THESIS

A PILOT STUDY: INHIBITION IN EATING BEHAVIOR OF CHILDREN THROUGH EATING IN THE ABSENCE OF HUNGER PARADIGM AND ELECTROENCEPHALOGRAPHY

Submitted by

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ABSTRACT

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Objective. Eating behaviors contribute to the issue of obesity. The purpose of this research is to determine if there is an association between neural inhibition and behavioral inhibition relating to eating in children (age 4-6 years). **Method**. Neural inhibition was measured via EEG recordings during go/no-go task using visual food cues. Behavioral inhibition was measured via the amount consumed in free access phase of Eating in the Absence of Hunger (EAH) protocol. Other outcome measures included: Child Feeding Questionnaire (CFQ), a survey developed based on the Picky Eating in Toddlers and Preschoolers Questionnaire, and body mass index (BMI) measurements. **Results**. This study found a significant correlation between BMI and kcal consumed in free access phase of EAH protocol (p=.006). This study did not find significant correlation between neural inhibition, as measured by N2 amplitude during no-go tasks, and kcal during free access phase. **Conclusions**. Neural processing of young children possibly varies from that of older children and adults. Further research should be conducted to determine the development of neural processing in children as well as the relationship between development and behavioral inhibition related to eating behaviors.

TABLE OF CONTENTS

THE ISSUE OF OBESITY

Childhood obesity is an epidemic in the United States. The most recent estimation of obesity prevalence among child and adolescents in the United States is 17% (Ogden et al., 2016). Obesity prevalence is based on body mass index (BMI) which is determined as a calculation of weight (pounds) divided by height squared (inches). These BMI values are interpreted via CDC growth charts which are based on national survey data. For children, obesity is defined as a BMI at or above the 95th percentile for children of the same age and sex. Overweight is defined as BMI between 85th and 95th percentile for children of the same age and sex (cdc.gov). Obesity is a major concern because this health condition has many negative health outcomes. According to the Center for Disease Control (2017), obesity during childhood can increase risk for sleep apnea (Institute of Medicine [IOM], 2012), type 2 diabetes (IOM, 2012; US Department of Health and Human Services [US DHHS], 2010; May, Kuklina, & Yoon, 2012), heart disease (US DHHS, 2010; May, Kuklina, & Yoon, 2012), hypertension, liver disease, orthopedic problems, and atherosclerosis (Freedman, Dietz, Srinivasan, & Berenson, 2007). Furthermore, childhood obesity can lead to obesity in adulthood (Gurnani, Birken & Hamilton, 2015; Puhl & Luedicke, 2012). I believe that if eating behaviors which contribute to overweight and obesity can be better understood, the rate and effect of this epidemic may be impacted.

In order to decrease the prevalence of obesity, the causes of the disease need to be addressed. Obesity is thought to be caused by genetics, environmental influences, and behavioral factors (Williams, Mesidor, Winters, Dubbert, & Wyatt, 2015; Gurnani, Birken & Hamilton, 2015; Rosemond et al., 2016). According to Biro & Wien (2010), environmental opportunities for energy intake coupled with limited energy expenditure are considered the most important factors contributing to the obesity epidemic.

Behavior also influences obesity. Lawrence et al. (2015) stated that individuals who show a strong reward-related response to food and low levels of self-control are more inclined to overeat and be overweight. This statement has been supported by evidence which has linked poor self-control to overeating and obesity. Poor self-control has been measured through motor response inhibition which looks at behavioral measures of impulsivity. Food-related impulsive behaviors contribute to overeating because individuals exhibit less self-control, or behavioral inhibition, and consume food simply because it is within the environment. Perhaps it is impulsive nature, or lack of inhibition, that contributes to obesity. Inhibition is the ability to "suppress inappropriate and unwanted actions" (Simmonds, Pekar ,& Mostofsky, 2008, p. 2). Regarding this research, suppression of the unwanted behavior of overeating is vital in maintaining a healthy weight in a food-rich environment.

Further supporting the link between inhibitory capacity and obesity, research has shown some individuals struggle to inhibit pleasure-based food intake (Ely, Winter & Lowe, 2013). Ely et al. (2013) hypothesized that modern food environments, which include an abundance of sustenance, contribute to non-homeostatic eating. Such eating is considered hedonic, meaning "eating beyond the point of nutritional repletion" (p. 1). Homeostatic eating and hedonic eating involve different processes of inhibition. During homeostatic eating, inhibition is a primarily autonomic process resulting in the removal of internal hunger cues. In contrast, hedonic eating involves inhibition which is voluntary and cognitively controlled. This type of inhibition is the voluntary control preventing consumption of food beyond nutritional replenishment.

Ely et al. (2013) found that female undergraduates (*M* age=20 y; *SD*=2.25) who become obese demonstrate low inhibitory activation as measured by neural inhibition in a go/no-go task. Jansen et al. (2009) found that female undergraduates (*M* age=19.1 y; *SD*=1.3) are likely to

overeat when they have low inhibitory control. Other studies have found that impulsive responses on go/no-go and stop signal tasks are positively correlated with BMI in adults (Bonato & Boland, 1983; Nederkoorn, Braet, Van Eijs, Tanghe & Jansen, 2006; Nederkoorn, Jansen, Mulkens & Jansen, 2007; Nederkoorn, Smulders, Havermans, Roefs & Jansen, 2006) and caloric intake (Guerrieri et al., 2007). Batterink, Yokum & Stice (2010) found that the BMI of adolescent girls (*M* age=15.7 y; *SD*=0.93) correlated positively with lower neural inhibition as evidenced by failure to inhibit response to no-go stimuli in a go/no-go task (N=35; r_s=0.50; p=0.002). Batterink et al. (2010) concluded that impulsivity is positively related to overeating as measured by BMI. Further research needs to be done to connect neural inhibition to BMI and behavioral inhibition in young children.

EXECUTIVE FUNCTIONING & INHIBITION

Executive functions can be described as "complex processing requiring the coordination of several subprocesses to achieve a particular goal" (Elliott, 2003, p. 49). Executive functions include memory, planning, verbal reasoning, problem solving, inhibition, mental flexibility, and multi-tasking (O'Brien, 2014). Crowe, Catroppa, and Anderson (2015) stated executive functions are used to monitor, self-regulate, set goals, plan to achieve goals, and respond to novel situations. One result of break down in executive control systems is disinhibition. If individuals are unable to appropriately select actions that are advantageous or are unable to withhold actions that are inappropriate, then they have difficulty maintaining control of their behaviors. This is referred to as response inhibition (Simmonds et al., 2008).

According to Hofmann, Schemichel, and Baddeley (2012) inhibition is the ability to deliberately inhibit prepotent responses when necessary. A prepotent response is a dominant or automatic response to a stimulus (Miyake et al., 2000). Barkley (1997) stated inhibition warrants the performance of four executive abilities including: internalization of speech, working memory, reconstitution, and self-regulation of affect-motivation-arousal. Without inhibition, however, these four abilities may be hindered. Ineffective inhibition is of interest to this research because it has been linked to overeating. In fact, Nederkoorn et al. (2010) concluded that ineffective inhibition causes overeating and is correlated with weight gain. This study of 74 female undergraduate students found decreased response inhibition was related to weight gain.

Response inhibition must be measured in order to determine if it correlates with other variables. One form of measuring response inhibition is through functional magnetic resonance imaging (fMRI). This technology can localize where inhibition takes place within the brain. Studies using fMRI have shown response inhibition reveals frontal lobe activation (Simmonds et

al, 2008). As fMRI is taking place, a participant can complete a task requiring inhibition. Such tasks include Stroop or stop-signal task (Hoffman et al., 2012). During the Stroop test individuals are prompted to read a list of colors which are printed in different colors than those denoted by the word (i.e. the word "red" printed in the color blue) (Stroop, 1992). Stop-signal tests involve two tasks called a stop task and a go task. These tests require participants to discriminate between two tasks (i.e. the letters X and O). When the stop task is presented an auditory tone is simultaneously presented which indicates the participant should inhibit their response to the task (Logan, Schachar, & Tannock, 1997).

Another measure of response inhibition is through an event-related potential produced via EEG during a go/no-go task using visual cues. A go/no-go task is similar to stop-signal task. However, it strictly uses visual cues to prompt the participant to inhibit. In a go/no-go task with visual cues, participants are asked to inhibit pressing the button when a specific no-go stimulus is presented (i.e. picture of orangutan) and to press a button when go stimuli are presented (i.e. picture of any other animal). Typically, 25% of trials will be no-go and 75% will be go with go trials being novel images and no-go trials being the same image.

The electroencephalography (EEG) measurement that has been defined as indicative of inhibition is the N2. Electroencephalography is a non-invasive method of analyzing electrical activity of the brain via communication between electrodes placed on the scalp of an individual and a computer. EEG is a unique data collection process in that it allows for data to be collected while an individual engages in a task so it can capture temporal processing involved in inhibition. The size of the N2 amplitude correlates with the amount of inhibition. Therefore, a smaller N2 amplitude communicates less inhibition and vice versa (Jodo & Kayama, 1992; Eimer, 1993). The current study will measure response inhibition in two ways. First, behavioral

inhibition related to eating will be measured using the Eating in the Absence of Hunger paradigm. Secondly, neural responses of inhibition will be measured with electroencephalograph techniques during go/no-go tasks with visual cues paradigm. Both these ways will be further described in subsequent sections.

Response inhibition has been negatively related to impulsivity (Batterink et al., 2010). Research also shows that adults who are low in behavioral inhibition are more significantly influenced by impulsive tendencies (Houben & Wiers, 2009; Payne, 2005; Hofmann, Friese & Roefs, 2009). Previous research has related response inhibition to impulsivity and impulsivity to behavioral inhibition. Therefore, this research will attempt to determine a relationship between response inhibition and behavioral inhibition in children by looking at EEG data as well as eating behaviors.

ELECTROENCEPHALOGRAPHY

Electroencephalography (EEG) is a technique used to measure electrical activity produced by the brain. It is done by placing an array of electrodes on the surface of the scalp. According to Forney (2011), EEG has been used to diagnose conditions (i.e, epilepsy, sleep disorders, and brain damage) and it has been used in research to study neural activity and human development.

When particular events or stimuli are presented, specific responses measured by EEG can be extracted. These responses are called event-related potentials (ERPs). An ERP is a very small voltage generated in the brain that occurs during sensory, motor, or cognitive events. Studying an ERP allows researchers to study how the brain responds to various stimuli (Sur & Sinha, 2009). Event-related potentials are represented by a series of peaks named by their polar deflection and latency. For example, an N100 component is a negative deflection that presents approximately 100 milliseconds after a stimulus.

Response inhibition can be examined via ERPs during go/no-go tasks. This requires participants to quickly press a button in response to "go trials," but to inhibit responding to other stimuli, or "no-go trials." When evaluating ERPs, visual go/no-go tasks generate a negative deflection between 180 and 350 milliseconds following the stimuli when looking at sites Fz, Cz and Pz (Lui et al., 2015). This is referred to as N200 or N2 (Bailey et al., 2014). Since N2 amplitude correlates positively with neural inhibition, a smaller N2 amplitude would indicate less inhibition (Jodo & Kayama, 1992; Eimer, 1993).

Visual go/no-go tasks have been studied using pictures of food. Studies have been done with adults using food cues that are a mix of high-calorie and low-calorie foods. Results examining the effects of caloric content of food items on behavioral inhibition are mixed (e.g.,

Meule & Kubler, 2014; Teslovich et al. 2014). In a study by Meule & Kubler (2014), no-go cues included both high and low-calorie foods. This study determined that inhibitory control in response to high-calorie food cues depended on self-reported impulsivity and reward sensitivity as measured by the Barratt Impulsiveness Scale (Spinella, 2007) and Food Cravings Questionnaire (Cepeda-benito, Gleaves, Williams & Erath, 2000). However, this did not hold true for low-calorie food cues. Therefore, measures of inhibition were likely skewed based on content of cues. In contrast, Teslovich et al. (2014) found that calorie level of food cues did not alter behavioral performance. High and low calorie food cues gave similar responses. Although findings are mixed about the impact of calorie level of no-go picture cues, this current research will use an image of Cheerios® as the no-go cue because it is neither a high nor a low-calorie food.

Another component seen when evaluating ERPs for go/no-go tasks is a positive deflection that is generally between 250 and 400 following the stimuli (Sur & Sinha, 2009). This is called the P300, or P3. The P3 component is said to represent stimulus evaluation and classification (Barry & Rushby, 2006). This means it is associated with the process of deciding whether the response to the stimulus was appropriate. Greater amplitude of P3 has been associated with increased attention (Sur & Sinha, 2009).

The use of food cues in go/no-go tasks has shown a significant inverse relationship between body mass index (BMI) and response inhibition (r_s=-0.54; p=0.001) in adolescent girls (*M* age=15.7 y; *SD*=0.93) (Batterink et al., 2010). Another study with female adults (*M* age=20.20 y; *SD*=2.64), which used a go/no-go task with food cues, associated low inhibitory control with higher food intake (Kakoschke, Kemps & Tiggemann, 2015). Furthermore, the magnitude of N2 has been positively correlated with BMI in female adults (*M* age=21.63 y;

SD=2.33) (Watson & Garvey, 2013). These relationships involving neural measures of inhibition and inhibitory control related to eating have not been reported in the literature for children. Thus, more research needs to be conducted in order to study the relationship between neural measures of inhibition and children's eating behavior.

EATING IN THE ABSENCE OF HUNGER PARADIGM

Fisher and Birch (1999) developed the eating in the absence of hunger (EAH) behavioral paradigm in order assess the relationship between mothers' restriction of children's access to highly palatable foods and children's eating behaviors when given free access to such foods. The purpose of this study was to understand: children's inhibitory responses to highly palatable foods, eating behaviors that may lead to excess caloric consumption and excess growth, and if maternal restriction of food related to children's over-consumption in the absence of hunger. In this original EAH study, information was gathered on 70 children between the ages 3-6-years-old. Children participated in this study immediately following consumption of a meal in order to minimize hunger. Children were required to validate they were "full" before they could participate in the "free access procedure." Validation of fullness was done with the aid of visuals to conceptualize fullness. Children were shown three cartoon figures depicting the status of a stomach: empty stomach, half empty stomach, and full stomach. If a child did not indicate they had a full stomach, then they did not participate in the free access procedure.

During the free access procedure, the child was given free access to toys and generous quantities of ten snack foods: popcorn, potato chips, pretzels, nuts, fig bars, chocolate chip cookies, fruit-chew candy, chocolate bars, ice cream, and frozen yogurt. The child was placed in front of containers holding substantial amounts of these snack foods and toys. The child was told he or she could play with the toys or eat any of the foods. Then the child was left alone for ten minutes. During this time, the researcher observed the child through a one-way mirror. After ten minutes, the researcher returned to the child and asked whether his or her parents restrict consumption of each of the ten foods. Food consumption was measured by comparing pre- and post- weights of the ten snack foods. The amount consumed measured in kilocalorie (kcal) was

the dependent variable for this study and can be thought of as the eating in the absence of hunger score for the purposes of the present study.

Other information collected during the study included: children's anthropometric data (height, weight, and skinfold measures), parental BMI as calculated based on self-reported height and weight, and paternal restriction of children's access to the ten snack foods.

Ultimately this study determined that, in comparison to boys, girls were more prone to eating desirable snack foods in the absence of hunger. Furthermore, maternal restriction of children's access to food related to their daughter's perceptions and consumption. Positive correlations were found between maternal restriction of children's access to snack foods and 1) girls' perception of restriction, and 2) girls' consumption of the foods. However, these relationships did not hold true for boys.

Since this study in 1999, there have been over 60 studies using the EAH paradigm with children;19 were published between 1999 and 2013 as reported in a systemic review by Lansigan et al (2015) and 43 more studies were published between 2013 and 2017 as determined by searching "eating in the absence of hunger" AND children in PubMed. Among those studies, various traits have been studied in relation to the eating in the absence of hunger paradigm. Nederkoorn et al. (2015) looked at the interaction between hunger, impulsivity, and consumption of food types in 66 children between ages 7 and 9 years. Results showed children who were more impulsive, as measured by score on a stop signal task, ate more high energy-dense foods than children with less impulsive behaviors. No differences between impulsivity and low- and medium-energy dense food consumption was found. Kral et al. (2012) compared caloric compensation and EAH in 47 sibling pairs between ages 5 and 12 years. Siblings were served the same dinner once per week for three weeks. Before the dinner, they consumed or did not

consume preloads that varied in energy density. When no preload was consumed, EAH was tested following dinner. Results showed that overweight/obese siblings undercompensated and overate after a high-energy dense preload. However, normal-weight siblings did not over or under compensate. Overweight/obese siblings consumed over a third more calories in the absence of hunger than their normal-weight siblings. A recent study by Emond, Lansigan, Ramanujam, & Gilbert-Diamond (2016) studied the effect of exposing children (age 2-5 years) to food advertisements while watching television on eating in the absence of hunger. This study used the EAH protocol as defined by Fisher and Birch (1999). This study did not find statistically significant differences between the kcals consumed in the EAH phase and age, BMI, or parental feeding restriction. However, this study found that consumption of foods shown in the television advertisements increased upon exposure to advertisements. These three studies show that relationships between eating in the absence of hunger and the variables of impulsivity and weight exist within some populations.

Several other studies have been conducted using the EAH protocol as defined by Fisher and Birth (1999). Those studies include: Birch & Fisher (2000), Fisher & Birch (2000), Fisher & Birch (2002), Birch, Fisher & Davison (2003), Shunk & Birch (2004), Francis & Birch (2005), and Francis, Ventura, Marini & Birch (2007). Of note, all seven of these studies used data from the same cohort of 197 girls who were between ages 4.6 - 6.4 years at the origin of the study and their mothers. This cohort was followed over a 4-year period. Among those studies, parental restrictive feeding practices were positively correlated with children's EAH score, that is the amount consumed in the absence of hunger (Birch & Fisher, 2000; Fisher & Birch, 2000; Fisher & Birch, 2002; Birch, Fisher & Davison, 2003; Francis & Birch, 2005). Children who were overweight and who received greater restriction had highest EAH scores at 4-year follow up

(Birch et al., 2003). Shunk and Birch (2004) found adiposity was positively correlated with EAH. The rate of increase in EAH scores over time was greatest among overweight girls compared to normal weight girls. Francis and Birch (2005) found daughters of overweight mothers showed EAH scores that positively associated with increase in adiposity during two and four year follow-ups. Lastly, Francis et al. (2007) found participants with parents who were both overweight had highest EAH scores and highest increase in EAH over 8-year follow-up.

Many studies have been conducted to compare children's eating behavior with their weight, impulsivity, BMI, and parental BMI. This study will be unique because it will study the relationship of children's neural inhibition, as measured through visual go/no-go task with food cues, with their eating behaviors during the EAH protocol in younger children (age 4-6 years).

PURPOSE

The purpose of this research is to determine if there is an association between neural inhibition and eating behavior inhibition among children (age 4-6 years). Within this research, three types of inhibition will be used: neural, motor response, and eating behavior. Neural inhibition is operationally defined as the amplitude of N2 in the event-related potential (ERP) from the electroencephalography (EEG). Motor response inhibition is the ability to inhibit response when shown the no-go food cue (Cheerios®) during the go/no-go paradigm. A diminished number of kcal consumed in the EAH paradigm is an indicator of eating behavior inhibition. This pilot study is important because it paves a new path in the field of research by looking at factors affecting eating that have never been directly considered in children ages 4-6 years. This study is novel because no previous studies have considered neural inhibition during a go/no-go task with food cues in relation to eating behavior inhibition during the EAH paradigm.

RESEARCH QUESTIONS

Question 1: Does the ability of a child (age 4-6 years) to inhibit motor response during a go/no-go visual task with food cues correspond with their ability to inhibit eating within the EAH paradigm?

Hypothesis 1.1: It is hypothesized that children (ages 4-6 years) who more frequently fail to inhibit motor response as measured by number of failures to inhibit (press button) on go/no-go visual tasks will consume a higher quantity of food within the EAH paradigm than those with less frequency of failure to inhibit motor response.

Hypothesis 1.2: It is hypothesized that children (age 4-6 years) who demonstrate less neural inhibition as measured by a smaller N2 amplitude on the ERP during the go/no-go visual task with food cues will consume a higher quantity of food within the EAH paradigm than those who demonstrate more neural inhibition.

Question 2: Does the quantity of food children (age 4-6 years) consumed within the EAH paradigm correlate with their average BMIs and average N2 amplitude?

Hypothesis 2: It is hypothesized that children (age 4-6 years) who consume a higher quantity of food in the EAH paradigm will have higher BMIs and smaller N2 amplitudes on average than children (age 4-6 years) who consume lower quantities of food in the EAH paradigm.

Question 3: Will children (age 4-6 years) with parents who score higher on the Restriction Subscale of the Child Feeding Questionnaire consume a higher quantity of food in the EAH paradigm and have smaller N2 amplitudes than children with parents who score lower on the Restriction Subscale of the Child Feeding Questionnaire?

Hypothesis 3.1: It is hypothesized that children (age 4-6 years) with parents who score higher on the Restriction Subscale of the Child Feeding Questionnaire will consume a higher quantity of food in the absence of hunger paradigm and have smaller average N2 amplitudes than children with parents who score lower on the Restriction Subscale of the Child Feeding Questionnaire.

Hypothesis 3.2: It is hypothesized that children with parents who more frequently restrict foods from the child as indicated by selecting "child likes but does not eat" on the Picky Eating in Toddlers and Preschoolers Questionnaire will eat higher quantity of foods within the EAH paradigm and have smaller N2 amplitudes than children whose parents do not restrict as many foods from their children.

METHODS

Participants

A total of 23 children between ages 4-6-years-old were recruited for this study via flyer and word of mouth. Recruitment also occurred via advertisements in "Source" and emails to CSU faculty. Inclusion criteria were: age (4-6 years of age), speaking English, normal or corrected vision and hearing.

Children were excluded from this study if they had disabilities (i.e. autism, attention deficit hyperactive disorder, cerebral palsy, Down syndrome, history of significant brain injury), if they had a condition known to affect eating (i.e. autism, cystic fibrosis, genetic disorders, significant difficulties with chewing or swallowing), if they took medications for development, medical, or psychiatric disorders, if they took medication that may affect body weight and eating, or if they had food allergies that affected ability to participate in this study. All participants took part in the study voluntarily. Prior to beginning the study, written consent was obtained from parents of the children and all children signed assent forms.

PROCEDURE

Each child participated in one study session at the Gifford Building at Colorado State University. Children began by taking part in an electroencephalographic (EEG) recording which lasted approximately 90 minutes including application and removal of EEG sensors. Then they ate a meal which lasted about 30 minutes. During the final activity of the study, the children were shown sweet and savory snacks and given 10 minutes to play with toys in a room where these snacks were available to eat. The total time required for the study was approximately 2.5 to 3 hours.

When the participant arrived with his/her parent, the parent was given 4 questionnaires to be filled out: child screening questionnaire (demographic information, developmental information, food allergies, etc.), the Behavioral Rating Inventory of Executive Function (BRIEF), the Child Feeding Questionnaire (CFQ), and a survey developed based on the Picky Eating in Toddlers and Preschoolers Questionnaire (modified to include 38 items which are the foods shown in the EEG task or offered to the children during free-access procedure).

During the EEG task, the child was seated in a comfortable chair and was shown the equipment. The child was given a brief description of the process. When the child was comfortable with the EEG process, the cap and metal EEG sensors were placed on the participant's head. Next two tasks requiring executive functioning were completed while the EEG was recorded. The tasks were counterbalanced. Thus, either the first or second task was the visual go/no-go with food cues, the task reported in this thesis. Participants were given 1-3 practice runs before beginning the testing phase. Each practice run included 12 trials. Participants were given feedback during practices if they were not performing properly and a second practice was issued if needed. No feedback was given during the task itself.

Following EEG recording, the child was offered a meal. He or she was given plenty of time to eat until he/she reported he/she was full, approximately 15-30 minutes. Then the child was given a 10-minute break. At the end of this break the child was asked if he/she was full. Finally, the child participated in the free access procedure of the EAH paradigm in which the child was left alone for 10 minutes with a plethora of palatable snack foods and a container of toys. The child was observed through a one-way mirror during the free access procedure.

Electrophysiological Paradigm

The food picture go/no-go paradigm consisted of a total of 320 trials presented over a period of about 10 minutes. This task was completed as the child responded by pressing a button to target items displayed on a computer monitor on a table in front of him/her. Stimuli were presented on a computer monitor positioned directly in front of the participant. A response pad was placed on the table in front of the participant. The participant was instructed to refrain from pressing the button on the response pad when a picture of Cheerios® was presented (i.e., no-go trials). The participant was instructed to press the button when any of the other 30 different food items (Appendix A) was presented (i.e., go trials) (Blechert, Meule, Busch, & Ohla, 2014). Of the 320 stimuli presented, 80 were Cheerios® (i.e., no-go trial). No-go stimuli made up 25% of the trials. Stimuli appeared on the screen for 300 ms. Stimuli were presented in a pseudorandom order with a random, variable interstimulus interval of 1400-1800 ms. During the paradigm, the participant was given three 30-second breaks. If the participant got restless or was not focusing on the tasks, other brief breaks were given as needed. During breaks the participant was encouraged to work on a maze on paper using colored pencils.

Electrophysiological Recording

EEG recordings were obtained using the BioSemi ActiveTwo system with a Lycra head cap (BioSemi, Inc., Amsterdam, The Netherlands). Active EEG were recorded from 32 Ag-AgCl sintered electrodes with locations on the scalp based on the American Electroencephalographic Society's 10/20 nomenclature guidelines (1994). Common Mode Sense (CMS) active electrode and a Driven Right Leg (DRL) passive electrode served as reference and ground respectively (http://www.biosemi.com/faq/cms&drl.htm). An additional pin-type Ag/AgCl electrode was placed at FCz for a total of 33 scalp sites. Electrodes were placed on the left and right earlobes for off-line referencing. Two bipolar electrooculograms (EOG) were used to account for vertical and horizontal eye movements. The vertical EOG was derived from electrodes placed on the supra- and infraorbital regions of the left eye. The horizontal EOG was derived from two electrodes placed on the left and right outer canthi. Data were sampled at a rate of 1024Hz. Electrode offsets were maintained at ±20mV throughout each session.

Electrophysiological Data Reduction

Using BrainVision Analyzer 2.0 software (www.brainproducts.com), data from the continuous EEG recording was re-referenced to the averaged voltage of the two earlobe electrodes. They were then filtered with a .1 to 30Hz bandpass filter. EEG data were segmented about each visual stimulus from 200 ms pre-stimulus to 800 ms post-stimulus onset. Baseline correction was performed on each segment using the EEG data from -200 – 0 ms relative to the stimulus onset. Segments were then divided into correct go and no-go trials. Correct no-go trials were any trials in which the button was not pressed following no-go stimuli (i.e., Cheerios®). Correct go trials were any trials in which the button was pressed following a go stimulus (i.e., all other food items). Trials in which a button was pressed after presentation of the no-go stimuli

(i.e., Cheerios®) were considered incorrect trials and were not included in the averaging process. A regression procedure used to remove eye blinks was applied to retained segments (Segalowitz, 1996). Following the regression procedure, segments were baseline corrected again using the -200 to 0 ms window. Segments then underwent an artifact rejection procedure to remove segments with voltages exceeding ±100μV.

Averaged ERPs for go and no-go segments retained after data reduction were calculated for each participant. The data was processed using a Matlab routine which allowed for automatic scoring and visual inspection of ERP components, and, when necessary, allowed for manual rescoring of components. All ERP component measurements were carried out by a trained research assistant. Baseline-to-peak amplitudes and latencies for the P2, N2, and P3 were measured using the Matlab routine.

The N2 ERP component was analyzed at Cz and the P3 ERP component was analyzed at Pz. The P2 component was analyzed at both Cz and Pz. Since peak-to-peak amplitudes were used, the value of P2 at Cz was used to measure peak-to-peak amplitude of N2. Furthermore, the value of P2 at Pz was used to measure peak-to-peak amplitude of P3. These sites were chosen based on previous literature and analysis of topographical view of data patterns within Analyzer 2.0 software. Previous EEG studies on participants within a similar age range analyzed data at Pz and Cz. Such studies include Lui, Zhu, Ziegler & Shi, 2015 (*M* age=4.87 years), Todd. Lewis, Meusel & Zelazo, 2008 (age=4-6 years), and Buss, Dennis, Brooker & Sippel, 2011 (age=4-6 years). The most positive peak between150-300 ms after stimulus onset was defined as P2. The most negative peak 250-450 ms after stimulus onset was defined as N2. The most positive peak 400-700 ms after stimulus onset was defined as P3. The N2 and P3 amplitudes were calculated using peak-to-peak measures. Specifically, the peak-to-peak N2 amplitude was calculated by

subtracting the preceding positivity (P2 amplitude) from the N2 amplitude. The peak-to-peak P3 amplitude was calculated by subtracting the preceding negativity (N2 amplitude) from the P3 amplitude.

EAH Paradigm

Parents' report of restriction in child feeding

Parents' report of restricting their children's access to foods was assessed based on the Restriction construct of the Child Feeding Questionnaire. The Restriction construct of the Child Feeding Questionnaire has been determined to have acceptable internal consistency of 0.73 (Birch et al., 2001). The Restriction construct includes five questions that assesses parental perception of how much they restrict what their children eat and if they intentionally keep foods out of their children's reach.

Standardized meal protocol

The EAH paradigm was used to measure children's responsiveness to the presence of palatable foods in the absence of hunger (Fisher & Birch, 1999). First, children were fed a pre-weighed standardized meal including: chicken nuggets (54 g, 189 kcal), butter roll (40 G, 80 kcal), carrots (30 g, 12 kcal), raisins (37 g, 118.9 kcal), graham crackers (31 g, 139.5 kcal) and milk 267 g, 124.5 kcal). The standardized meal consisted of a total of 663.9 kcal. The children were supervised while eating this meal and were encouraged to eat until they were full. Each item of the meal was weighed after consumption to derive the energy content consumed. Then children were interviewed to indicate the extent to which they were full. This was done by showing the child a paper with three figures. Each figure was the outline of a person with a stomach that was: 1) empty, 2) partially full, or 3) full. Children were told what each figure represented and then asked to choose the one that represented their hunger level. According to

the standardized protocol, children who indicate their stomachs are empty would be excluded from the data analysis. However, for this current study none of the children indicated their stomachs were empty. Therefore, no children were excluded from data analysis do to this criterion.

Free-access protocol

Children were then presented with highly palatable snack foods and toys. The snack foods included: potato chips (58 g, 327 kcal), pretzels (39 g, 149 kcal), Skittles (66 g, 269 kcal), Hershey's chocolate bars (66 g, 363 kcal), Oreo cookies (70 g, 270 kcal), donuts (53 g, 233 kcal), and Honey Buns (50 g, 212 kcal). Snack foods were pre-weighed and a total of 1823 kcal was offered. The researcher told the child he/she could play with the toys or eat any of the foods while the researcher stepped out of the room to complete some work. The researcher then observed the child through a one-way mirror for while the child was left alone. After five minutes, the researcher stepped into the room to remind the child of the directions. After a total of ten minutes, the researcher returned to the room to inform the child that the task was finished. Palatable snack items were weighed after consumption. Energy consumption was calculated based on the weight of each food consumed. The weight was measured in grams then the total energy consumed was calculated according to previously mentioned values based upon manufacturer nutrition information. The dependent variable in this study was kcal consumed during the free access protocol.

Statistical Analysis

Research Question 1

In order to address hypothesis 1.1 which relates motor response inhibition to quantity of food consumed, Pearson product-moment correlation will be used to analyze the data. Pearson

product-moment correlation will also be used to address hypothesis 1.2 which relates inhibition to quantity of food consumed.

Research Question 2

Hypothesis 2.1 relates quantity of food consumed to BMI. This hypothesis will be analyzed through use of Pearson product-moment correlation.

Research Question 3

Hypothesis 3.1 relates Restriction Subscale score to quantity of food consumed. This will be addressed through analysis with Pearson product-moment correlation. Hypothesis 3.2 relates parental restriction to quantity of food consumed. This will be addressed through Pearson product-moment correlation.

Data were analyzed using IBM SPSS (version 24).

RESULTS

Participant Demographics

Twenty-three participants were recruited during 2016. Two participants were excluded because researchers were piloting the tasks and needed to make changes after the first two participants. One participant was excluded due to failure to complete the EEG task. Thus, data from 20 participants were used for statistical analyses. Participants were 45% female (n=9) and 55% male (n=11). Participants were between 4-6-years-old (*M* age=5.55 years; *SD* = 0.88). Average BMI of participants was 16.3 (*SD*=1.4). According to CDC Body-Mass-Index-for-Age Percentiles for girls and boys ages 2-20, respectively, 15 participants were healthy weight, 4 were overweight, and 1 was obese (See Table 1).

Table 1. Participant Demographics and BMI

Subject	*Age (Years)*	*BMI*	*BMI-for-Age Category*	*Gender*
103	5.28	17.0	Overweight	M
105	6.49	15.3	Healthy weight	F
106	5.06	15.6	Healthy weight	M
107	6.09	15.5	Healthy weight	M
108	6.04	16.9	Healthy weight	M
109	4.48	16.6	Healthy weight	F
110	4.58	17.2	Overweight	F
111	6.58	15.4	Healthy weight	F
112	5.98	16.3	Healthy weight	F
113	4.18	16.4	Healthy weight	M
114	5.84	15.8	Healthy weight	M
115	4.77	13.9	Healthy weight	M
116	6.72	15.3	Healthy weight	F
117	4.39	15.5	Healthy weight	F
118	5.38	14.8	Healthy weight	M
119	6.14	15.4	Healthy weight	M
120	6.85	18.6	Overweight	M
121	5.85	20.2	Obese	M
122	6.23	17.7	Overweight	F
123	3.99	16.8	Healthy weight	F
Mean	5.55	16.3		
SD	+/-0.88	+/-3.27		

Note. BMI calculated as weight (kg)/height(m)^2. BMI-for-age category determined according to CDC Body-Mass-Index-for-Age Percentiles for girls and boys ages 2-20, respectively (www.cdc.gov/growthcharts/data). Less than 5th percentile is underweight, 5th- 85th percentile is healthy weight, 85th-95th percentile is overweight, and greater than 95th percentile is obese.

Electrophysiological Data

N2 and P3 differences between go and no-go trials

EEG data analysis does not include participants with fewer than 29 correct no-go trials because participants with few correct no-go trials are assumed to perform inhibition by chance rather than intentionally (See Table 2). Three participants were excluded due to this issue. Results from ERPs were different than expected because mean N2 amplitude of correct no-go trials were not more negative than those of correct go trials as reported in the literature for go/no-go paradigms (Jodo & Kayama, 1992; Eimer, 1993). Although the mean N2 amplitude of correct go trials was more negative (-14.24 μV) than the mean N2 amplitude of correct no-go trials (-12.67 μV), a paired sample *t* test showed that no statistically significant difference was found between the N2 amplitude of correct go trials and those of correct no-go trials ($t(16) = -1.612$, $p=0.127$). After inspecting the grand average ERPs (see Figure 1), we decided it would be important to measure the P3 component. Interestingly, statistically significant difference was found between the P3 amplitude of correct go trials and correct no-go trials ($t(16) = 2.877$, $p = 0.011$). The mean P3 amplitude of correct no-go trials (17.93 μV) was significantly larger than P3 amplitude of correct go trials (14.26 μV; Figure 1).

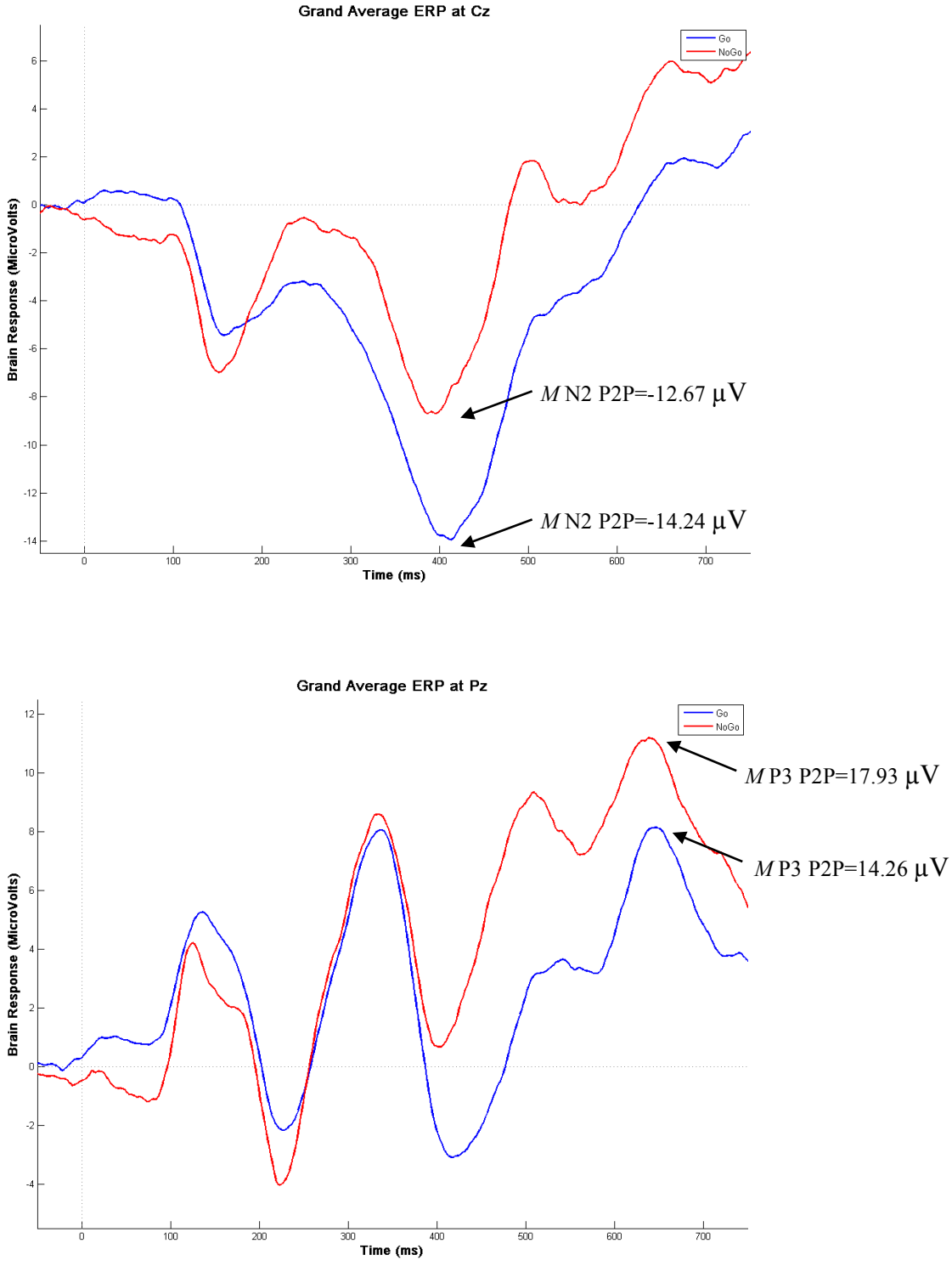

Figure 1. Grand average ERPs for N2 at Cz and P3 at Pz..

Note. N2 P2P=Peak-to-peak N2 amplitude. P3 P2P=Peak-to-peak P3 amplitude.

Table 2. Correct number of go and no-go trials per participant after artifact rejection.

Subject	*Number of Correct Go Trials*	*Number of Correct No-Go Trials*
103	93	42
105	168	33
106	205	17
107	185	37
108	183	37
109	70	34
110	159	42
111	166	29
112	19	54
113	189	10
114	213	64
115	151	36
116	191	71
117	136	65
118	75	33
119	188	61
120	178	39
121	37	17
122	195	49
123	100	32
Mean	145.05	40.25
SD	+/-58.52	+/-16.53

EAH Data

Weight of food consumed during meal protocol and free access protocol was measured as amount (g) served minus amount (g) remaining. Weight of food consumed was calculated into kcal consumed via specific calculations for each food according to nutrition labels (See Table 3). Children consumed an average of 300.0 kcal (*SD*=134.8) during the meal protocol and an average of 206.9 kcal (*SD*=127.9) during the free access protocol (See Table 4).

Table 3. Gram to kcal conversions

	Food Type	*Food Brand*	*kcal/g*
Standardized Meal	Graham cracker	Kroger	4.444
	Milk (lowfat)	Horizon organic	0.466
	Chicken nuggets	Kroger	2.368
	Dinner roll	King Soopers	2.667
	Carrots	Crispak	0.412
	Raisins	Kroger	3.214
Free Access	Powdered donuts	Little Debbie	4.151
	Honey Buns	Little Debbi	4.600
	Potato chips	Lays	5.714
	Pretzels	Snyder's	3.667
	Skittles	Skittles	4.000
	Oreos	Oreos	4.706
	Chocolate bars	Hershey's	4.884

Table 4. Kcal consumed during standardized meal and free access protocol.

Subject	*Free Access Kcal Consumed*	*Standardized Meal Kcal Consumed*
103	179	256
105	91	24
106	259	357
107	357	326
108	257	394
109	278	283
110	68	253
111	37	203
112	184	236
113	97	419
114	275	351
115	13	5
116	237	293
117	166	296
118	113	360
119	431	305
120	294	631
121	499	410
122	152	366
123	153	226
Mean	206.9	300.0
SD	+/-127.9	+/-134.8

Picky Eating and Child Feeding Questionnaire Data

The Picky Eating questionnaire was scored according to the number of time the parent selected "child eats but does not like." The CFQ was scored according to the questions from the Restriction construct which were answered on a Likert scale (never=1, rarely=2, some of the time=3, most of the time=4, always=5). See Table 5 for results on both questionnaires.

Table 5. Parental restriction questionnaire scores.

Subject	*CFQ Score*	*Picky Eating Score*
103	19	2
105	18	0
106	20	17
107	19	1
108	21	0
109	23	1
110	18	1
111	18	0
112	21	5
113	23	0
114	20	3
115	19	3
116	16	0
117	17	0
118	21	0
119	25	4
120	13	0
121	11	0
122	20	10
123	21	1
Mean	19.15	2.4
SD	+/-3.3	+/-4.2

Relationship Between Motor Response Inhibition and Eating Behavior Inhibition

Hypothesis 1.1 was not supported because motor response inhibition was not significantly correlated with eating behavior inhibition. The percent of incorrect no-go trials was not significantly correlated with free access kcal consumed (r=-0.132, p=0.615), suggesting there was no relationship between the behavioral performance on the go/no-go task and free access kcal consumed.

Hypothesis 1.2 was not supported because neural inhibition was not significantly correlated with eating behavior inhibition. The amplitude of N2 during no-go trials was not significantly correlated with free access kcal consumed (r=0.026, p=0.920), indicating there was no relationship between the N2 amplitude of the no-go condition and free access kcal consumed.

Furthermore, post hoc analysis also failed to show significant correlation between P3 amplitude on no-go condition and free access kcal consumed (r=-0.280, p=0.277).

Relationship Between Eating Behavior Inhibition, BMIs, and Neural Inhibition

Hypothesis 2.1 was partially supported because eating behavior inhibition was correlated with BMI, but not with neural inhibition. Free access kcal consumed significantly correlated with BMI (r=0.467, p=0.038). However, regression analysis used to examine the effect of BMI and N2 amplitude on free access kcal consumed showed neither of the predictors significantly accounted for variance in free access kcal consumed (overall model: $F(2,14) = 0.353$, p=0.709, adjusted R^2 = -0.088; BMI: $\beta = 0.225$, $t(14) = 0.834$, $p = 0.418$; N2 amplitude: $\beta = 0.032$, $t(14) = -0.117$, $p = 0.908$). Furthermore, regression analysis used to examine the effect of BMI and P3 amplitude on free access kcal consumed showed neither of the predictors significantly accounted for variance in free access kcal consumed (overall model: $F(2,14) = 0.868$, p=0.441, adjusted R^2 = -0.017; BMI: $\beta = 0.181$, $t(14) = 0.712$, $p = 0.488$; P3 amplitude: $\beta = -0.254$, $t(14) = -0.998$, $p = 0.335$).

Relationship Between Restriction, Eating Behavior Inhibition, and Neural Inhibition

Hypothesis 3.1 was not supported because restriction was not significantly correlated with eating behavior inhibition according to the Child Feeding Questionnaire. There was no significant correlation between restriction subscale score and free access kcal consumed (r=-0.180, p=0.447). Furthermore, regression analysis used to examine the effect of restriction subscale score and N2 amplitude of no-go trials on free access kcal consumed showed neither of the predictors significantly accounted for variance in free access kcal consumed (overall model: $F(2,14) = 0.588$, p=0.569, adjusted R^2 = -0.054; restriction subscale score: $\beta = 0.277$, $t(14) = 1.079$, $p = 0.299$; N2 amplitude: $\beta = 0.035$, $t(14) = 0.135$, $p = 0.895$). Similarly, regression model

used to examine effect of restriction subscale and P3 amplitude of no-go trials on free access kcal consumed was not significant (overall model: $F(2,14) = 1.165$, p=0.340, adjusted $R^2 = 0.020$; restriction subscale score: $\beta = 0.255$, $t(14) = 1.027$, $p = 0.322$; P3 amplitude: $\beta = -0.259$, $t(14) = -1.042$, $p = 0.315$).

Hypothesis 3.2 was not supported because eating behavior inhibition was not significantly correlated with restriction according to the Picky Eating Questionnaire. No significant correlation was found between free access kcal consumed and count of selecting "child likes but does not eat" on Picky Eating questionnaire (r=0.069, p=0.772) Furthermore, a regression model used to examine the effect of "child likes but does not eat" count and N2 amplitude for no-go trials on free access kcal consumed showed neither of the predictors significantly accounted for variance in free access kcal consumed (overall model: $F(2,7) = 0.021$, p=0.980, adjusted $R^2 = -0.139$; "child likes but does not eat" count: $\beta = 0.050$, $t(7) = 0.178$, $p = 0.861$; N2 amplitude: $\beta = 0.012$, $t(7) = 0.044$, $p = 0.966$). Similarly, a model used to examine effect of "child likes but does not eat" count and P3 amplitude for no-go trials on free access kcal consumed showed neither of the predictors significantly accounted for variance in free access kcal consumed (overall model: $F(2,7) = 0.594$, p=0.565, adjusted $R^2 = 0.053$; "child likes but does not eat" count: $\beta = -0.009$, $t(7) = -0.035$, $p = 0.972$; P3 amplitude: $\beta = -0.282$, $t(7) = -1.070$, $p = 0.303$).

Pearson correlations demonstrated meal kcal consumed were significantly correlated with BMI (r=0.588, p=0.006), indicating that the more kcal children ate during the meal, the greater their BMI. Similarly, the meal kcal consumed were also significantly correlated with free access kcal consumed (r=0.526, p=0.017), suggesting that the more kcal children ate during the meal, the more kcal they consume during the free access protocol. A regression analysis was used to

examine the effect of BMI on free access kcal consumed after controlling for the meal kcal consumed. The results revealed that after controlling for the effect of meal kcal consumed on free access kcal consumed (β = 0.385, t(17) = 1.550, p=0.139), BMI did not significantly predict free access kcal consumed (β = 0.240, t(17) = 0.968, p=0.347; the overall model: F(1,17) = 3.902, p=0.04, adjusted R^2=0.234.)

To investigate whether there is a relationship between ERPs and BMI after controlling for meal kcal consumed and free access kcal consumed, a series of hierarchical regression analyses were used. Controlled variables, meal kcal consumed and free access kcal consumed, were used to predict BMI in model one. The N2 amplitude of no-go condition was added in model two. Finally, the P3 amplitude of no-go condition was added in model three. While both N2 and P3 amplitudes were not significant predictors of BMI, the results showed meal kcal consumed was significant in predicting BMI in all the three models (Table 6). This suggests that the more kcal children consumed in the meal, the greater their BMI.

Table 6. Summary of Regression Analyses and Coefficients Evaluating the Contributions of ERPs in BMI. (dependent variable is BMI for all models)

	Model Statistics				***Change Statistics***		***Standardized Coefficient for each model***		
	R^2	Adj R^2	*F*	*p*	*F*	*P*	IVs	β	*p*
Model 1	.453	.375	5.792	.015	5.792	0.015	meal Kcal consumed	.780	.006
							free access Kcal consumed	-.233	.353
Model 2	.453	.327	3.593	.043	0.012	0.915	meal Kcal consumed	.793	.014
							free access Kcal consumed	-.239	.371
							N2 amplitude	-.025	.915
Model 3	.467	.289	2.629	.087	0.309	0.588	meal Kcal consumed	.810	.015
							free access Kcal consumed	-.211	.450
							N2 amplitude	.039	.884
							P3 amplitude	.142	.588

Note. DVs = dependent variables; IVs = independent variables; Adj R^2 = Adjusted R^2; Sig = significant; *F* = *F* value; *p* = *p* value; β = standardized coefficient

DISCUSSION

Results of the present study failed to find significant correlation between N2 amplitude and free access kcal consumed, P3 amplitude and free access kcal consumed, picky eating questionnaire and free access kcal consumed, and restriction subscale from the Child Feeding Questionnaire and free access kcal consumed. However, results of the present study found significant positive relationship between free access kcal consumed and BMI. When controlling for kcal consumed during the standardized meal, the significant relationship no longer held true between BMI and free access kcal consumed. Notably, the kcal consumed during the standardized meal significantly predicted BMI when controlling for free access kcal consumed.

As mentioned previously, relationships involving neural inhibition and behavioral inhibition have rarely been reported in the literature for young children. One study looked at a different measure of neural inhibition in children (age: $M = 9$ years; $SD = 0.6$) and kcal consumed in a bogus taste test (Guerrieri, Nederkoorn, & Jansen, 2008). Guerrieri and colleagues used a stop signal task to measure neural inhibition in which children were presented with a series of trials: 75% "go trials" and 25% "stop trials." During the go trials, the children pressed a certain button as fast as possible depending on the stimulus (i.e. right for X and left for O). At the same time, a tone served as a stop signal and told the child not to press the button in response to the stimulus. Following the task the children were presented with food that was either varied (different textures, tastes and colors) or monotonous (same textures, tastes, and colors). Then they were asked to rate the food based on how much they liked it. The study by Guerrieri et al. (2008) was different than the present study because it used an auditory stop signal to cue inhibition, whereas the present study used a visual no-go cue. The study measured motor response inhibition via the stop signal reaction time, or the amount of time taken for stop delay

minus reaction time. Increased stop signal reaction time was assumed to be associated with decreased motor response inhibition. Furthermore, the population for Guerrieri et al. (2008) was older than the present study (*M* age=9 years). Despite these differences, the results of this study support those of the present study. Ultimately, Guerrieri et al. (2008) and this present study discovered no correlation between neural inhibition and food consumption. Guerrieri et al. (2008) and the present study provide evidence that perhaps neural processes of inhibition are not necessarily correlated with behavioral inhibition related to eating behaviors in children. More research should be done to validate the findings of these studies among children.

Currently the go/no-go paradigm with food visual cues used in this present study is being administered to adults (ages 18-25 years) in the Brainwaves Research Lab at Colorado State University. Thus far 8 adults have participated in two sessions of the study. Over the 16 sessions, *t*-tests show N2 amplitude is significantly greater for no-go condition than for go condition (p=0.002). These results directly contrast the results from the present study. In fact, the present study found that N2 amplitude, on average, was smaller for no-go than for go among children ages 4-6-years-old. Findings from the study with adults validate the robustness of the paradigm because results are consistent with results in the literature among go/no-go paradigms for adults (Jodo & Kayama, 1992; Eimer, 1993). The study on adults used the exact same paradigm as the present study which leads to the conclusion that perhaps neural inhibition is not seen in this paradigm among children as young as 4-6 years old, but this neural response may develop with age.

Studies using go/no-go visual cues that look at the difference in amplitude among N2 for go vs. no-go trials have found mixed results. Grabell (2014) administer a go/no-go paradigm with zoo cues to 37 typically developing preschool-age children (age: M = 4.83 years, SD =

0.65). In paired-sample *t*-tests, Grabell (2014) found amplitude of N2 was marginally smaller for no-go trials than for go trials at Cz (p =0.08). These findings are substantial in supporting the findings of the present study because they both include typically developing preschool children and they are both looking at N2 at Cz. On the other hand, Vuellier et al. (2016) administered a go/no-go paradigm with zoo cues to 18 slightly older children (age: M = 8.42 years; SD = 0.42) and found that N2 was significantly greater in the no-go trials than the go trials (p=0.005). The studies reported here point to the existence of a shift in N2 amplitude for go vs. no-go trials as children develop, with younger children having larger N2 amplitude during go trials and older children having larger N2 amplitude during no-go trials. Results of the present study support the idea that perhaps younger children have larger N2 amplitudes during go trials. Further research needs to be done to investigate this potential shift during development.

Interestingly, this present study found that average P3 amplitude was larger for no-go trials than for go trials. In the literature, P3 has been associated with evaluation of outcome (Piispala et al., 2017; Barry & Rushby, 2006). Considering the association between P3 and evaluation, results of the present study suggest more effort is put into evaluation of outcomes for no-go trials than for go trials. This indicates that children use more effort to evaluate their behavioral response to trials that require motor response inhibition than to those that do not. This finding confirms that neural processing in young children is different for trials requiring inhibition than those that do not. This difference in processing does not appear until a later component, the P3, rather than appearing in the N2. The adult study in process in this lab demonstrates that adults show a larger N2 in trials with inhibited responses. The P3 is also larger in these no-go trials compared to the go trials.

There is some literature relating parental restriction and free access kcal consumed among young children. A recent study by Emond, Lansigan, Ramanujam, & Gilbert-Diamond (2016) studied the effect of exposing children (age 2-5 years; *M* = 4.1 years; *SD* = 0.9) to televised food advertisements on eating in the absence of hunger. This study used the EAH protocol as defined by Fisher and Birch (1999). Emond et al. (2016) did not find statistically significant differences between the kcal consumed in the EAH phase and parental feeding restriction among the control or experiment group. Average kcal consumed among children exposed to advertisements was 126.8. Average kcal consumed among children not exposed to advertisements was 97.3. Parental feeding restriction was measured according to the same scale used in this present study: The Child Feeding Questionnaire. The study by Emond et al. (2016) varies from the present studied because Emond et al. slightly modified the Child Feeding Questionnaire by combining two items that assessed food as a reward into one item. Despite this alteration, the findings of Emond et al. (2016) align with the present study in that neither found significant differences between kcal consumed in free access phase and parental feeding restriction.

On the other hand, another study found some opposing conclusions. A study by Fisher and Birch (1999) accessed maternal restriction of children's access to foods in children ages 3-5-years-old (*M* age=5.0 years; *SD*=0.1). Participants included boys (n=40) and girls (n=30). Fisher and Birch (1999) measured restriction via 9 questions about each of the 10 foods used in the experiment. Such questions included some similar to those used in the present study (i.e. "do you keep the food out of reach?" and "do you monitor the child's consumption of the food?") This study found that maternal restriction of children's access to foods was positively correlated with consumption of foods in girls but not boys. With girls, greater levels of restriction were

associated with increased kcal consumed in an unrestricted setting. However, restriction was not associated with kcal consumed in boys. Notably, there were no gender differences in energy intake. Overall, an average of 215 kcal was consumed in the unrestricted setting among boys and girls. Fisher and Birch (1999) suggest the different relationship between restriction and consumption seen between boys and girls may be influenced by a difference in responsiveness to restriction between boys and girl. Authors also suggest perhaps the differences between genders is influenced by parents granting less autonomy to girls than boys. Therefore, perhaps girls' consumption is more affected by an unrestricted environment which is more novel to them than to boys. The results of Emond et al. (2016), Fisher and Birch (1999), and this present study show inconsistencies about the relationship between parental restriction and amount of kcal consumed in unrestricted environment for young children. It is possible some inconsistencies between study results are attributed to the use of parental restriction measures that vary somewhat. Another possible reason for differing results between studies is different analysis of the results. Fisher and Birch (1999) found a positive correlation between restriction and kcal consumption for girls, but not for boys. Emond et al. (2016) and the present study did not specifically analyze the effect of gender. Further research should be done among this population to determine whether a relationship exists between restriction and consumption.

Studies in the past have shown a positive correlation between BMI and kcal consumed in the absence of hunger. Francis and Birch (2005) looked at data from a longitudinal study of 197 5-year-old girls and found EAH consumption was associated with BMI change from age 5 to age 9. However, a study on younger children by Emond et al. (2016) failed to find significant relationship between free access kcal consumption and BMI in children 2-5-years-old. Again, research has shown different patterns between younger children and older children. Perhaps this

is due to variance among average BMI increasing with age. This is demonstrated by Francis and Birch (2005) who found as age increased, variance of BMI also increased from 5-years-old (*M* BMI=15.8; *SD*=1.6) to 7-years-old (*M* BMI=16.3; *SD*=2.1) to 9-years-old (*M* BMI=17.8; *SD*=2.9). Children who are older may have more variance in average BMI which allows for the possibly of higher correlation between BMI and other factors, including kcal consumed. Therefore, it is possible that among older children a more significant correlation may be found between BMI and kcal consumed. Further research should be done to investigate if this pattern exists and if so, why.

IMPLICATIONS FOR OCCUPATIONAL THERAPY PRACTICE

Occupational therapy practitioners can play an important role in affecting eating behaviors in children. Results from this pilot study provide groundwork for the following ways in which occupational therapists can address eating behaviors in children:

- Establish healthy routines and habits at a young age, especially if young children have a lack of neural processing relating to inhibition.
- Provide family education regarding proper healthy habits and routines related to eating to further support the development of children at a time when neural processes are not yet fully developed.
- Modify the environment to make it optimal for making healthy decisions, especially in the absence of fully developed neural processing related to inhibition.

LESSONS LEARNED IN THIS FEASIBILITY STUDY

In line with the nature of feasibility studies, this present study brought about many obstacles that are reported here to benefit the process of future research. These challenges were found in the EEG/ERG portion of this study because this was the first study, to our knowledge, that used this visual go/no-go paradigm with food cues. Thus, there was no literature reporting parameter that should be considered for this ERP paradigm.

One challenge that arose was the development of two go/no-go patterns that varied in number of go vs. no-go trials. This issue arose due to a problem in the way the paradigm was programmed. Randomization did not reset following practice runs. Therefore, once the actual go/no-go task was started, some children were at one point in the sequence of randomized cues, while others were at a different point. The point in the sequence at which children began the go/no-go task was dependent on how many practices the child completed before the task. This issue was fixed following this pilot study so future research will not have the same problem. Ultimately. the development of two sets of go/no-go patterns created the problem of fewer no-go trials for some participants. Fewer no-go trials creates the opportunity for increased mean amplitude. This is due to the decrease of latency variance associated with decreased sample size of individual trials in the averaged ERP per individual participants. This concept is called latency jitter (Ouyang, Sommer, & Zhou, 2016). If there is a smaller sample of trials, then the mean amplitude will not be attenuated as much as it would with a larger sample of trials. This leads to the possibility of a smaller trial sample having a larger mean amplitude. Although this present study determined no-go trials had a smaller amplitude than go trials, this issue is documented to inform future studies of possible issues associated with fewer trials.

Some challenges arose during data analysis. One challenge was accounting for the variance among number of correct go and no-go trials between participants. One method attempted to control for this variance was a calculation to normalize the data based on the number of trials included. This proved ineffective. Inclusion criteria for amount of no-go trials was established to ensure exclusion of participants with motor responses that were random rather than representative of motor inhibition. For participants with a small number of trials, each trial had an exponential-like effect on results. The effect of sample size, or in this case number of trials, appears to asymptote around 30 trials. Therefore, those with less than about 30 correct no-go trials were excluded from the present study. No previous literature has reported such criteria. Thus, it is not possible to determine if other EEG/ERP studies using this type of paradigm may have had similar issues. Another difficulty was establishing N2 and P3 windows during peak picking as it was not widely stated in the literature for this population. We used visual inspection of the grand average ERP (i.e., average of all participant ERPs) to determine the window. This technique is common among studies exploring novel EEG/ERP paradigms such as this study.

LIMITATIONS

Small sample size is one limitation of this research. Furthermore, although data was also recorded for a go/no-go paradigm using zoo cues which have been previously studied in the literature, only the unique go/no-go food cues were analyzed in this research. Future studies should be done to compare the ERP results of the go/no-go food cues with the go/no-go zoo cues. One method that was not attempted to account for the differences in amount of correct trials between participants was down-sampling. This could be explored in future research to determine if present results hold true given alternate analytical methods.

CONCLUSION

This study demonstrates that children ages 4-6-years-old within this population may have different neural processing related to inhibition than older children as well as adults. This study supported the findings of previous literature that amount consumed in the absence of hunger among children within this population correlates positively with BMI. However, this study determined that when controlling for kcal consumed during the standardized meal, no significant correlation was found between free access kcal and BMI. Future studies should be completed to further research the correlation of neural inhibition and behavioral inhibition related to eating behavior in young children.

REFERENCES

Bailey, N. W., Hoy, K. E., Maller, J. J., Segrave, R. A., Thomson, R., Williams, N., . . . Fitzgerald, P. B. (2014). An exploratory analysis of Go/Nogo event-related potentials in major depression and depression following traumatic brain injury. *Psychiatry Research, 224*(3), 324-334. doi:10.1016/j.pscychresns.2014.09.008

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.

Barry, R.J. & Rushby, J.A. (2006). An orienting reflex perspective on anteriorisation of the P3 of the event-related potential. *Experimental Brain Research, 173*, 539–545.

Batterink, L., Yokum, S., & Stice, E. (2010). Body mass correlates inversely with inhibitory control in response to food among adolescent girls: an fMRI study. *Neuroimage, 52*(4), 1696-1703. doi:10.1016/j.neuroimage.2010.05.059

Batterink, L., Yokum, S., & Stice, E. (2010). Body mass correlates inversely with inhibitory control in response to food among adolescent girls: an fMRI study. *Neuroimage, 52*(4), 1696-1703. doi:10.1016/j.neuroimage.2010.05.059

Birch, L. L., & Fisher, J. O. (2000). Mothers' child-feeding practices influence daughters' eating and weight. *American Journal of Clinical Nutrition, 71*(5), 1054-1061.

Birch, L. L., Fisher, J. O., & Davison, K. K. (2003). Learning to overeat: maternal use of restrictive feeding practices promotes girls' eating in the absence of hunger. *American Journal of Clinical Nutrition, 78*(2), 215-220.

Birch, L. L., Fisher, J. O., Grimm-Thomas, K., Markey, C. N., Sawyer, R., & Johnson, S. L. (2001). Confirmatory factor analysis of the Child Feeding Questionnaire: A measure of parental attitudes, beliefs and practices about child feeding and obesity proneness. *Appetite, 36*(3), 201–210.

Biro, F. M., & Wien, M. (2010). Childhood obesity and adult morbidities. *The American Journal of Clinical Nutrition, 91*(5), 1499S-1505S. doi:10.3945/ajcn.2010.28701B

Blechert J, Meule A, Busch NA and Ohla K (2014). Food-pics: an image database for experimental research on eating and appetite. Front. Psychol. 5:617. doi: 10.3389/fpsyg.2014.00617

Bonato, D. P., & Boland, F. J. (1983). Delay of gratification in obese children. *Addictive Behaviors, 8*(1), 71-74. doi:http://dx.doi.org/10.1016/0306-4603(83)90059-X

Buss, K.A., Dennis, T.A., Brooker, R.J., Sippel, L.M. (2011). An ERP study of conflict monitoring in 4-8-year old children: Associations with temperament. *Developmental Cognitive Neuroscience, 1*(2), 131-140.

Cepeda-Benito, A., Gleaves, D. H.,Williams, T. L., & Erath, S. A. (2000). The development and validation of the state and trait Food-Cravings Questionnaires. *Behavior Therapy, 31*, 151–173.

Crowe, L. M., Catroppa, C., & Anderson, V. (2015). Chapter 41 - Sequelae in children: developmental consequences. In G. Jordan & M. S. Andres (Eds.), *Handbook of Clinical Neurology* (Vol. Volume 128, pp. 661-677): Elsevier.

Eimer, M. (1993). Effects of attention and stimulus probability on ERPs in a Go/Nogo task. *Biological Psychology, 35*(2), 123-138. doi:10.1016/0301-0511(93)90009-W

Elliott, R. (2003). Executive functions and their disorders. *British Medical Bulletin, 65*, 49-59.

Ely, A. V., Winter, S., & Lowe, M. R. (2013). The generation and inhibition of hedonically-driven food intake: behavioral and neurophysiological determinants in healthy weight

individuals. *Physiology & Behavior, 121*, 25-34. doi:10.1016/j.physbeh.2013.03.026
Emond, J.A., Lansigan, R.K., Ramanujam, A., & Gilbert-Diamond, D. (2016). Randomized exposure to food advertisements and eating in the absence of hunger among preschoolers. *Pediatrics, 138*(6), 1-8.
Fisher, J. O., & Birch, L. L. (1999). Restricting access to foods and children's eating. *Appetite, 32*(3), 405-419.
Fisher, J. O., & Birch, L. L. (2000). Parents' restrictive feeding practices are associated with young girls' negative self-evaluation of eating. *Journal of American Dietetic Association, 100*(11), 1341-1346. doi:10.1016/s0002-8223(00)00378-3
Fisher, J. O., & Birch, L. L. (2002). Eating in the absence of hunger and overweight in girls from 5 to 7 y of age. *The American Journal of Clinical Nutrition, 76*(1), 226-231.
Forney, E. M. (2011). Electroencephalogram classification by forecasting with recurrent neural networks. *Masters Thesis, Department of Computer Science, Colorado State University, Fort Collins, CO.*, 1-73.
Francis, L. A., & Birch, L. L. (2005). Maternal weight status modulates the effects of restriction on daughters' eating and weight. *International Journal of Obesity, 29*(8), 942-949. doi:10.1038/sj.ijo.0802935
Francis, L. A., Ventura, A. K., Marini, M., & Birch, L. L. (2007). Parent overweight predicts daughters' increase in BMI and disinhibited overeating from 5 to 13 years. *Obesity (Silver Spring), 15*(6), 1544-1553. doi:10.1038/oby.2007.183
Freedman, D.S., Dietz, W.H., Srinivasan, S.R., Berenson, G.S., (2007). Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Stud*y. The Journal of Pediatrics, 150*(1), 12-17.
Guerrieri, R., Nederkoorn, C., & Jansen, A. (2008). The interaction between impulsivity and a varied food environment: Its influence on food intake and overweight. *International Journal of Obesity, 32*, 708-714.
Guerrieri, R., Nederkoorn, C., Stankiewicz, K., Alberts, H., Geschwind, N., Martijn, C., & Jansen, A. (2007). The influence of trait and induced state impulsivity on food intake in normal-weight healthy women. *Appetite, 49*(1), 66-73.
Gurnani, M., Birken, C., & Hamilton, J. (2015). Childhood obesity: causes, consequences, and management. *Pediatric Clinics of North America*, *62*(4), 821-840.
Hofmann, W., Friese, M., & Roefs, A. (2009). Three ways to resist temptation: The independent contributions of executive attention, inhibitory control, and affect regulation to the impulse control of eating behavior. *Journal of Experimental Social Psychology, 45*(2), 431-435. doi:10.1016/j.jesp.2008.09.013
Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends In Cognitive Sciences, 16*(3), 174-180. doi:10.1016/j.tics.2012.01.006
Houben, K., & Wiers, R. W. (2009). Response inhibition moderates the relationship between implicit associations and drinking behavior. *Alcohol Clinical Experimental Resesarch, 33*(4), 626-633. doi:10.1111/j.1530-0277.2008.00877.x
Institute of Medicine, *Accelerating progress in obesity prevention: solving the weight of the nation*. 2012, Washington, DC: National Academies Press.
Jansen, A., Nederkoorn, C., van Baak, L., Keirse, C., Guerrieri, R., & Havermans, R. (2009). High-restrained eaters only overeat when they are also impulsive. *Behaviour Research and Therapy, 47*(2), 105-110. doi:10.1016/j.brat.2008.10.016
Jodo, E., & Kayama, Y. (1992). Relation of a negative ERP component to response inhibition in

a Go/No-go task. *Electroencephalography and Clinical Neurophysiology, 82*(6), 477-482.
Kakoschke, N., Kemps, E., & Tiggemann, M. (2015). Combined effects of cognitive bias for food cues and poor inhibitory control on unhealthy food intake. *Appetite, 87*, 358-364. doi:10.1016/j.appet.2015.01.004
Kral, T. V., Allison, D. B., Birch, L. L., Stallings, V. A., Moore, R. H., & Faith, M. S. (2012). Caloric compensation and eating in the absence of hunger in 5- to 12-y-old weight-discordant siblings. *American Journal of Nutrition, 96*(3), 574-583. doi:10.3945/ajcn.112.037952
Lamm, C., Walker, O. L., Degnan, K. A., Henderson, H. A., Pine, D. S., McDermott, J. M., & Fox, N. A. (2014). Cognitive control moderates early childhood temperament in predicting social behavior in 7-year-old children: An ERP study. *Developmental Science, 17*, 667-681. doi:10.1111/desc.12158
Lawrence, N. S., O'Sullivan, J., Parslow, D., Javaid, M., Adams, R. C., Chambers, C. D., . . . Verbruggen, F. (2015). Training response inhibition to food is associated with weight loss and reduced energy intake. *Appetite, 95*, 17-28. doi:10.1016/j.appet.2015.06.009
Liu, Q., Zhu, X., Ziegler, A., & Shi, J. (2015). The effects of inhibitory control training for preschoolers on reasoning ability and neural activity. *Scientific Reports, 5*(14200), 1-10.
Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science, 8*(1), 60-64. doi:10.1111/j.1467-9280.1997.tb00545.x
May, A.L., Kuklina, E.V., Yoon, P.W. (2012). Prevalence of Cardiovascular Disease Risk Factors Among US Adolescents, 1999−2008. *Pediatrics, 129*(6), 1035-1041.
Meule, A., & Kübler, A. (2014). Double trouble. Trait food craving and impulsivity interactively predict food-cue affected behavioral inhibition. *Appetite, 79*, 174-182. doi:10.1016/j.appet.2014.04.014
Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. *Cognitive Psychology, 41*(1), 49-100. doi:10.1006/cogp.1999.0734
Nederkoorn, C., Braet, C., Van Eijs, Y., Tanghe, A., & Jansen, A. (2006). Why obese children cannot resist food: the role of impulsivity. *Eating Behaviors, 7*(4), 315-322.
Nederkoorn, C., Dassen, F. C. M., Franken, L., Resch, C., & Houben, K. (2015). Impulsivity and overeating in children in the absence and presence of hunger. *Appetite, 93*, 57-61. doi:10.1016/j.appet.2015.03.032
Nederkoorn, C., Houben, K., Hofmann, W., Roefs, A., & Jansen, A. (2010). Control yourself or just eat what you like? Weight gain over a year is predicted by an interactive effect of response inhibition and implicit preference for snack foods. *Health Psychology: Official Journal of The Division Of Health Psychology, American Psychological Association, 29*(4), 389-393. doi:10.1037/a0019921
Nederkoorn, C., Jansen, E., Mulkens, S., & Jansen, A. (2007). Impulsivity predicts treatment outcome in obese children. *Behaviour Research and Therapy, 45*(5), 1071-1075.
Nederkoorn, C., Smulders, F. T. Y., Havermans, R. C., Roefs, A., & Jansen, A. (2006). Impulsivity in obese women. *Appetite, 47*(2), 253-256.
Ogden, C.L., Carroll, M.D., Lawman, H.G., Fryar, C.D., Kruszon-Moran, D., Kit, B.K., Flegal, K.M. (2016). Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *JAMA, 315*(21), 2292-2299.

O'Brien, L. M. (2014). Chapter 29 - Cognitive and Behavioral Consequences of Obstructive Sleep Apnea A2 - Sheldon, Stephen H. In R. Ferber, M. H. Kryger, & D. Gozal (Eds.), *Principles and Practice of Pediatric Sleep Medicine (Second Edition)* (pp. 231-238). Philadelphia: Content Repository Only

Ouyang, G., Sommer, W., Zhou, C., (2016). Reconstructing ERP amplitude effects after compensating for trial-to-trial latency jitter: A solution based on a novel application of residue iteration decomposition. *International Journal of Psychophysiology, 109*, 9-20.

Payne, B. K. (2005). Conceptualizing control in social cognition: how executive functioning modulates the expression of automatic stereotyping. *Journal of Personality And Social Psychology, 89*(4), 488-503.

Piispala, J., Maatta, S., Paakkonen, A., Bloigu, R., Kallio, M., & Jansson-Verkasalo, E. (2017). Atypical brain activation in children who stutter in a visual go/nogo task: An ERP study. *Clinical Neurophysiology, 128,* 194-203.

Puhl, R.M., Luedicke, J. (2012). *Weight-based victimization among adolescents in the school setting: Emotional reactions and coping behaviors*. *Journal of Youth and Adolescence, 41*(1), 27-40.

Rosemond, T.N., Blake, C.E., Buff, S.M., Blake, E.W., Dunn, B.L., Browne, T., Bell, B.A., & Iachini, A.D. (2016). Sensitizing future health professionals to determinants on childhood obesity. *American Journal of Preventative Medicine, 51*(1), 106-113.

Shunk, J. A., & Birch, L. L. (2004). Girls at risk for overweight at age 5 are at risk for dietary restraint, disinhibited overeating, weight concerns, and greater weight gain from 5 to 9 years. *Journal of American Dietetic Association, 104*(7), 1120-1126. doi:10.1016/j.jada.2004.04.031

Simmonds, D. J., Pekar, J. J., & Mostofsky, S. H. (2008). Meta-analysis of Go/No-go tasks demonstrating that fMRI activation associated with response inhibition is task-dependent. *Neuropsychologia, 46*(1), 224-232.

Spinella, M. (2007). Normative data and a short form of the Barratt Impulsiveness Scale. *The International Journal of Neuroscience, 117,* 359–368.

Stroop, J. R. (1992). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology: General, 121*(1), 15-23. doi:10.1037/0096-3445.121.1.15

Sur, S., & Sinha, V. K. (2009). Event-related potential: An overview. *Industrial Psychiatry Journal, 18*(1), 70-73. doi:10.4103/0972-6748.57865

Taylor, B. K., Gavin, W. J., & Davies, P. L. (2016). The test-retest reliability of the contingent negative variation (CNV) in children and adults. *Developmental Neuropsychology*. doi:10.1080/87565641.2016.1170835

Teslovich, T., Freidl, E. K., Kostro, K., Weigel, J., Davidow, J. Y., Riddle, M. C., . . . Mayer, L. (2014). Probing behavioral responses to food: development of a food-specific go/no-go task. *Psychiatry Research, 219*(1), 166-170. doi:10.1016/j.psychres.2014.04.053

Todd, R.M., Lewis, M.D., Meusel, L., Zelazo, P.D. (2008). The time course of social-emotional processing in early childhood: ERP responses to facial affect and personal familiarity in a go-nogo task. *Neuropsychologia, 46*(2), 595-613.

Watson, T. D., & Garvey, K. T. (2013). Neurocognitive correlates of processing food-related stimuli in a Go/No-go paradigm. *Appetite, 71*, 40-47. doi:10.1016/j.appet.2013.07.007

Williams, E. P., Mesidor, M., Winters, K., Dubbert, P. M., & Wyatt, S. B. (2015). Overweight and Obesity: Prevalence, Consequences, and Causes of a Growing Public Health Problem. *Current Obesity Reports*, *4*(3), 363-370.

US Department of Health and Human Services. *The Surgeon General's Vision for a Healthy and Fit Nation*. Rockville, MD: US Dept., Health and Human Services; 2010.

APPENDIX A

Food cues images included the following items:

- Cheerios®
- Rice
- Apple
- Corn
- Kit Kat bars
- French fries
- Chicken
- Sliced bread
- Bread rolls
- Spaghetti noodles
- Ravioli
- Orange
- Green grapes
- Banana
- Strawberries
- Carrots
- Peas
- Spinach
- Cucumber
- Cookie
- Cupcake
- Ice cream
- Cake
- Tortilla chips
- Popcorn
- Crackers
- Cheetos
- Hot dogs
- Sliced sandwich meat
- Cheese
- Pork chop
- Potato chips
- Pretzels
- Chocolate bars
- Oreos
- Skittles
- Honey buns
- Powdered donuts