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# Does Inhibitory Control Training Reduce Weight and Caloric Intake in Adults with Overweight and Obesity? A Pre-Registered, Randomized Controlled Event-Related Potential Study

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Event-Related Potential (ERP) Study


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ABSTRACT

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Overweight and obesity are prevalent public health problems that impact physical, mental, and social health. Many studies have evaluated weight loss treatments, but most individuals are unsuccessful at maintaining weight loss long-term. Behavioral and cognitive interventions may be effective in promoting weight loss and weight loss maintenance. One cognitive intervention that has shown potential success in reducing weight and caloric intake is inhibitory control training (ICT). ICT involves trainings where individuals are asked to repeatedly withhold dominant responses to unhealthy or high-calorie food images in an effort to increase food-related inhibitory control abilities. Reductions in caloric intake or weight may occur after as little as one week of ICT; however, it is unclear how more frequent ICT sessions promote weight loss and reduce caloric intake. Further, studies on food-specific ICT are generally poorly powered and it is unclear how ICT affects underlying cognitive and neural mechanisms. One way to measure inhibitory control processes is through the N2 component of the scalp-recorded event-related potential (ERP). The amplitude of the N2 ERP component tends to be larger (i.e., more negative) when an individual inhibits a dominant response during go/no-go tasks compared to non-inhibition go trials. I conducted a quasi-randomized controlled trial where 100 individuals with overweight or obesity were assigned to either a generic (active control; $n = 48$) or food-specific ICT (experimental group; $n = 52$). ICTs were completed four times per week for four weeks. Weight and caloric intake were obtained at baseline, immediately after four-weeks of ICT, and at a 12-week follow-up. Participants also completed a high-calorie and a neutral go/no-go task while electroencephalogram (EEG) was recorded at each visit. Results from mixed model analyses suggest that neither weight, caloric intake, nor N2 ERP component amplitude towards high-calorie foods changed at post-testing or at the 12-week follow up for either group. Regression analyses suggest that individuals with lower baseline levels of inhibition may show greater weight loss and reductions in caloric intake after a generic ICT, while individuals with higher baseline levels of inhibition may show greater weight loss and reductions in caloric intake after a food-specific ICT. Self-report ratings indicated the appetitive drive towards food decreased over the course of the study, particularly for individuals with higher levels of baseline inhibition. Overall, generic- or food-specific ICT did not affect weight, caloric intake, or food-specific N2 ERP amplitude. Food-specific ICT may be more effective in reducing caloric intake and weight for individuals with larger inhibition responses to food stimuli, while generic ICT may be more effective in reducing caloric intake and weight for individuals with smaller inhibition responses to food stimuli. ICT may also be targeting other mediating processes, such as the appetitive value of food, as opposed to improving food-specific inhibitory control.

Keywords: inhibitory control training, food-related inhibitory control, N2 ERP, weight, caloric intake

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Obesity and overweight are prominent public health concerns in the United States, with over two-thirds of the United States adult population being obese or overweight (Flegal et al., 2016; Flegal et al., 2010). The deleterious effects of overweight and obesity on physical health are well-documented, such as increased risk for cardiovascular disease (Avenell et al., 2004; Calle et al., 1999), Type II diabetes (Chan et al., 1994; Colditz et al., 1995), multi-cause cancer (Calle et al., 1999; Flegal et al., 2007; Reeves et al., 2007; Renehan et al., 2008) and death (Calle et al., 1999; Flegal et al., 2007; Kivimäki et al., 2008; Thorpe & Ferraro, 2004). There are also multiple mental health risks associated with overweight and obesity. For example, individuals with obesity are more likely to experience body image dissatisfaction (Grilo et al., 1994; Schwartz & Brownell, 2004). Individuals with overweight or obesity may also have lower levels of self-esteem (Puhl & Heuer, 2009) and higher rates of depression than normal-weight individuals, particularly in females and in cases of extreme obesity (Luppino et al., 2010; Onyike et al., 2003; Zhao et al., 2009). Overweight and obesity affect social functioning as well, such that individuals with obesity are more likely than normal-weight individuals to experience loneliness (Mushtaq et al., 2014). Individuals with overweight and obesity also face discrimination in the workplace (Puhl & Heuer, 2009; Spahlholz et al., 2016) and are the recipients of negative stereotype judgments (Puhl & Heuer, 2009) and weight teasing (Anderson, 2003; Puhl & Heuer, 2009).

Overweight and obesity are also associated with a high economic burden. Finkelstein et al., (2009) estimated that the direct costs of adult obesity (e.g., doctor visits, medications, etc.) in

the United States was about $147 billion—approximately 10% of all medical spending. An additional $66 billion was estimated as the indirect costs of adult obesity (e.g., absenteeism, disability, etc.). In another estimate, Tsai et al., (2011) suggested the direct costs associated with both overweight and obesity were about $113.9 billion—approximately 5-10% of all medical spending. Research has also consistently shown that individuals with overweight and obesity have higher medical costs than normal-weight individuals (Finkelstein et al., 2003; Gorsky et al., 1996; Thompson & Wolf, 2001). As the number of individuals with overweight and obesity are expected to keep increasing (Bell et al., 2011; Wang et al., 2012), the medical costs associated with overweight and obesity will rise (Lehnert et al., 2013) and nearly double over the next decade (Wang et al., 2012). Specifically, Wang et al. estimates that in 2030, approximately $860 to $956 billion will be spent on overweight- and obesity-related medical costs (approximately 16-18% of all medical spending). It is clear that overweight and obesity are economic burdens that impact physical, mental, and social wellbeing. As such, it is important to develop and systematically assess treatments that may be beneficial in preventing and reducing the prevalence of obesity and overweight (Kumanyika et al., 2002).

Modest reductions in weight (i.e., around 5% of body weight) can improve health risks associated with overweight and obesity (Cohen & Cohen, 2015; Jensen et al., 2014). A 5-10% reduction in body weight is associated with improved glucose levels (Jensen et al., 2014; Wing et al., 1987), blood pressure (Jensen et al., 2014), and cholesterol levels/lipid profiles (Goldstein, 1992; Jensen et al., 2014). Weight loss not only decreases the risk for obesity-related comorbidities, such as diabetes (Norris et al., 2005; Sheard, 2003; Sjostrom et al., 1999), but also decreases mortality risk (Ma et al., 2017; Williamson et al., 1995). Weight-loss is also associated with increased health-related quality of life (Fontaine et al., 1999; Hageman et al., 2019) and the

improvements in health can become long-term benefits if even modest weight loss is maintained (Rueda-Clausen et al., 2015; Thomas, 1995).

Weight loss is difficult and complex, as there are multiple factors to consider when treating individuals with overweight and obesity, such as metabolic health (Kramer et al., 2013), diet (Matarese & Pories, 2014; Schwartz, 2016), social relationships (Christakis & Fowler, 2007) and lifestyle factors (e.g., levels of physical activity and sleep; Chastin et al., 2015; Alamuddin & Wadden, 2016). In an attempt to become more precise and clarify what factors are differentially beneficial in promoting and sustaining weight loss, the National Weight Control Registry (http://www.nwcr.ws/) analyzed data from over 10,000 individuals who lost 30 pounds and maintained that weight loss for at least a year (Klem et al., 1997). The two most common practices identified among these individuals were: 1) self-monitoring food intake and weight, and 2) consuming fewer calories (i.e., 1300-1400 kcals per day compared to ~2,000), with a low proportion of calories coming from fat (20-25% of energy intake; Klem et al., 1997). As such, many weight-loss interventions have focused on targeting diet and reducing caloric intake in order to improve weight management (Bazzano et al, 2014; Lai et al., 2014; Matarese & Pories, 2014; Schwartz, 2016). In fact, Matarese and Pories (2014) estimate that there are over 1,000 different weight loss diet plans. While there are mixed results as to what specific diet intervention is best, reducing caloric intake and maintaining diet adherence generally seem to be key aspects to improving weight loss, at least in the short term (Matarese & Pories, 2014; Schwartz, 2016).

Not all diet interventions are successful at reducing weight and even after individuals lose weight, many are unsuccessful at maintaining their weight loss (Elfhag & Rössner, 2019; Jensen et al., 2014). For individuals with overweight and obesity, dietary interventions show a maximal

weight loss at six months from onset of the intervention and then show slow weight regain over time, mainly due to intervention adherence decreasing (Jensen et al., 2014). The American Heart Association (AHA), American College of Cardiology (ACC) and Obesity Society (TOS) together evaluated the evidence for weight loss interventions (Jensen et al., 2014) and found that individuals see a 4-12 kg reduction from initial weight at six months following intervention onset, a 4-10 kg reduction from initial weight at one year from intervention onset, and a 3-4 kg reduction from initial weight at two years from intervention onset. Difficulty in adhering to weight loss protocols that focus on reducing caloric intake may account for the poor rates of weight loss maintenance (Laddu et al., 2011). In addition, some diets, such the very low-calorie diet (~900 calories a day), are not meant for long-term adherence due to potential health complications (Cannon & Kumar, 2009; Strychar, 2006). Therefore, when individuals transition from a very low-calorie diet to a low-calorie diet, there is an increase in weight (Wadden et al., 1994).

Interventions that help individuals adhere to diet plans and improve dietary decisions could assist in obtaining and maintaining weight loss. One class of interventions that could promote weight loss and weight-loss maintenance are behavioral and cognitive interventions (Hainer et al., 2008). Cognitive and behavioral interventions reduce weight by approximately 8% on average in a six-month period (Laddu et al., 2011; McLean et al., 2015). Cognitive-based interventions (i.e., cognitive restructuring, problem solving, etc.) in particular are suggested as possible treatments to improve adherence and weight loss (Kelley et al., 2016; Laddu et al., 2011). There has also been a call to better understand how cognitive functioning is related to and/or altered with weight loss (McLean et al., 2015). In terms of regulating energy balance,

interventions involving self-control may be particularly important for long-term weight loss and

weight maintenance success (Hainer et al., 2008).

One cognitive aspect that is related to self-control and has gained recent attention is

inhibitory control. Inhibitory control refers to the ability to withhold a dominant or automatic

response in order to correctly respond to environmental demands or task-relevant information

(Ko & Miller, 2013). Inhibitory control may be related to diet in multiple ways, such as

withstanding cravings, suppressing the urge to eat palatable foods, or withholding from engaging

in emotional eating (Blundell & Gillett, 2001; Davis et al., 2007; Guerrieri et al., 2007; Jasinska

et al., 2012). Studies utilizing self-report and behavioral measurements (e.g., reactions times,

accuracy) of inhibitory control suggest that individuals who have lower inhibitory control

abilities eat more when presented with food than individuals who have higher levels of inhibitory

control (Guerrieri et al., 2007; Jansen et al., 2009). Lower levels of inhibitory control are also

associated with increased intake of palatable foods that are high in fat and sugar (Appelhans et

al., 2011; Hall, 2012). As such, it may be difficult for individuals with lower levels of inhibitory

control to withhold from palatable, unhealthy foods, which may be associated with subsequent

weight gain.

Given the relationship between inhibitory control and food intake, it may be possible for

inhibitory control to be utilized and trained to improve eating habits and weight. Studies of

individuals with attention deficit/hyperactive disorder (ADHD) suggest that neurocognitive

trainings can change and improve inhibitory control. For example, children with ADHD who

underwent neurofeedback training showed increased brain activity in inhibitory control regions,

such as the left superior parietal lobe and right middle frontal gyrus, after training compared to a

waitlist control group (Beauregard & Levesque, 2006) and an active control group (Baumeister

et al., 2018). In addition, children with ADHD showed improvements in inhibitory control, as measured by parental reports and neuropsychological tests, after an executive function training (Dovis et al., 2015; Johnstone et al., 2012). These results suggest inhibitory control is a cognitive domain that can be modified with training.

In the diet and weight-control literature, multiple studies have tested inhibitory control training (ICT) in acute, single-session laboratory experiments (e.g., Adams et al., 2017; Chen et al., 2019; Forman et al., 2016; Houben & Jansen, 2011, 2015; van Koningsbruggen et al., 2014; Veling et al., 2013). For example, in two separate studies, Houben and Jansen (2011, 2015) found that individuals who were asked to continually inhibit responses to chocolate pictures during a go/no-go task ate less when presented with chocolate afterwards compared to individuals who were asked to occasionally inhibit or always respond to chocolate pictures. In another study, individuals who inhibited responses to images of sweet foods selected fewer sweet foods from an *ad libitum* dispenser immediately following the training compared to individuals who inhibited responses to everyday objects (van Koningsbruggen et al., 2014). Low emotional eaters who completed an ICT tailored to salty snacks also consumed fewer salty snacks in the seven days after the ICT compared to the seven days before (Forman et al., 2016). These results suggest that inhibitory control can be trained and utilized in as quickly as one session to reduce short-term food intake.

Further, ICT may be particularly beneficial to populations who have difficulties in implementing healthy eating behaviors. For example, in individuals diagnosed with an eating disorder, such as binge eating disorder (BED), ICT helps reduce weight (Preuss et al., 2017), decrease binges, and improve inhibitory control (Giel et al., 2017). ICT has also been effective in reducing food intake for chronic dieters (Veling et al., 2011), individuals with restrained eating

tendencies (Adams et al., 2017; Lawrence et al., 2015), and individuals with higher appetites

compared to those with lower appetites (Veling et al., 2013). In individuals who demonstrate

lower baseline levels of inhibitory control, ICT may be more effective since these individuals

have the most room to improve their inhibitory control abilities (Houben, 2011; Forman et al.,

2019). In sum, ICT can be an effective intervention for individuals who have habits and

tendencies that have decreased their ability to obtain healthy diet and weight-loss outcomes.

Meta-analyses on food-related ICT further support the hypothesis that utilizing food

stimuli during ICT is related to decreased food consumption after training (Allom et al., 2015;

Jones et al., 2016). Results suggest that not only do objective (e.g., laboratory taste tests) and

subjective (e.g., self-report) measures of food intake decrease following ICT, but also unhealthy

food choices decrease in response to ICT (Allom et al., 2015 standardized mean difference =

0.37; Jones et al., 2016 standardized mean difference = 0.33). As far as mechanisms,

questionnaire, behavioral, and functional magnetic resonance imaging (fMRI) data suggest that

part of the success of ICT may be due to ICT reducing the hedonic, motivational, or reinforcing

value of food (Houben & Giesen, 2018; Veling et al., 2017). The devaluation of rewarding food

may reduce conflict between dominant response tendencies to eat rewarding foods and goals to

reduce unhealthy food consumption (De Pretto et al., 2019). Overall, results suggest that similar

to other behavioral/cognitive interventions, ICT may promote healthy dietary decisions and aid

in weight loss and weight loss maintenance.

However, the Allom et al. (2015) and Jones et al. (2016) meta-analyses issue a word of

caution, as the majority of studies included in the meta-analyses only had single sessions, small

samples sizes, and were conducted in healthy young adults (i.e., not individuals with overweight

or obesity trying to lose weight). A recent *p*-curve analysis (an assessment of the distribution of

significant *p*-values in published literature in order to measure selective reporting, power, and effect size) on food-related ICT supports these cautions. In the analysis, Carbine and Larson (2019) found that current published food-related ICT studies have small sample sizes (approximately 28 participants per group on average) that are only powered at about 7% to 18% to detect small effects ($d = .04$ to $.25$). The *p*-curve results also suggest researchers may be selectively reporting and publishing only significant results in the food-related ICT literature, while non-significant results are infrequently published (i.e., there is likely a file-drawer problem in the ICT literature). Some published studies have reported no significant ICT effects on weight loss or eating behaviors (Adams et al., 2017; Poppelaars et al., 2018; Turton et al., 2018) and it's possible that the significant effect of ICT are due to impulsivity being induced in the control group (e.g., having participants respond to foods) instead of inhibition being improved in the experimental group (Adams et al., 2017). Studies with larger sample sizes of individuals with overweight and obesity and testing the effect of food-related ICT to a comparable control group (i.e., inhibiting to neutral stimuli) beyond a single session are needed to better understand the magnitude of the effects of ICT on food intake and weight.

While clinical samples (e.g., BED; Preuss et al., 2017) have seen success in reducing weight when using more long-term (e.g., multiple trainings a week for multiple weeks) as opposed to acute (e.g., single laboratory session) ICT interventions, there is limited research examining the longer-term effects of food-specific ICT in psychiatrically healthy individuals (Allom et al., 2015; Carbine & Larson, 2019). Lawrence et al. (2015) had 84 individuals (about 42 individuals per group) across the body mass index (BMI) spectrum complete four, 10-minute-long, generic or food-specific ICT sessions over the course of one week. They found that individuals in the food-specific, but not generic, ICT group reduced their caloric intake (about a

220.4 kcals decrease across two weeks) and lost weight immediately after the intervention

(approximately 0.67 kgs of weight loss across 2 weeks) and at a six-month follow-up

(approximately 2.21 kgs of weight loss). Similarly, Oomen et al. (2018) found in 41 normal-

weight individuals (about 20 per group) that those who completed a food- relative to a non-food

ICT once per day for six days reduced their food consumption during a laboratory taste test.

Veling et al. (2014) conducted a four-week intervention where 113 participants (about 28 per

group) across the BMI spectrum completed either a food-specific or generic ICT paired with

either a diet or control intervention once a week (the overall intervention lasted about 25

minutes). They found that individuals in the food-specific ICT group lost weight, while those in

the generic ICT group only lost weight if the trainings were supplemented with the diet intention.

Further, individuals with a higher BMI lost 1.04 more kgs of weight in the food ICT group, while

individuals with a lower BMI lost 0.15 more kgs of weight in the generic ICT group, suggesting

a disproportionate increase in weight loss for individuals with overweight or obesity in the food-

specific ICT compared to the generic ICT. Finally, Stice et al. (2017) implemented one 50-

minute ICT session per week for four weeks in 47 individuals with overweight or obesity (about

23 individuals per group). They found individuals in the food-specific versus generic ICT group

not only had greater reductions in reward and attention to high-calorie food images, but also

showed greater reductions in body fat percentage after the four weeks (about 1%, although the

reduction in body fat was not significant at 6-month follow-up).

Although the extant findings on food-related ICT for weight loss are promising, most

studies are still relatively short-term (i.e., last only one week or have less than six ICT sessions)

and are heterogeneous in terms of how long each ICT session lasts, how frequently ICTs are

implemented, and the BMIs of participants. The benefit from completing an ICT multiple times a

week for multiple weeks in a psychiatrically-healthy overweight/obese sample is unclear. The longest, most consistent ICT study to date conducted exclusively in individuals with overweight and obesity was done by Forman et al. (2019). In their study, 106 participants (about 26 in each group) completed a gamified or non-gamified ICT that involved food or neutral stimuli once per day for 6 weeks and then one training per week for two weeks (for a total of 44 trainings). The authors found that while inhibitory control performance improved for participants who received the food compared to generic ICT intervention, individuals in the food ICT group did not lose more weight than the generic ICT group. However, individuals in the food ICT group who showed the greatest improvement in inhibitory control (as measured by changes in response times) across the first three weeks of the study lost more weight at the end of the trainings. While adherence was excellent for the both groups (88.8%), which shows promise for using ICT as a consistent intervention, the food ICT in this study was specifically targeted at reducing consumption of sweet foods (not high-calorie or unhealthy foods in general). Further, all individuals were participating in a no-added-sugar diet, and sample sizes in each arm of the study were still relatively small (about 26 people per group). As such, more long-term and higher-powered ICT studies that that are not paired with a specific diet are needed in order to robustly test if ICT is an effective weight loss intervention to use on its own (Carbine & Larson, 2019; Wolz et al., 2020).

It is also unclear if the benefits of ICT persist after the trainings are complete. An examination of ICT with a longer-term follow-up is needed in order to assess how long benefits last, whether it is days to weeks to months. Knowing how long ICT-related benefits persist will inform the research and clinical community on if ICT can promote sustainable diet, weight, and behavioral changes by itself or as an augmentation to other treatments. Testing how more

frequent ICT sessions affect weight and diet over a longer period (i.e., weeks to months) has

important implications for weight management interventions. If successful, the easy to

administer ICT could aid in caloric intake and weight management, and may assist individuals

who initially are struggling with controlling their diet (Forman et al., 2019; Houben, 2011).

Consequently, the first aim of my dissertation was to determine if a more consistent and long-

term ICT (i.e., multiple days per week for four weeks) improved caloric intake and weight

outcomes in individuals with overweight and obesity and if those improvements persisted for 12-

weeks after the intervention was complete.

     Not only are the long-term benefits of ICT uncertain, but it is still unclear how ICT

paradigms influence neural indices of inhibitory control towards food (Allom et al., 2015).

Psychophysiological and neuroimaging research to date indicates that neural indices of inhibitory

control are associated with food stimuli and changes in weight. In the adult fMRI literature,

inhibiting to high- compared to low-calorie foods elicits a larger hemodynamic response in left

frontal and parietal brain regions, which are involved in inhibitory control (Carbine et al., 2018).

These results suggest that withholding responses specifically to high-calorie foods may

necessitate increased recruitment of inhibitory control resources. A meta-analysis on

neuroimaging measures of inhibitory control in individuals with obesity found that individuals

with lower BMIs and greater weight loss over time demonstrate increased activation in inhibitory

control brain regions (Lavagnino et al., 2016), although the inhibitory control activity was not

specific to food stimuli. In a sample of normal-weight individuals, individuals with obesity, and

successful weight losers (i.e., individuals who had maintained a weight loss of at least 30 pounds

over three years), the successful weight losers showed greater activation in inhibitory control

regions, such as the left superior frontal lobe, when viewing food images compared to normal-

weight individuals and individuals with obesity (McCaffery et al., 2009). Together, results

suggest that additional recruitment of inhibitory control resources in the brain may be necessary

to manage food intake and maintain weight loss.

An additional way to observe neural indices of food-related inhibitory control is through

components of the scalp-recorded event-related potential (ERP). ERPs are changes in the brain's

electrical activity recorded from scalp-electrodes on an electroencephalography (EEG) net

(Luck, 2014). The amplitude of an ERP gets larger or smaller depending on how people process

or respond to a stimulus (Rugg & Coles, 1995). There are multiple ERP components associated

with a variety of brain processes, such as visual processing, emotional processing, or higher-level

cognitive processing (e.g., Folstein & Van Petten, 2008; Hajcak et al., 2010; Krolak-Salmon et

al., 2001). One ERP component, the N2 component, is a negative deflection in the ERP

waveform that occurs around 200 ms after a stimulus has been presented (Folstein, & Van

Petten, 2008). The N2 ERP component is a multifaceted waveform that reflects different

cognitive processes depending on the instructions and task at hand. The N2 ERP component is

consistently elicited during multiple cognitive tasks, such as oddball paradigms, stop signal

tasks, flanker tasks, Stroop tasks, and go/no-go tasks. As such, there is strong evidence that

changes in N2 ERP amplitude reflects changes in attention, conflict monitoring, and inhibitory

control (Folstein, & Van Petten, 2008). In terms of inhibitory control, the amplitude of the N2

ERP component is larger when individuals withhold a dominant or prepotent response,

particularly on go/no-go tasks (i.e., when individuals withhold their response to the no-go

stimulus), providing support that the N2 ERP component serves as an indicator or measurement

of inhibitory control processes (Folstein, & Van Petten, 2008).

The amplitude of the N2 ERP component is larger (i.e., more negative) when individuals inhibit responses from food cues as opposed to neutral cues (Watson & Garvey, 2013), suggesting there is an increased recruitment of neural resources when individuals must withhold dominant responses to food. Inhibiting responses to high-calorie foods compared to low-calorie foods also results in a larger N2 ERP component amplitude (Carbine et al., 2017), possibly because the more rewarding and palatable nature of high-calorie foods requires increased inhibitory control to withhold dominant responses (Appelhans et al., 2011; Hall, 2012). Additionally, individuals with a larger N2 ERP amplitude to high-calorie foods tend to consume fewer calories and carbohydrates, suggesting they are recruiting the necessary cognitive resources to manage their food intake (Carbine et al., 2017), although this effect has not always been consistent (Carbine et al., 2018).

In terms of food-related ICT, the magnitude of theta waves, or neural oscillations between 4-8 hertz that increase when inhibiting responses to stimuli (Harper et al., 2014) and may reflect similar regulatory processes as the N2 ERP component (Cavanagh & Frank, 2014), increases towards foods previously paired with a no-go response (van de Vijver et al., 2018). At the end of an ICT go/no-go paradigm, electrophysiological activity in the orbito-cingulate around 200 ms after stimulus presentation increased when inhibiting to rewarding versus aversive food stimuli (De Pretto et al., 2019). These two results suggest it is possible that ICT may affect neural indices of inhibitory control, particularly those that are related to the N2 ERP component, by increasing activation of inhibitory control resources.

Results showing how ICT affects psychophysiological measures of inhibition are not consistent. Two studies by Aulbach et al. (2020) and Blackburne et al. (2016) found that ICT did not affect the amplitude of the N2 ERP component, supporting the idea that ICT may not affect

top-down cognitive processes (Veling et al., 2017). It is important to note that Blackburne et al. (2016) used an auditory go/no-go task, and thus did not specifically test changes in the N2 ERP component towards food-specific stimuli. While Aulbach et al. (2020) measured the N2 ERP component during food go/no-go tasks, they tested the effects of a single training session (i.e., comparing N2 ERP amplitude at the beginning of the go/no-go task to the end). As such, research is still needed to see how consistent, long-term, food-specific ICT affects neural indices of food-related inhibitory control, as measured by the N2 ERP component.

Understanding how ICT may influence neural indices of inhibitory control is important, since neural indices of food-related inhibitory control may relate to food intake (Carbine et al., 2017, 2018). Changes in neural indices of inhibitory control could elucidate potential underlying mechanisms as to why ICT works (Jones et al., 2016). For example, if ICT improves eating habits, examining the N2 ERP component could elucidate if the improved eating habits are because ICT aids individuals in successfully recruiting more inhibitory control resources. Thus, the second aim of my dissertation was to measure how neural indices of inhibitory control change immediately after ICT and at a 12-week follow-up. For my third aim, I tested if neural indices of inhibitory control at baseline predicted the weight loss and caloric intake changes that occur after ICT. As ERP measures seem to consistently detect differences in inhibitory control between low- and high-calorie foods (Carbine et al., 2018), I utilized EEG technology and, specifically, the N2 ERP component as a neural marker/measurement of food-related inhibitory control.

In summary, I tested how more frequent and long-term ICT affects weight, caloric intake, and neural indices of inhibitory control in psychiatrically-healthy individuals with overweight and obesity. My project helps lay the foundation for future research to understand how ICT on its

own or paired with other interventions, like diet plans, improve weight loss, weight maintenance, and the overall health of individuals. The specific research questions and hypotheses for my dissertation were as follows:

**Research Question 1:** Compared to a generic ICT (active control), does a food-specific ICT completed multiple times a week over four weeks alter weight and caloric intake in individuals with overweight and obesity, and do these changes persist at a 12-week follow-up?

**Hypothesis:** Individuals in the food-specific ICT compared to generic ICT will exhibit greater weight loss and greater reductions in caloric intake from baseline to immediately after ICT, and at a 12-week follow-up (Lawrence et al., 2015; Veling et al., 2014).

**Research Question 2:** Do neural indices of inhibitory control, as measured by N2 ERP amplitude, change from pre- to post-treatment due to a generic ICT or food-specific ICT, and do these changes persist at a 12-week follow-up?

**Hypothesis:** Immediately after ICT and at the 12-week follow-up, individuals in the food-specific ICT compared to the generic ICT will exhibit larger N2 ERP difference amplitudes, suggesting they are more successful at recruiting necessary food-related inhibitory control resources (Carbine et al., 2017; van de Vijver et al., 2018).

**Research Question 3:** Do neural indices of smaller or larger food-related inhibitory control at baseline relate to changes in weight and caloric intake after ICT?

**Hypothesis:** Individuals with smaller N2 ERP difference amplitudes at baseline will show greater reductions in caloric intake and weight, particularly if they were in

the food-specific ICT, as individuals who demonstrate lower inhibition to food may benefit the most from ICT (Forman et al., 2019; Houbon, 2011) and, therefore, show greater "success", as indicated by greater reductions in weight and caloric intake.

## Method

All experimental protocols, materials, and data are posted to the Open Science Framework (OSF; https://osf.io/szxua/). Study protocols followed CONSORT reporting guidelines for clinical trials (Altman et al., 2001; see Figure 1), were registered on clinicaltrials.gov prior to beginning data collection, and all hypotheses, methods, and the analysis plan were pre-registered on OSF prior to data collection (posted here: https://osf.io/39wv6).

### Power Analyses

Sample size calculations are important to not only determine if a study is powered enough to detect effects of interest (Larson & Carbine, 2017), but to ensure that presented effects are not inflated (Button et al., 2013). As changes in weight and caloric intake across multiple ICT sessions were my main outcomes of interest, I conducted three power analyses in G*Power (v3.1) based on data and effect sizes reported by Lawrence et al. (2015) and Veling et al. (2014).

First, based on the effect size reported in Lawrence et al. (2015), I conducted a two-group, three-measurements, within-between interaction power analysis based on the observed Time x Group interaction of weight- $\eta_p^2$=.08 (a difference of 0.84 kg between ICT groups at the 2-week follow-up), which was converted into an $F$ effect size of .29. To achieve 80% power, a sample of 60 participants was needed to detect the effects of interest (alpha = .05). Second, based off Lawrence et al. (2015), I conducted a two-group, three-measurements, within-between

interaction power analysis based on the observed Time x Group interaction for caloric intake-$\eta_p^2$=.06 (a difference of about 240 kcals between ICT groups during the intervention week), which was converted into an *F* effect size of .25. To achieve 80% power, a sample of 80 participants was needed to detect the effects of interest (alpha = .05). Finally, based off Veling et al. (2014), I conducted a two-group, three-measurements, within-between interaction power analysis based on the observed weight loss difference between ICT groups in adults with higher BMIs- $\eta_p^2$=.07 (higher BMI group lost 1.04 more kilograms of weight in the food ICT while the lower BMI group lost 0.15 more kilograms of weight in the generic ICT), which was converted into an *F* effect size of .27. To achieve 80% power, a sample of 68 participants was needed to detect the effects of interest (alpha = .05). As published effect sizes are likely to be inflated (Ioannidis, 2005), I chose to follow the larger sample size estimate of 80 participants. In anticipating a conservative ~20% drop out rate or losing individuals with bad EEG/ERP data that do not have sufficient reliability, I planned on recruiting at least 100 participants to have a minimum of 80 participants used in the final analyses.

**Participants**

Two-hundred and forty individuals were recruited and assessed for eligibility, of which 135 were excluded for not meeting inclusion criteria, leaving 105 participants who were enrolled in the study (see Figure 1 for the CONSORT diagram). Participants were recruited *via* flyers posted on Brigham Young University's campus and throughout the local community. All participants were between the ages of 18 and 45 in order to account for possible confounding effects of cognitive and hormonal changes associated with menopause and with increased age (e.g., Berent-Spillson et al., 2010; Faubion et al., 2015; Ryan et al., 2014). All participants were individuals with overweight (25 kg/m$^2$ $\leq$ BMI < 30 kg/m$^2$) or obesity (30 kg/m$^2$ $\leq$ BMI), as I

aimed to recruit individuals who would benefit from a modest amount of weight loss, and ICT

may be more effective for individuals with higher BMIs (Veling et al., 2014).

In order to decrease potential confounding variables due to psychological and health

factors, participants had to meet the following inclusion criteria (see Figure 1): no current

diagnosis of a psychological disorder (e.g., major depressive disorder, general anxiety disorder)

and no history of a neurological disorder (e.g., epilepsy, stroke), learning disability, ADHD,

eating disorder (e.g., anorexia nervosa, bulimia nervosa, binge eating disorder), or

metabolic/chronic disease (e.g., cardiovascular disease, Type II diabetes); not pregnant or

lactating; not currently participating in a weight-loss diet; and no a head injuries that resulted in a

loss of consciousness. In order to account for exercise habits, potential participants were

excluded if they were avid exercisers (i.e., participated in at least 20 minutes of vigorous

physical activity more than three times a week; Carbine et al., 2017). Exclusion criteria were

assessed *via* phone calls by trained research assistants after individuals expressed interest in

participating in the study.

Out of the 105 participants, five were excluded after starting study participation after

reasons for not meeting inclusion criteria were discovered (two for currently being on a weight-

loss diet, one for having a chronic disease, one for previously being diagnosed with an eating

disorder, and one for being normal-weight at baseline) leaving a final enrollment of 100

participants. Of the 100 final participants ($M_{age}$ = 28.05; $SD_{age}$ = 7.56; 53 female; see Table 1 for

participant demographics by group), 100% completed the baseline visit, 87% completed the four-

week visit, and 84% completed the 12-week visit (see Figure 1).

**General Procedures Overview**

The study design is outlined in Figure 2. The study checklist is also posted to OSF and can be found here: https://osf.io/z7m92/. Participants completed a baseline lab session, a four-week long ICT period, a follow-up lab session immediately after the ICTs were completed (i.e., four-week visit), and a lab session 12-weeks after trainings were completed (i.e., 12-week visit). In order to reduce potential confounding effects from exercise (Hanlon et al., 2012) and sleep (St-Onge et al., 2014), participants were asked to refrain from consuming caffeine and participating in vigorous physical activity 24 hours before and get at least 7 hours of sleep the night before their lab sessions. Participants were also asked to stop eating and drinking (besides water) by 9pm the night before if they came in during the morning (i.e., 7-10am) and four hours before if they came in during the afternoon or evening in order to control for hunger effects (Carbine et al., 2017). The fasting requirements also allowed me to collect neural responses to food at a time when participants were about to consume their next meal of the day. Participants completed all lab sessions at the same time of day to control for time of day effects (i.e., the baseline, four-week, and 12-week lab visits were approximately at the same time of day for each individual participant).

During the baseline lab session, participants completed a questionnaire that assessed basic health and demographic information, the Dutch Eating Behavior Questionnaire (DEBQ; van Strien et al., 1986), and the Power of Food Scale (Cappelleri et al., 2009; Lowe et al., 2009). Height and weight were measured by a physician scale (Detecto). Participants also completed six visual analog scales (VAS) to assess hunger. They then completed two go/no-go tasks while EEG data were collected. Finally, participants were read a standard explanation that described the ICT protocol and goals (explanation can be found here: https://osf.io/6ya23/), and

information to set up the trainings (e.g., what business days they would like to complete trainings on; see Inhibitory Control Training Protocol section below) was gathered.

The four-week and 12-week lab sessions were identical to the baseline visit, except participants did not complete the basic health and demographic questionnaire again (height, weight, DEBQ, and Power of Food Scale were still obtained) and did not have the ICT explanation re-read to them. For all lab sessions, research assistants were blinded to participant ICT assignment. In the days immediately following the baseline, four-week, and 12-week lab visits, participants completed three dietary recalls using the Automated Self-Administered 24-hour Dietary Recall program (ASA24; Subar et al., 2012), for a total of nine recalls over the course of the study (see ASA24 Dietary Recalls section below).

**Inhibitory Control Training Protocol**

There were two arms for the ICT protocol: a food-specific ICT group and a generic ICT group. In the food-specific ICT group, participants inhibited responses to high-calorie foods, while participants in the generic ICT group utilized a task where they inhibited responses to everyday items (see Inhibitory Control Training Tasks section). I strived for a similar proportion of male and female participants in each arm. As ICTs may work better for individuals with lower levels of inhibitory control (Forman et al., 2019; Houben, 2011), I also strived to have a similar proportion of individuals with high and low levels of inhibitory control in the two ICT groups to ensure that results were not due to one intervention having a larger proportion of individuals with lower levels of inhibitory control. Then, regression analyses to test if baseline levels of inhibitory control actually influenced weight and caloric intake outcomes were conducted (see Statistical Analyses section). Thus, the assignment of participants to ICT group was quasi-random based on sex and baseline inhibitory control (see specific procedures in the next paragraph below;

randomization sheet can be found here: https://osf.io/bhqsp/). In the final sample of 100

participants, 52 participants were quasi-randomly assigned to the food-specific ICT (24 high

inhibitory control; 27 females) and 48 participants were quasi-randomly assigned to the generic

ICT (20 high inhibitory control; 26 females). A *chi*-square analyses revealed the proportion of

individuals with low or high baseline inhibition responses did not differ between ICT groups

($\chi^2(1) = 0.20$; $p = .65$) and the proportion of males and females did not differ between ICT

groups ($\chi^2(1) = 0.05$; $p = .82$).

To assign participants to either a high or low inhibitory control response, a median split

was performed on N2 ERP no-go amplitude data from a previous sample of 214 participants

across the BMI spectrum (median = -2.22 µv; Carbine et al., 2017, 2018), who completed the

same high-calorie go/no-go task that was used in this study (see EEG Inhibitory Control Tasks

section). After the baseline lab session, ERP analyses (see EEG Data Acquisition and

Assessment section) were conducted on participants' data from the high-calorie go/no-go task to

get initial food-specific inhibitory control levels, which was then used (based on the -2.22 µv

median) to label participants with either a high or low baseline inhibitory control response and

assign participants to an ICT group. As previous research has suggested that 10 or more trials are

necessary to produce reliable N2 ERP amplitude when using food stimuli (Carbine et al., 2018),

participants were required to have at least 10 useable trials for their baseline data to be

considered reliable. If participants' baseline session was unreliable, they returned within one

week to redo the high-calorie go/no-go task ($n = 2$).

Utilizing an experimental group (i.e., food-specific ICT) and an active control group (i.e.,

generic ICT) is a strength of the current design and replicates the interventions implemented by

Lawrence et al. (2015) and Veling et al. (2014). Interventions that use a non-contact control

group as opposed to an active control group may undermine the inferences one can make from an intervention, as the effect could be due to a number of reasons such as a placebo effect or a training effect (Boot et al., 2013). Further, the two groups must be similar enough that any differences observed can be attributed to the effects of interest (Boot et al., 2013). Using the generic ICT as an active control (as opposed to using a non-contact control group) means potential differences between groups can be attributed to improving food-specific inhibitory control as opposed to improving inhibitory control in general.

Based off Veling et al. (2014), who did a month-long intervention, and Lawrence et al. (2015), who had participants complete the ICTs four times a week, my ICT intervention lasted for four-weeks where participants were instructed to complete a 10-minute ICT on a mobile device (i.e., iPhone or iPad) outside of the lab for four days of the week. In order to increase compliance, participants were allowed to choose four out of five business days each week to complete the ICT. Even if participants did not complete all required training sessions, I still gathered post-treatment and follow-up data on as many participants as possible to be used in the final analyses (White et al., 2012). Follow-up analyses using data only from participants who completed on average three out of the four days each week (75% adherence) was also completed (see Statistical Analyses section below), as a 75% adherence rate is a similar requirement in other behavioral interventions (Krebs et al., 2010; Kirk et al., 2007; LeCheminant et al., 2007; LeCheminant et al., 2014).

Participants were randomly assigned one of 16 ICT tasks to complete each training day (randomization sheet can be found here: https://osf.io/5r4mu/). To help participants complete the ICT, an automated text or email message (whichever the individual participant indicated was their preference) was sent at 9:00am each business day with instructions on how to complete one

of the 16 trainings and then again at 5:00pm to remind participants to complete the ICT if they

had not yet completed the training. In order to improve adherence, for each week participants

had an adherence rate of at least 75%, their name was entered to win an iPad (maximum of four

entries). The trainings were administered *via* the Paradigm for Mobile app on an IOS device

(http://www.paradigmexperiments.com/). Participants were provided an iPad mini for use if they

did not have access to their own IOS device.

**Inhibitory Control Training Tasks**

Task parameters were consistent with those used by Lawrence et al. (2015). For both the

generic and food-specific ICT tasks, there were six blocks of 40 trials each, for a total of 240

trials per training. Per block, fifty percent of trials were go trials and 50% were no-go trials.

During the task, a picture on a white background then appeared on the screen for 1250ms with

two gray boxes, one on the bottom right side of the screen and one of the bottom left side of the

screen. A blank white screen then appeared for 1250ms as an inter-stimulus interval. Pictures

indicating a go trial appeared as normal while pictures indicating a no-go trail were framed in a

bold black box (see Figure 3 for example stimuli). Participants were instructed on go trials to

indicate as quickly and accurately as possible what side of the screen the picture appeared on by

pressing the right or left side box. For no-go trials, participants were instructed to withhold their

response and to not hit either box. Half of go trials appeared on the right side of the screen and

half on the left. Similarly, half of no-go trials appeared on the right side of the screen and half on

the left. The order of go and no-go trials and the side of the screen they appeared were random.

Stimuli for all ICT tasks were selected from the Food-Pics database, a large picture

database normed in 1,988 people that contains food and non-food items (Blechert et al., 2014). In

the Food-Pics database, there are 315 non-food pictures and 568 food pictures, with provided

weight and calorie content of the pictured foods. One of the benefits of using a large and standardized database is that participants did not see the same pictures too frequently, helping prevent burnout and habituation. In a previous study sample collected from our lab (Carbine et al., 2020), food pictures from the Food-Pics database were quantified as high-calorie foods if they had a caloric density of at least 3 kcal/g (e.g., chocolate, donuts, cake), resulting in 180 "high-calorie" foods. Food pictures were quantified as low-calorie foods if they had a caloric density of 1 kcal/g or less (e.g., apples, celery, carrots), resulting in 200 "low-calorie" foods. A group of 100 undergraduates then rated the 380 pictures as high- or low-calorie foods. One-hundred and twelve pictures were accurately classified as high-calorie foods 95% of the time or better and 114 pictures were accurately classified as low-calorie foods 95% of the time or better. From these pictures, 100 high-calorie foods and 100 low-calorie foods were randomly selected to be used in the final food ICT task (see list of stimuli here: https://osf.io/zau2y/).

For the food-specific ICT task, 10 low-calorie, 10 high-calorie, and 20 non-food pictures were randomly selected for each of the 16 trainings. The high- and low-calorie pictures were selected from 200 food pictures as described above. The non-food pictures came from a random selection of 200 non-food items from the animals, tools, kitchen, and household item categories of the Food-Pics database (so that there were the same number of food and non-food items; see list of stimuli here: https://osf.io/25msr/). It is important to note that the food-packaging category (e.g., the wrapper of a candy bar, pizza box, … etc.) was not used, as it contained relevant food stimuli that could have confounded results. High-calorie foods were always presented as no-go trials (i.e., always surrounded by a bold black box). Similarly, low-calorie foods were always presented as go trials. Fifty percent of the non-food items were randomly presented as go trials and 50% were randomly presented as no-go trials, providing variance to the task and making the

purpose of the task less obvious. Each picture was shown once per block (i.e., each picture was shown 6 times in one task). For the generic ICT task, only the non-food pictures were used as go and no-go trials. Specifically, 40 non-food pictures were randomly selected for each of the 16 tasks from the 200 non-food items. Fifty-percent were randomly selected as go trials and 50% were randomly selected as no-go trials. Besides picture stimuli, the generic and food-specific ICT tasks were identical.

**EEG Inhibitory Control Tasks**

For EEG assessment during lab sessions, participants completed a high-calorie food go/no-go task to assess food-related inhibitory control and a neutral go/no-go task to assess general inhibitory control (i.e., not specific to food stimuli). For the food-specific go/no-go task, participants were asked to respond with a button press of their right index finger when they saw a low-calorie food (go stimuli) and refrain from responding when they saw a high-calorie food (no-go stimuli). The task was identical to that used in previous studies (Carbine et al., 2017, 2018). For the neutral go/no-go task, participants were asked to respond with a button press of their right index finger when they saw a picture of an office/household object (go stimuli) and refrain from responding when they saw a picture of a flower/leaf (no-go stimuli). Other than the use of different go and no-go stimuli, the two tasks were identical. The tasks consisted of two blocks with 100 trials each. In order to establish a pre-potent response toward go trials, 70% of trials were go trials and 30% of trials were no-go trials, as is commonly used in ERP go/no-go tasks (e.g., Benikos et al., 2013; Ramos-Loyo et al., 2013). Go and no-go trials were randomly presented. Stimuli were presented for 500 milliseconds on a white background with an inter-stimulus black fixation cross on a white background varying in presentation duration between

1200 and 1400 milliseconds. Tasks were presented using E-prime 2.0 (Psychology Software Tools, Inc., 2012) and order was counterbalanced across participants and sessions.

For the EEG food go/no-go task, 120 pictures were provided from Killgore et al. (2003), who have used the same pictures in multiple studies (e.g., Killgore & Yurgelun-Todd, 2005, 2007; Killgore et al., 2013). A separate group of 26 undergraduates previously rated the pictures as either high- or low-calorie foods (Christensen, 2014). Only food pictures that were accurately classified as high- or low-calorie foods 95% of the time or better were used in the final task, resulting in 38 high-calorie food pictures and 38 low-calorie food pictures. For the low-calorie food pictures, there are 13 vegetables (e.g., carrots, broccoli) and 25 fruits (e.g., apples, oranges). For the high-calorie food pictures, there are 16 desserts (e.g., cake, ice cream), 15 high-calorie dinner meals (e.g., hamburgers, hot dogs), and 7 high-calorie breakfast meals (e.g., waffles, pancakes; stimuli are available upon request).

For the object go/no-go tasks, 38 pictures of flowers/leaves were randomly selected from the 42 pictures in the flower/leaf category of the Food-Pics database (which was not used in the generic ICT task). Thirty-eight pictures of office/household items were selected from the 45 pictures in the "office" and "other" categories of the Food-Pics database (also not used in the generic ICT task; see list of stimuli here: https://osf.io/8fmwx/). For both tasks, pictures were randomly selected during stimulus presentation. As there are 70 go trials per block but only 38 go pictures, after all 38 pictures were shown once, the pictures were repeated again with random selection. Consequently, some pictures were shown more than others, but in a random fashion. The use of different stimuli and task parameters for the lab tasks and the ICT tasks ensured that any changes seen at lab visits were not due to learning the specific task, but due to changes in

inhibitory control. Specifically, using different food pictures may help clarify if the effects from the food-specific ICT generalize.

**EEG Data Acquisition and Assessment**

EEG data were recorded using an Electrical Geodesics, Inc. NA300 amplifier system (20K nominal gain, bandpass = 0.10-100 Hz) and a high-density 128-channel EEG net with equidistant passive Ag/AgCl electrodes. During collection, electrode impedances will be kept below 50kΩ (as suggested by the manufacturer). Data were referenced online to the vertex electrode (Cz) and digitized continuously at 250 Hz. During off-line data analyses, data were digitally high-pass filtered with a first order 0.1 Hz and digitally low-pass filtered at 30 Hz (2 Hz roll off; FIR) in NetStation (version 4.5.7). ERP data were then segmented from 200ms before stimulus onset to 1,000ms after stimulus onset and exported into EGIS format to read into the ERP PCA Toolkit for artifact correction (Dien, 2010).

Bad channels were flagged if the fast average amplitude exceeded 100 microvolts (µV) or if the differential average amplitude exceeded 50µV, and channel data was interpolated using the nearest neighbor approach (i.e., six surrounding electrodes; Dien, 2010). For artifact removal, independent components analysis (ICA) was used to remove eye blinks (Dien, 2010). Specifically, if ICA components correlated at .90 or higher with either a template created from previous food go/no-go data collected in in my lab or a template provided by the toolkit, the component was removed (Dien et al., 2010). Data were then re-referenced to an average reference offline and the 200ms window before stimulus onset was used for baseline correction. All analyses are consisted with previous work from my lab examining food-related N2 ERP amplitude (Carbine et al., 2017, 2018).

Given that N2 ERP component amplitude tends to peak around 200–350 ms after stimulus onset (Clayson & Larson, 2013; Folstein & Van Petten, 2008), the N2 ERP component was extracted as the mean amplitude between 200 and 300ms (Carbine et al., 2017). Mean amplitude is a more reliable estimate of ERP measures compared to peak or adaptive mean analyses (Clayson et al., 2013). In addition, N2 ERP data were averaged over four frontocentral electrodes (6, 7, 107, and Cz; see Larson et al., 2011 for electrode montage) in order to improve signal reliability relative to using a single electrode (Clayson & Larson, 2013). Average windows and electrode sites were chosen *a priori* and are consistent with multiple previous studies analyzing the N2 ERP component (e.g., Clawson et al., 2013; Clayson et al., 2011; Clayson & Larson, 2012, 2013), including those examining food-related inhibitory control (Carbine et al., 2017, 2018).

Reliability of the N2 ERP component was assessed using the ERP Reliability Analysis Toolbox v0.4.8 (Clayson & Miller, 2017a). Dependability estimates (a generalizability theory [G-theory] analogue of reliability; see Baldwin et al., 2015 for review and formulas) was calculated for go and no-go trials for each task and each session. The results indicate the number of trials needed in order to have strong estimates of dependability for each ERP estimate. I chose *a priori* that each subject would need enough trials to obtain a dependability estimate of .70 or better to be included (Clayson & Miller, 2017b). ERP data from participants with less than the number of specified trials were not used in analyses and were considered missing data. All participants had reliable data for the baseline high-calorie task, as ERP data from that task was needed to assign participants to their ICT group. For the other tasks and visits, five participants had unreliable data for the baseline neutral task, three for the four-week high-calorie task, four for the neutral four-week task, five for the 12-week high-calorie task, and three for the 12-week

neutral task. Table 2 reports the final dependability estimates, 95% credible intervals, trial cut off values, mean/range values for number of trials, and average noise values from the final sample by task and trial.

**Dutch Eating Behavior Questionnaire**

As ICT may be more effective for restrained eaters (Jones et al., 2016; Lawrence et al., 2015), the DEBQ was administered in order to describe restrained eating tendencies in my sample. The DEBQ is a 33-item questionnaire that assesses restrained eating in addition to external and emotional eating styles (means for restrained eating subscale are reported in Table 3; van Strien et al., 1986). The three-factor structure of the DEBQ has strong internal consistency when used to assess eating habits in men, women, lean individuals, and individuals with obesity (all Cronbach's $\alpha > .79$; Wardle, 1987). As such, the three-factor structure and scoring conventions were used. The external validity of the DEBQ is also strong, as it has accurately identified eating characteristics in women on diets (Wardle, 1987) and predicted weight gain in middle-aged women over a three-year time period (Tucker & Bates, 2009). Internal consistency for average DEBQ scores were excellent in my sample (baseline $\alpha = .91$; four-week $\alpha = .89$; 12-week $\alpha = .92$).

**Power of Food Scale**

As devaluing food stimuli may be a potential outcome of food-specific ICT (Veling et al., 2017), the Power of Food Scale was administered in order to assess the appetitive drive individuals have to consume food. The Power of Food Scale is a 15-item questionnaire that assesses thoughts, feelings, and motivations to consume palatable food in a food-abundant environment (Lowe et al., 2009). The three-factor structure of the Power of Food Scale assesses appetitive drive for food in three environments: when food is available, when food is physically

present, and when food is tasted. The three-factor structure has demonstrated good reliability in

normal-weight individuals and individuals with overweight and obesity from clinical,

undergraduate, and web-based samples (CFIs > .94, all Cronbach's $\alpha$ > .81; Cappelleri et al.,

2009; Lowe et al., 2009). Therefore, the three-factor structure and scoring convention was used.

Individuals with higher Power of Food Scale scores also tend to consume more when chocolate

is present (Forman et al., 2007) and more dessert when full (Levitsky & Shen, 2008), supporting

the validity of the Power of Food Scale as a measure of appetitive drive to consume food. Due to

the high correlation between the three factors, the use of an overall average Power of Food Scale

score to assess appetitive drive is acceptable (see Table 3 for overall means; Lowe et al., 2009).

Internal consistency for average Power of Food Scale scores were also excellent in my sample

(baseline $\alpha$ = .92; four-week $\alpha$ = .91; 12-week $\alpha$ = .92).

**Visual Analog Scales**

To measure hunger levels before the EEG portion, I used six visual analog scales. The six

questions ask about current levels of hunger, fullness (reverse scored), desire to eat, how much

you could eat, urge to eat, and preoccupation with thoughts of food (scale posted here:

https://osf.io/uv6n5/). Each question consists of a 100 mm line, anchored from "Not at all" to

"Extremely" on each end (Blundell et al., 2010; Stratton et al., 1998). Participants were asked to

indicate with a mark where they fell on the scale. The measured distances to the marks on each

of the six scales were then averaged for an overall measure of hunger (means for each visit

reported in Table 3). A review from Stubbs et al. (2007) found VAS measurements reliably and

consistently predict meal initiation, amount eaten, and were sensitive to experimental

manipulations. For example, the correlation between hunger ratings and same hour energy intake

was $r$=.50. However, VAS measurements are best when combined with other measures of eating

behavior. A sleep VAS was also administered, which simply asked participants to indicate their quality of sleep the previous night on a 100mm line, anchored from "Very low" to "Very high".

**ASA24 Dietary Recalls**

The ASA24 is an online automated multiple-pass dietary recall system developed by the National Cancer Institute where participants record their food intake for the previous day (Subar et al., 2012). The ASA24 is based on the U.S. Department of Agriculture multiple pass recall method, where individuals are required to review the food they ate the previous day at multiple points during the recall. As such, the ASA24 asks several questions throughout the recall in order to obtain comprehensive information on the amount, type, and time of food consumption. The ASA24 system also uses pictures of food to ensure the right food and portion size is selected. At a 95% confidence interval, healthy-weight participants using a multiple pass recall system underreport food intake by less than 3% (Moshfegh et al., 2008). The ASA24 itself has been validated against interviewer-administered multiple-pass recalls in terms of accuracy of food recall (Kirkpatrick et al., 2014). Using the ASA24 is beneficial, as the web-based program can be done from multiple locations and the ASA24 itself requires minimal participant burden compared to other methods, like dietary records (Shim et al., 2014). In addition, previous research has found that food intake from averaged daily self-reports, but not a weighed food buffet, was related to N2 ERP component amplitude (Carbine et al., 2018), suggesting neural responses to food cues may have a stronger relationship to food intake measures like the ASA24.

As previously mentioned, participants recorded their food intake at multiple points throughout the study. Specifically, participants were asked to complete three recalls at baseline, three recalls immediately following the four-week long ICT, and three recalls at the 12-week follow-up, for a total of nine recalls. For each period, participants were asked to complete a

recall for the day they came into the lab, and then a randomly assigned weekday (Monday-

Thursday) and randomly assigned weekend (Friday-Saturday; Christensen, 2014), so that all

participants had two weekdays and one weekend for their recall. If a recall was not completed,

the type of day that was missed (weekday or weekend) was randomly assigned to make up for

the missed recall. For all recalls, participants were unaware of what day they are recording until

they were contacted *via* a text message to log in and record the food they ate the previous day.

If a participant's recorded caloric intake was less than their resting metabolic rate, they

were contacted to ensure that the recall was accurate. If inaccurate, participants were assigned to

record another randomized day. Resting metabolic rate is the energy required for an individual to

perform necessary and basic body functions at rest (Sabounchi et al., 2013). The Mifflin-St. Jeor

equation was used to calculate resting metabolic rate (Mifflin et al., 1990). Compared to other

resting metabolic rate equations, the Mifflin-St. Jeor tends to have the lowest error rates

(Frankenfield et al., 2003, 2005), particularly for individuals with overweight and obesity (Weijs,

2008). Average caloric intake was calculated separately for the baseline, four-week post-

intervention, and 12-week time periods. Recalls that were low in calorie count and deemed

inaccurate, and recalls that were recorded more than 14 days after the session date were excluded

from averages. As such, seven participants are missing ASA24 data who completed the second

session, and two participants are missing ASA24 data who completed the third session (ASA24

data for the first session was obtained for all 100 participants). Means and standard deviations for

average caloric intake for each session are presented in Table 3.

**Statistical Analyses**

All statistical analyses were conducted in Stata v.16 and code for all analyses are posted

to OSF: https://osf.io/8xfd3/. All participants were used in the mixed model analyses (as mixed

models allow one to estimate missing data with restricted maximum likelihood [REML]) and participants with complete baseline and four-week visit data were used in regression analyses. Please refer to Figure 1 and the Results section for final sample sizes with each analysis.

To test my first research question whether ICT influences dietary intake and weight, a 2-training group (food-specific, generic) x 3-session (baseline, four-week, 12-week) mixed model was used to analyze changes in average caloric intake and weight. Analyses were similar to Lawrence et al. (2015) who utilized a comparable training group by time model. As a reference, Lawrence et al. found that participants in the food-specific ICT lost about 1.5 lbs (.67 kg) of weight in two weeks and consumed about 220 kcals less during the week-long intervention. Their results are consistent with the Centers for Disease Control and Prevention (CDC) guidelines that recommend losing approximately one pound per week (CDC, 2015) and the AHA/ACC/TOS guidelines that recommend reducing caloric intake by at least 500 kcals per week for healthy weight loss (Jensen et al., 2014). Therefore, in order for my findings to be clinically-meaningful and follow recommended guidelines, I expected at the four-week visit that individuals in the food ICT would show a reduction of approximately four pounds (1.81 kg) in weight and around 500 kcals in food intake, and that these reductions would persist or be larger at the 12-week visit.

For the weight and caloric intake mixed models, I also included three *a priori* follow-up group comparisons in each model: comparing the food-specific ICT to the generic ICT group at baseline, comparing the food-specific ICT to the generic ICT group at four-weeks, and comparing the food-specific ICT to the generic ICT group at 12-weeks. Specifically, I tested if the food-specific ICT group had greater weight loss and consumed fewer calories at the four-week visit and 12-week visit, but didn't show any differences at the baseline visit. Such a pattern

would support my hypotheses that food-specific compared to a generic ICT produced changes in weight and caloric intake immediately after the training and that these changes persist even once training is complete. As there were only three comparisons that were planned *a priori*, I did not conduct a multiple comparisons correction to these analyses.

For my second aim examining how ICT is associated with N2 ERP amplitude, I first conducted a 3-session (baseline, four-week, 12-week) x 2-task (high-calorie, neutral) x 2-trial (go, no-go) mixed model to ensure that my task was successful at eliciting an inhibitory response (i.e., main effect of trial, with no-go trials have more negative N2 ERP amplitude than go trials). As a note for the N2 ERP amplitude, I expected no-go amplitude specifically would change but go amplitude would remain the same, as ICT should be targeting the inhibitory control processes and not the response selection processes. If only the no-go trials change after ICT, it would demonstrate the specificity of ICT in improving inhibitory control processes. For this assumption to be correct, a significant Session x Trial interaction or Session x Task x Trial interaction would need to be observed in the model, showing the specificity of no-go amplitude, but not go amplitude, changing over time and potentially for just the high-calorie task.

For my next mixed model, I calculated the N2 ERP difference amplitude (no-go N2 ERP amplitude minus go N2 ERP amplitude) for each task, so that a larger (i.e., more negative) amplitude reflected a larger inhibitory response. I used a 2-training group (food-specific, generic) x 3-session (baseline, four-week, 12-week) x 2-task (high-calorie, neutral) mixed model to assess how N2 ERP amplitude changed over time due to ICT. As a note, if there was a main effect or significant interaction involving group in this analysis, and a significant interaction involving trial in the above analysis, my plan was to conduct a 2-training group (food-specific, general) x 3-session (baseline, four-week, 12-week) x 2-task (high-calorie, neutral) mixed model on no-go

N2 ERP amplitude only, to see how food-specific and generic ICT differentially affected no-go amplitude over time.

For the N2 ERP difference amplitude mixed model, I also included three follow-up planned group comparisons: comparing N2 ERP difference amplitude between the food-specific and generic ICT groups at baseline, comparing N2 ERP difference amplitude between the food-specific and generic ICT groups at four-weeks, and comparing N2 ERP difference amplitude between the food-specific and generic ICT groups at 12-weeks. These planned comparisons would confirm that there were no significant differences between groups at baseline, that the food-specific ICT group would have a larger N2 ERP difference amplitude at the four-week visit, and that this difference would be maintained at the 12-week visit. Having no group differences at baseline but then significant group differences at the follow-up visits would support my hypotheses that food-specific compared to a generic ICT produced changes in inhibitory control immediately after the training and that these changes persist once training is complete. Again, no multiple comparison corrections were applied to the *a priori* follow-up group comparisons.

I also hypothesized that due to ICT, no-go trial accuracy on the lab session go/no-go tasks would improve and correct go trial reaction times (RTs) would be faster following the ICT. To test these hypotheses, I conducted a 2-training group (food-specific, generic) x 3-session (baseline, four-week, 12-week) x 2-task (high-calorie, neutral) mixed model to assess no-go accuracy on the lab session tasks. For RTs, I conducted a 2-training group (food-specific, generic) x 3-session (baseline, four-week, 12-week) x 2-task (high-calorie, neutral) mixed model on correct go trial RTs. For all mixed model analyses, subject was modeled as a random effect and all other predictor variables were fixed effects. Identity structure for residuals was used in all

models, based off best fit using the Akaike's Information Criteria (AIC) and Bayesian

Information Criteria (BIC). REML was used as the estimation method in all models and the

Satterthwaite approximation was used to estimate degrees of freedom (which takes into account

the structure of the covariance of residuals matrix when estimating degrees of freedom;

Satterhwaite, 1946). Cohen's $f^2$ for multilevel models was used as an estimate of effect size and

calculated in Stata (code posted at https://osf.io/zauvw/), according to the method described by

Selya et al. (2012). For significant interactions, follow-up comparisons without multiple

comparison corrections were used for decomposition. Cohen's $d$ was calculated as an effect size

for follow-up group comparisons and Cohen's $d_z$ was calculated as an effect size for follow-up

within-subject comparisons.

Finally, regression analyses were performed to see how neural indices of inhibitory

control relate to changes in food intake and weight. Baseline high-calorie N2 ERP difference

amplitude, sex (males = 1, females = 0), ICT group (food-specific = 1, generic = 0) and a

Training Group x N2 ERP Amplitude interaction were entered to predict weight loss from the

baseline to the four-week visit (i.e., baseline minus four-week weight). Baseline high-calorie N2

ERP difference amplitude, baseline weight, sex (males = 1, females = 0), ICT group (food-

specific = 1, generic = 0) and a Training Group x N2 ERP Amplitude interaction were entered to

predict reductions in caloric intake from the baseline to the four-week visit (i.e., baseline minus

four-week caloric intake).

As stated in the OSF pre-registration, outliers in regression analyses were identified as

values 1.5 interquartile ranges below the first quartile or above the third quartile and excluded in

order for model assumptions to be met. All final models met basic assumptions for normality of

residuals (assessed by graphs, P-P norm plots, Q-Q norm plots, and Shapiro-Wilk test) and

homoscedasticity of residuals (assessed by graphs, White's test and the Breusch-Pagan test).

Variance inflation factor (VIF) scores are reported as measures of multicollinearity, adjusted $R^2$,

$\Delta R^2$, and Cohen's $f^2$ are reported as measures of effect sizes, and unstandardized $b$-weights are

reported. Only participants who had weight, caloric intake, and high-calorie N2 ERP amplitude

data for the first and second session were included in the regression analyses.

The mixed model analyses allowed me to use all participants who have observed data,

even if the participant did not complete all lab sessions (i.e., came in for baseline and four-week

visit, but not the 12-week visit). White et al. (2012) suggest that for mixed model analyses,

analyzing all participants with any observed data is acceptable if the missing at random

assumption is met. The missing at random assumption was assessed using two *chi*-square

analyses (one with sex and one with ICT group) and three point-biserial correlations (one with

weight, one with age, and one with no-go N2 ERP amplitude on the high-calorie task), to see if

there is a relationship between the sex, ICT assignment, weight, age, or baseline food-related

inhibitory control of participants and participants who dropped out of the study or had unreliable

(i.e., missing) N2 ERP data.

**Sensitivity Analyses**

To determine how robust the effect of ICT was, I subsequently conducted two sensitivity

analyses. The first was an intent-to-treat sensitivity analysis where I used the last observation

carried forward (LOCF) method to account for participants with missing four-week and 12-week

data. Analyses were repeated as above to see how departure from full study participation affected

results. Although average percent compliance on the ICTs for both groups was high (food-

specific *M*[*SD*]: 86.25% [.17]; generic: 83.72% [.21]; overall: 85.01% [.19]), a second sensitivity

analysis was conducted on only participants who on average completed three out of the four

weekly ICT sessions (75%) in order to see if adherence to ICT changed the overall pattern of results. Notably, both of these sensitivity analyses were determined *a priori* and are presented along with initial findings so as not to increase Type I error, but to ensure that the initial analyses are robust to the inevitable dropout and missing data that occurs in intervention research (Thabane et al., 2013).

**Exploratory Analyses**

Since ICT may influence individuals to devalue food stimuli (Veling et al., 2017), I also conducted planned exploratory analyses examining how a food-specific and generic ICT affected appetitive drive to consume food, as measured by the Power of Food Scale (Cappelleri et al., 2009; Lowe et al., 2009). I first conducted a 2-training group (food-specific, generic) x 3-session (baseline, four-week, 12-week) mixed model to analyze changes in overall Power of Food Scale score. In the mixed model analysis, subject was modeled as a random effect and all other predictor variables were fixed effects. Identity structure for residuals was used, REML was used as the estimation method, Satterthwaite approximation was used to estimate degrees of freedom, and Cohen's $f^2$ for multilevel models was used as an estimate of effect size. I also included three planned comparisons in the model: comparing the food-specific ICT to the generic ICT group at baseline, comparing the food-specific ICT to the generic ICT group at four-weeks, and comparing the food-specific ICT to the generic ICT group at 12-weeks. Seeing no differences between groups at baseline but a lower appetitive drive in the food-specific ICT at the four-week and 12-week visit would support the idea that ICT may help individuals to devalue food.

Finally, I conducted a multiple linear regression model, with baseline high-calorie N2 ERP difference amplitude, sex (males = 1, females = 0), ICT group (food-specific = 1, generic = 0) and a Training Group x N2 ERP Amplitude interaction predicting changes in appetitive drive

from the baseline to the four-week visit (i.e., baseline minus four-week Power of Food Scale total score). For regression analyses, as before, IQR was used to identify potential outliers and the final model met basic assumptions for homoscedasticity and normality of residuals. VIF scores are reported as measures of multicollinearity, adjusted $R^2$, $\Delta R^2$, and Cohen's $f^2$ are reported as measures of effect sizes, and unstandardized $b$-weights are reported.

**Deviations from OSF Pre-Registration**

Five changes were made to the current study that differ from the pre-registration (https://osf.io/39wv6). Changes were made prior to data analyses and were done to improve methods and analyses. First, participants with a BMI over 40 kg/m$^2$ (who were originally excluded) were recruited for study participation in order to make the sample more representative of individuals with overweight and obesity in today's population (Flegal et al., 2016). Such a change aligns with ICT research conducted exclusively in individuals with overweight and obesity (Forman et al., 2019). Second, an additional point-biserial correlation to test the missing at random assumption was added to make sure missing data did not correlate with baseline food-related inhibitory control responses, as measured by the high-calorie N2 ERP no-go amplitude. Third, as mixed models do not produce partial-eta squared effect sizes, Cohen's $f^2$ for multilevel models was used instead as a measure of effect size for the mixed model analyses.

The fourth and fifth changes involve changes to the sensitivity analyses. Originally for the LOCF analyses, participants with missing four-week and 12-week data were excluded, and the four-week visit data was carried forward for missing 12-week data only. However, intent to treat analyses are typically implemented so that all participants who are missing data or who dropped from the study are included in the analyses (Gupta, 2011), not just those with only one timepoint of missing data. Therefore, the goal of the intent to treat analyses was not met by

excluding individuals missing both four-week and 12-week data. Research also suggests that the intent to treat LOCF method (i.e., carrying baseline observations forward for multiple visits) and LOCF method (i.e., requiring participants have a baseline observation and one other data point) produce similar results (Elobeid et al., 2009). Finally, for the 75% adherence training, original analyses were only going to include individuals who had 75% adherence rates and completed all sessions. However, given mixed models can estimate missing data and regression analyses only depended on baseline and four-week data, the 75% adherence analyses now include individuals who completed at least 75% of the data and had at least baseline and four-week visit data.

## Results

### Assumptions of Randomness of Missing Data

*Chi*-square results revealed that missing data from participants who discontinued participation in the study or had missing/unreliable N2 ERP amplitude data did not significantly differ by biological sex ($\chi^2(1) = 0.70$; $p = .40$) or ICT group assignment ($\chi^2(1) = 0.08$; $p = .77$). Point-biserial correlations indicated that missing or unreliable N2 ERP component amplitude data was not related to baseline weight ($r_{pb} = -.004$; $p > .05$), age in years ($r_{pb} = -.19$; $p > .05$), or baseline food-related inhibitory control, as measured by baseline no-go amplitude on the high-calorie task ($r_{pb} = .14$; $p > .05$). These analyses suggest that the missing at random assumption to use mixed models is met for my data.

### First Hypothesis: Changes in Weight and Caloric Intake

#### *Mixed Model Predicting Weight Changes*

Means and standard deviations for participant weight as a function of group across the three sessions are reported in Table 1. The main effect of training group ($F[1, 98.03] < 0.001$; $p = .98$; $f^2 = .03$), main effect of session ($F[2, 167.24] = 2.85$; $p = .06$; $f^2 = .06$), and Training

Group x Session interaction ($F$[2, 167.24] = 2.25; $p$ = .11; $f^2$ = .03) were non-significant, suggesting that ICT, regardless of group, did not produce changes in weight immediately after the training or 12-weeks after completion. Table 4 contains the results of the *a priori* follow-up group comparisons, which similarly suggested that weight did not differ between ICT training groups at any of the sessions ($p$s > .82). In addition to not being statistically significant, results were not clinically meaningful, as the difference in weight between the baseline and four-week visit, baseline and 12-week visit, and four-week and 12-week visit were all less than 1.81 kg (see Table 1; largest difference was approximately 0.6 kgs).

*Mixed Model Predicting Caloric Intake Changes*

Means and standard deviations for caloric intake as a function of group across the three sessions are reported in Table 3. Similar to the weight analyses, the main effect of training group ($F$[1, 97.36] = 0.22; $p$ = .64; $f^2$ = .01), main effect of session, ($F$[2, 165.89] = 2.16; $p$ = .12; $f^2$ = .03), and Training Group x Session interaction ($F$[2, 165.89] = 0.47; $p$ = .63; $f^2$ = .01) were all non-significant. Results suggest that ICT, regardless if it is food-specific or generic, does not reduce average daily caloric intake over a time period of four to 12 weeks. Table 4 contains the results of the *a priori* follow-up group comparisons, which similarly suggested that average daily caloric intake did not differ between ICT training groups at any of the sessions ($p$s > .50). In addition to not being statistically significant, results were not clinically meaningful, as the difference in caloric intake between the baseline and four-week visit, baseline and 12-week visit, and four-week and 12-week visit were all less than 500 kcals (see Table 3; largest difference was approximately 150 kcals).

**Second Hypothesis: Changes in N2 ERP Amplitude and Behavioral Data**

*Mixed Model with Trial Predicting N2 ERP Amplitude Changes*

Waveforms for the N2 ERP amplitude by group, session, task, and trial are presented in

Figure 4. Scalp distributions for the no-go N2 ERP amplitude by group, session, and task are

presented in Figures 5 - 7. Means and standard deviations for N2 ERP amplitudes as a function

of group across the three sessions are reported in Table 5. The 3-session (baseline, four-week,

12-week) x 2-task (high-calorie, neutral) x 2-trial (go, no-go) model revealed a main effect of

trial ($F[1, 156] = 10.46$; $p = .001$; $f^2 = .18$), with no-go trials eliciting a larger (i.e., more

negative) N2 ERP amplitude than go trials, as expected. There was also a main effect of task

($F[1, 417.81] = 45.26$; $p < .001$; $f^2 = .15$), with the high-calorie task overall having larger (i.e.,

more negative) amplitudes than the neutral task.

For interactions, there was a significant Task by Trial interaction ($F[1, 516] = 68.05$; $p <$

$.001$; $f^2 = .15$). A follow-up pairwise comparison revealed that the difference between go and

no-go trials was larger on the high-calorie task than neutral task ($t[99] = 9.15$; $p < .001$; $d_z =$

$0.80$). Finally, there was also a significant Session x Task x Trial interaction ($F[2, 516] = 3.61$; $p$

$= .03$; $f^2 = .01$). Follow-up pairwise comparisons revealed that on the high-calorie task, the go

amplitude became more negative from the baseline to four-week visit ($t[86] = 2.38$; $p = .02$; $d_z =$

$0.21$), but this was not the case for no-go amplitude ($t[86] = 0.86$; $p = .39$; $d_z = 0.09$), and this

pattern did not exist for the neutral task ($ts[86] < 1.68$; $ps > .09$).

### Mixed Model with Group Predicting N2 ERP Amplitude Changes

Results of the 2-training group (food-specific, generic) x 3-session (baseline, four-week,

12-week) x 2-task (high-calorie, neutral) mixed model are presented in Table 6. Briefly, results

revealed a main effect of task, with the N2 ERP difference amplitude being larger (i.e., more

negative) on the high-calorie than neutral task, as also evidenced in the previous mixed model.

There was also a significant Task by Session interaction. Follow-up pairwise comparisons

revealed that while the N2 ERP difference amplitude on the high-calorie task did not change

over time ($ts[86] < 1.88$; $ps > .06$), the N2 ERP difference amplitude on the neutral task got

larger (i.e., more negative) from the baseline to the 12-week visit ($t[86] = 2.35$; $p = .02$; $d_z =$

0.15). No other main effects or interactions were significant ($ps > .50$). Table 4 contains the

results of the *a priori* follow-up group comparisons, which similarly revealed that N2 ERP

difference amplitude did not differ between ICT training groups at any of the sessions ($ps > .27$).

### Mixed Model Predicting No-Go Trial Accuracy

Means and standard deviations for no-go accuracy as a function of group across the three

sessions are reported in Table 7. Results of the 2-training group (food-specific, generic) x 3-

session (baseline, four-week, 12-week) x 2-task (high-calorie, neutral) mixed model are

presented in Table 6. In sum, while all main effects and two-way interactions were not

significant, there was a significant Training Group x Task x Session interaction ($F[2, 265] =$

3.96; $p = .02$; $f^2 = .03$). Follow-up comparisons revealed that while ICT groups did not differ on

the neutral task ($ps > .05$), individuals in the food-specific ICT group were more accurate than

the generic ICT group on the high-calorie task during the four-week visit ($t[85] = 2.11$; $p = .04$; $d$

$= 0.43$), but this difference did not exist during the baseline ($t[98] = 0.02$; $p = .86$; $d = 0.04$) or

12-week visit ($t[82] = -0.15$; $p = .88$; $d = 0.01$). Results suggest that after ICT, individuals in a

food-specific compared to generic ICT group may be more accurate at withholding dominant

responses to high-calorie food cues, but this difference does not persist at the 12-week follow-up.

### Mixed Model Predicting Correct Go Trial RTs

Results of the 2-training group (food-specific, generic) x 3-session (baseline, four-week,

12-week) x 2-task (high-calorie, neutral) mixed model are presented in Table 6. Means and

standard deviations for RTs across the three sessions are reported in Table 7. The only significant

effect was a main effect of task, which showed that individuals overall were faster on responding to office and tool items on the neutral task compared to the low-calorie food images on the high-calorie task, suggesting there was increased attention to the food compared to neutral images (Carbine et al., 2017).

**Third Hypothesis: Relationship between Baseline Inhibitory Control, Weight, and Caloric Intake**

*Regression Analysis Predicting Weight Change*

Eighty-four participants had high-calorie N2 ERP difference amplitude and weight data for both the baseline and four-week visit. Two weight difference scores (IQRs > 1.85) and two baseline high-calorie N2 ERP difference amplitudes (IQRs > 2.19) were identified as outliers and removed from analyses. All normality and homoscedasticity assumptions were met after outliers were removed. The final sample size for the regression analyses predicting changes in weight was 80 (food-specific ICT $n = 42$; generic ICT $n = 38$). Results of the regression analysis are presented in Table 8. The only significant effect was a Training Group x N2 ERP Amplitude interaction (*b*-weight = -0.90; $p = .03$). Results suggest that while individuals with *lower* baseline inhibition levels lost more weight in the generic ICT group, individuals with *higher* baseline inhibition levels lost more weight in the food-specific ICT group. Marginal means show that in the generic ICT group, individuals with lower inhibition levels lost approximately 1.07 kgs of more weight than those with higher inhibition levels. In the food-specific ICT group, individuals with higher inhibition levels lost approximately 1.00 kgs of more weight than those with lower inhibition levels. Thus, while statistically significant, these changes in weight were not clinically meaningful (i.e., they were less than a 1.81 kg weight change).

*Regression Analysis Predicting Caloric Intake Change*

Seventy-six participants had high-calorie N2 ERP difference amplitude and average caloric intake data for both the baseline and four-week visit. One calorie intake difference score (IQR = 1.61), one baseline weight score (IQR = 3.61) and two baseline high-calorie N2 ERP difference amplitudes (IQRs > 2.27) were identified as outliers and removed from analyses. All normality and homoscedasticity assumptions were met after outliers were removed. The final sample size for the regression predicting changes in caloric intake was 72 (food-specific ICT $n$ = 36; generic ICT $n$ = 36). Results of the regression analysis are presented in Table 8. Baseline high-calorie N2 ERP difference amplitude was a significant predictor of changes in caloric intake ($b$-weight = 187.82; $p$ = .045; see Figure 8), with smaller (i.e., more positive) N2 ERP difference amplitude at baseline predicting the greatest reduction in average daily caloric intake between the two sessions. There was also a Training Group x N2 ERP amplitude interaction ($b$-weight = -264.21; $p$ = .048). Results suggest that while individuals with *lower* baseline inhibition levels had the greatest caloric reductions in the generic ICT group, individuals with *higher* baseline inhibition levels had the greatest caloric reductions in the food-specific ICT group. Marginal means show that in the generic ICT group, individuals with lower inhibition levels reduced their caloric intake by approximately 240.05 kcals more than those with higher inhibition levels. In the food-specific ICT group, individuals with higher inhibition levels reduced their caloric intake by approximately 230.78 kcals more than those with lower inhibition levels. Thus, while statistically significant, these changes in weight were not clinically meaningful (i.e., they were less than a 500 kcal change). All other effects were non-significant ($p$s > .10).

**Sensitivity Analyses**

Final numbers for all sensitivity analyses (i.e., carrying the last observation forward for missing data and only using individuals who completed at least 75% of the ICTs) are presented in Figure 1. Tables (as referred to below) present the results for sensitivity analyses. For clarity, only results that changed are presented below.

*First Hypothesis: Changes in Weight and Caloric Intake*

Please see Tables 9-12. When using LOCF, the main effect of session in the weight mixed model analyses became statistically significant ($p = .04$; see Table 9). Follow-up pairwise comparisons revealed that there was an increase in weight from the baseline to 12-week visit (0.67 kgs; $t[99] = 2.11$; $p = .04$; $d_z = 0.20$) and from the four-week visit to the 12-week visit (0.75 kgs; $t[99] = 2.38$; $p = .02$; $d_z = 0.21$).

*Second Hypothesis: Changes in N2 ERP Amplitude and Behavioral Data*

Please see Tables 13-16. When using LOCF, the Session by Task by Trial interaction on N2 ERP amplitude trial data was no longer statistically significant ($p = .20$; see Table 13). For the N2 ERP difference amplitude mixed model, the Session by Task interaction was no longer statistically significant ($p = .10$; see Table 14). For accuracy results, the Group by Task interaction became significant ($p = .04$; see Table 14). Follow-up comparisons were not significant ($t$s[98] < 1.91; $p$s > .06), but the pattern of results suggest that the food-specific ICT group was more accurate than the generic ICT group on the neutral task but not high-calorie task.

When only including individuals who completed at least 75% of the trainings, the Session by Task by Trial interaction on N2 ERP amplitude trial data was no longer statistically significant ($p = .13$; see Table 15). For the N2 ERP difference amplitude mixed model, the

Session by Task interaction was no longer significant ($p = .09$; see Table 16). For accuracy, the

Group by Session by Task interaction was no longer significant ($p = .06$; see Table 16).

### *Third Hypothesis: Relationship Between Baseline Inhibitory Control, Weight, and Caloric Intake*

Please see Tables 17-18. When using LOCF, the Training Group x N2 ERP Amplitude

interaction predicting weight change was no longer statistically significant ($p = .052$; see Table

17). When only including individuals who completed at least 75% of the trainings, the Training

Group x N2 ERP Amplitude interaction predicting weight change was no longer statistically

significant ($p = .06$; see Table 18). For the regression analysis predicting caloric intake change,

the effect of baseline high-calorie N2 ERP difference amplitude ($p = .11$) and the Training Group

x N2 ERP Amplitude interaction ($p = .09$) were no longer statistically significant when only

including individuals who completed at least 75% of the trainings.

### Exploratory Analyses

### *Mixed Model Predicting Power of Food Scale*

Means and standard deviations for Power of Food Scale scores as a function of group

across the three sessions are reported in Table 3. Similar to the caloric intake analyses, the main

effect of training group was not significant ($F[1, 98.08] = 3.17$; $p = .08$; $f^2 = .01$); however, there

was a main effect of session ($F[2, 169.92] = 7.38$; $p < .001$; $f^2 = .09$). Follow-up pairwise

comparisons revealed that there was a decrease in Power of Food Scale scores from the baseline

to four-week visit ($t[86] = 2.38$; $p = .02$; $d_z = 0.27$) and 12-week visit ($t[83] = 3.79$; $p < .001$; $d_z = 0.34$). The Training Group x Session interaction was not significant ($F[2, 169.92] = 0.35$; $p = .71$; $f^2 = .01$). Results suggest that, regardless of training type (generic or food-specific), ICT

may help individuals reduce their appetitive drive towards food during the trainings and after

when the trainings are not currently being completed.

### Regression Analysis Predicting Power of Food Scale Change

Eighty-four participants had high-calorie N2 ERP difference amplitude and Power of

Food Scale data for both the baseline and four-week visit. Four Power of Food Scale difference

scores were identified as outliers (|IQRs| > 2.56) and one baseline N2 ERP difference amplitude

(IQR = 2.50) were identified as outliers and removed from analyses. After dropping outliers, all

assumptions for the linear regression were met. The final sample size for the regression analysis

predicting Power of Food Scale change was 79. Results of the regression analysis are presented

in Table 8. The only significant results was the effect of baseline N2 ERP difference amplitude

during the high-calorie go/no-go task was significant ($b$-weight = -0.20; $p$ = .002; see Figure 9),

with individuals who had a larger high-calorie N2 ERP amplitude at baseline showing the

greatest reduction in appetitive drive between the two sessions.

### Discussion

The main goals of my dissertation were to test if a food-specific compared to generic

ICT, completed four times per week over four weeks in individuals with overweight and obesity,

was associated with changes in weight, caloric intake, and neural indices of inhibitory control, as

measured by the N2 ERP component. I hypothesized that from baseline to the four-week and 12-

week visits, individuals in the food-specific compared to generic ICT would exhibit increased

weight loss, greater reductions in caloric intake, and larger N2 ERP difference amplitudes to

food-cues. I also aimed to test if baseline levels of food-related inhibitory control related to

changes in weight and caloric intake after ICT. I hypothesized that individuals with a lower

baseline food-specific inhibitory response (i.e., more positive N2 ERP difference amplitude) would benefit the most from ICT and therefore show greater reductions in weight and caloric.

My first hypothesis testing changes in weight and caloric intake following ICT was not supported. Individuals in the food-specific ICT compared to generic ICT group did not lose more weight or have greater reductions in caloric intake immediately after four-weeks of trainings or at the 12-week follow-up. Weight results are inconsistent with a handful of studies that found decreases in weight after ICT (Lawrence et al., 2015; Preuss et al., 2017; Stice et al., 2017; Veling et al., 2014), but are consistent with Forman et al. (2019), who did not observe a change in weight. Studies that showed an effect on weight loss typically had participants complete four ICTs over the course of a week or a month, and the length of those trainings varied from 10 to 50 minutes (Lawrence et al., 2015; Stice et al., 2017; Veling et al., 2014). Participants in studies where the individuals lost weight also varied from having individuals across the BMI spectrum (Lawrence et al., 2015; Veling et al., 2014), to only including individuals with overweight and obesity (Stice et al., 2017), to including individuals with BED (Preuss et al., 2017). In the studies that did not show an effect on weight (Forman et al., 2019 and the current study), participants completed more sessions of ICT (16 to 44 10-minute long ICTs) over a longer time course of four to eight weeks and only included psychiatrically-healthy individuals with overweight or obesity.

Taking all the studies together, it may be that a reduction in weight is seen after only a few ICT sessions (e.g., Lawrence et al., 2015; Stice et al., 2017; Veling et al., 2014), but those effects do not persist during a longer, consistent ICT intervention with more sessions, particularly in a sample of only individuals with overweight or obesity (e.g., Forman et al., 2019 and the current study). Follow-up results from Stice et al. (2017) support this idea, as the

reduction in body fat percentage did not persist at a six-month follow-up. In addition, ICT may be a feasible and beneficial intervention for clinical populations that are characterized by impulsive eating (Preuss et al., 2017; Schag et al., 2013). However, the many differences in intervention length, frequency of ICT implementation, and participant characteristics and BMI across ICT studies conducted to date make it difficult to know if there is a dose-response relationship between ICT and weight loss, what the most effective dose is, and who that dose is most effective for.

My findings that individuals in the food-specific ICT group did not have a greater reduction in caloric intake than those in the generic ICT group are consistent with some published results (e.g., Adams et al., 2017; Poppelaars et al., 2018; Turton et al., 2018), but differ from the majority of published findings in the food ICT literature (e.g., Adams et al., 2017; Chen et al., 2019; Forman et al., 2016; Houben & Jansen, 2011, 2015; van Koningsbruggen et al., 2014; Veling et al., 2013). However, as noted by Allom et al. (2015) and Carbine and Larson (2019), these studies generally consist of a single laboratory session or only last one week and do not test if ICT can promote more long-term caloric intake reduction, like the current study.

One reason I may have not observed a difference in caloric intake by group was due to the duration of the study and the 12-week follow-up. Perhaps there is a short-term effect of ICT (e.g., Adams et al., 2017; Chen et al., 2019; Forman et al., 2016; Houben & Jansen, 2011, 2015; van Koningsbruggen et al., 2014; Veling et al., 2013), which participants habituate to with longer-term training (e.g., the current study). Since increased ICT practice should be associated with more weight loss or less caloric intake if inhibitory control processes are, in fact, being improved, the habituation of ICT effects support the concept that neural circuits and inhibitory control processes are not being "trained" or enhanced with ICT (Veling et al., 2017). A second

reason for the differences between the published literature and the current results that did not find a change in caloric intake by group may be due my use of an active control condition. Some studies have used as their comparison training a task that has individuals respond to (rather than inhibit toward) high-calorie or palatable food images (e.g., Houben & Jansen, 2011, 2015), potentially increasing impulsivity in the comparison group as opposed to increasing inhibitory control in the experimental group (Adams et al., 2017). Since my generic ICT did not use food stimuli and required participants to respond and withhold responses at the same rate as those in the food-specific ICT, food-related impulsivity would not have been induced in the generic ICT group, which ensured that significant results would have been due to changes in food-specific inhibitory control.

Interestingly, the regression analysis predicting changes in weight showed that individuals with lower inhibition responses to high-calorie foods at baseline lost more weight than those with higher-inhibition responses to high-calorie foods at baseline. Adding to this result was a significant Training Group by N2 ERP Amplitude interaction, which was also observed in the regression analysis predicting changes in caloric intake. These results suggest that for both the reductions in weight and caloric intake, individuals with *lower* baseline inhibitory responses (i.e., more positive N2 ERP difference amplitudes) saw the greatest reductions in the *generic* ICT group, while individuals with *higher* baseline inhibitory control responses (i.e., more negative N2 ERP difference amplitudes) saw the greatest reductions in the *food-specific* ICT group.

As ICT may be more effective for individuals with lower levels of inhibitory control (Forman et al., 2019; Houben, 2011), it is possible that individuals with a lower baseline inhibitory response profit from even just a generic ICT, as they would benefit from an increase in

inhibitory control functioning overall and not just specifically towards food stimuli, and as such see decreases in weight and caloric intake. Individuals with a higher baseline inhibitory response, on the other hand, may need an ICT that specifically targets food-related inhibitory control in order to see any improvements in caloric intake or weight, as their overall inhibitory control functioning may be at a ceiling. The interaction also helps explain why no effects involving ICT group were significant in the mixed model analyses, as reductions depended on baseline levels of inhibition, and my ICT groups had similar proportions of individuals with higher and lower levels of inhibitory control due to using baseline N2 ERP amplitude to help assign individuals to an ICT group.

When looking at marginal means, the changes in weight from the baseline to four-week visit between individuals with lower- and higher-inhibition responses was approximately 1.07 kgs in the generic ICT group and approximately 1.00 kgs in the food-specific ICT group. The changes in caloric intake from the baseline to four-week visit between individuals with lower and higher inhibition responses was approximately 240.05 kcals for the generic ICT group and approximately 230.78 kcals for the food-specific ICT group. These changes in weight and caloric intake do not meet the clinically meaningful reductions of approximately 500 calories per day (CDC, 2015) and 1.81 kgs over four-weeks (Jensen et al., 2014). Therefore, while ICT may significantly reduce food intake and weight in laboratory-based interventions under some conditions (e.g., Houben & Jansen, 2011, 2015; Lawrence et al., 2015; current study), it remains unclear if ICT can produce meaningful reductions in weight and caloric intake in clinical settings beyond a four-week period. It is also important to note that the significant interactions did not hold in the sensitivity analyses. As such, replication is needed to confirm that results are not spurious.

Current results suggest that ICT does not affect N2 ERP measurements of inhibitory control processes. While there was a larger N2 ERP difference amplitude when inhibiting towards high-calorie food images compared to neutral images (which replicates previous findings that high-calorie foods necessitate an increased inhibitory response; Carbine et al., 2017, 2018; Watson & Garvey, 2013), neither N2 ERP component no-go amplitude nor N2 ERP difference amplitude on the high-calorie task changed during the course of the study. In fact, it seemed that N2 ERP component difference amplitude during the neutral task showed the most change overtime (although these results did not hold in sensitivity analyses). My results align with other studies that suggest the N2 ERP component amplitude does not change due to ICT (Aulbach et al., 2020; Blackburne et al., 2016) and expand on previous findings by showing that neither a single ICT session (Aulbach et al., 2020) nor multiple ICT sessions over the course of four weeks have an effect on food-related N2 ERP amplitude. Behavioral data also did not change over time due to ICT. While individuals were slower when responding to food images compared to neutral image (paralleling previous results that individuals were slower at responding to high-calorie compared to low-calorie images; Carbine et al., 2017), RTs did not significantly change from baseline to four-week to 12-week visits. Individuals in the food-specific compared to generic ICT group were more accurate at withholding responses to food images at the four-week visit, but this change did not persist at the 12-week visit and did not hold in the 75% adherence sensitivity analyses. Taking the behavioral and N2 ERP component amplitude results together, it seems my measurements of inhibitory control did not change over the course of the study due to ICT.

Results in the literature are mixed as whether inhibitory control improves due to ICT (Forman et al., 2019) or does not change due to ICT (Ooomen et al., 2018). How inhibitory

control was measured varied between studies, from assessing error rates (Ooomen et al., 2018), to reaction times (Forman et al., 2019), to psychophysiological indices of inhibitory control in addition to behavioral data (current study). The time between the baseline visit and when changes in inhibitory control were measured also differed from six days (Ooomen et al., 2018), to three weeks (Forman et al., 2019) to four weeks (current study). The heterogeneity of the literature makes is difficult to know if inhibitory control processes are affected, to what extent they are affected, and how long those changes last. As it stands, it may be that shorter interventions (i.e., one week) are not long enough to see changes in inhibitory control, but any changes that do occur in inhibitory control do not last beyond a three- to four-week period.

While inhibitory control processes in my study did not change due to ICT, the Power of Food Scale scores in participants did appear to change due to ICT. The Power of Food Scale, which measures the appetitive drive towards food, decreased over the course of the trainings, regardless of ICT group, and the decrease in Power of Food Scale scores persisted at the 12-week visit. The finding that Power of Food Scale decreased due to ICT while neural and behavioral measures of inhibitory control did not change aligns with other research suggesting that ICT alters the rewarding value of food as opposed to inhibition processes per se (Houben & Giesen, 2018; Veling et al., 2017). fMRI results also found that a week-long ICT intervention decreased attentional and reward responses to food stimuli (Stice et al., 2017). The reduction in reward value of food following ICT may be beneficial, as it would make inhibiting to hedonic foods easier by decreasing the conflict one experiences when having to inhibit towards foods that are appetizing (De Pretto et al., 2019). This particular aspect of ICT could be very beneficial in improving adherence to weight-loss diets (Jensen et al., 2014). Specifically, low calorie diets, with a low proportion of calories coming from fat, seem to be effective in reducing weight (Klem

et al., 1997) but have low adherence rates (Laddu et al., 2011). If ICT could help individuals decrease the rewarding value of foods that are high in fat and sugar, those foods may become less of a temptation to eat while on a diet, which could then help improve adherence, weight loss, and weight loss maintenance.

Interestingly, regression analyses in the current study showed that individuals with a higher baseline food-related inhibitory response (i.e., more negative N2 ERP difference amplitude) had the greatest decrease in appetitive drive towards food, as measured by the Power of Food Scale. While increased N2 ERP amplitude is thought to be indicative of a larger inhibitory response, it may be that the function of the N2 ERP component specifically is to signal that increased inhibitory control resources are needed to override a prepotent response (Folstein & Van Petten, 2008). In order to appropriately respond to the heighted recruitment signal, individuals with a larger N2 ERP difference amplitude to high-calorie foods may reduce the rewarding or appetitive value of food to a greater extent. The reduction in appetitive value would then help decrease the conflicting response that the N2 ERP component amplitude was reflecting- having to inhibit towards foods that are rewarding and desirable (De Pretto et al., 2019).

It is important to note that weight loss is multifactorial, as there are many variables that need to be considered when an individual is trying to lose weight. For example, what is the current overall health of the individual? Do they have comorbid chronic diseases, or what does their metabolic health look like (Kramer et al., 2013)? How are their social relationships, and do they have individuals who will support them on their weight-loss journey (Christakis & Fowler, 2007)? What does their current diet look like and are there special dietary needs that need to be considered (Matarese & Pories, 2014; Schwartz, 2016)? What lifestyle habits does the individual

have? How much sleep are they getting and how are their physical activity levels (Chastin et al., 2015; Alamuddin & Wadden, 2016)? Food-related inhibitory control is only one aspect of many that may help improve weight loss efforts. As previously stated, current results suggest that ICT does not produce clinically-meaningful changes in caloric intake and weight. Pairing ICT with another intervention as an adjuvant that addresses one of the many other factors that affects weight loss may prove to be the most beneficial and result in successful and clinically meaningful reductions in caloric intake and weight. Future studies specifically testing ICT as an adjuvant instead of primary treatment are, therefore, needed.

Further, the current regression results showing that ICT was effective depending on baseline levels of inhibitory control and the type of ICT administered lend to the idea that different weight-loss interventions may be more effective for different people (i.e., personalized or "precision" treatment options). A copious amount of research has tried to understand what lifestyle factors and behaviors, health habits, characteristics, personalities, and mental health traits are related to successful weight-loss and weight-loss maintenance after behavioral and surgical weight-loss interventions (e.g., Bond et al., 2009; Coleman et al., 2010; Fuglestad et al., 2012; Klem, 2000; Klem et al., 2000; Rafiei & Gill, 2018; Robinson et al., 2014; Soini et al., 2015; Sullivan et al., 2007). It is important to assess both the characteristics of individuals who are successful at weight loss, and whether certain interventions work better for certain individuals (i.e., are personalized). Interventions that are able to adapt and adjust to an individual participant's needs, circumstances, and characteristics may be more beneficial for weight loss and maintenance (Stead et al., 2015). Clinicians could then use information on what interventions work best for different people in order to better guide their patients to an intervention that may be more effective for that particular patient. Further, steps could be taken

to screen individuals on certain characteristics (for example, food-related inhibitory control

levels) that may help clinicians and individuals know what interventions might lead to the

greatest success in reducing weight and caloric intake. The current study itself shows that it is

feasible to obtain baseline measures on neural indices of inhibitory control, which could then

help indicate if a food-specific or generic ICT intervention would be effective in reducing caloric

intake and weight for an individual.

As with any study, there were weaknesses in the current project. Self-reported dietary

recalls, such as the ASA24, tend to underestimate total food intake, particularly for individuals

with overweight and obesity (Macdiarmid & Blundell, 1997), and are less reliable than other

methods for assessing food intake, such as weighing food (Nydahl et al., 2009). It is possible the

results with caloric intake would have been stronger, weaker, or not present if a different or more

reliable measure of food intake was used. However, the ASA24 dietary recalls were chosen

because data from ASA24 dietary recalls have previously been shown to relate to N2 ERP

amplitude (Carbine et al., 2017, 2018) when other measures of food intake, such as an *ad libitum*

buffet, have not (Carbine et al., 2018). As another weakness, individuals in the study were asked

to maintain their current exercise levels and to not participate in a weight loss diet during the

course of the study. However, no follow-up questions were administered to ensure these

guidelines were followed.

In addition, while individuals were told that the ICTs were aimed at improving their

ability to withhold dominant responses to food, they were not given practical information on how

the trainings would apply to their daily food choices (e.g., when they have strong desires to eat

high-calorie foods, these trainings may help manage those cravings or urges). While this was

intentionally done so that results would reflect on if ICT directly affects inhibitory control and

therefore weight and caloric intake outcomes, further instruction and training on the role of

inhibitory control and how to implement ICT in everyday situations could improve the success of

ICTs (Lawrence et al., 2015). Finally, this study did not have a separate arm that implemented a

weight-loss diet (e.g., a calorie allotment diet). We were not able to draw any conclusions on

how ICT may improve adherence to, or generally influence, weight-loss diets or how ICT could

be used as an adjuvant treatment, which could be one of the major benefits of cognitive

interventions (Kelley et al., 2016; Laddu et al., 2011).

The current study also had many strengths. First, adherence to the trainings was excellent

(overall average = 85%). These findings not only show that ICT is a very feasible intervention to

implement on mobile devices outside of laboratory settings (Lawrence et al., 2015), but gives

confidence that observed changes in weight, caloric intake, or inhibitory control outcomes would

have been due to the ICT intervention. Second, my project is one of the largest, well-powered

studies on ICT and weight/caloric intake to date that consistently implemented ICTs over a time

period longer than a week. Average sample sizes in previous ICT studies were approximately 56

participants total (about 28 participants per group; Carbine & Larson, 2019), while the current

study had 100 participants (approximately 50 participants per group) in mixed model analyses

and 72 (36 participants per group) in the smallest regression analysis. Such projects are needed to

clarify if ICTs have an effect on caloric intake and weight outcomes, given the low power and

small effect sizes seen in the literature (Carbine & Larson, 2017). Third, as previously

mentioned, the use of an active control group allowed me to ensure that results were not due to a

placebo or training effect (Boot et al., 2013) or eliciting impulsivity in my control group (Adams

et al., 2017). Along those lines, my two groups were very well matched in terms of age, sex,

education, and BMI (see Table 1), which ensures that findings were not due to confounding

participant characteristics. Finally, collecting N2 ERP amplitude data both during a food and generic go/no-go task was a strength, as it provided specificity in my study design to test if food-specific and not just general inhibitory control changed due to ICT. Measuring the N2 ERP component also strengthened ICT group assignment, as it ensured equal proportions of individuals with low and high inhibition responses were in each arm of the study and did not confound study results.

There are multiple future directions and studies to be conducted on food-specific ICT in order to understand how ICT can be an effective weight-loss intervention in a clinical setting. First, as noted above, ICT needs to be examined as an adjuvant intervention with other successful weight-loss interventions, such as a calorie-restricted diet. Using ICT as an adjuvant would allow researchers and clinicians to assess if the reduction in food reward value that ICT offers helps individuals increase adherence to a diet, which would promote additional benefits in weight loss and caloric intake reductions. Second, although improvements have been made to test the long-term feasibility and utility of ICT (Forman et al., 2019 and the current study), long-term ICT studies that span multiple months are needed. Research suggests that in diet-based interventions, maximal weight-loss is observed at six months from intervention onset (Jensen et al., 2014). Therefore, having an ICT study that spans the course of six months would help researchers and clinicians know where ICT stands compared to more traditional weight-loss and other cognitive interventions that have been assessed at a six-month period (Laddu et al., 2017; McLean et al., 2015). Having multiple testing points throughout the six-month period would also be beneficial in order to see if and when changes in caloric intake, weight, and inhibitory control occur due to ICT. Finally, if ICT does in fact change attention and appetitive drive towards food, studies that measure the underlying neural processes associated with attention and motivation

may help clarify what cognitive processes ICT targets. In the ERP field, two ERP components known as the P3 and the late positive potential are functionally suggested to serve as indicators of attention and, specifically, attention that is driven by emotions or motivation (Hajcak et al., 2010; Schupp et al., 2006). Examining how these ERP components change over the course of an ICT intervention may help elucidate what cognitive mechanisms ICT targets and why changes in the N2 ERP component are not being observed from baseline ICT to post-ICT intervention.

To summarize and conclude, current findings indicate that multiple ICT sessions, whether food-specific or generic, over the course of a four-week period do not affect overall weight loss, caloric intake, or recruitment of inhibitory control resources. These findings suggest that ICT may not directly affect inhibitory control functions and associated health outcomes. A generic ICT may be more effective for individuals with lower levels of inhibitory control while a food-specific ICT may be more effective for individuals with higher levels of inhibitory control, although the effects may not be large enough to assist with clinically meaningful caloric reductions and weight loss. However, ICT decreases appetitive drive towards food, even after trainings are complete. The decrease in appetitive drive may lend to ICT being an effective adjuvant intervention for weight-loss diets where adherence is low, and such a possibility should be tested with future research, in addition to testing how effect ICT is at a six-month period.

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**Table 1**

*Participant Demographics by Group*

| | Food-Specific ICT $n = 52$ | Generic ICT $n = 48$ | Total $n = 100$ |
|---|---|---|---|
| Age: Mean (SD) | 28.50 (8.13) | 27.56 (6.95) | 28.05 (7.56) |
| Sex: Number of females (%) | 27 (52%) | 26 (54%) | 53 (53%) |
| Ethnicity: Number of Caucasian (%) | 44 (85%) | 37 (77%) | 81 (81.0%) |
| Ethnicity: Number of Hispanic (%) | 5 (10%) | 9 (19%) | 14 (14%) |
| Ethnicity: Number of Other (%) | 2 (4%) | 3 (6%) | 5 (5%) |
| Education (years): Mean (SD) | 16.04 (2.93) | 16.02 (2.28) | 16.03 (2.63) |
| Baseline BMI (kg/m$^2$): Mean (SD) | 32.27 (5.69) | 32.71 (5.03) | 32.48 (5.36) |
| Baseline weight (kg): Mean (SD)[a] | 96.37 (21.34) | 95.50 (15.42) | 95.95 (18.65) |
| Four-Week weight (kg): Mean (SD)[a] | 96.82 (22.55) | 95.07 (14.95) | 95.97 (18.57) |
| 12-Week weight (kg): Mean (SD) | 96.32 (22.04) | 95.65 (14.52) | 96.00 (18.64) |

*Note.* ICT = inhibitory control training. For education, 12 = completed high school. SD= standard deviation

[a]These averages contain outliers that were removed in regression analyses

**Table 2**

*N2 ERP Component Dependability Estimates*

| Trial Type | Dependability | 95% C.I. | Trial Cut Off | Mean (SD) Trials | Trial Range | Noise Values |
|---|---|---|---|---|---|---|
| Baseline: High-calorie go | 0.96 | (0.95, 0.97) | 12 | 121.47 (21.12) | 29 - 139 | 0.74 |
| Baseline: High-calorie no-go | 0.92 | (0.90, 0.94) | 10 | 47.07 (9.38) | 13 - 60 | 1.17 |
| Baseline: Neutral go | 0.97 | (0.96, 0.98) | 9 | 123.71 (16.92) | 43 - 140 | 0.67 |
| Baseline: Neutral no-go | 0.92 | (0.90, 0.94) | 10 | 47.51 (7.81) | 17 - 59 | 1.09 |
| Four-Week: High-calorie go | 0.97 | (0.97, 0.98) | 8 | 122.93 (18.35) | 50 - 140 | 0.66 |
| Four-Week: High-calorie no-go | 0.94 | (0.92, 0.96) | 8 | 49.50 (8.15) | 17 - 60 | 1.09 |
| Four-Week: Neutral go | 0.98 | (0.97, 0.98) | 7 | 123.25 (18.55) | 64 - 140 | 0.68 |
| Four-Week: Neutral no-go | 0.93 | (0.91, 0.95) | 8 | 47.05 (8.95) | 20 - 59 | 1.11 |
| 12-Week: High-calorie go | 0.97 | (0.96, 0.98) | 9 | 118.51 (26.95) | 20 - 140 | 0.76 |
| 12-Week: High-calorie no-go | 0.93 | (0.90, 0.95) | 9 | 46.59 (10.46) | 9 - 59 | 1.31 |
| 12-Week: Neutral go | 0.98 | (0.97, 0.98) | 7 | 122.28 (19.14) | 26 - 140 | 0.69 |
| 12-Week: Neutral no-go | 0.93 | (0.91, 0.95) | 9 | 46.99 (9.48) | 10 - 59 | 1.14 |

*Note*. CI = credible interval.

**Table 3**

*Participant Questionnaire and Dietary Data by Group*

| | Food-Specific ICT | Generic ICT | Total |
|---|---|---|---|
| | Mean (SD) | Mean (SD) | Mean (SD) |
| Food VAS (cm): Baseline | 5.79 (1.71) | 6.00 (1.42) | 5.89 (1.56) |
| Food VAS (cm): Four-Week | 5.65 (1.80) | 5.96 (1.55) | 5.80 (1.68) |
| Food VAS (cm): 12-Week | 5.91 (1.81) | 5.76 (1.76) | 5.84 (0.60) |
| Sleep VAS (cm): Baseline | 6.50 (1.89) | 6.50 (1.87) | 6.50 (1.78) |
| Sleep VAS (cm): Four-Week | 6.88 (1.82) | 6.60 (1.53) | 6.74 (1.68) |
| Sleep VAS (cm): 12-Week | 6.80 (1.77) | 6.03 (2.15) | 6.43 (1.99) |
| Restrained eating (DEBQ): Baseline | 2.78 (0.63) | 2.60 (0.44) | 2.69 (0.55) |
| Restrained eating (DEBQ): Four-Week | 2.76 (0.69) | 2.78 (0.54) | 2.77 (0.62) |
| Restrained eating (DEBQ): 12-Week | 2.80 (0.64) | 2.75 (0.55) | 2.78 (0.60) |
| Power of Food Scale average: Baseline | 3.06 (0.87) | 3.28 (0.84) | 3.17 (0.86) |
| Power of Food Scale average: Four-Week | 2.93 (0.84) | 3.14 (0.72) | 3.03 (0.79) |
| Power of Food Scale average: 12-Week | 2.84 (0.89) | 3.08 (0.73) | 2.96 (0.82) |
| Caloric intake (kcals): Baseline | 2,377.94 (719.93) | 2,285.92 (709.58) | 2,333.77 (712.87) |
| Caloric intake (kcals): Four-Week | 2,220.81 (656.31) | 2,231.40 (591.19) | 2,226.10 (620.66) |
| Caloric intake (kcals): 12-Week | 2,309.23 (794.88) | 2,121.00 (631.92) | 2,220.03 (701.35) |

*Note.* SD= standard deviation. VAS = Visual Analog Scale. cm = centimeters. DEBQ = Dutch Eating

Behavior Questionnaire.

**Table 4**

*A Priori Group Comparisons*

|  | Baseline Visit Food vs Generic ICT | Four-Week Visit Food vs Generic ICT | 12-Week Visit Food vs Generic ICT |
|---|---|---|---|
| Weight: | | | |
| *t*-statistic (*p*-value) | 0.22 (.82) | 0.04 (.97) | 0.19 (.85) |
| Caloric intake: | | | |
| *t*-statistic (*p*-value) | 0.67 (.51) | -0.17 (.87) | 0.68 (.50) |
| N2 ERP amplitude: | | | |
| *t*-statistic (*p*-value) | 0.29 (.78) | 0.04 (.97) | 1.10 (.27) |

*Note*. ICT = inhibitory control training.

**Table 5**

*Participant N2 ERP Amplitude Data by Group*

| | Food-Specific ICT | Generic ICT | Total |
|---|---|---|---|
| | Mean (SD) | Mean (SD) | Mean (SD) |
| High-calorie go (µv): Baseline | -1.82 (2.22) | -1.88 (2.16) | -1.85 (2.18) |
| High-calorie go (µv): Four-week | -2.43 (2.64) | -2.38 (2.12) | -2.40 (2.39) |
| High-calorie go (µv): 12-Week | -2.50 (2.40) | -2.13 (2.33) | -2.32 (2.36s) |
| High-calorie no-go (µv): Baseline | -2.65 (2.23) | -2.54 (2.67) | -2.60 (2.44) |
| High-calorie no-go (µv): Four-Week | -2.85 (2.71) | -2.85 (2.38) | -2.85 (2.54) |
| High-calorie no-go (µv): 12-Week | -3.14 (2.64) | -2.49 (2.69) | -2.84 (2.67) |
| High-calorie difference (µv): Baseline | -0.83 (1.13) | -0.66 (1.15) | -0.75 (1.14) |
| High-calorie difference (µv): Four-Week | -0.37 (1.05) | -0.51 (0.96) | -0.44 (1.00) |
| High-calorie difference (µv): 12-Week | -1.74 (2.69) | -1.85 (2.15) | -1.79 (2.43) |
| Neutral go (µv): Baseline | -1.47 (2.38) | -1.73 (2.14) | -1.60 (2.26) |
| Neutral go (µv): Four-week | -1.78 (2.73) | -2.04 (2.27) | -1.91 (2.51) |
| Neutral go (µv): 12-Week | 0.40 (1.13) | 0.30 (1.29) | 0.11 (1.21) |
| Neutral no-go (µv): Baseline | -1.05 (2.44) | -1.37 (2.48) | -1.21 (2.45) |
| Neutral no-go (µv): Four-Week | -1.60 (2.58) | -1.62 (2.38) | -1.61 (2.26) |
| Neutral no-go (µv): 12-Week | -1.66 (2.75) | -1.71 (2.09) | -1.68 (2.44) |
| Neutral difference (µv): Baseline | 0.45 (1.21) | 0.41 (1.06) | 0.43 (1.13) |
| Neutral difference (µv): Four-Week | 0.20 (1.11) | 0.29 (1.22) | 0.24 (1.15) |
| Neutral difference (µv): 12-Week | 0.02 (1.22) | 0.14 (1.15) | 0.07 (1.18) |

*Note*. SD= standard deviation. µv = microvolt. Difference = no-go minus go.

**Table 6**

*N2 ERP Amplitude and Behavioral Data Mixed Models*

|  | N2 ERP Amplitude | No-Go Trial Accuracy | Correct Go Trial RT |
|---|---|---|---|
|  | $F(f^2)$ | $F(f^2)$ | $F(f^2)$ |
| Main effect: Task | 82.78 (.23)*** | 0.27 (.05) | 6.89 (.05)** |
| Main effect: Session | 0.42 (.03) | 1.63 (.04) | 0.59 (.02) |
| Main effect: ICT group | 0.46 (.01) | 2.02 (.04) | 0.64 (.02) |
| Task x Session | 4.48 (.02)** | 0.80 (.04) | 0.32 (.02) |
| Task x Group | 0.07 (.003) | 2.69 (.04) | 0.92 (.02) |
| Session x Group | 0.44 (.01) | 0.49 (.03) | 0.13 (.01) |
| Group x Session x Task | 0.67 (.003) | 3.96 (.03)* | 1.83 (.01) |

*Note*. RT = reaction time. ICT = inhibitory control training. $f^2$ = Cohen's $f^2$ effect size.

* $p < .05$. ** $p < .01$. *** $p < .001$.

**Table 7**

*Participant Behavioral Data by Group*

|  | Food-Specific ICT | Generic ICT | Total |
|---|---|---|---|
|  | Mean (SD) | Mean (SD) | Mean (SD) |
| High-calorie no-go accuracy (%): Baseline | 0.89 (0.07) | 0.89 (0.09) | 0.89 (0.08) |
| High-calorie no-go accuracy (%): Four-Week | 0.90 (0.08) | 0.86 (0.12) | 0.88 (0.10) |
| High-calorie no-go accuracy (%): 12-Week | 0.90 (0.08) | 0.89 (0.09) | 0.89 (0.09) |
| High-calorie correct go RT (ms): Baseline | 420.94 (61.77) | 424.08 (47.80) | 422.45 (55.25) |
| High-calorie correct go RT (ms): Four-Week | 419.05 (46.86) | 424.32 (52.77) | 421.59 (49.58) |
| High-calorie correct go RT (ms): 12-Week | 418.31 (40.53) | 421.06 (57.81) | 418.65 (49.44) |
| Neutral no-go accuracy (%): Baseline | 0.90 (0.08) | 0.86 (0.09) | 0.88 (0.09) |
| Neutral no-go accuracy (%): Four-Week | 0.89 (0.07) | 0.87 (0.09) | 0.88 (0.08) |
| Neutral no-go accuracy (%): 12-Week | 0.91 (0.06) | 0.87 (0.09) | 0.89 (0.09) |
| Neutral correct go RT (ms): Baseline | 409.44 (51.93) | 423.08 (47.53) | 415.99 (50.08) |
| Neutral correct go RT (ms): Four-Week | 417.01 (54.56) | 416.67 (54.95) | 416.85 (54.43) |
| Neutral correct go RT (ms): 12-Week | 412.83 (44.35) | 420.87 (65.84) | 416.75 (55.68) |

*Note*. SD= standard deviation. % = percent. RT = reaction time. ms = milliseconds.

**Table 8**

*Linear Regressions Predicting Weight, Calorie, and Power of Food Scale Change*

| | $b$ | $t$ | VIF | $\Delta R^2$ | $F$ | df | Adj. $R^2$ | Cohen's $f^2$ |
|---|---|---|---|---|---|---|---|---|
| Model predicting changes in weight | | | | | 3.74** | 4, 75 | 0.12 | 0.14 |
| Sex | -0.35 | -0.90 | 1.00 | .00 | | | | |
| Training group | 0.56 | 1.16 | 1.49 | .00 | | | | |
| Baseline high-calorie N2 difference amplitude | 0.50 | 1.78 | 2.03 | .04 | | | | |
| N2 ERP by training group interaction | -0.90 | -2.27* | 2.62 | .05 | | | | |
| Model predicting changes in caloric intake | | | | | 1.64 | 5, 66 | 0.04 | 0.04 |
| Sex | 88.57 | 0.62 | 1.25 | .00 | | | | |
| Baseline weight | -7.43 | -1.67 | 1.28 | .03 | | | | |
| Training group | -93.38 | -0.59 | 1.57 | .04 | | | | |
| Baseline high-calorie N2 difference amplitude | 187.82 | 2.04* | 2.00 | .04 | | | | |
| N2 ERP by training group interaction | -264.21 | -2.01* | 2.72 | .04 | | | | |
| Model predicting changes in Power of Food Scale | | | | | 3.84** | 4, 74 | 0.13 | 0.15 |
| Sex | -0.12 | -1.39 | 1.01 | .01 | | | | |
| Training group | -0.01 | -0.12 | 1.48 | .00 | | | | |
| Baseline high-calorie N2 difference amplitude | -0.20 | -3.18** | 2.16 | .13 | | | | |
| N2 ERP by training group interaction | 0.09 | 1.08 | 2.77 | .01 | | | | |

*Note.* Difference = no-go minus go.

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

**Table 9**

*Weight and Caloric Intake LOCF Mixed Models*

|  | Weight (kgs) | Calorie (kcal) |
|---|---|---|
|  | $F(f^2)$ | $F(f^2)$ |
| Main effect: Session | 3.39 (.06)* | 2.68 (.03) |
| Main effect: ICT group | 0.00 (.03) | 0.14 (.004) |
| Session x Group | 2.52 (.03) | 0.35 (.003) |

*Note*. LOCF = last observation carried forward. ICT = inhibitory control training.

$f^2$ = Cohen's $f^2$ effect size.

* $p < .05$.

**Table 10**

*A Priori Group Comparisons for LOCF Mixed Models*

|  | Baseline Visit Food vs Generic ICT | Four-Week Visit Food vs Generic ICT | 12-Week Visit Food vs Generic ICT |
|---|---|---|---|
| Weight: | | | |
| *t*-statistic (*p*-value) | 0.22 (.82) | 0.06 (.96) | -0.15 (.88) |
| Caloric intake: | | | |
| *t*-statistic (*p*-value) | 0.67 (.51) | -0.05 (.96) | 0.35 (.73) |
| N2 ERP amplitude: | | | |
| *t*-statistic (*p*-value) | -0.26 (.79) | -0.33 (.74) | -1.23 (.22) |

*Note*. ICT = inhibitory control training.

**Table 11**

*Weight and Caloric Intake 75% Adherence Mixed Models*

|  | Weight (kgs) | Calorie (kcal) |
|---|---|---|
|  | $F(f^2)$ | $F(f^2)$ |
| Main effect: Session | 2.94 (.06) | 1.87 (.04) |
| Main effect: ICT group | 0.11 (.02) | 1.76 (.02) |
| Session x Group | 1.26 (.02) | 0.93 (.01) |

*Note.* % = percent. ICT = inhibitory control training. $f^2$ = Cohen's $f^2$ effect size.

* p < .05.

**Table 12**

*A Priori Group Comparisons for 75% Adherence Mixed Models*

|  | Baseline Visit<br>Food vs Generic ICT | Four-Week Visit<br>Food vs Generic ICT | 12-Week Visit<br>Food vs Generic ICT |
|---|---|---|---|
| Weight: | | | |
| *t*-statistic (*p*-value) | 0.46 (.65) | 0.36 (.72) | 0.17 (.7) |
| Caloric intake: | | | |
| *t*-statistic (*p*-value) | 1.82 (.07) | 0.48 (.63) | 1.01 (.31) |
| N2 ERP amplitude: | | | |
| *t*-statistic (*p*-value) | -0.30 (.76) | 0.14 (.89) | -0.87 (.39) |

*Note*. ICT = inhibitory control training.

**Table 13**

*N2 ERP Amplitude Trial Data LOCF Mixed Model*

| | N2 ERP Amplitude |
|---|---|
| | $F(f^2)$ |
| Main effect: Trial | 14.09 (.21)*** |
| Main effect: Task | 57.32 (.18)*** |
| Main effect: Session | 1.89 (.01) |
| Trial x Task | 105.97 (.18)*** |
| Trial x Session | 0.26 (.01) |
| Task x Session | 0.21 (.01) |
| Session x Task x Trial | 1.59 (.01) |

*Note*. LOCF = last observation carried forward. ICT = inhibitory control training. $f^2$ = Cohen's $f^2$ effect size.

* p < .05. ** p < .01. *** p < .001.

**Table 14**

*N2 ERP Amplitude and Behavioral Data LOCF Mixed Models*

|  | N2 ERP Amplitude | No-Go Trial Accuracy | Correct Go Trial RT |
|---|---|---|---|
|  | $F(f^2)$ | $F(f^2)$ | $F(f^2)$ |
| Main effect: Task | 132.01 (.27)*** | 0.19 (.04) | 10.03 (.05)*** |
| Main effect: Session | 0.26 (.02) | 1.69 (.03) | 0.66 (.02) |
| Main effect: ICT group | 0.67 (.01) | 1.93 (.04) | 0.70 (.02) |
| Task x Session | 2.32 (.01) | 0.82 (.03) | 0.21 (.02) |
| Task x Group | 0.03 (.003) | 4.19 (.04*) | 0.98 (.02) |
| Session x Group | 0.44 (.01) | 0.41 (.02) | 0.05 (.01) |
| Group x Session x Task | 0.82 (.003) | 3.42 (.02)* | 1.64 (.01) |

*Note*. LOCF = last observation carried forward. ICT = inhibitory control training. $f^2$ = Cohen's $f^2$ effect size.

* p < .05. ** p < .01. ***p < .001.

**Table 15**

*N2 ERP Amplitude Trial Data 75% Adherence Mixed Model*

|  | N2 ERP Amplitude |
|---|---|
|  | $F(f^2)$ |
| Main effect: Trial | 16.15 (.18)*** |
| Main effect: Task | 36.10 (.14)*** |
| Main effect: Session | 1.96 (.01) |
| Trial x Task | 51.47 (.14)*** |
| Trial x Session | 0.50 (.01) |
| Task x Session | 0.05 (.01) |
| Session x Task x Trial | 2.03 (.01) |

*Note.* % = percent. ICT = inhibitory control training. $f^2$ = Cohen's $f^2$ effect size.

\* p < .05. \*\* p < .01. \*\*\**p* < .001.

**Table 16**

*N2 ERP Amplitude and Behavioral Data 75% Adherence Mixed Models*

|  | N2 ERP Amplitude | No-Go Trial Accuracy | Correct Go Trial RT |
|---|---|---|---|
|  | $F(f^2)$ | $F(f^2)$ | $F(f^2)$ |
| Main effect: Task | 63.67 (.24)*** | 0.68 (.05) | 5.96 (.06)* |
| Main effect: Session | 0.47 (.03) | 1.48 (.03) | 0.88 (.02) |
| Main effect: ICT group | 0.23 (.02) | 1.36 (.04) | 0.06 (.03) |
| Task x Session | 2.42 (.04) | 0.95 (.03) | 0.43 (.02) |
| Task x Group | 1.76 (.02) | 2.56 (.04) | 2.38 (.03) |
| Session x Group | 0.36 (.01) | 0.59 (.02) | 0.45 (.02) |
| Group x Session x Task | 1.42 (.01) | 3.78 (.01) | 2.06 (.02) |

*Note.* % = percent. ICT = inhibitory control training. $f^2$ = Cohen's $f^2$ effect size.

* p < .05. ** p < .01. *** p < .001.

**Table 17**

*LOCF Linear Regressions Predicting Weight and Calorie Change*

| | $b$ | $t$ | VIF | $\Delta R^2$ | $F$ | df | Adj. $R^2$ | Cohen's $f^2$ |
|---|---|---|---|---|---|---|---|---|
| Model predicting changes in weight | | | | | 2.42 | 4, 89 | 0.06 | 0.06 |
| Sex | -0.16 | -0.50 | 1.01 | .00 | | | | |
| Training group | 0.24 | 0.58 | 1.63 | .06 | | | | |
| Baseline high-calorie N2 difference amplitude | 0.27 | 1.20 | 2.01 | .02 | | | | |
| N2 ERP by training group interaction | -0.62 | -1.97 | 2.77 | .03 | | | | |
| Model predicting changes in caloric intake | | | | | 1.72 | 5, 86 | 0.04 | 0.04 |
| Sex | 137.75 | 1.44 | 1.29 | .01 | | | | |
| Baseline weight | -3.38 | -1.17 | 1.30 | .01 | | | | |
| Training group | -71.81 | -0.66 | 1.69 | .04 | | | | |
| Baseline high-calorie N2 difference amplitude | 142.02 | 2.41* | 2.07 | .04 | | | | |
| N2 ERP by training group interaction | -184.21 | -2.23* | 2.87 | .04 | | | | |

*Note.* LOCF = last observation carried forward. Difference = no-go minus go.

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

**Table 18**

*75% Adherence Linear Regressions Predicting Weight and Calorie Change*

| | $b$ | $t$ | VIF | $\Delta R^2$ | $F$ | df | Adj. $R^2$ | Cohen's $f^2$ |
|---|---|---|---|---|---|---|---|---|
| Model predicting changes in weight | | | | | 2.17 | 4, 63 | 0.07 | 0.08 |
| Sex | 0.20 | 0.51 | 1.00 | .00 | | | | |
| Training group | 0.30 | 0.60 | 1.56 | .07 | | | | |
| Baseline high-calorie N2 difference amplitude | 0.42 | 1.42 | 2.04 | .03 | | | | |
| N2 ERP by training group interaction | -0.81 | -1.93 | 2.78 | .04 | | | | |
| Model predicting changes in caloric intake | | | | | 1.44 | 5, 56 | 0.03 | 0.03 |
| Sex | 97.40 | 0.65 | 1.25 | .00 | | | | |
| Baseline weight | -7.09 | -1.52 | 1.26 | .02 | | | | |
| Training group | -7.00 | -0.04 | 1.54 | .03 | | | | |
| Baseline high-calorie N2 difference amplitude | 171.11 | 1.64 | 2.25 | .02 | | | | |
| N2 ERP by training group interaction | -247.40 | -1.74 | 2.99 | .03 | | | | |

*Note.* % = percent.

* $p < .05$.

**Figure 1**

*Overview of Participant Recruitment and Analyses*
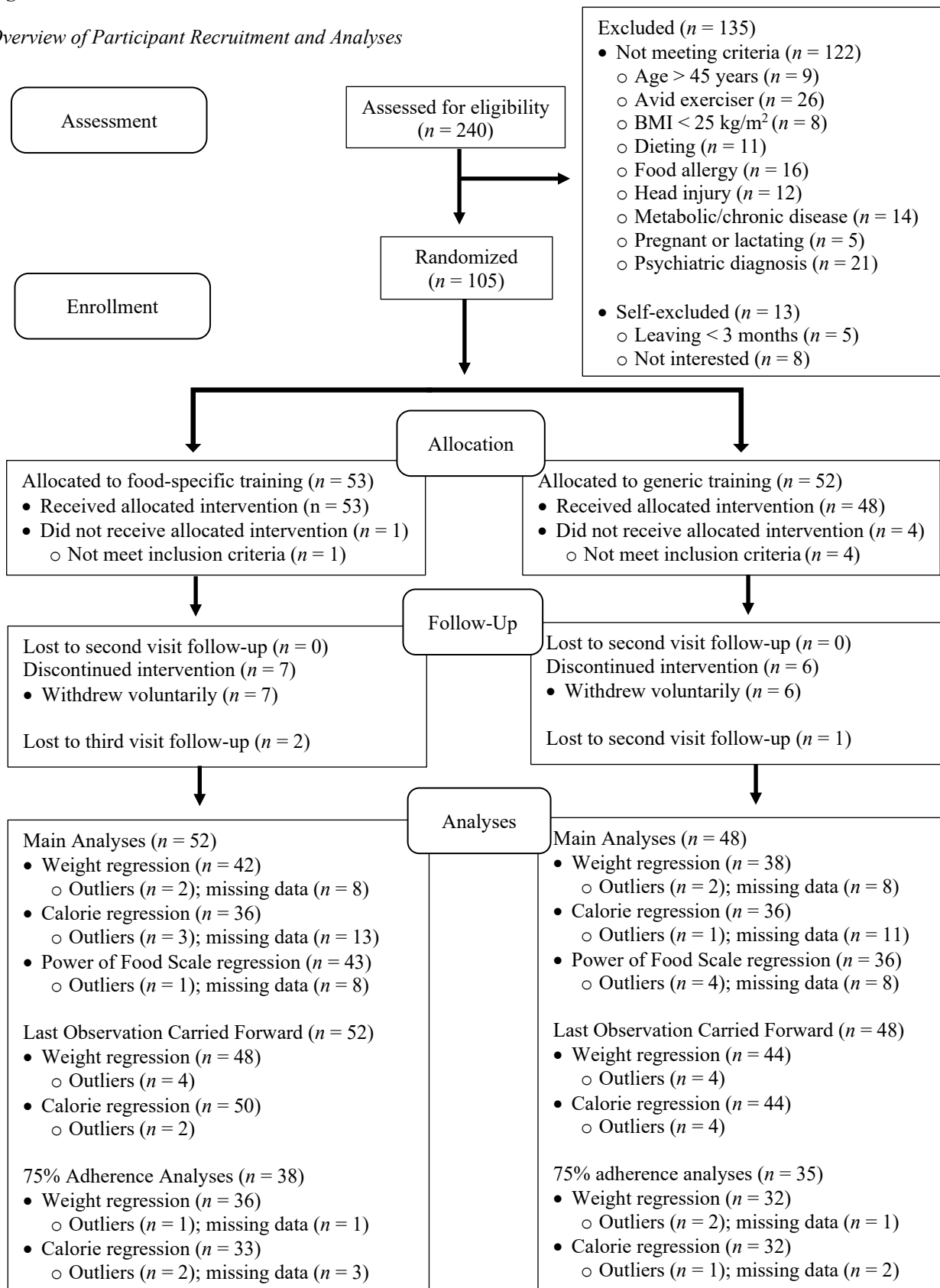
**Figure 2**

*Overview of Experimental Protocol*

```
┌─────────────────────────────────────────────────────┐
│                  Baseline Visit:                     │
│                                                       │
│                   -Consent Form                       │
│     -Demographics, DEBQ, and Power of Food Scale      │
│            -Height and Weight Measurements            │
│                 -Food and Sleep VAS                   │
│      -EEG Protocol: High-Calorie and Neutral Tasks    │
│                 -Three ASA24 recalls                  │
└─────────────────────────────────────────────────────┘
                          ↓
┌─────────────────────────────────────────────────────┐
│              Inhibitory Control Training:             │
│                                                       │
│    -Assignment to food-specific or generic training   │
│                   -Four-weeks long                    │
│             -One task a day, four days a week         │
└─────────────────────────────────────────────────────┘
                          ↓
┌─────────────────────────────────────────────────────┐
│                  Four-Week Visit:                     │
│                                                       │
│             -DEBQ and Power of Food Scale             │
│            -Height and Weight Measurements            │
│                 -Food and Sleep VAS                   │
│      -EEG Protocol: High-Calorie and Neutral Tasks    │
│                 -Three ASA24 recalls                  │
└─────────────────────────────────────────────────────┘
                          ↓
┌─────────────────────────────────────────────────────┐
│               12-Week Waiting Period:                 │
│                                                       │
│                   -No trainings                       │
│                  -No dietary recalls                  │
└─────────────────────────────────────────────────────┘
                          ↓
┌─────────────────────────────────────────────────────┐
│                  12-Week Visit:                       │
│                                                       │
│             -DEBQ and Power of Food Scale             │
│            -Height and Weight Measurements            │
│                 -Food and Sleep VAS                   │
│      -EEG Protocol: High-Calorie and Neutral Tasks    │
│                 -Three ASA24 recalls                  │
└─────────────────────────────────────────────────────┘
```

*Note.* DEBQ = Dutch Eating Behavior Questionnaire. VAS = visual analog scale. EEG = electroencephalography. ASA24 = Automated self-administer 24-hour dietary recall.

**Figure 3**

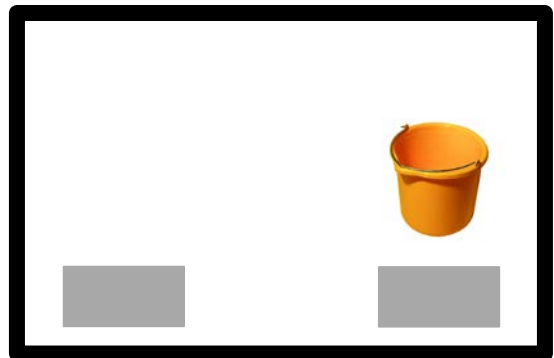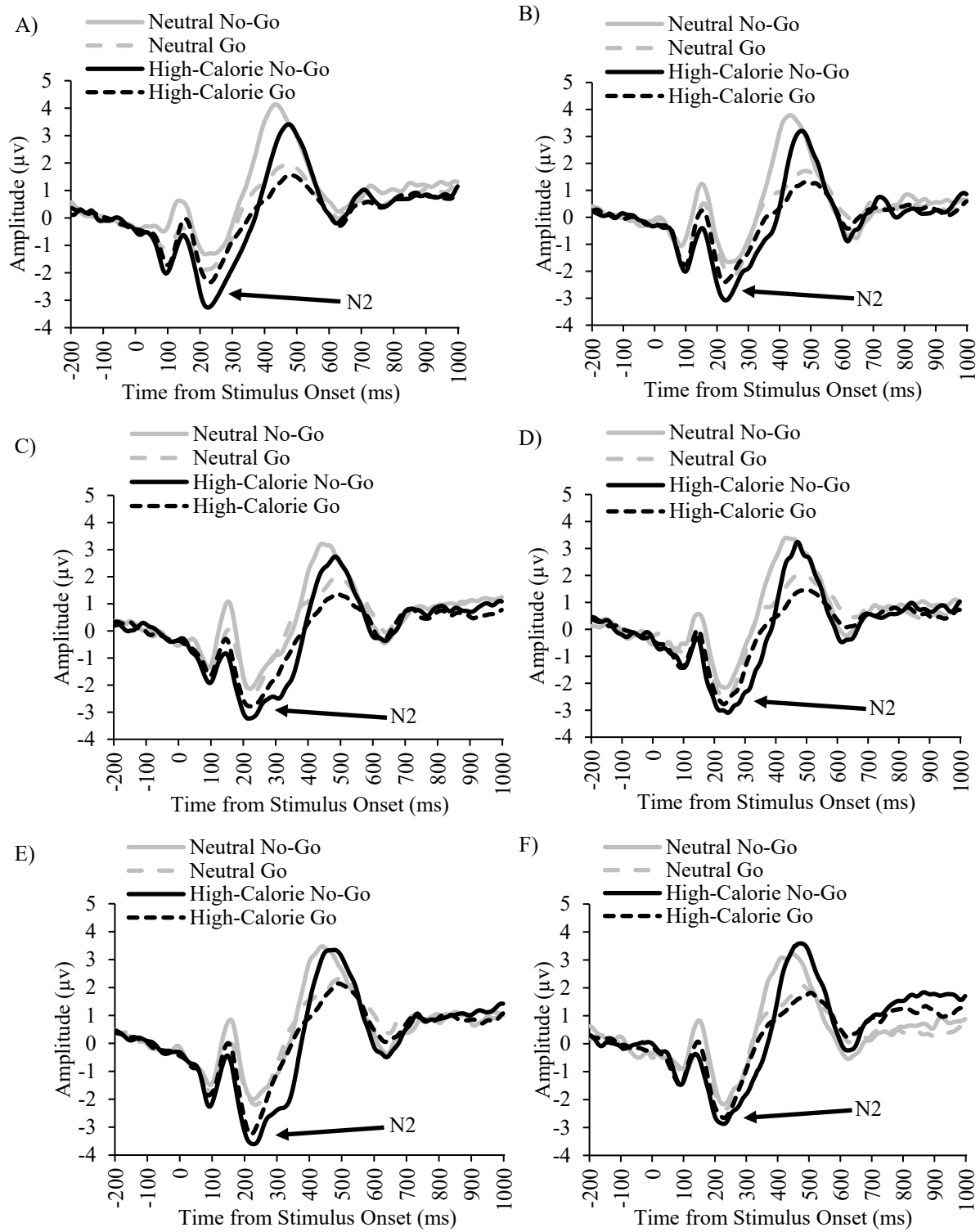*Examples of Stimuli for ICT Tasks*

A)



B)



C)



D)



*Note.* A) Food-specific go trial. B) Food-specific no-go trial. C) Generic go-trial. D) Generic no-go trial.
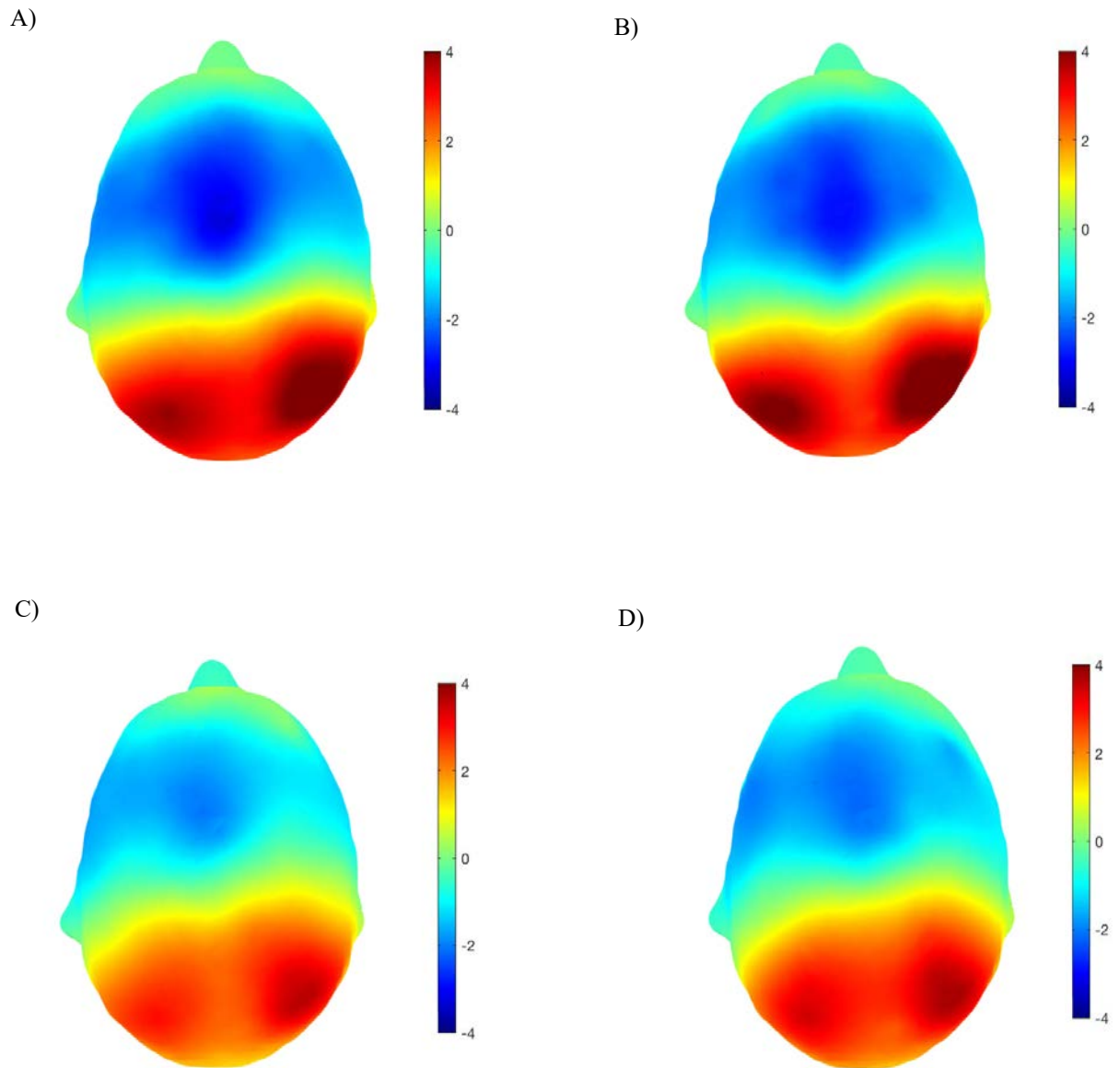
**Figure 4**

*N2 ERP Waveforms by Group and Session*



*Note.* A) Food-specific ICT baseline visit. B) Generic ICT baseline visit. C) Food-specific ICT four-week visit.

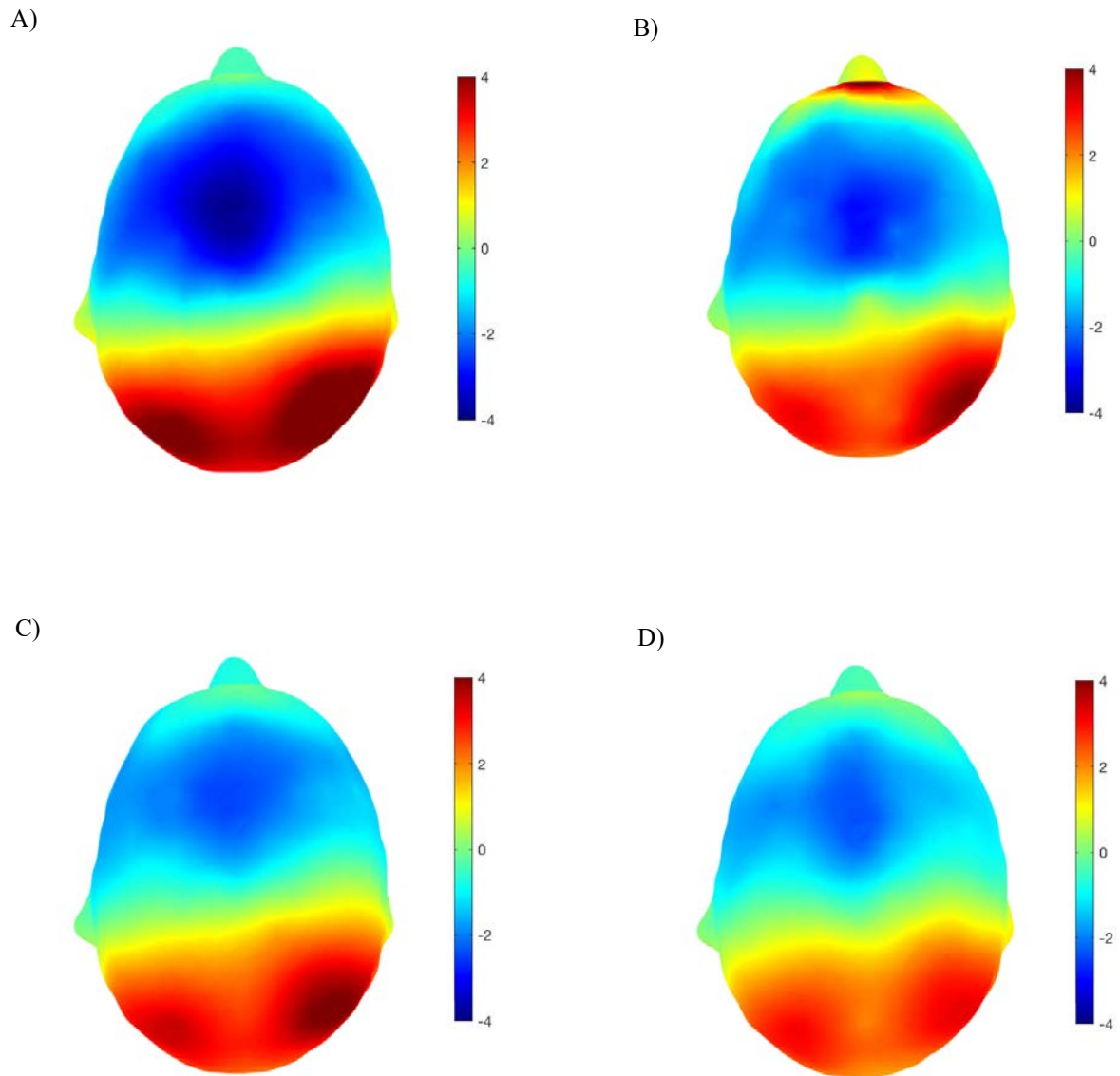D) Generic ICT four-week visit. E) Food-specific ICT 12-week visit. F) Generic ICT 12-week visit.

**Figure 5**

*Scalp Distributions of No-Go N2 ERP Amplitude at Baseline Visit*



*Note.* A) High-calorie task for the food-specific ICT. B) High-calorie task for the generic ICT. C) Neutral task for the food-specific ICT. D) Neutral task for the generic ICT.

**Figure 6**

*Scalp Distributions of No-Go N2 ERP Amplitude at Four-Week Visit*
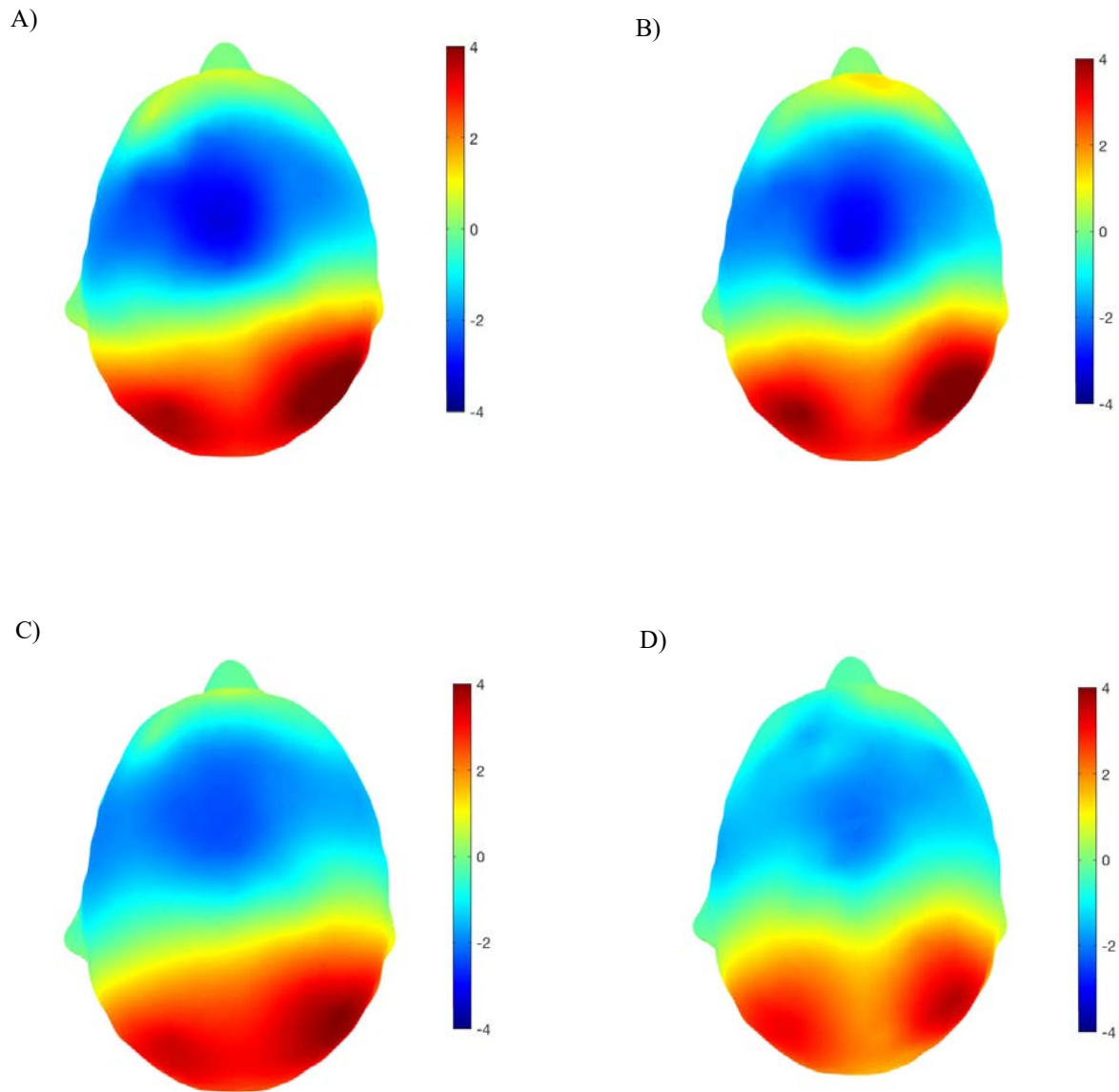
A)

B)

C)

D)



*Note.* A) High-calorie task for the food-specific ICT. B) High-calorie task for the generic ICT. C) Neutral task for the food-specific ICT. D) Neutral task for the generic ICT.

**Figure 7**

*Scalp Distributions of No-Go N2 ERP Amplitude at 12-Week Visit*

A)

B)

C)

D)

*Note.* A) High-calorie task for the food-specific ICT. B) High-calorie task for the generic ICT. C) Neutral task for the food-specific ICT. D) Neutral task for the generic ICT.
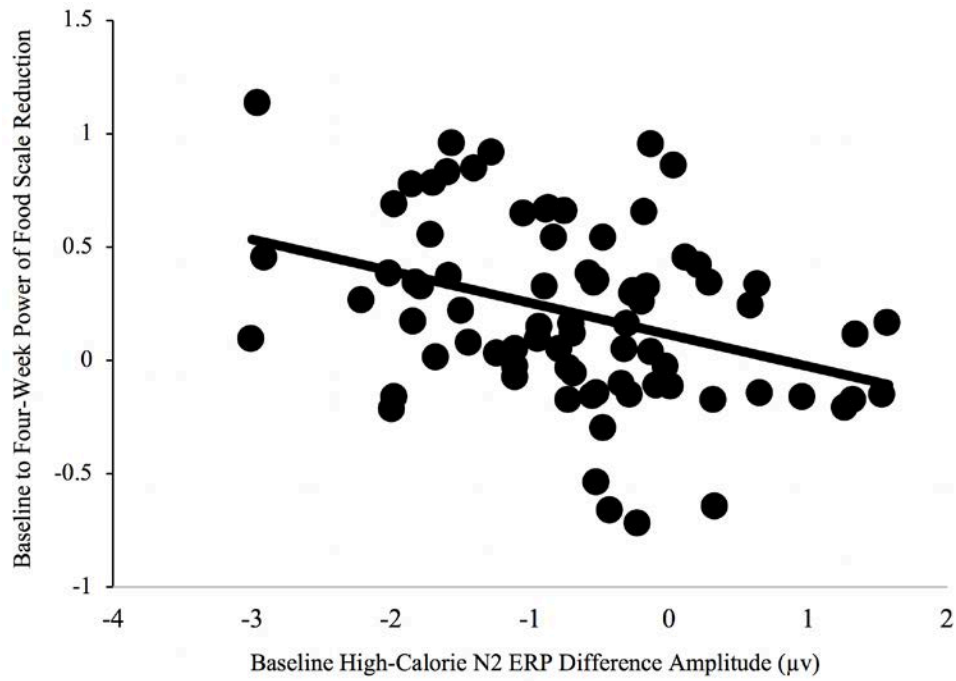
**Figure 8**

*Baseline to Four-Week Caloric Reductions by Baseline High-Calorie N2 ERP Difference Amplitude*
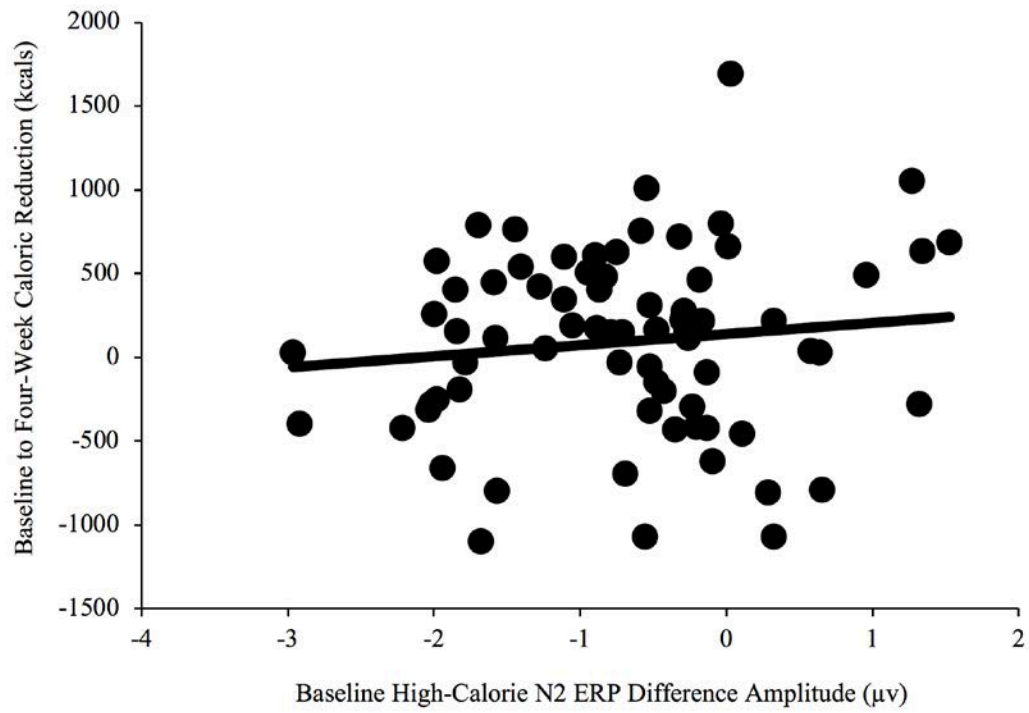


*Note.* Difference = no-go minus go.

**Figure 9**

*Baseline to Four-Week Power of Food Scale Reductions by Baseline High-Calorie N2 ERP Difference Amplitude*



*Note.* Difference = no-go minus go.