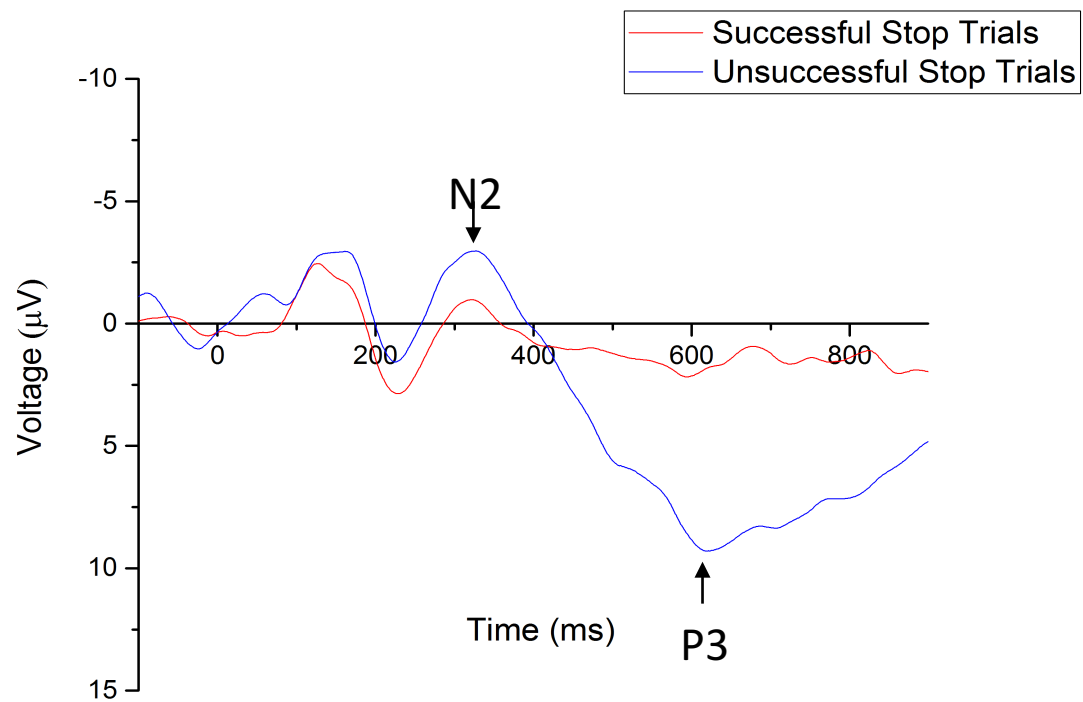


Figure 3

Grand Average Waveforms at the Central-parietal ROI for Successful Stop Trials and Unsuccessful Stop Trials Across Groups

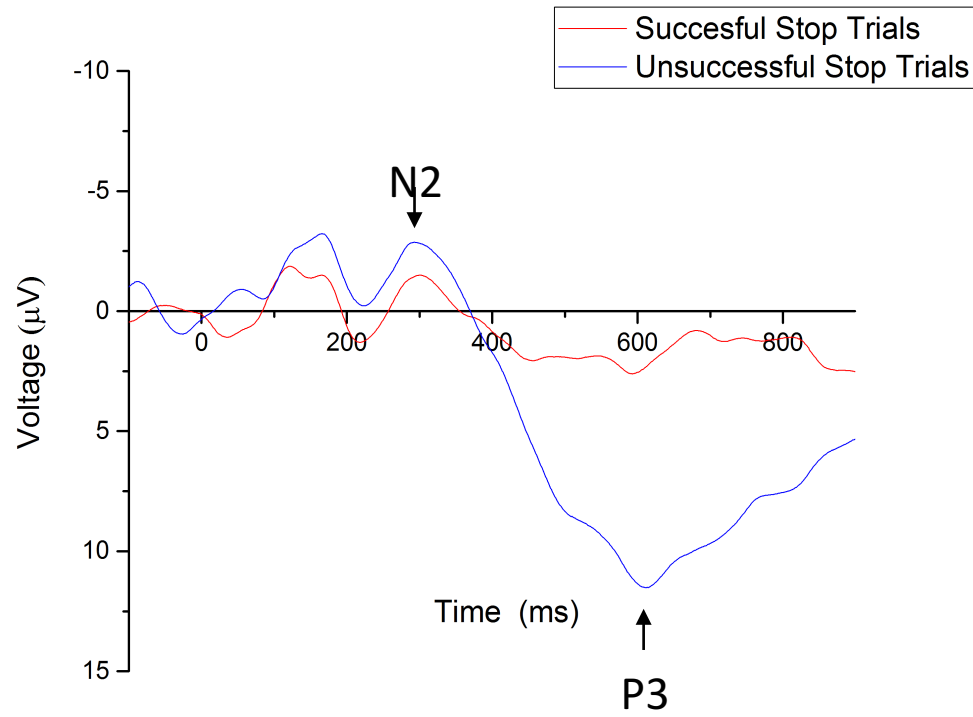


Figure 4

Grand Average Waveforms at the Frontal-central ROI for Successful Stop Trials Between Groups

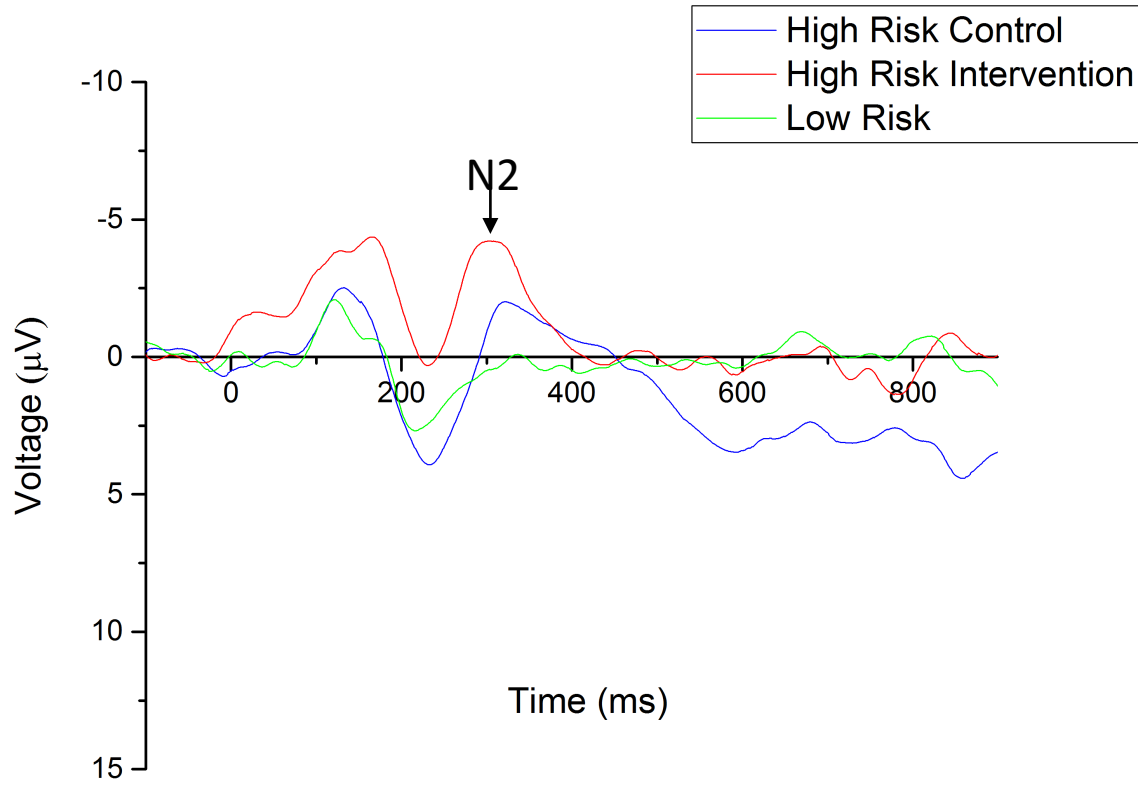


Figure 5

Grand Average Waveforms at the Frontal-central ROI for Unsuccessful Stop Trials Between Groups

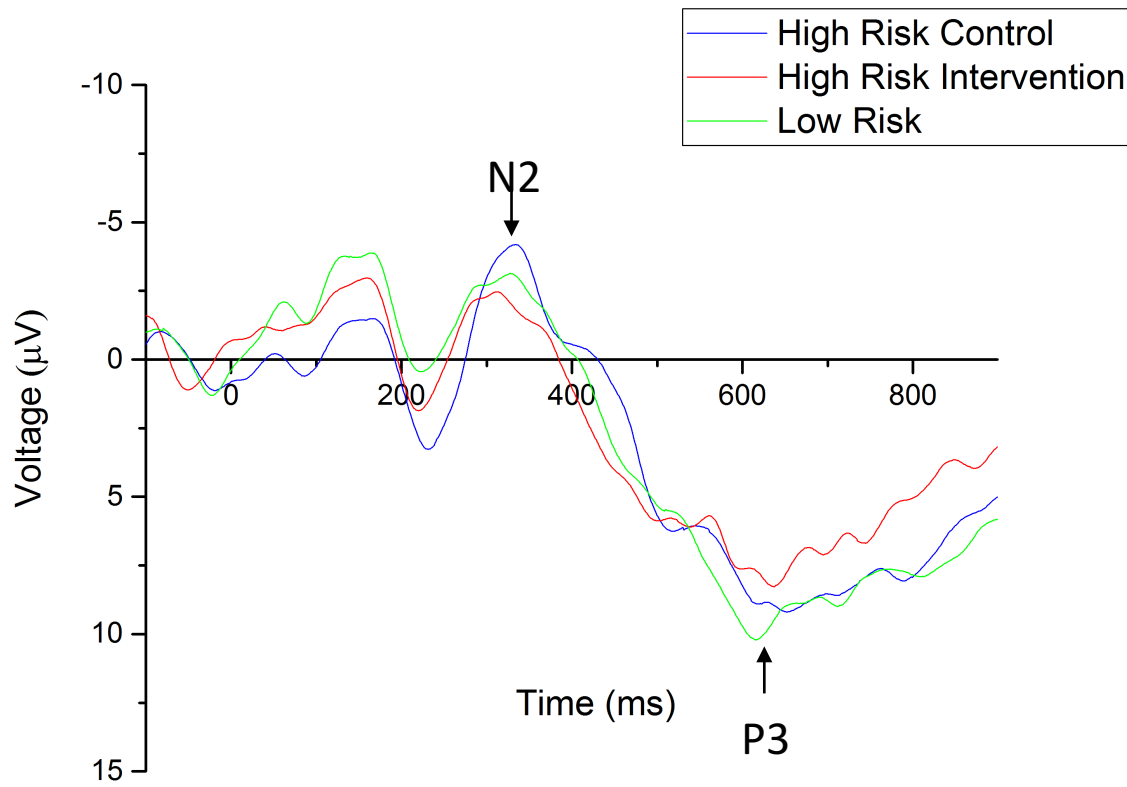


Figure 6

Grand Average Waveforms at the Central-parietal ROI for Successful Stop Trials Between Groups

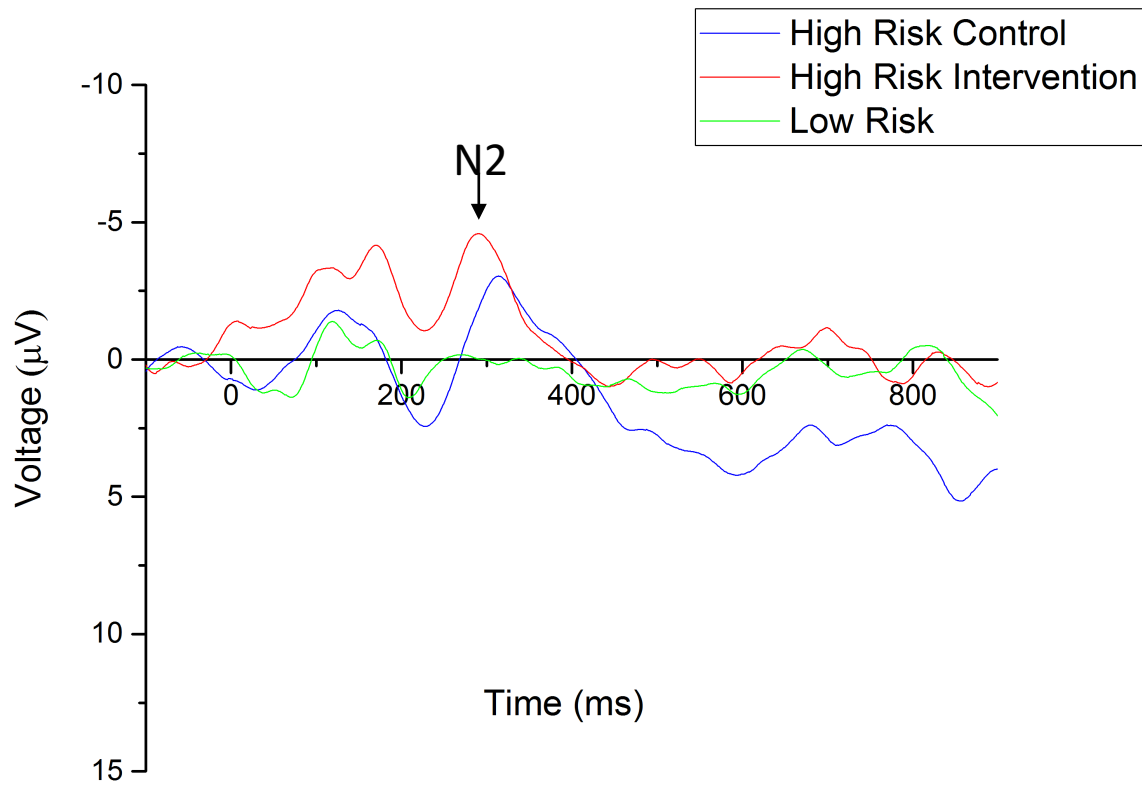
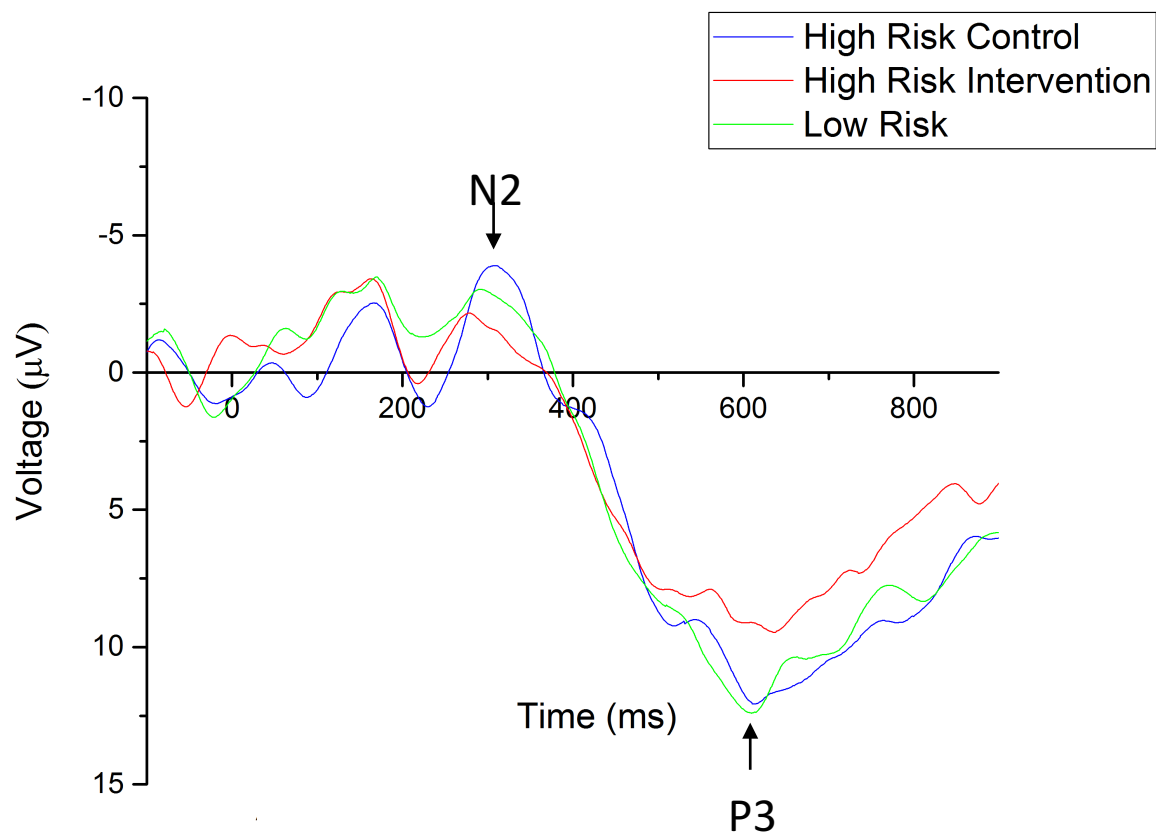


Figure 7

Grand Average Waveforms at the Central-parietal ROI for Unsuccessful Stop Trials Between Groups



Chapter 4

DISCUSSION

Inhibitory control is a self-regulatory process that influences performance in social and academic arenas (Kochanska, Murray, and Coy, 1997). Poor inhibitory control places one at risk for the development of later psychopathology (Campbell, & von Stauffenberg, 2009; Oosterlaan, & Sergeant, 1996; Salas, Fuentes, Bernedo, Garcia-Martin, 2016; Utendale, & Hastings, 2011). As sensitive parenting plays a significant role in promoting strong self-regulatory abilities, it is important to use interventions that target parenting in order to prevent deficits in inhibitory control and protect against later psychopathology (Kochanska, Murray, & Harlan, 2000; Rueben, et al. 2016). Therefore, the current study analyzed the effects of an attachment-based parenting intervention implemented in infancy on inhibitory control, with inhibitory control assessed by ERP and behavioral methodology, in 8-year-old children.

In this study we hypothesized that children whose parents received the intervention and children in the low risk comparison group would perform better on behavioral measures of inhibitory control than children in the high risk control group. The results reveal that the low risk comparison group exhibited a significantly faster SSRT than the high risk control group (DEF). In contrast, no differences were found between the intervention group and the low risk comparison group or high risk control group. These results highlight that children in the high risk control group perform

significantly worse at an inhibitory control task than children in the low risk comparison group.

With regards to ERP, we hypothesized that children in the high risk control group would demonstrate more blunted ERP amplitudes than children in the high risk intervention group and the low risk comparison group. Our results did not support this hypothesis. Specifically, no differences were found in ERP amplitudes between the intervention group, low risk comparison group, and high risk control group.

It is possible that the failure to find differences is a result of frontal lobe development. In a meta-analysis, Romine and Reynolds (2005) found the frontal lobe continues to develop throughout childhood, adolescence, and into early adulthood. As a result, it is possible that as the children in this study age and frontal lobe development progresses, differences will emerge between the high risk intervention group, high risk control group, and low risk comparison group.

4.2 N2 and P3 Components

Typically, when the SSRT task is completed by adults, a larger amplitude is produced for the N2 component when failing to inhibit a behavioral response (USST) than when successfully inhibiting a behavioral response (SST; Dimoska, Johnstone, & Barry, 2006). This finding is expected when considering the hypothesis that the N2 component represents the “flag” that indicates a conflict; therefore when inhibition fails this “flag” is larger than when inhibition is successful (Kok, 1986). In contrast, for the frontal P3 component a larger amplitude is produced for successful inhibition than unsuccessful inhibition (Dimoska, Johnstone, & Barry, 2006; Kok, 1986). This is

consistent with the view that the P3 component represents the “brake” or stopping, and, therefore, successful stopping would produce a larger amplitude than unsuccessful stopping (Rubia et al., 1999). However, our results showed no differences between SST and USST amplitudes for the N2 component and a larger USST amplitude than SST amplitude for the P3 component. Furthermore, our findings indicate a larger P3 amplitude in the central-parietal region than in the frontal-central region, which contradicts previous SSRT findings (see Falkenstein, Hoorman, & Hohnsbein, 1999) indicating a larger P3 in the frontal-central region than in the central-parietal region. Although these results are not consistent with previous findings, Johnstone, Barry, and Clarke (2007) and Johnstone and colleagues (Johnstone, et al. 2007) also found larger P3 amplitudes for children in the parietal ROI than in the frontal or central ROI. Furthermore, whereas, the “flag” and “brake” model refers to the P3 as solely an indication of stopping (Kok, 1986) there is an alternative theory concerning the nature of the P3 component that may be consistent with our pattern of results. Kok (1999) describes two distinct P3 components, a P3 labeled a novelty P3 in response to non-target stimuli (the stop signal) primarily located in the frontal-central region and a target P3 in response to target stimuli primarily located parietally. It is possible that the P3 component that emerged in this study is the target P3 that is elicited when one is presented with task related stimuli (the go signal). In contrast, the fact that the novelty P3 is not elicited may be a result of the fact that the frontal lobe is continuing to develop at this age (Romine and Reynolds, 2005).

4.2 N1 and P2 Components

Interestingly, additional components (N1 and P2) to the N2 and P3 components emerged in our data. Pliszka, Liotti, and Woldroff (2000) hypothesize that these N1 and P2 components might represent early orienting to the “go” stimulus. Further research needs to be conducted in order to replicate these findings and determine the nature of these early components.

4.3 Strengths and Limitations

There are multiple strengths of the current study including the nature of this sample; the makeup of this sample is an ethnically diverse one, which increases the generalizability of the results. Furthermore, to our knowledge, this is the first study to measure SSRT performance using behavioral and ERP methodologies in a high risk sample. This is a significant contribution as these high risk populations are more likely than low risk populations to develop deficits in inhibitory control (McDermott, et al., 2013; Pears et al., 2010; Skowron, et al. 2014).

There are also several limitations to consider. Whereas the overall sample size is large for a study of this kind, the sample sizes of the individual groups are unbalanced, and in some cases, small. This limitation is particularly relevant when considering the behavioral data from this study. Further research should be conducted using a larger sample size. In addition, future studies should analyze inhibitory control using ERP methodologies in an older high-risk sample. As the young age of the current sample may be responsible for the inconsistency between current findings and

the robust literature, conducting this study with older children may produce results that are more consistent.

In conclusion, our study demonstrates that children who did not receive the intervention performed significantly worse than children in the low risk comparison group on behavioral measures of inhibitory control. These results indicate that children in the high risk control group produced SSRTs farthest from the low risk comparison group.

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Appendix
IRB APPROVAL LETTER



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TO: Mary Dozier, PhD
FROM: University of Delaware IRB (HUMANS)

STUDY TITLE: [547621-10] Intervening Early with Neglected Children: Key Middle Childhood Outcomes

SUBMISSION TYPE: Continuing Review/Progress Report

ACTION: APPROVED
APPROVAL DATE: February 20, 2017
EXPIRATION DATE: February 14, 2018
REVIEW TYPE: Full Committee Review

Thank you for your submission of Continuing Review/Progress Report materials for this research study. The University of Delaware IRB has APPROVED your submission. This approval is based on an appropriate risk/benefit ratio and a study design wherein the risks have been minimized. All research must be conducted in accordance with this approved submission.

This submission has received Full Committee Review based on the applicable federal regulation.

Please remember that informed consent is a process beginning with a description of the study and insurance of participant understanding followed by a signed consent form. Informed consent must continue throughout the study via a dialogue between the researcher and research participant. Federal regulations require each participant receive a copy of the signed consent document.

Please note that any revision to previously approved materials must be approved by this office prior to initiation. Please use the appropriate revision forms for this procedure.

All SERIOUS and UNEXPECTED adverse events must be reported to this office. Please use the appropriate adverse event forms for this procedure. All sponsor reporting requirements should also be followed.

Please report all NON-COMPLIANCE issues or COMPLAINTS regarding this study to this office.

Please note that all research records must be retained for a minimum of three years.

Based on the risks, this project requires Continuing Review by this office on an annual basis. Please use the appropriate renewal forms for this procedure.

If you have any questions, please contact Maria Palazuelos at (302) 831-8619 or mariapj@udel.edu. Please include your study title and reference number in all correspondence with this office.