EXAMINING PERSONALITY AND TASK-BASED MEASURES OF IMPULSIVITY IN RELATION TO OBESITY AND FOOD ADDICTION

by

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(Under the Direction of Steven R. H. Beach and James MacKillop)

ABSTRACT

There is increasing interest in impulsivity as a determinant of obesity, but with mixed findings. "Food addiction" (FA), a more proximal eating variable, may strengthen this link in a way that parallels drug addiction. The current study sought to examine interrelationships among impulsivity (multiply-defined), obesity, and FA to better understand how these constructs operate independently and together. Community- and university-recruited adults ($N = 181$) of all weight classes completed a biometric screening to generate Body Mass Index (BMI), the *Yale Food Addiction Scale* (YFAS) to capture addiction-like eating behavior, and self-report and task-based measures of impulsivity. The results generally suggested stronger zero-order associations between the impulsivity indices and YFAS than BMI, and supported FA as a presumptive mediator connecting the impulsivity variables and obesity. Food addiction may be one potential pathway to obesity for impulsive individuals.

INDEX WORDS: Obesity; Food Addiction; Impulsivity; Delay Discounting; UPPS-P Impulsive Behavior Scale; Behavioral Inhibition

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CHAPTER 1

INTRODUCTION

Obesity is a complex condition that is not well understood, despite an alarming rise in global rates over the past four decades (Finucane et al., 2011). In the United States, current prevalence rates estimate that 17% of youth and over one third of adults are obese (Ogden, Carroll, Kit, & Flegal, 2014). Rising obesity rates are associated with substantial increases in healthcare costs, negative physical health consequences (e.g., diabetes, cancer, cardiovascular disease), and psychosocial challenges (e.g., weight-related stigma and discrimination, depression) (Faith et al., 2011; Gearhardt et al., 2012; World Health Organization, 2000; Yach, Stuckler, & Brownell, 2006). At a biological level, obesity results when calorie consumption consistently exceeds calorie expenditure (i.e., a "caloric imbalance"), thus causing excess calories to be retained in the body's fat cells (National Institutes of Health, 2014). However, this explanation does not account for *why* a caloric imbalance exists for an increasing number of individuals. In other words, it is important to better identify meaningful subtypes of or pathways to obesity because "obesity" simply describes the end result (i.e., excess body weight) of an amalgamation of various risk factors, processes, and consumption patterns, all of which differ for each individual.

Societal-level factors can partially explain overall weight gain trends. The modern, westernized food environment, often described as "obesogenic" in nature, is characterized by large portion sizes; highly palatable, energy dense food items; and ubiquitous exposure to food cues via advertising and commercial availability (Hill & Peters, 1998; Wardle, 2007). Additionally, work, school, home, and leisure environments encourage a sedentary lifestyle (Hill & Peters, 1998). Together, these societal characteristics can result in a positive caloric imbalance, leading to obesity over time, but not everyone living in an obesogenic environment struggles to maintain a healthy weight. Understanding the

characteristics and behaviors that predispose some individuals to overconsumption and unhealthy eating is a person-level strategy for identifying points of intervention (Jasinska et al., 2012).

1.1 Examining Obesity Using Drug Addiction Perspectives

A burgeoning body of research has begun to identify food intake patterns that resemble the consumption patterns observed for drugs of abuse. These findings have led some to believe that food, or certain types of food like those high in fat or sugar content, can precipitate a process that resembles addictive behavior (Gearhardt, Corbin, & Brownell, 2009). Animal and human studies have provided preliminary evidence to support "food addiction" (FA) as a construct. For example, rodent model studies showed associations between high-sugar and high-fat diets and increases in "behaviors that resemble addiction" such as "binge eating, compulsive food-seeking, and withdrawal symptoms…accompanied by concomitant neural changes" (for a review, Avena, 2010; Ziauddeen & Fletcher, 2013, p. 23). Similarly, compulsive overeaters and those who abuse drugs exhibited behavioral parallels, which included loss of control, tolerance and withdrawal, cravings, and relapse (for a review, Davis & Carter, 2009). Additionally, brain imaging studies demonstrated shared disruptions in dopaminergic signaling in brain reward and motivation circuits for obese and drug addicted individuals, as well as shared changes in brain regions associated with craving for both food and drugs (for a review, Volkow, Wang, Fowler, Tomasi, & Baler, 2012).

1.2 Food Addiction in Relation to Obesity

Increasing interest in food addiction provides an additional eating-related variable of interest, which represents a more specific, and perhaps clinically relevant, phenotype for study than obesity (Avena, Bocarsly, Hoebel, & Gold, 2011; Davis et al., 2011). Gearhardt et al. (2009) developed the *Yale Food Addiction Scale* (YFAS) in order to operationalize this construct for further evaluation. Over two dozen

studies have examined YFAS diagnosis (i.e., endorsing at least three of seven total symptoms, in addition to clinically significant impairment or distress) and symptom scores (i.e., a summation of the number of symptoms endorsed out of seven total symptoms) in association with a variety of biometric, demographic, dietary, and eating behavior variables (for a meta-analytic review, Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014). Based on published YFAS studies, Pursey et al. (2014) estimated that 24.9% of overweight and obese individuals and 11.1% of healthy weight individuals met YFAS diagnostic criteria for food addiction. Higher YFAS symptom scores positively correlated with adiposity (Pedram et al., 2013), negative health indicators (Flint et al., 2014), and poorer weight loss outcomes in some (e.g., Clark & Saules, 2013) but not all studies (Lent, Eichen, Goldbacher, Wadden, & Foster, 2014). Importantly, although food addiction is associated with obesity, the two conditions are not one and the same. Consuming foods in a way that leads to weight gain is not necessarily indicative of food addiction, not all obese individuals exhibit addiction-like eating behavior, and some individuals may experience addiction-like eating but maintain a healthy weight due to biological factors or compensatory strategies (Gearhardt et al., 2012). As such, obesity appears to be the end result of a variety of different processes, and food addiction may be one particularly problematic pathway to obesity for some individuals (Ziauddeen & Fletcher, 2013). An understanding of how these two constructs are related is needed in order to clarify this pathway and to identify underlying traits and vulnerabilities that contribute to these conditions.

1.3 Impulsivity and Addiction

In the domain of drug addiction, one major determinant of addictive behavior is impulsivity. In general, impulsivity is thought of as a pattern of under controlled behavior or a tendency to act out in response to impulses, something that makes self-control more difficult (Evenden, 1999; Hofmann, Friese, & Strack, 2009). However, impulsivity is a broad construct with no single, widely agreed upon definition across fields of study. Instead, impulsivity is increasingly considered to be multidimensional in nature (Bari & Robbins, 2013; Evenden, 1999). Factor analytic and correlational approaches have suggested three broad,

somewhat overlapping domains of impulsivity (de Wit, 2008; MacKillop et al., 2015; Meda et al., 2009; Reynolds, Ortengren, Richards, & de Wit, 2006). These domains include (1) "impulsive personality traits," which refer to dispositional tendencies toward impulsive behavior and are measured using self-report questionnaires such as the *UPPS-P Impulsive Behavior Scale* (Cyders et al., 2007; Whiteside & Lynam, 2001); (2) "impulsive action," which refers to deficits in inhibitory response control and is measured using tasks such as the Go/No-go task; and (3) "impulsive choice," which refers to impulsive decision-making and is measured using delay discounting tasks. Because impulsivity involves multiple unique processes, its components have not always correlated, or have correlated weakly (Bari & Robbins, 2013; Cyders & Coskunpinar, 2011; Jentsch et al., 2014). Additionally, not all impulsive individuals develop problems, certain impulsive processes may be more important than others for each individual person and at different stages of problem behavior (e.g., initiation versus maintenance), and these processes may interact in a way that contributes to problem severity and chronicity (Dawe & Loxton, 2004; de Wit, 2008).

Individuals with substance use disorders are often characterized by stronger impulsive tendencies, which have consistently demonstrated a robust, bi-directional relationship with substance use: high impulsivity appears to function as a risk and maintenance factor for drug abuse, and persistent drug use can lead to increased impulsivity (Perry & Carroll, 2008). In this context, impulsive personality traits provide a useful framework for understanding dispositional differences in the general ability to resist strong impulses, impulsive action affords an explanation for the addicted individual being unable to suppress a behavioral impulse to use drugs (Fillmore & Weafer, 2013), and impulsive choice reflects the individual sacrificing long-term physical and psychosocial health for short-term drug effects (Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Stein & Madden, 2013). Human and animal studies have provided evidence of these associations across impulsivity domains (for a review, Jentsch et al., 2014). Overall, self-reported impulsivity was greater among current and past drug addicted individuals and those with a family history of addictive behavior, relative to healthy controls (Meda et al.,

2009), and, in general, the UPPS-P traits associated with problematic drug use (for a review, Miller & Lynam, 2013). Negative Urgency and Positive Urgency demonstrated the greatest difference between drug-dependents and controls (Fischer & Smith, 2008; Smith et al., 2007; Verdejo-García, Bechara, Recknor, & Pérez-García, 2007; Zapolski, Cyders, & Smith, 2009). In contrast, Sensation Seeking was associated with use and frequency of use, but not with problems associated with using substances (Cyders, Flory, Rainer, & Smith, 2009; Smith et al., 2007; Spillane, Smith, & Kahler, 2010). Additionally, a number of studies showed greater difficulty intentionally inhibiting action for drug addicted individuals relative to healthy controls across numerous drugs of abuse (for a review, Fillmore & Weafer, 2013; Smith, Mattick, Jamadar, & Iredale, 2014). A recent meta-analysis compared Go/No-go task performance across various psychological disorders and found a small-to-medium effect for individuals with addictive disorders demonstrating poorer response inhibition relative to controls (Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014). Similarly, a meta-analysis by MacKillop et al. (2011) identified an overall medium magnitude effect size for comparisons of delay discounting between those who met clinical or subclinical criteria for addiction and control groups, with drug addicted individuals consistently demonstrating greater impulsive discounting across a range of substances (i.e., alcohol, tobacco, stimulants, marijuana, opiates, as well as poly-substance abuse). In sum, various dimensions of impulsivity have demonstrated consistent and robust relationships with substance use and abuse.

1.4 Impulsivity and Obesity

Weight is managed, in part, via self-monitoring and cognitive control over food choices (Johnson, Pratt, & Wardle, 2012). Additionally, overconsumption of palatable foods is more likely to occur among those who are more sensitive to food rewards *and* who have lower impulse control (Appelhans et al., 2011). As with drug addiction, impulsive personality features relate to eating behavior via their influence on thought, feeling, and behavior patterns characterized by low levels of inhibition and self-control (Mobbs,

Crépin, Thiéry, Golay, & Van der Linden, 2010). Impulsive action is also a potentially important process for understanding eating behavior in the current food environment because constant exposure to palatable food cues might trigger a relatively automatic response to engage in appetitive behavior (Ames et al., 2014). Without strong inhibitory control processes, an individual may habitually reach for a desired food item when faced with relevant cues. Lastly, impulsive choice (delay discounting) is thought to relate to eating decisions that sacrifice the long-term goals of health and achieving a desired body weight for the short-term satisfaction of eating tasty food items (Johnson et al., 2012). Those who consistently make poor dietary decisions at the detriment of their long-term health and weight goals may be at the highest risk for obesity and associated morbidities.

A number of studies have examined obesity's association with various impulsivity domains. However, only a few studies to date examined UPPS-P subscales in relation to BMI. Results were generally mixed. One study found higher levels of Urgency and (lack of) Premeditation among obese and overweight participants relative to healthy controls (Mobbs et al., 2010), whereas another did not observe a direct relationship between impulsive personality traits and obesity, but demonstrated a significant association between Urgency and snacking behavior (Churchill & Jessop, 2011). Murphy et al. (2014) found a small correlation between (lack of) Premeditation and BMI, but no significant direct association with any of the other UPPS-P subscales. Importantly, Murphy et al. (2014) did observe an indirect relationship between obesity and the UPPS-P Negative Urgency and (lack of) Perseverance subscales via food addiction, highlighting the importance of characterizing more proximal eating-related variables.

Findings from impulsive action studies also demonstrated mixed results. For example, one study found impaired motor response inhibition in obese individuals relative to healthy controls (Mole et al., 2014); however, another study did not find a relationship between BMI and this variable (Loeber et al., 2012). Notably, although a study by Jasinska et al. (2012) did not observe a significant zero-order association between obesity and impulsive action, structural equation modeling techniques showed an indirect relationship between these variables via unhealthy eating (e.g., overeating in response to negative mood states). Many of the impulsive action findings are difficult to compare across studies due to

differences in tasks administered (e.g., Go/No-go tasks that assessed prepotent response inhibition versus Stop Signal Tasks that assessed the ability to stop an already initiated response) and outcome variables within tasks (e.g., for Go/No-go tasks, commission errors [to capture impulsive action], versus omission errors and reaction time [which assess attentional aspects of executive functioning]).

Stronger evidence exists for a positive relationship between delay discounting and obesity, as number of studies found greater discounting among those with higher BMIs (e.g., Bickel et al., 2014; Buono, Whiting, & Sprong, 2015; Epstein et al., 2013; Fields, Sabet, & Reynolds, 2013; Jarmolowicz et al., 2014; Lawyer, Boomhower, & Rasmussen, 2015; Mole et al., 2014). However, certain studies have only observed this relationship for females (Weller, Cook, Avsar, & Cox, 2008) and for food rewards but not monetary rewards (Schiff et al., 2015). Inconsistent findings may relate to differences across studies in terms of the delay discounting task reward amounts and temporal delays assessed. Additionally most of these studies did not examine delay discounting and obesity in conjunction with measures of unhealthy or pathological eating behavior. Notably, the Fields et al. (2013) study was conducted with an adolescent sample and found an additive relationship between BMI and impulsivity. Obese adolescents demonstrated greater impulsivity on two measures (delay discounting and a measure of sustained attention) than healthy weight controls, and overweight adolescents were more impulsive on one measure (delay discounting) than healthy weight controls. Greater impulsive delay discounting may be an early risk marker for obesity.

In sum, these studies identified significant links between impulsivity and obesity, but with some mixed findings overall, suggesting that this relationship may be more complex.

1.5 Impulsivity and Food Addiction

Relative to the impulsivity and obesity literature, very few studies to date have examined impulsivity and food addiction, and results have been mixed. Pivarunas and Conner (2015) examined the UPPS-P as a predictor of food addiction and found that Negative Urgency positively predicted food addiction symptom

count while (lack of) Premeditation demonstrated a negative relationship with food addiction symptom count. Although this sample was large (N = 878), it solely consisted of undergraduate students, and did not consider obesity status. Findings from the aforementioned Murphy et al. (2014) study conflicted with the Pivarunas and Conner (2015) results, as Murphy et al. (2014) showed significant positive zero-order correlations between YFAS symptom count and all UPPS-P subscales, except for Sensation Seeking. Again, this study (Murphy et al., 2014) identified food addiction as a mediator of the relationship between self-reported impulsivity (i.e., Negative Urgency and [lack of] Perseverance subscales of the UPPS-P) and BMI, though this was also an undergraduate only sample with just 5% of participants in the obese weight range. Other studies examined food addiction in relation to a different impulsivity self-report measure, the *Barratt Impulsiveness Scale* (BIS; Patton, Stanford, & Barratt, 1995). Most recently, Raymond and Lovell (2015) found strong, positive zero-order correlations between all three BIS subscales and YFAS score. Additionally, food addiction and the BIS Non-planning subscale in conjunction were significant predictors of BMI, though all participants in this sample had type 2 diabetes and 79% were obese.

To our knowledge, only two studies examined multiple impulsivity dimensions in relation to food addiction. Davis et al. (2011) examined these constructs with a community sample of obese adults. Those who met criteria for food addiction were compared with age- and weight-matched controls on a delay discounting task and the BIS. Food addiction positive participants (i.e., 3+ on the YFAS) exhibited significantly greater impulsivity on both impulsivity measures than the non-food addicted group. This was the first study to provide evidence that subgroups of obese individuals can be distinguished by impulsivity and that impulsivity levels differ by food addiction status. It was also the only study to examine delay discounting in conjunction with food addiction. Meule, Lutz, Vögele, and Kübler (2012) utilized a median split for the number of food addiction symptoms endorsed to group participants into high and low food addiction groups, which they then contrasted for performance on a behavioral inhibition task and on the BIS. The groups did not differ on impulsive action, and only one BIS scale (i.e.,

Attentional Impulsivity, or lack of task focus and restlessness) was significantly greater for the high food addiction group. Importantly, this undergraduate only sample was all female and all healthy weight, and the categorical representation of food addiction could have limited the ability to detect a relationship between behavioral inhibition and varying levels of food addiction.

In sum, these studies provided preliminary support for a relationship between food addiction and certain impulsivity dimensions. However, strong conclusions cannot be drawn due to the small number of studies conducted and the limitations noted below.

1.6 Limitations of the Current Literature

Overall, the existing literature on impulsivity in relation to obesity and/or food addiction is limited in a number of ways. For example, the majority of the studies reviewed above utilized relatively homogenous samples (e.g., all female, undergraduate only, restricted BMI ranges, recruited due to a specific medical condition). Also, several studies utilized a categorical analytic strategy, which has clinical utility, but neglects those who may go on to become obese without intervention, and, as noted above, limits power and resolution. Additionally, obesity may not be the ideal unit of analysis because there is substantial variability among obese individuals and because obesity is a condition that develops over a relatively long period of time. Furthermore, although a number of studies looked at impulsivity and obesity, few studies included a measure of eating behavior, and even fewer specifically measured food addiction. As several studies demonstrated (Churchill & Jessop, 2011; Davis et al., 2011; Jasinska et al., 2012; Murphy et al., 2014), more proximal eating related variables, such as food addiction, can explain how mechanistic processes, like impulsivity, actually confer risk for or contribute to the maintenance of obesity among impulsive individuals. Moreover, given general consensus that impulsivity is a multidimensional construct with certain dimensions differentially contributing to various addiction processes, these studies were limited by not considering all relevant dimensions in the same sample. Additional research is needed with samples that allow for examination of the interrelationships between multiple dimensions of

impulsivity, obesity, and food addiction. This will be important for understanding the pathways by which impulsivity might lead to obesity and food addiction's role in this relationship.

CHAPTER 2

CURRENT STUDY

Following from the preceding literature review, if certain patterns of food consumption among obese individuals mimic patterns of drug consumption among drug-addicted individuals, then the same underlying traits and vulnerabilities should operate in both groups. Although impulsivity is not the only pathway to drug addiction, as noted above, it is one major factor that places a subset of individuals at greater risk for drug-related problems and also contributes to the maintenance of the condition. Thus, we expected impulsivity to relate to obesity similarly for individuals who endorse a greater addiction-like relationship with food. Stated differently, the literature suggests that some individuals (those with fewer self-control resources) develop a compulsive relationship with a particular commodity (food or drug of choice), which increases the likelihood that those individuals will end up in a disordered state (obese or drug addicted).

The overall purpose of the current study was to expand on prior studies that have examined the interrelationships between impulsivity, food addiction, and obesity in order to gain a greater understanding of self-regulation and dysregulated eating. The first aim was to examine interrelationships among several different impulsivity measures commonly utilized in addiction research, food addiction, and obesity. The second aim was to determine whether and to what degree impulsivity predicts food addiction and obesity, when food addiction and obesity are considered independently. The third aim was to examine how impulsivity relates to food addiction and obesity, when food addiction and obesity are considered together, in models that test all three constructs for indirect pathways of influence. The study addressed limitations of previous studies by employing multiple impulsivity measures for a more comprehensive assessment of the construct, by concurrently examining the interrelationships among the impulsivity variables and eating- and weight-related variables, and by doing so in a more diverse sample

that includes both genders and participants across the BMI range. Based on this perspective, the specific hypotheses were as follows: (1) impulsivity (multiply defined), BMI, and food addiction were all expected to significantly associate with each other; (2) impulsivity was expected to be more highly associated with food addiction than with BMI; and (3) the predicted pattern in hypothesis two was expected to be further substantiated by food addiction partially mediating the relationship between impulsivity and obesity.

CHAPTER 3

METHOD

3.1 Participants

Participants were recruited via two sources: (1) community advertisements soliciting individuals who were interested in participating in a research study on eating behavior in exchange for $36 and (2) an undergraduate human subjects research pool soliciting research participation in exchange for three hours of research credit. Participants were required to be between the ages of 18-55, to have at least an eighth grade education, and, if female, to not be pregnant or have given birth in the past nine months. A total of 208 individuals enrolled; however, the final sample consisted of 181 participants. Participants were removed from the original sample for missing or incomplete data for one or more study measures (n = 16), for being red-flagged by research assistants during the participation session due to uncooperative behavior or failure to comply with protocol instructions (n = 3), and for greater than two invalid responses on the delay discounting control items (see below) (n = 8).

Participants (N = 181) were community adults (48%; n = 87) and university undergraduates (52%; n = 94) from a large, Southeastern university town. Women comprised 71% of the sample (n = 129). The majority of the sample was Caucasian (63%; n = 114). Twenty three percent of participants reported their ethnicity as Black or African American (n = 42), 8% as Asian (n = 14), 6% as mixed race (n = 10), and <1% as American Indian or Alaskan Native (n = 1). The modal age of participants was 18, with a median age of 20 and a mean age of 24.8 years. The majority of participants reported being full- or part-time students at the time of data collection (73.5%; n = 132). Per the World Health Organization's (2000) graded weight classification scheme, 4% of participants (n = 7) were underweight (BMI < 18.50), 45% were healthy weight (BMI = 18.50 - 24.99), 20% were overweight (BMI = 25.00 - 29.99), and 32%

were obese (BMI > 29.99). The modal number of YFAS symptoms endorsed was 1, with median score of 2 and a mean of 2.16 symptoms. Thirty five percent of the sample endorsed three or more YFAS symptoms (n = 63). Sample characteristics are presented in Table 1.

3.2 Measures

Demographics Assessment.

This self-report questionnaire consisted of standard demographic questions about gender, age, race, income, and other demographic variables.

Biometric Assessment.

Participant weight, percent body fat, and percent body water were measured with a digital scale. Participant height and participant waist, hip, neck circumferences were measured using a standard tape measure. Participant body mass index was calculated from participant weight and height using the following formula: BMI = weight (lb)/[height (in)]^2 x 703.

Eating Behavior Assessment.

Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009). This 27-item, self-report questionnaire was designed to assess for difficulty controlling palatable food (e.g., sweets, starches, and fatty foods) intake over the past 12 months. Individual items map on to one of seven substance dependence diagnostic symptoms adapted from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (American Psychiatric Association [DSM-IV-TR], 2000). The YFAS offers two scoring options: a continuous total symptom count (0 - 7) and a dichotomous diagnostic version. A participant meets diagnostic criteria when he/she endorses three or more symptoms and clinically significant impairment or distress. A total symptom count score was the primary YFAS variable used in the current analyses. Higher scores reflected greater levels of addictive eating behavior. For the

current sample, the aggregate Cronbach's alpha for the 20 items that appear on the seven symptom scales was adequate (α = .74).

Impulsivity Assessment.

UPPS-P Impulsive Behavior Scale (UPPS-P; Cyders et al., 2007; Whiteside & Lynam, 2001). This 59-item, self-report questionnaire was designed to quantify personality characteristics associated with impulsivity. Participants rated how much they agreed with statements about the way that people think and act (e.g., "I often get involved in things I later wish I could get out of.") on a 4-point likert scale ranging from 1 ("agree strongly") to 4 ("disagree strongly"). The UPPS-P items each mapped on to one of five subscales, and each of the subscales captured a different motivation for engaging in impulsive behavior: Negative Urgency (NU; i.e., a tendency to act rashly when experiencing negative emotions), (lack of) Perseverance (PSV; i.e., an inability to sustain attention and motivation in order to complete tasks), (lack of) Premeditation (PMD; i.e., a tendency to act without thinking), Positive Urgency (PU; i.e., a tendency to act rashly when experiencing positive emotions), and Sensation Seeking (SS; a tendency to seek out and enjoy novel or exciting activities). Subscale scores were calculated as the average of the available items. Higher scores indicated greater self-reported impulsive characteristics. All subscales demonstrated adequate Cronbach's alpha levels (NU, α = .88; PRM, α = .86; PSV, α = .80; PU, α = .92; SS, α = .88).

Go/No-go Task (GNG; Kiehl, Liddle, & Hopfinger, 2000). This computer-based behavioral task measured the ability to inhibit inappropriate responses when presented with two different stimuli. It consisted of one block of 80 trials, during which participants were to press a button on their keyboard every time the letter "X" (i.e., the "go" signal; 85% of trials) appeared on the computer screen, and to not respond when the letter "K" (i.e., the "no-go" signal; 15% of trials) appeared. Task instructions requested that participants respond as quickly and accurately as possible. Commission Error Rate (CER; i.e., the percentage of "no-go" trials for which the participant failed to inhibit a response) was used in analyses as the primary measure of impulsive action. Higher CERs reflected greater levels of difficulty inhibiting a

prepotent response. The GNG task also produced two additional variables used in analyses, Omission Error Rate (OER; i.e., the percentage of "go" trials for which the participant failed to respond) and Go Trial Reaction Time (Go Trial RT; i.e., average time taken to submit a response for "go" trials only), which reflected lapses in attention control and processing speed, respectively. Although these two additional variables are distinct from impulsivity, as currently conceptualized, these variables underwent the same analytic procedures as the "true" impulsivity variables and are reported in conjunction with them.

Monetary Delay Discounting. This task measured impulsive decision making using 27 preconfigured items from the *Monetary Choice Questionnaire* (MCQ; Kirby, Petry, & Bickel, 1999), a reliable and validated measure for assessing an individual's discounting preferences, and an iterative 90-item delay discounting task (DDT; Amlung, Sweet, Acker, Brown, & MacKillop, 2014). Both discounting measures were used to obtain a more thorough assessment of discounting preferences. The 27 items assessed discounting preferences across three delayed reward magnitudes, small ($25 - $35), medium ($50 - $60), and large ($75 - $85). It comprised dichotomous choice items that asked the participant to choose between a smaller, immediate reward and a larger, delayed reward (e.g., "Would you rather have $11 today, or $30 in 7 days?"). The 90-item delay discounting task also comprised dichotomous choice items, but choice preferences for a smaller reward ($10 - $99) today were always assessed relative to the same $100 reward at varying delays (one day, one week, two weeks, 1 month, 2 months, 3 months, six months, or one year). Additionally, this task presented a larger number of repeated decisions and covered all possible choice preferences, or permutations, in a randomized order. Temporal discounting rates, or *k* values were generated for each MCQ magnitude and for the DDT. A *k* value reflected the rate at which a particular reward lost its subjective value based on its temporal delay, for that participant. Higher *k* values represented a more impulsive decision-making profile and a stronger preference for smaller, immediate rewards. For the MCQ, *k* values were estimated using the using the method detailed by Kirby et al. (1999). Each participant's responses within each of the three reward magnitudes were used to estimate discounting-rate parameters (i.e. *k* values) for each magnitude. For the

DDT, each participant's responses within each of the eight temporal delays were used to estimate indifference points, or the point at which the subjective value of the smaller, sooner reward was roughly equal to the larger, later reward. The average indifference point for each delay was then converted into a hyperbolic discounting function for each participant using the equation described by Mazur (1987). The *k* values were skewed, as is common, and were log10 transformed to improve normality.

3.3 Procedure

The University of Georgia Institutional Review Board approved all study procedures. Community participants were recruited using radio, print, and bus ads around the community. University participants were recruited via an online, research opportunity listing Website sponsored by the university. Interested community participants completed a brief telephone screen to assess for inclusion and exclusion criteria prior to attending the laboratory session. University participants were not screened prior to the laboratory visit; however, the online sign-up information explicitly noted the inclusion criterion and stated that documentation of age would be required prior to participation.

Upon arrival at the laboratory, participants underwent written informed consent during which they were told that identifying information would be removed from their data and that they were free to withdraw from participation at any time without penalty or loss of benefits to which they were entitled. Prior to beginning the study session, participant eligibility was verified by interview with trained research assistants.

All questionnaires and behavioral tasks were completed on a desktop computer in a private laboratory room. Questionnaire data was collected using Qualtrics Research Suite, a secure Internet survey service. Behavioral tasks were conducted using E-Prime 2.0, a software suite for computerized experiment design, data collection, and analysis. Study participation was organized around assessment blocks, within which measures were randomized for pseudo-counterbalancing. The first block contained demographic-, medical-, personal history-, and impulsivity-related questionnaires and behavioral tasks. A

second block contained eating- and food-related measures, to avoid drawing attention to eating and weight-related material prior to the independent variables. Biometric data was collected by trained research assistants at the conclusion of the study session, again to ensure that awareness of body measurements did not significantly influence responding on assessments. Participants were offered four breaks at scheduled times throughout the study session. The study session concluded after participants were debriefed and provided with laboratory contact information for future questions or concerns about their study participation.

3.4 Data Analysis

First, given high correlations among *k* values in previous studies (e.g., Amlung & MacKillop, 2014; Vanderbroek, Acker, Palmer, de Wit, & MacKillop, 2015), discounting indices were examined to determine the appropriateness of consolidation via principal component analysis (PCA) using oblique, direct oblimin rotation. Second, Pearson correlation coefficients were generated to examine the uncorrected patterns of relationships in this sample. For demographic variables that were significantly correlated with BMI and/or YFAS score, partial correlations were used to examine the independent effects of each demographic variable. Demographic variables that remained significantly correlated with BMI and/or YFAS score after the effects of other variables were partialed out were entered as covariates in all subsequent analyses. Third, hierarchical regressions were used to test whether each impulsivity variable significantly associated with BMI and/or YFAS score. Separate regressions were run for BMI and for YFAS score as dependent variables. Covariates were entered in step one, and the impulsivity variable of interest was entered in step two. Lastly, exploratory mediation analyses were conducted to integrate significant regression findings. Specifically, if an impulsivity variable significantly associated with YFAS score in the regression model, that impulsivity variable was entered as the independent variable (IV) in a mediation model with YFAS score as the mediator, BMI as the dependent variable (DV), and significantly associated demographic variables as covariates. If an impulsivity variable was

significant in a regression model with BMI, but not in a regression model with YFAS score, it was not examined for mediation due to its lack of association with the proposed mediator. If an impulsivity variable was significant in a regression model with YFAS score, but was not significantly correlated with BMI or significant in a regression model with BMI, the impulsivity variable was still retained in mediation models. Although this is not intuitive, the contemporary perspective asserts that failing to examine indirect effects, when a total effect is not present precludes a deeper understanding of mechanisms by which the IV potentially effects the DV (Hayes, 2009). Additionally, this relaxed approach is particularly useful in cases where the proposed causal process is thought to be temporally distal from the DV, thus exerting a smaller effect due to more links in the causal chain and competing or random factors (Shrout & Bolger, 2002). Mediation analyses were completed using Preacher and Hayes' (2008) SPSS INDIRECT macro. This macro estimated direct and total effects and then inferred the indirect effect of the IV on the DV through the mediator (see Figure 1). Indirect effects were tested with Preacher and Hayes' (2004, 2008) bootstrapping technique using the recommended 5000 bootstrap resamples with replacement and 95% bias-corrected confidence intervals (CIs). A significant indirect effect (i.e., mediation) was detected when the bootstrap-derived percentile CI did not contain zero. Bootstrap-based mediation model testing methods have been recommended over others because they allow for higher power and better Type I error control, and do not assume a normal distribution (Hayes, 2009; Preacher & Hayes, 2004, 2008).

Table 1: Sample Characteristics

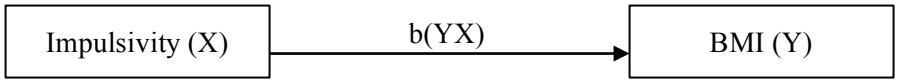
Variable	%/Mean (*SD*)		
	Overall Sample	Community Recruitment[a]	University Recruitment[b]
Demographic Variables			
Sex (% Female)	71.3	63.2	78.7
Age	24.80 (9.45)	30.83 (10.29)	19.21 (3.07)
Race (% Caucasian)	63.0	51.7	73.4
Years of Education	13.62 (2.12)	14.44 (2.40)	12.86 (1.47)
Income (Median)	$60,000 - 74,999	$30,000 – 44,999	$75,000 – 89,999
Biometric Variables			
Height (in.)	66.41 (3.64)	66.97 (3.70)	65.89 (3.53)
Weight (lb.)	176.29 (61.43)	209. 12 (69.90)	145.92 (29.09)
Body Fat (%)	29.42 (13.35)	35.00 (15.06)	24.49 (9.23)
Waist-to-Hip Ratio	.89 (.09)	.92 (.10)	.85 (.07)
Neck Circumference (in.)	14.47 (2.06)	15.52 (2.26)	13.50 (1.21)
Weight and Eating Variables			
Body Mass Index	28.01 (9.06)	32.76 (10.37)	23.62 (4.35)
YFAS Symptom Count	2.16 (1.45)	2.52 (1.54)	1.83 (1.28)
Impulsivity Variables			
UPPS-P			
Negative Urgency	2.22 (.56)	2.22 (.56)	2.22 (.57)
(Lack of) Premeditation	1.89 (.48)	1.84 (.49)	1.93 (.47)
(Lack of) Perseverance	1.85 (.46)	1.85 (.48)	1.85 (.43)
Positive Urgency	1.80 (.56)	1.79 (.57)	1.81 (.55)
Sensation Seeking	2.79 (.63)	2.60 (.67)	2.96 (.54)
Go/No-go Task			
Commission Error Rate	.35 (.19)	.32 (.20)	.38 (.19)
Omission Error Rate	.04 (.08)	.06 (.09)	.02 (.05)
Go Trial Reaction Time	334.35 (73.46)	352.96 (84.46)	317.13 (56.77)
PCA *k*	.00 (1.00)	.04 (1.08)	-.04 (.93)

Note: *SD* = standard deviation; in. = inches; lb. = pounds; YFAS = *Yale Food Addiction Scale*; UPPS-P = *UPPS-P Impulsive Behavior Scale;* PCA *k* = delay discounting factor score from principal component analysis.

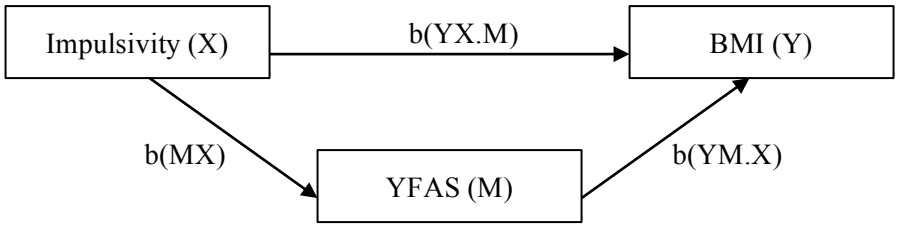
N = 181; [a]*n* = 87; [b]*n* = 94.

Figure 1: Food Addiction as a Mediator of the Relationship Between Impulsivity and BMI

A. Direct Pathway



B. Indirect Pathway



Note: Age and income were controlled in all analyses. YFAS = *Yale Food Addiction Scale* total symptom count; BMI = body mass index; X = independent variable; Y = dependent variable; M = mediator; YX = direct effect of X on Y; YX.M = direct effect adjusting for the mediator; MX = effect of X on M; YM.X = indirect (mediating) effect. The indirect effect was calculated by multiplying b(MX) and b(YM.X).

CHAPTER 4

RESULTS

4.1 Preliminary Analyses

For the delay discounting data, PCA *k* accounted for 79.86% of the variance. PCA *k* was used in all subsequent analyses; see Table 2 for delay discounting descriptives and intercorrelations among *k* values.

Interrelationships among demographic variables, BMI, and YFAS score are presented in Table 3. BMI and YFAS score were significantly positively correlated and showed the same general pattern of association with demographic variables. Both BMI and YFAS score were significantly positively associated with age and non-Caucasian race status, and negatively associated with income. Age and income, and race and income, were significantly negatively intercorrelated. Education was also associated with age and income, but not with BMI or YFAS score. Gender was not significantly associated with BMI, YFAS score, or any other demographic variables in the current sample.

Because age, income, and race correlated with BMI and YFAS score, these relationships were examined using zero-order partial correlations in order to explore the relationship between each demographic variable and each DV while controlling for the effects of the other two demographic variables. Age, but not income or race, demonstrated a statistically significant zero-order correlation with BMI (age, r [177] = .36, p <.001; income, r [177] = -.08, p = .32); race, r [177] = .12, p = .12). Income, but not age or race, demonstrated a statistically significant zero-order correlation with YFAS score (age, r [177] = .06, p = .42; income, r [177] = -.19, p <.05); race, r [177] = .12, p = .13). Given these patterns, both age and income were entered as covariates in all subsequent analyses, for consistency across analyses. Of note, recruitment strategy was correlated with BMI (r = -.51; p < .001) and YFAS score (r = -.24; p < .01), but when entered as a third covariate, along with age and income in the regression and

mediation analyses that follow, all results were unchanged. Thus, for the sake of parsimony and replicability, recruitment strategy was not included as a control variable in the results reported here.

Interrelationships among BMI, YFAS score, and impulsivity variables are presented in Table 4. BMI was negatively associated with UPPS-P Sensation Seeking and positively associated with GNG Omission Error Rate and Go Trial Reaction Time. YFAS score was also negatively associated with UPPS-P Sensation Seeking. YFAS score was positively associated with UPPS-P Negative Urgency, (lack of) Perseverance, and Positive Urgency, and with PCA *k.* All UPPS-P scales were significantly intercorrelated, with the exception of Sensation Seeking, which was only associated with (lack of) Premeditation and Positive Urgency. The strongest association was observed between the Negative Urgency and Positive Urgency subscales. GNG Commission Error Rate, Omission Error Rate, and Go Trial Reaction Time were all significantly intercorrelated. Commission Error Rate was negatively associated with Go Trial Reaction Time and positively associated with Omission Error Rate. PCA*k* was not significantly associated with any other impulsivity variables, except for a small correlation with GNG Omission Error Rate. Additionally, GNG Commission Error Rate was associated with UPPS-P (lack of) Perseverance. Omission Error Rate was associated with UPPS-P Positive Urgency.

4.2 Primary Analyses

Impulsive Personality Traits.

In order to test whether impulsivity would associate with BMI and YFAS score over and above covariates, individual hierarchical regressions were conducted with each impulsivity variable predicting each outcome variable in step two, controlling for the effects of age and income in step one. For YFAS regressions, age and income together accounted for 9.6% of the variance in YFAS score (F [2, 178] = 9.46, p <.001, R^2 = .10). For BMI regressions, age and income together accounted for 22% of the variance in BMI (F [2, 178] = 25.04, p <.001, R^2 = .22). This covariate model applied to all subsequent regressions. None of the UPPS-P scales were significant in regressions with BMI after accounting for age

and income. However, a different pattern emerged for YFAS regressions. Three of the five UPPS-P scales were significant in regressions with YFAS score. Specifically, Negative Urgency accounted for an additional 3.1% of the variance in YFAS score (F [1, 177] = 6.24, p <.05, R^2 = .03), (lack of) Perseverance accounted for a further 2% of the variance in YFAS score (F [1, 177] = 3.91, p <.05, R^2 = .02), and Positive Urgency accounted for an additional 3.9% of the variance in YFAS score (F [1, 177] = 8.06, p <.01, R^2 = .04), in each scale's respective regression. Higher levels of Negative Urgency, (lack of) Perseverance, and Positive Urgency were associated with higher levels of food addiction. Total variance accounted for in each significant regression was as follows: Negative Urgency, 12.7% (total R^2 = .13); (lack of) Perseverance, 11.6% (total R^2 = .12); Positive Urgency, 13.5% (total R^2 = .14).

Impulsive Action.

Go/No-go Commission Error Rate was not significantly associated with BMI or with YFAS after controlling for age and income in step one. However, the two additional GNG variables were significant in their respective regressions, but only for models with BMI. Omission Error Rate accounted for a further 2.2% of the variance in BMI (F [1, 177] = 5.14, p <.05, R^2 = .02), and Go Trial Reaction Time accounted for an additional 6.6% of the variance in BMI (F [1, 177] = 16.46, p <.001, R^2 = .07). Higher Omission Error Rates and longer Go Trial Reaction Times were associated with higher BMI. Total variance accounted for in each significant BMI regression was as follows: Omission Error Rate, 24.2% (total R^2 = .24); Go Trial Reaction Time, 28.6% (total R^2 = .29).

Impulsive Choice.

The delay discounting component variable, PCA k, was not significant in the BMI regression but was significant in the YFAS regression, accounting for an additional 2.3% of the variance in YFAS score (F [1, 177] = 4.69, p <.05, R^2 = .02). Total variance accounted for in this regression was 11.9% (total R^2 = .12). Higher discounting of delayed rewards was associated with higher levels of food addiction. For illustrative purposes, Figure 2 presents a graphical depiction of medium magnitude discounting curves for

individuals who endorsed 0-1 YFAS symptoms (n = 79) and those who endorsed three or greater YFAS symptoms (n = 63), putatively reflecting the presence of food addiction.

4.3 Integrative Analyses

Four impulsivity variables, UPPS-P Negative Urgency, (lack of) Perseverance, Positive Urgency, and PCA *k,* were significantly associated with food addiction in regressions, and thus, were tested in mediation models to examine the indirect effect of impulsivity on BMI by way of food addiction. As in regression analyses, all models included age and income as covariates. Although none of these impulsivity variables were directly associated with BMI, the tests of indirect effects demonstrated that there were significant indirect effects on BMI for all models, as indicated by bias-corrected CIs for all models that did not include zero. These results are presented in Table 6. Higher levels of Negative Urgency, (lack of) Perseverance, Positive Urgency, and higher discounting of delayed rewards were associated with higher BMI via higher levels of food addiction. This demonstrates a significant contribution of food addiction to the effect of impulsivity on BMI, and implicates food addiction as a presumptive mediator in each relationship.

Table 2: Monetary Discounting Task Descriptive Statistics and Intercorrelations Between PCA *k* and individual DDT and MCQ *k* values

	M (SD)	1.	2.	3.	4.	5.
1. PCA *k*	.00 (1.00)	-	-	-	-	-
2. DDT *k*	-1.81 (.87)	.86***	-	-	-	-
3. MCQ *k*: $30	-1.53 (.66)	.88***	.66***	-	-	-
4. MCQ *k*: $55	-1.73 (.70)	.93***	.74***	.77***	-	-
5. MCQ *k*: $80	-1.95 (.76)	.90***	.67***	.72***	.82***	-

Note: The associations between the individual indices and PCA *k* reflect component loadings. Monetary amounts listed reflect the average reward amount within the MCQ magnitude; *M* = mean; *SD* = standard deviation; PCA *k* = delay discounting factor score from principal component analysis; DDT = 90-item delay discounting task; MCQ = *Monetary Choice Questionnaire*.

***$p<.001$.

Table 3: Zero-order Correlations Among Demographic Variables, BMI, and Food Addiction

	1.	2.	3.	4.	5.	6.	7.
1. Gender	-	-	-	-	-	-	-
2. Age	-.13	-	-	-	-	-	-
3. Race	-.02	.11	-	-	-	-	-
4. Education	-.08	.32***	-.04	-	-	-	-
5. Income	.12	-.52***	-.37***	-.20**	-	-	-
6. BMI	-.04	.50***	.19*	.08	-.33***	-	-
7. YFAS	.07	.20**	.21**	-.06	-.31***	.34***	-

Note: BMI = Body Mass Index; YFAS = *Yale Food Addiction Scale* total symptom count.

***p<0.001; **p<.01; *p<.05.

Table 4: Zero-order Correlations Among BMI, Food Addiction, and Impulsivity Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. BMI	-	-	-	-	-	-	-	-	-	-	-
2. YFAS	.34***	-	-	-	-	-	-	-	-	-	-
3. UPPS-P: NU	.12	.20**	-	-	-	-	-	-	-	-	-
4. UPPS-P: PRM	-.05	.08	.38***	-	-	-	-	-	-	-	-
5. UPPS-P: PSV	.14	.18*	.45***	.39***	-	-	-	-	-	-	-
6. UPPS-P: PU	.08	.24**	.71***	.44***	.38***	-	-	-	-	-	-
7. UPPS-P: SS	-.30***	-.21**	.00	.25***	-.13	.17*	-	-	-	-	-
8. GNG: CER	-.10	-.03	.13	.06	.16*	.08	-.09	-	-	-	-
9. GNG: OER	.24**	.10	.14	-.10	-.02	.22**	-.06	.17*	-	-	-
10. GNG: Go Trial RT	.39***	.07	.02	-.13	-.12	.09	.00	-.50**	.28***	-	-
11. PCA *k*	.04	.21**	.01	.01	-.08	.04	-.10	.00	.15*	.02	-

Note: BMI = Body Mass Index; YFAS = *Yale Food Addiction Scale* total symptom count; UPPS-P = *UPPS-P Impulsive Behavior Scale*; NU = Negative Urgency; PRM = Premeditation; PSV = Perseverance; PU = Positive Urgency; SS = Sensation Seeking; GNG = Go/No-go Task; CER = commission error rate; OER = omission error rate; Go Trial RT = average reaction time on "Go" trials; PCA *k* = delay discounting factor score from principal component analysis.

***p<0.001; **p<.01; *p<.05.

Table 5: Hierarchical Regressions Predicting Food Addiction and BMI from Impulsivity Variables, with Age and Income as Covariates

	YFAS			BMI		
	B (SE)	**β**	***p***	**B (SE)**	**β**	***p***
	Impulsive Personality Traits					
UPPS-P Negative Urgency						
Age	.01 (.01)	.03	.71	.36 (.08)	.38	<.001
Income	-.14 (.04)	-.28	<.01	-.40 (.24)	-.13	.09
NU	.45 (.18)	.18	<.05	.87 (1.08)	.05	.42
UPPS-P (lack of) Premeditation						
Age	.01 (.01)	.07	.44	.37 (.08)	.39	<.001
Income	-.14 (.04)	-.28	<.01	-.41 (.24)	-.13	.09
PRM	.27 (.22)	.09	.21	-.11 (1.26)	-.01	.93
UPPS-P (lack of) Perseverance						
Age	.01 (.01)	.05	.52	.37 (.07)	.39	<.001
Income	-.13 (.04)	-.26	<.01	-.36 (.24)	-.12	.13
PSV	.45 (.23)	.14	<.05	1.96 (1.33)	.10	.14
UPPS-P Positive Urgency						
Age	.01 (.01)	.06	.45	.37 (.08)	.39	<.001
Income	-.12 (.04)	-.25	<.01	-.38 (.24)	-.12	.12
PU	.52 (.18)	.20	<.01	.89	.06	.42
UPPS-P Sensation Seeking						
Age	.00 (.01)	.01	.96	.33 (.08)	.34	<.001
Income	-.13 (.04)	-.27	<.01	-.38 (.24)	-.12	.12
SS	-.32 (.18)	-.14	.07	-1.93 (1.02)	-.14	.06
	Impulsive Action					
GNG Commission Error Rate						
Age	.01 (.01)	.05	.52	.37 (.08)	.39	<.001
Income	-.14 (.04)	-.28	<.01	-.40 (.24)	-.13	.10
CER	.05 (.54)	.01	.93	-2.02 (3.13)	-.04	.52
GNG Omission Error Rate						
Age	.01 (.01)	.05	.56	.35 (.07)	.37	<.001
Income	-.13 (.04)	-.27	<.01	-.35 (.24)	-.11	.14
OER	.76 (1.37)	1.37	.58	17.86 (7.88)	.15	<.05
GNG Go Trial Reaction Time						
Age	.01 (.01)	.05	.53	.31 (.07)	.32	<.001
Income	-.14 (.04)	-.28	<.01	-.34 (.23)	-.11	.14
Go Trial RT	-3.60 E-5 (.00)	.00	.98	.03 (.01)	.27	<.001
	Impulsive Choice					
PCA*k*						
Age	.01 (.01)	.06	.48	.37 (.08)	.39	<.001
Income	-.12 (.04)	-.24	<.01	-.42 (.24)	-.14	.09
PCA *k*	.23 (.10)	.16	<.05	-.21 (.62)	-.02	.74

Note: YFAS = *Yale Food Addiction Scale* total symptom count; BMI = Body Mass Index; UPPS-P = *UPPS-P Impulsive Behavior Scale*; NU = Negative Urgency; PRM = (lack of) Premeditation; PSV = (lack of) Perseverance; PU = Positive Urgency; SS = Sensation Seeking; GNG = Go/No-go Task; CER = commission error rate; OER = omission error rate; Go Trial RT = average reaction time for "go" trials; PCA *k* = delay discounting factor score from principal component analysis.

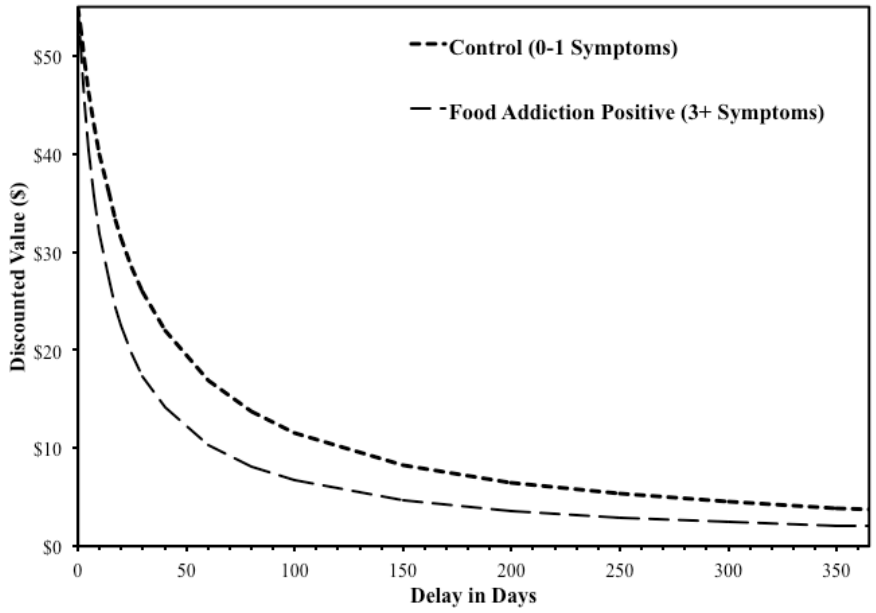
Table 6: Food Addiction as a Mediator of the Relationship Between Impulsivity and BMI

Mediation Relationship *X* → *M* → *Y*	Direct and Total Effects		Adj. R^2	Indirect Effect	Bootstrapping	
					Lower BC 95% CI	Upper BC 95% CI
Model 1: UPPS-P NU → YFAS → BMI	b(YX) b(MX) b(YM.X) b(YX.M) Age Income	.87 .45* 1.53*** .18 .36*** -.19	.26***	.70	.23	1.56
Model 2: UPPS-P PSV → YFAS → BMI	b(YX) b(MX) b(YM.X) b(YX.M) Age Income	1.96 .45* 1.48*** 1.29 .36*** -.17	.26***	.67	.06	1.65
Model 3: UPPS-P PU → YFAS → BMI	b(YX) b(MX) b(YM.X) b(YX.M) Age Income	.89 .52** 1.54*** .09 .36*** -.19	.26***	.80	.25	1.72
Model 4: PCA *k* → YFAS → BMI	b(YX) b(MX) b(YM.X) b(YX.M) Age Income	-.21 .23* 1.61*** -.57 .36*** -.23	.26***	.36	.05	.87

Note: Number of bootstrapped resamples = 5000. YFAS = *Yale Food Addiction Scale* total symptom count; BMI = Body Mass Index; X = independent variable; M = mediator; Y = dependent variable; YX = direct effect of X on Y; MX = direct effect of X on M; YX.M = direct effect adjusting for the mediator; YM.X = indirect (mediating) effect; BC = bias-corrected; CI = confidence interval; UPPS-P = *UPPS-P Impulsive Behavior Scale*; NU = Negative Urgency; PSV = Perseverance; PU = Positive Urgency; PCA *k* = delay discounting factor score from principal component analysis.

***p<0.001; **p<.01; *p<.05.

Figure 2: Overall Differences in Discounting for Individuals Who Reported Minimal Versus Clinically Significant Levels of Food Addiction



Note: This figure presents overall medium magnitude (average reward amount of $55) MCQ discounting curves for participants who endorsed minimal (i.e., 0-1) YFAS symptoms relative to participants who endorsed clinically significant (i.e., three or more) YFAS symptoms.

CHAPTER 5

DISCUSSION

The purpose of the current study was to expand on previous research that investigated associations between impulsivity, food addiction, and obesity, in order to gain a greater understanding of how these constructs operate independently and together. Results were generally consistent with the proposed hypotheses. As predicted, higher impulsivity was associated with higher levels of food addiction, but contrary to expectations, higher impulsivity was not associated with greater BMI. Additionally, impulsivity associated more strongly with food addiction than with BMI when these constructs were considered independently, and certain impulsivity domains were related to BMI only through their relation to addictive-like eating behavior when all three constructs were considered together. Notably, these relationships held for some but not all impulsivity domains assessed. Specific findings, implications, and future directions are discussed below.

Although none of the impulsivity variables were directly associated with BMI in regression models, mediation analyses demonstrated indirect associations between four of the impulsivity variables and BMI via food addiction. Specifically, results supported food addiction as a mediator of the relationships between BMI and a tendency to act rashly when experiencing strong emotions (Negative Urgency and Positive Urgency), a tendency towards difficulty persisting in and completing tasks and goals ([lack of] Perseverance), and greater discounting of delayed monetary rewards (PCA *k*). These findings were consistent with and expanded upon the Murphy et al. (2014) results. Findings from these two studies suggest that individuals who tend to behave rashly when feeling particularly bad may be more likely to compulsively consume food as a way to cope with negative mood states; those who tend to behave rashly when feeling particularly good may be more likely to overindulge or lose control over their eating when experiencing positive emotions; those who tend to experience difficulty sticking with a task

or goal may find it difficult to control appetitive urges and to persist in dieting efforts over time; and those who tend to chose immediate gratification at the expense of a greater long-term reward may be more likely to give in to food urges at the expense of long-term health outcomes. Importantly, both studies showed that these impulsivity variables only relate to obesity because of their association with addiction-like eating behavior. The delay discounting finding is particularly important to highlight because the current study is the to provide empirical support for a pathway by which delay discounting contributes to obesity. Although a number of previous studies identified a relationship between delay discounting and obesity, only one (Davis et al., 2011) examined discounting in relation to food addiction, and this study focused on subtyping obese individuals by food addiction status and then examining group differences (e.g., discounting preferences), rather than exploring processes by which these constructs are related. The current study did not support any clear associations between impulsive action and food addiction or obesity, but this was not surprising given previous mixed findings for Go/No-go tasks (e.g., Jasinska et al., 2012; Loeber et al., 2012; Meule et al., 2012; Mole et al., 2014). Task inconsistency across studies could also account for these contradictory findings, or it may be that this impulsivity domain is less important than others when considering food addiction. Although speculative, another possibility is that impulsive action is only relevant for particular stages (e.g., beginning stages of weight gain) or levels of problem behavior (e.g., very high levels of food addiction), which the current study was not designed to detect.

The current findings have a number of theoretical and clinical implications. In conjunction with the previously reviewed literature, the current results support impulsivity as a determinant of overconsumption disorders, expanding beyond drug addiction. Less clear is why impulsivity relates to overconsumption. One possibility is that weak or impaired prefrontal regulation, putatively underlying executive functioning and self-regulatory capacity, leads to poor inhibition and decision making (Feil et al., 2010; Fitzpatrick, Gilbert, & Serpell, 2013). For example, executive control allows the individual to disengage from tempting stimuli in the environment and weigh the pros and cons of a decision before acting, so poor executive control may increase the probability of reacting to rewarding stimuli (Martin &

Davidson, 2014). Along these lines, another possibility is that impulsivity in disorders of overconsumption shares the same overactive subcortical reward processing in regions such as the ventral tegmental area, nucleus accumbens, and amygdala (Koob & Volkow, 2010). Evenden (1999) and, more recently, Jentsch et al. (2014) reviewed studies published on biological mechanisms of impulsivity. Both concluded, based on human and animal psychopharmacological research, that different impulsivity domains involve unique but interacting neural pathways and neurochemical substrates. Differential neurobiological activity across impulsivity domains and processes is consistent with the variable patterns of association observed in the current study. However, additional research spanning different levels of analysis (e.g., genetic, neurobiological, behavioral, interpersonal) is needed in order to clarify the reasons why impulsivity relates to overconsumption disorders.

In addition to implications for impulsivity as a determinant for addiction, the current results also have implications for food addiction as a construct. As stated in the introduction, if certain patterns of food consumption among obese individuals mimic certain patterns of drug consumption among drug-addicted individuals, then similar underlying traits and vulnerabilities should operate in both groups. The current study showed that higher levels of impulsivity in some domains are associated with higher levels of addiction-like eating behavior and, subsequently, obesity. This finding is consistent with general patterns of association between impulsivity and addictive behavior in the drug addiction literature. The delay discounting finding, in particular, provides strong support for the food addiction construct because delay discounting is one of the most robust correlates of a range of addictive behaviors (MacKillop et al., 2011).

The food addiction construct should also have utility, meaning that it should aid in identifying, developing, and adapting appropriate obesity prevention and intervention programs (Ziauddeen & Fletcher, 2013). The current results support the utility of the food addiction construct because food addiction links impulsivity to obesity, thus identifying impulsive processes that might serve as relevant obesity treatment targets. A process level treatment approach is particularly important given substantial evidence that the weight loss method of choice, dieting by means of calorie restriction, is effective in the

short-term, but counterproductive in the long-term, with the majority of dieters regaining most of, all of, or more than their pre-diet weight (Mann et al., 2007). Targeting the basic risk factors and processes that contribute to overconsumption patterns (e.g., impulsivity, in the case obese individuals who report high levels of food addiction) may be a more fruitful long-term strategy. A relevant conceptual review by Ashe, Newman, and Wilson (2015) lends credence to the idea that individual traits and processes are important to consider when identifying when, how, and with whom to employ particular intervention tactics. The Ashe et al. (2015) review discusses mindfulness (i.e., objectively attending to the present moment to assess and experience reactions to temptations rather than impulsively acting on them) and distraction (i.e., diverting attention away from a temptation to provide time for urges to dissipate) techniques for treating drug addiction, but proposes that delay discounting level moderates the effectiveness of these strategies. Specifically, Ashe et al. (2015) suggest that, for steep discounters experiencing strong urges, attending to tempting stimuli (mindfulness) may increase urges, whereas shifting attention away from tempting stimuli (distraction) should decrease urges; however, when urges are manageable, mindfulness may be a better strategy than distraction for promoting long-term change.

The current study findings suggest that four forms of impulsivity are particularly relevant for obesity interventions: (1) delayed reward discounting; (2, 3) general emotional reactivity, for both positive and negative mood states; and (4) poor ability to persist in goal pursuit. A small, but growing, literature provides preliminary evidence that reducing impulsivity, or strengthening self-control, reduces overconsumption of drugs and/or food. For example, several studies have shown that episodic future thinking training (EFT), a process that involves imagining future events (e.g., achieving a health goal) rather than focusing on the anticipated pleasure of an immediate decision (e.g., calorie consumption), can improve eating habits and reduce discounting of delayed rewards (Daniel, Stanton, & Epstein, 2013; Dassen, Jansen, Nederkoorn, & Houben, 2016; O'Neill, Daniel, & Epstein, 2016). Another approach shown to reduce delay discounting among stimulant addicts (Bickel, Yi, Landes, Hill, & Baxter, 2011) and problem drinkers (Houben, Wiers, & Jansen, 2011), but not yet tested with addictive-like eating behavior or obesity, is working memory training. This approach proposes to improve executive

functioning ability via completing repeated seriates of cognitive training tasks, thus strengthening a number of cognitive processes including inhibitory control.

Treatment approaches may also target multiple impulsive processes simultaneously. For example, a combined behavioral intervention study (i.e., cognitive behavioral therapy, physical activity programming, and dietary counseling) for overweight adolescents found the greatest post-intervention BMI reduction for adolescents who also showed greater reductions in Negative Urgency (measured via the UPPS-P) and greater cognitive inhibitory control improvement (measured via changes on the Iowa gambling, Stroop, and letter number sequencing tasks) (Delgado-Rico et al., 2012). These results hint that greater behavioral control in response to negative emotions and greater cognitive control have an effect on weight; however, these findings require replication due to the cross-sectional nature of the data and the lack of a treatment control group. Elements of treatment approaches for other disorders (e.g., drug addiction) should also hold promise for reducing impulsive tendencies that contribute to overconsumption. For example, individuals who tend to cope with intense emotions by eating may benefit from emotion regulation or distress tolerance skills to learn to recognize and respond to emotional arousal in a more adaptive, and less impulsive, way. Difficulty persisting in health- and weight-related goal pursuit could be addressed via motivational interviewing techniques to increase self-efficacy and motivation for change and from cognitive behavioral strategies to improve goal-setting and self-monitoring, identify trigger situations for overeating, and generate alternative rewarding activities or plan strategies to cope with urges to consume. Continuing to identify distinct pathways to obesity will be useful for identifying obesity risk factors and vulnerabilities to target during obesity interventions and, eventually, for developing personalized intervention plans tailored to an individual's risk profile.

The current study addresses a number of limitations of the existing literature. This is the first study to examine food addiction, obesity, and all three impulsivity domains in the same sample, providing a more comprehensive snapshot of relations among these constructs. Additionally, the current study results are more generalizable than previous studies due to a more inclusive sample (e.g., greater gender balance, multiple recruitment sources, all BMI ranges) and dimensional characterization of all variables,

which also allowed for greater power and resolution. Lastly, the current study moved beyond BMI and obesity status as proxy eating markers and assessed self-reported eating behavior. This strategy allowed for a better understanding *how*, not just *if*, impulsivity confers risk for or contributes to obesity. It is important for future studies to continue focusing on food addiction and other more proximal eating related variables that can link basic processes to obesity. It will also be useful to develop more sensitive and specific indicators of obesity, as BMI remains a relatively course indicator of health status.

Limitations of the current study are also important to consider. The current study is cross-sectional in nature, and the temporal directionality of the pathway model presented above is based on theoretical assumptions. Thus, no causal inferences can be drawn. Future studies should investigate this presumptive pathway longitudinally and from early childhood. Although the diversity of the current sample is a strength, different recruitment sources and the inclusive recruitment approach also led to demographic differences (i.e., age and income) that had to be controlled for in analyses. Additionally, this recruitment strategy led to a low level of reported addictive-like eating, relative to average YFAS scores in clinical samples. Importantly, it is likely that the current findings would be amplified, and some relationships (e.g., the nearly significant zero-order association between UPPS-P Sensation Seeking and YFAS score) might reach significance, in a sample reporting higher average levels of food addiction. A final limitation is that an updated version of the YFAS*,* the *Yale Food Addiction Scale 2.0* (YFAS 2.0; Gearhardt, Corbin, & Brownell, 2016) was published after the current study concluded. The YFAS 2.0 was designed to reflect changes to substance use disorder diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (American Psychiatric Association [DSM-5], 2013) and now includes 11 symptoms, rather than 7. It also provides an updated diagnostic scoring approach with graded severity level cut-offs (mild, moderate, severe) in addition to the symptom count scoring method. Importantly, the YFAS 2.0 preliminary validation report demonstrated a high level of association between YFAS and YFAS 2.0 symptom count scoring results ($r = .78$; $p < .001$), suggesting that both measures are capturing the same construct (Gearhardt et al., 2016). Nonetheless, future food addiction

studies may benefit from adopting the updated measure to ensure similar characterization symptomology across addictive disorders (Gearhardt et al., 2016).

There are a few final considerations to note. First, a substantial proportion of variance remains unexplained in the models tested. The current study does not champion impulsivity as a major explanatory variable for obesity at large. Deficits in self-regulatory processes are just one person-level factor that seemingly place individuals high on impulsivity at greater risk for addictive eating behavior. A whole host of other individual (e.g., differences in appetitive and metabolic processes, genetic vulnerabilities, physical activity level) and environmental (e.g., level of access to healthy food options, modeling of healthy/unhealthy eating practices) factors may be more important to consider, or may interact with impulsivity in unique ways to promote obesity. Future studies should continue to identify addiction-specific and other, more general processes that might show a stronger direct association with obesity. Second, the majority of obese individuals in the current sample did not report clinically significant levels of food addiction. This is consistent with existing data (Pursey et al., 2014) and provides additional evidence that food addicted obese individuals appear to be a unique group of obese persons with greater impulsive tendencies (Davis et al., 2011; Murphy et al., 2014). A continued focus on specific characteristics of this subgroup may provide further support for food addiction theory and could provide greater evidence for a clinically relevant obesity phenotype that might benefit from specialized treatment approaches. Finally, also consistent with existing data (Pursey et al., 2014), a number of non-obese participants reported high levels of impulsivity and/or high levels of food addiction, relative to the study average for these variables. It is possible that these individuals will eventually develop obesity; however, it is equally possible that certain protective factors (e.g., activity level, dietary choices) are operating against weight gain for these individuals. Future studies should test these possibilities, as protective characteristics could eventually be leveraged for obesity interventions.

In conclusion, the current study results lend support for impulsivity (i.e., certain impulsive personality traits and greater discounting of delayed rewards) as one pathway to obesity, but only through the influence of food addiction. This pathway parallels that observed in the drug addiction literature and

provides evidence in support of the food addiction construct. The current findings also provide support for impulsivity as a process underlying disorders of overconsumption extending beyond drug addiction.

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