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Multidimensional Assessment of Impulsivity in Relation to Obesity and Food Addiction

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Abstract

Based on similarities between overconsumption of food and addictive drugs, there is increasing interest in “food addiction,” a compulsive eating pattern defined using symptoms parallel to substance use disorders. Impulsivity, a multidimensional construct robustly linked to drug addiction, has been increasingly examined as an obesity determinant, but with mixed findings. This study sought to clarify relations between three major domains of impulsivity (i.e., impulsive personality traits, discounting of delayed rewards, and behavioral inhibition) in both obesity and food addiction. Based on the association between impulsivity and compulsive drug use, the general hypothesis was that the impulsivity-food addiction relation would be stronger than and responsible for the impulsivity-obesity relation. Using a cross-sectional dimensional design, participants ($N = 181$; 32% obese) completed a biometric assessment, the *Yale Food Addiction Scale* (YFAS), the *UPPS-P Impulsive Behavior Scales*, a Go/NoGo task, and measures of monetary delay discounting. Results revealed significantly higher prevalence of food addiction among obese participants and stronger zero-order associations between impulsivity indices and YFAS compared to obesity. Two aspects of impulsivity were independently significantly associated with food addiction: (a) a composite of Positive and Negative Urgency, reflecting proneness to act impulsively during intense mood states, and (b) steep discounting of delayed rewards. Furthermore, the results supported food addiction as a mediator connecting both urgency and delay discounting with obesity. These findings provide further evidence linking impulsivity to

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food addiction and obesity, and suggest that food addiction may be a candidate etiological pathway to obesity for individuals exhibiting elevations in these domains.

Keywords

obesity; food addiction; impulsivity; delay discounting; urgency

Obesity is a complex condition and, despite an alarming rise in global rates over the past four decades, its etiology is not well understood (Finucane et al., 2011). Defined as a body mass index (BMI) of 30 or above, current prevalence rates indicate that 17% of youth and over 33% of adults in the United States are obese (Flegal, Kruszon-Moran, Carroll, Fryar, & Ogden, 2016; Ogden et al., 2016). Rising obesity rates are associated with substantial increases in healthcare costs, negative physical health consequences, and psychosocial challenges (Gearhardt et al., 2012; Yach, Stuckler, & Brownell, 2006). Societal-level factors, such as the modern, westernized food environment (i.e., large portion sizes, highly palatable and energy dense food items), may partially explain overall weight gain trends, but person-level variables are also putatively influential in the development of obesity. Furthermore, there is increasing interest in leveraging insights on the causes of drug addiction to inform obesity.

Examining Obesity Using Insights from Drug Addiction

A growing literature has begun to identify food intake patterns that resemble the consumption patterns observed for addictive drugs, leading some to believe that food, or certain types of food, like those high in fat, sugar, and salt, can give rise to an equivalent syndrome (Gearhardt, Corbin, & Brownell, 2009). Food addiction provides an novel syndrome that potentially represents a more specific, and perhaps clinically relevant, eating phenotype for study than obesity (Avena, Bocarsly, Hoebel, & Gold, 2011; Davis et al., 2011). Animal and human studies provide preliminary evidence to support the “food addiction” (FA) construct. For example, rodent models show associations between high-sugar and high-fat diets and increases in binge eating and compulsive food-seeking, accompanied by complimentary neurobiological changes (Avena, 2010). Similarly, compulsive overeaters and those who abuse drugs exhibit behavioral parallels, which include loss of control, tolerance, cravings, and relapse (Davis & Carter, 2009). Additionally, brain imaging studies demonstrate 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 (Volkow, Wang, Fowler, Tomasi, & Baler, 2012). The *Yale Food Addiction Scale* (YFAS; Gearhardt et al., 2009) was developed to operationalize a food addiction syndrome. Compared to healthy weight individuals, significantly more overweight and obese individuals meet YFAS diagnostic criteria for food addiction (Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014) but, although food addiction is associated with obesity, the empirical literature suggests that the two conditions are by no means identical (Gearhardt et al., 2012). One possibility is that obesity is an end result of a variety of different processes and food addiction may be one particularly problematic pathway to obesity for some individuals.

Given similarities between addiction-like eating behavior and drug addiction, a broad hypothesis is that similar processes may be operating across the two conditions. 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 increasingly considered to be multidimensional in nature (Bari & Robbins, 2013; Evenden, 1999). Factor analytic and correlational approaches suggest 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 (a) “impulsive personality traits,” or dispositional tendencies toward impulsive behavior, typically measured using self-report questionnaires such as the *UPPS-P Impulsive Behavior Scales* (Cyders et al., 2007; Whiteside & Lynam, 2001); (b) “impulsive action,” or deficits in behavioral inhibition, typically measured using tasks such as the Go/No-go task; and (c) “impulsive choice,” or impulsive decision-making, typically measured as relative preference for smaller immediate rewards compared to larger delayed rewards (i.e., delay discounting or delay of gratification). Because impulsivity involves multiple unique processes, its components (both within and across domains) do not always correlate or correlate weakly (Bari & Robbins, 2013; Cyders & Coskunpinar, 2011; Jentsch et al., 2014). Importantly, in each of these domains, numerous studies provide evidence of associations between impulsivity measures and aspects of substance use disorders (for reviews, see Jentsch et al., 2014; MacKillop et al., 2011; Miller & Lynam, 2013). There are some nuances to these relations. Not all impulsive individuals develop problem outcomes, 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). Despite these differential relations, however, individuals with substance use disorders can be broadly characterized as having stronger impulsive tendencies in a number of domains (Jentsch et al., 2014; MacKillop et al., 2011; Miller & Lynam, 2013; Perry & Carroll, 2008).

Impulsivity, Obesity, and Food Addiction

A number of studies have also examined various impulsivity domains in relation to obesity, although overall results have been mixed. For example, the few studies examining impulsive personality traits and obesity have only found significant direct associations between greater BMI and Urgency (i.e., tendency to act rashly when experiencing intense emotions) (Mobbs, Crépin, Thiéry, Goley, & Van der Linden, 2010) and greater BMI and (lack of) Premeditation (i.e., tendency to act without thinking) (Mobbs et al., 2010; Murphy, Stojek, & MacKillop, 2014), but these relations do not hold across all studies (Churchill & Jessop, 2011). Associations between impulsive action and obesity are even less consistent, with some results showing greater impairment in motor response inhibition for obese than for healthy individuals (Mole et al., 2014), and others not finding evidence for a direct association between impulsive action and BMI (Lawyer, Boomhower, & Rasmussen, 2015; Loeber et al., 2012). Stronger evidence exists for a positive relation between obesity and indices of delay discounting (i.e., tendency to prefer smaller sooner rewards to larger later

rewards). A recent meta-analysis found steeper discounting of both monetary and food rewards to be a consistent feature of obesity across studies (Amlung, Petker, Jackson, Balodis, & MacKillop, 2016). Interestingly, recent studies have found mindfulness training reduces discounting, albeit selectively for discounting of food (Hendrickson & Rasmussen, 2013; Hendrickson & Rasmussen, 2016), suggesting its potential as a treatment target. In sum, with the exception of delay discounting, the direct link between obesity and impulsivity is ambiguous.

The existing mixed findings may be because BMI is simply a measure of body composition and does not capture motivational aspects of eating behavior. In turn, following from the link between impulsivity and drug addiction, impulsivity may be theorized to relate to obesity most directly via a greater addiction-like relationship with food. This hypothesis has been addressed by a small number of studies that examined the relations between all three constructs (i.e., impulsivity, food addiction, and obesity) and are generally supportive. For example, one study suggested that subgroups of obese individuals can be distinguished by impulsivity (i.e., delay discounting and impulsive personality traits) and that impulsivity levels differ by food addiction status (Davis et al., 2011). However, the design of this study exclusively included obese individuals. Additionally, food addiction was found to mediate the relation between BMI and certain impulsive personality traits (i.e., Negative Urgency and [lack of] Perseverance; Murphy et al., 2014), although behavioral measures of impulsivity were not examined. These studies provide initial evidence that impulsivity operates in a similar way across disorders of overconsumption and that some individuals (those with elevations in aspects of impulsivity) experience 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). However, strong conclusions about the interrelations among impulsivity, food addiction, and obesity cannot yet be drawn due to the small number of studies and a number of methodological limitations in those to date.

Current Study

Overall, the existing literature on impulsivity in relation to obesity and food addiction is relatively nascent and is particularly limited to the extent that few studies have concurrently examined both obesity and food addiction. Moreover, given an increasing consensus that impulsivity is a multidimensional construct with dimensions differentially contributing to addiction processes, a further limitation is that most studies have been relatively narrow in the scope of the assessment of impulsivity. Finally, most studies have had a relatively restricted range of obesity levels. The current study sought to examine interrelations among food addiction, obesity, and several different impulsivity measures commonly utilized in drug addiction research, while addressing limitations of prior work. Specifically, the study sought to more comprehensively address this question by using measures from all three domains of impulsivity (i.e., impulsive personality traits, impulsive action, and impulsive choice) and operationalizing obesity using a factor analytic composite measure of body composition (i.e., body mass index; percent adiposity [body fat]; and waist, hip, and neck circumferences). The first aim was to determine whether and to what degree different facets of impulsivity were associated with food addiction and obesity independently. Then, where

significant associations were present, the second aim was to examine models that test all three constructs for indirect pathways of influence. Specifically, drawing on the drug addiction literature, the hypothesis was that food addiction would partially mediate the relation between indices of impulsivity, in particular those associated with reward valuation and affect regulation, and obesity. This pattern of relations would tentatively (pending confirmation of this model via longitudinal design in future studies) suggest an etiological pathway from self-regulatory deficits leading to compulsive eating patterns and in turn leading to obesity, not the other way around.

METHOD

Participants

Participants were 208 adults recruited from the general community (48%) and undergraduates in the University of Georgia human subjects research pool (52%). Sample characteristics are in Table 1 and characteristics by recruitment source are in supplementary materials. 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. The final sample consisted of 181 participants as individuals were not included for missing or incomplete data for one or more key study measures ($n = 16$), being flagged by research assistants during the participation session due to uncooperative behavior or failure to comply with protocol instructions ($n = 3$), or greater than two invalid responses on the delay discounting control items (see below) ($n = 8$). Percent adiposity could not be collected for four participants due to digital scale malfunction. Per the World Health Organization, 32% of the sample was obese ($BMI > 29.99$). Of the overall sample, 34.8% endorsed three or more food addiction symptoms and 6.6% were positive for food addiction (i.e., endorsed three or more YFAS symptoms *and* significant impairment or distress). Food addiction positive status significantly differed by obesity status (non-obese = 3.23%; obese = 14.04%; $\chi^2 [1] = 7.37, p = .007$). For a three-hour assessment, community participants received \$36 and university students received three hours of research credit.

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 and percent adiposity were measured with digital scales (Ozeri Touch 440 lbs – ZB13-W2; Tanita – BF-680W). Participant height and participant waist, hip, and neck circumferences were measured using a standard tape measure. Participant body mass index was calculated from participant weight and height using the following formula: $BMI = \text{weight (lb)} / [\text{height (in)}]^2 \times 703$. Given high correlations among biometric variables (Table 2), a principal component analysis using oblique, direct oblimin rotation was conducted for consolidation and for a more comprehensive measure of body composition. This analysis included BMI, percent adiposity, and waist, hip, and neck circumferences. The obesity composite accounted for 84.67% of variance in these five variables.

Food Addiction Symptoms—*Yale Food Addiction Scale* (YFAS; Gearhardt et al., 2009) is a 27-item, self-report questionnaire designed to assess food addiction 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 in order to increase power and to map the food addiction severity continuum. Higher scores reflected greater levels of addictive eating behavior. Internal reliability was good in the current sample ($\alpha = .74$).

Impulsive Personality Traits—*UPPS-P Impulsive Behavior Scales* (UPPS-P; Cyders et al., 2007; Whiteside & Lynam, 2001) is a 59-item, self-report questionnaire designed to quantify personality characteristics associated with impulsivity. The UPPS-P has five subscales: (a) Negative Urgency, tendency to act rashly when experiencing negative emotions; (b) (lack of) Perseverance, inability to sustain attention and motivation to complete tasks; (c) (lack of) Premeditation, tendency to act without thinking; (d) Positive Urgency, tendency to act rashly when experiencing positive emotions; and (e) Sensation Seeking, tendency to seek out and enjoy novel or exciting activities. Given a high degree of association between the Negative and Positive Urgency subscales ($r = .71, p < .001$), an Urgency composite, reflecting emotional reactivity, was created using the mean of the two. The Urgency composite was used in all subsequent analyses. Internal reliability was good: Urgency composite, $\alpha = .94$; Premeditation, $\alpha = .86$; Perseverance, $\alpha = .80$; Sensation Seeking, $\alpha = .88$.

Impulsive Action—Go/No-go Task (GNG; Kiehl, Liddle, & Hopfinger, 2000) is a computer-based behavioral task that measured the ability to inhibit prepotent 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. Commission error rate (the percentage of “No-go” trials for which the participant failed to inhibit a response) was used as the primary measure of impulsive action. The GNG task also produced two additional variables used in analyses, omission error rate (the percentage of “Go” trials for which the participant failed to respond) and go trial reaction time (average time taken to submit a response for “Go” trials only), which reflected lapses in attention control and processing speed, respectively.

Impulsive Choice—Delay discounting decision making was assessed using two measures, the 27-item *Monetary Choice Questionnaire* (MCQ; Kirby, Petry, & Bickel, 1999), a reliable and validated measure for assessing an individual’s discounting preferences using preconfigured items, and an iterative 90-item delay discounting task (DDT; Amlung, Sweet, Acker, Brown, & MacKillop, 2014). Two discounting measures were used for a higher resolution assessment. The MCQ assesses discounting preferences across three delayed

reward magnitudes, small (\$25 – \$35), medium (\$50 – \$60), and large (\$75 – \$85). The 90-item 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. For the MCQ, k values were estimated using the inferential method detailed by Kirby and colleagues (1999). For the DDT, each participant's responses within each of the eight temporal delays were used to estimate indifference points, or the points at which the subjective value of the smaller, sooner reward was approximately equal to the larger, later reward. The average indifference point for each delay was then used to generate a hyperbolic discounting function for each participant using the equation described by Mazur (1987). The k values were skewed, as is common, and were \log_{10} transformed to improve normality. Given very high correlations among k values (Table 2), the four k values consolidated via principal component analysis using oblique, direct oblimin rotation, as has been used successfully previously (Amlung & MacKillop, 2014; Vanderbroek, Acker, Palmer, de Wit, & MacKillop, 2015). This also provides a measure of delay discounting across multiple reward magnitudes. The delay discounting composite accounted for 79.86% of the variance among the four k values.

Procedure

The University of Georgia Institutional Review Board approved all study procedures. Community participants were recruited using radio, print, and bus ads. 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 and an interview with trained research assistants for verification of study eligibility. All questionnaires and behavioral tasks were completed on a desktop computer in a private laboratory room. The measures were counterbalanced by participant and biometric data were collected at the conclusion of the session to ensure that awareness of body measurements did not influence performance.

Data Analysis

First, Pearson correlation coefficients were generated to examine the uncorrected patterns of relations in this sample. For demographic variables that were significantly correlated with the obesity composite and/or YFAS score, partial correlations were used to examine the independent effects of each demographic variable. Demographic variables that remained significantly correlated with the obesity composite and/or YFAS score after the effects of other variables were partialled out were entered as covariates in all subsequent analyses. Second, hierarchical regressions were used to test whether each impulsivity variable was significantly associated with the obesity composite and/or YFAS score. Separate regressions

were run for the obesity composite and for YFAS score as dependent variables (DVs). Covariates were entered in an initial step, and the impulsivity variable of interest was entered in a second step. Third, combined and mechanistic analyses were conducted to integrate the individual significant regression findings. Specifically, impulsivity variables that were statistically significant in individual regressions were entered simultaneously in a combined regression to determine the relative strength of each association. Additionally, mediational analyses were conducted to test whether the relation between an index of impulsivity and food addiction mediated the relation between the index and obesity. These analyses were applied to the indices that were significantly associated with food addiction in the combined regression. This was the case even if a significant association with obesity was not present because a significant direct effect may not be present for a number of reasons and failing to examine indirect effects precludes the evaluation of mechanistic relations (Hayes, 2009; Kenny & Judd, 2014). 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. Indirect effects were tested with Preacher and Hayes' (2004, 2008) bootstrapping technique using the recommended 5,000 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).

RESULTS

Preliminary Analyses

Interrelations among demographic variables, the obesity composite, and YFAS score are presented in Table 3. The obesity composite and YFAS score were significantly positively correlated and showed the same general pattern of association with demographic variables. Both the obesity composite and YFAS score were significantly positively associated with age and non-White race status, and negatively associated with income. Age and income, and race and income, were significantly negatively intercorrelated. Education was also significantly associated with age (positive association) and income (negative association), but not with the obesity composite or YFAS score. Gender was not significantly associated with the obesity composite, YFAS score, or any other demographic variables in the current sample. Because age, income, and race all correlated with the obesity composite and YFAS score, these relations were examined using partial correlations in order to explore the relation 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 the obesity composite (age, $r[173] = .44$, $p < .001$; income, $r[173] = -.09$, $p = .26$; race, $r[173] = .14$, $p = .07$). 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, $r[177] = .11$, $p = .13$). 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 age, income,

the obesity composite, and YFAS score, but when entered as a third covariate, along with age and income, in the regressions and mediation analyses that follow, all results were unchanged. Therefore, for the sake of parsimony and replicability, recruitment strategy was not included as a control variable.

Interrelations among the obesity composite, YFAS score, and impulsivity variables are presented in Table 4. The obesity composite was negatively associated with Sensation Seeking and positively associated with omission error rate and go trial reaction time. YFAS score was also negatively associated with Sensation Seeking. YFAS score was positively associated with the Urgency composite, (lack of) Perseverance, and the delay discounting composite. All UPPS-P scales were significantly intercorrelated, with the exception of Sensation Seeking, which was only associated with (lack of) Premeditation. Go/No-go Task 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. The delay discounting composite was not significantly associated with any other impulsivity variables, except for a small correlation with omission error rate. Additionally, commission error rate demonstrated a small association with (lack of) Perseverance, and omission error rate was associated with the Urgency composite.

Primary Analyses

Regression results are presented in Table 5. Two of the four UPPS-P scales were significant in regressions with YFAS score, including the Urgency composite ($p < .01$, $R^2 = .04$) and (lack of) Perseverance ($p < .05$, $R^2 = .02$) in each scale's respective regression. Higher levels of Urgency and (lack of) Perseverance were associated with higher levels of food addiction. Total variance accounted for in each significant regression was as follows: Urgency composite, 13.7% (total $R^2 = .14$); (lack of) Perseverance, 11.6% (total $R^2 = .12$). Sensation Seeking was the only significant UPPS-P scale in regressions with the obesity composite after accounting for age and income ($p < .05$, $R^2 = .02$). Higher levels of Sensation Seeking were associated with lower obesity composite values. In terms of impulsive action, Go/No-go commission error rate was not significantly associated with the obesity composite after controlling for age and income. The two additional GNG variables were significantly related to the obesity composite: omission error rate ($p < .05$, $R^2 = .02$) and go trial reaction time ($p < .001$, $R^2 = .05$). Higher omission error rates and longer go trial reaction times were associated with higher obesity composite values. No GNG variables were significantly related to YFAS scores.

In terms of impulsive choice, the delay discounting composite variable was not significant in the obesity composite regression but was significant in the YFAS regression ($p < .05$, $R^2 = .02$). Higher discounting of delayed rewards was associated with higher levels of food addiction. For illustrative purposes, Figure 1 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 ($n = 63$).

Integrative Analyses

Because multiple facets of impulsivity were significantly associated with our index of food addiction (YFAS), we entered the Urgency composite, (lack of) Perseverance, and the delay discounting composite simultaneously in a combined regression to determine unique effects of each impulsivity variable on YFAS scores. Although Sensation Seeking had a trend level association with food addiction, it was not included in this combined model because it did not meet the established threshold for significance of $p < .05$. The Urgency composite ($B = .46$, $SE = .22$, $\beta = .16$, $p < .05$) and the delay discounting composite ($B = .24$, $SE = .10$, $\beta = .17$, $p < .05$), but not (lack of) Perseverance ($B = .28$, $SE = .25$, $\beta = .09$, $p = .26$), remained significant. Table 6 presents results from a combined food addiction regression with the Urgency and delay discounting composite variables (and without [lack of] Perseverance).

Given these combined regression findings, the Urgency composite and the delay discounting composite were tested in mediation models to examine the indirect effect of impulsivity on body composition by way of food addiction. Although neither of these impulsivity variables were directly associated with the obesity composite, the tests of indirect effects demonstrated that there were significant indirect effects on the obesity composite for both models, as indicated by bias-corrected CIs for all models that did not include zero (see Table 7). Higher levels of Urgency and higher discounting of delayed rewards were associated with higher obesity composite values via higher levels of food addiction.

DISCUSSION

The purpose of the current study was to extend previous research that investigated associations between impulsivity, food addiction, and obesity to gain a greater understanding of their interrelations. Results were generally consistent with the proposed hypothesis. Individual regressions with each impulsivity variable independently predicting food addiction and, separately, obesity, detected significant positive associations between three impulsivity variables (Urgency composite, [lack of] Perseverance, and delay discounting composite) and food addiction, and a significant negative association between one impulsivity variable (Sensation Seeking) and obesity. When all three variables significant in food addiction regressions were entered in the same model, (lack of) Perseverance was no longer significant, indicating that a tendency to act rashly when experiencing strong emotions (Urgency composite) and greater discounting of delayed monetary rewards (delay discounting composite) were the only distinct facets of impulsivity associated with food addiction. In addition, mediation analyses revealed indirect effects between these two impulsivity variables and obesity by way of food addiction. Specifically, the results supported food addiction as a mediator of the relations between both a tendency to act rashly when experiencing strong emotions and greater discounting of delayed monetary rewards in relation to obesity.

These findings suggest that individuals who tend to behave rashly when feeling particularly strong emotions may be more likely to compulsively consume food as an emotion regulation strategy that involves actively using food as a positive and negative reinforcer (i.e., to cope with negative or to modulate positive mood states). Previous studies examining impulsivity among eating disordered populations have found similar associations (e.g., Claes et al.,

2015). Additionally, the delay discounting finding suggests that those who tend to choose 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. These individuals may be generally oriented towards feeling good in the present, and food consumption may be one strategy for achieving this. Importantly, this study showed that these impulsivity variables only relate to obesity because of their association with addiction-like eating behavior. The delay discounting finding is particularly worth highlighting because the current study is the first to provide empirical support for a presumptive food addiction pathway by which delay discounting contributes to obesity. Although a number of previous studies identified a relation 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 Sensation Seeking findings also deserve mention. Contrary to expectations, food addiction and obesity were associated with low (rather than high) Sensation Seeking. It may be that eating is an enjoyable, but not highly arousing and stimulating experience, such that those who are risk averse but also reward-driven gravitate towards palatable food consumption, and those who are risk seeking and reward-driven might seek out experiences involving greater levels of arousal and stimulation than eating offers (e.g., illicit drug consumption, sky diving).

The results did not support any clear associations between impulsive action (i.e., commission errors on the behavioral inhibition task) and food addiction or obesity, but previous studies have been mixed and this converges with several negative findings (Jasinska et al., 2012; Loeber et al., 2012; Meule, Lutz, Vögele, & Kübler, 2012). 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. Interestingly, omission error rate, an index of inattention, and reaction time were significantly associated with obesity, but not food addiction. These findings may reflect deficits in other forms of executive function, but equally could represent challenges that come with larger body size.

The current findings lend additional support for impulsivity as a determinant of disorders of overconsumption beyond drug addiction. Less clear is what the underlying mechanisms are that relate to overconsumption. One possibility is that weak or impaired prefrontal cortex activity, 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). However, this is fundamentally an empirical question and future studies including groups

with pharmacological addictions, food addiction, and no addiction will be necessary to examine similarities and differences directly.

The current results also speak to the relation between food addiction and obesity to an extent. In this cohort, individuals with obesity were significantly more likely to meet criteria for food addiction, although the majority of obese individuals in the current sample did not report clinically significant levels of food addiction. This finding is consistent with existing data (Pursey et al., 2014) and provides additional evidence that food-addicted obese individuals appear to be a discrete group of obese persons who report compulsive eating patterns that parallel addictive drug use and who exhibit greater impulsivity in several domains (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 recent data (Pursey et al., 2014), some non-obese participants reported high levels of food addiction. It is possible that these individuals will progress to obesity over time; however, it is equally possible that certain protective factors (e.g., activity level, dietary choices) are operating against weight gain for these individuals and that food addiction may be a stable syndrome and not an obesity prodrome. Future longitudinal studies will be necessary to fully disentangle the relation between these two domains. If these speculations hold, protective characteristics against progression from food addiction to obesity could eventually be leveraged for obesity interventions.

These findings should be considered in the context of the study's strengths and limitations. This is the first study to concurrently examine food addiction, obesity, and three major domains of impulsivity, providing a relatively comprehensive perspective. An additional strength of the study was the use of composite variables for obesity via multiple biometric indices and for delay discounting via four reward magnitudes. High correlations and factor loadings indicate that the findings would be very similar with either an exclusive focus on BMI as the measure of obesity or the discounting function from any of the four discounting magnitudes, but the composites further demonstrate the generality of the findings, meaning that the results are unlikely to be specific to any individual measure in either domain. The inclusive sample and dimensional characterization of all variables are additional strengths, permitting greater resolution of interrelations within this sample. On the other hand, a clear limitation of the current study is that it was cross-sectional in nature. The temporal directionality of the pathway models was based on theoretical assumptions, and no causal inferences can be drawn. Future studies should investigate this presumptive pathway longitudinally. In addition, the different recruitment sources are also a pertinent methodological consideration, although this was mitigated to an extent by incorporating pertinent demographic differences into the analyses. The non-assessment of eating disorders is also a limitation that should be considered in future studies. Finally, delay discounting can be assessed for both money and consumable reinforcers, such as food (e.g., Hendrickson & Rasmussen, 2013; Hendrickson & Rasmussen, 2016), but the current study only assessed the former. As a result, differences in the strength of the relations between money and food discounting and the eating-related variables could not be examined.

In sum, the current study provides further support for the hypothesis that certain facets of impulsivity - acting rashly during intense mood states and steeply discounting future rewards – are relevant to obesity, but that the relation is an indirect one, by way of associations with food addiction. Whether these reflect longitudinal etiological processes is not clear, but the findings underscore the need for further work untangling and dismantling these relations to improve our understanding of the causes of obesity, and to further clarify the relation between obesity and food addiction.

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GENERAL SCIENTIFIC SUMMARY

Applying insights on drug addiction to overconsumption of food, this study investigated multiple forms of impulsivity in relation to obesity and “food addiction,” a novel syndrome with parallel symptoms to substance use disorders. The results revealed that two aspects of impulsivity - proneness to act out during high levels of emotion and steep discounting of future rewards - were significantly associated with food addiction. Mechanistic analyses suggested that these relations were responsible for the associations between the impulsivity variables and obesity.

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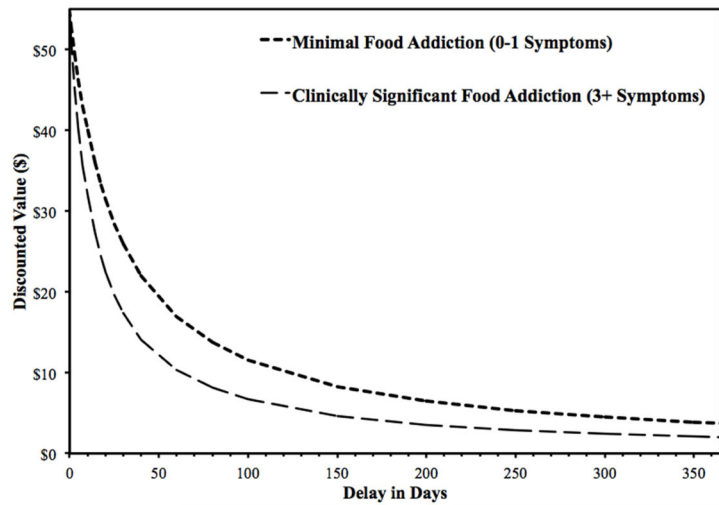


Figure 1. Illustrative Delay Discounting Curves for Monetary Rewards in Individuals Who Reported Minimal Versus Clinically Significant Levels of Food Addiction
Note: Delay discounting of future medium magnitude monetary rewards (average reward amount of \$55) in participants who endorsed minimal (i.e., 0–1) YFAS symptoms and those who endorsed clinically significant (i.e., three or more) YFAS symptoms.

Table 1

Sample Characteristics

Variable	%/Mean (SD)
Demographic	
Sex (% Female)	71.3
Age	24.80 (9.45)
Race	
White	63.0
Black/African American	23.2
Asian/Pacific Islander	7.7
American Indian/Alaskan Native	.6
Mixed	5.5
Years of Education	13.62 (2.12)
Income (Median)	\$60,000 – 74,999
Biometric	
Height (in.)	66.41 (3.64)
Weight (lb.)	176.29 (61.43)
Body Fat (%) ^b	29.42 (13.35)
Waist-to-Hip Ratio	.89 (.09)
Neck Circumference (in.)	14.47 (2.06)
Weight and Eating	
Body Mass Index	28.01 (9.06)
Obesity Status	31.5
YFAS Symptom Count	2.16 (1.45)
Food Addiction Positive Status	6.6
Impulsivity	
UPPS-P	
Urgency ^a	2.01 (.52)
Premeditation (lack of)	1.89 (.48)
Perseverance (lack of)	1.85 (.46)
Sensation Seeking	2.79 (.63)
Go/No-go Task	
Commission Errors	.35 (.19)
Omission Errors	.04 (.08)
Go Trial Reaction Time	334.35 (73.46)
Delay Discounting	
DDT <i>k</i> : \$100	-1.81 (.87)
MCQ <i>k</i> : \$30	-1.53 (.66)
MCQ <i>k</i> : \$55	-1.73 (.70)
MCQ <i>k</i> : \$80	-1.95 (.76)

Notes. For discounting variables, monetary amounts listed reflect the average reward amount within the DDT or MCQ magnitude. *SD* = standard deviation; in. = inches; lb. = pounds; Obesity Status = percent of sample/subsample with BMI > 29.99; YFAS = *Yale Food Addiction Scale*; Food

Addiction Positive Status = percent of sample/subsample endorsing three or more YFAS symptoms and significant impairment or distress; UPPS-P = *UPPS-P Impulsive Behavior Scale*; DDT = 90-item delay discounting task; MCQ = *Monetary Choice Questionnaire*.

^aA composite variable was used consisting of the mean of UPPS-P Negative Urgency and Positive Urgency scale scores.

N = 181;

^b*n* = 177.

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Principal Component Loadings and Zero-order Correlations among Variables for the Obesity and Delay Discounting Composite Variables. Loadings are in columns 1 and 7

Table 2

Biometric	1.^a	2.	3.^a	4.	5.
1. Obesity ^d	-	-	-	-	-
2. BMI	.97***	-	-	-	-
3. Body Fat % ^d	.87***	.85***	-	-	-
4. Waist Circumference	.97***	.93***	.77***	-	-
5. Hip Circumference	.96***	.89***	.85***	.91***	-
6. Neck Circumference	.83***	.79***	.52***	.85***	.70***
Monetary Discounting	7.	8.	9.	10.	11.
7. Delay Discounting	-	-	-	-	-
8. DDT k: \$100	.86***	-	-	-	-
9. MCQ k: \$30	.88***	.66***	-	-	-
10. MCQ k: \$55	.93***	.74***	.77***	-	-
11. MCQ k: \$80	.90***	.67***	.72***	.81***	-

Notes. The associations between the individual variables and the obesity or delay discounting composite reflect component loadings. Monetary amounts listed reflect the average reward amount within the DDT or MCQ magnitude. *M* = mean; *SD* = standard deviation; DDT = 90-item delay discounting task; MCQ = *Monetary Choice Questionnaire*.

N = 181;

^a *n* = 177.

*** *p* < .001.

Zero-Order Correlations Among Demographic Variables, the Obesity Composite, and Food Addiction

Table 3

	1.	2.	3.	4.	5.	6. ^b
1. Gender	-	-	-	-	-	-
2. Age	-.13	-	-	-	-	-
3. Race	-.02	.11	-	-	-	-
4. Education	-.08	.32 ^{***}	-.04	-	-	-
5. Income	.12	-.52 ^{***}	-.37 ^{***}	-.20 ^{**}	-	-
6. Obesity ^{a,b}	-.03	.53 ^{***}	.21 ^{**}	.11	-.38 ^{***}	-
7. Food Addiction (YFAS)	.07	.20 ^{**}	.21 ^{**}	-.06	-.31 ^{***}	.31 ^{***}

Note. YFAS = Yale Food Addiction Scale total symptom count.

^a A composite variable was used consisting of BMI, percent adiposity, and waist, hip, and neck circumferences.

N= 181;

^b n = 177.

* p<.05;

** p<.01;

*** p<.0001.

Table 4

Zero-order Correlations among Indices of Obesity, Food Addiction, and Impulsivity

	1. ^a	2.	3.	4.	5.	6.	7.	8.	9.
1. Obesity ^{a,b}	-	-	-	-	-	-	-	-	-
2. Food Addiction (YFAS)	.31 ^{***}	-	-	-	-	-	-	-	-
3. Urgency ^a	.09	.24 ^{**}	-	-	-	-	-	-	-
4. Premeditation (lack of)	-.09	.08	.44 ^{***}	-	-	-	-	-	-
5. Perseverance (lack of)	.08	.18 [*]	.45 ^{***}	.39 ^{***}	-	-	-	-	-
6. Sensation Seeking	-.33 ^{***}	-.21 ^{**}	.09	.25 ^{***}	-.13	-	-	-	-
7. GNG Commission Errors	-.10	-.03	.12	.06	.16 [*]	-.09	-	-	-
8. GNG Omission Errors	.25 ^{***}	.10	.20 ^{**}	-.10	-.02	-.06	.17 [*]	-	-
9. GNG Go Trial RT	.39 ^{***}	.07	.06	-.14	-.12	.01	-.50 ^{***}	.28 ^{***}	-
10. Delay Discounting ^a	.08	.21 ^{**}	.03	.01	-.09	-.10	.00	.15 [*]	.02

Notes. YFAS = *Yale Food Addiction Scale* total symptom count; GNG = Go/No-go Task; RT = reaction time.

^a A composite variable was used (Obesity = BMI, percent adiposity, and waist, hip, and neck circumferences; Urgency = UPPS-P Negative Urgency and Positive Urgency scale scores; Delay Discounting = the four individual delay discounting indices).

N = 181;

^b n = 177.

* p < .05;

** p < .01;

*** p < 0.001.

Hierarchical Regressions Predicting Food Addiction and the Obesity Composite from Impulsivity Variables, with Age and Income as Covariates

Table 5

	Food Addiction			Obesity ^{a,b}		
	B (SE)	β	p	B (SE)	β	p
<i>Impulsive Personality Traits</i>						
Urgency ^d						
Age	.01 (.01)	.04	.59	.05 (.01)	.46	<.001
Income	-.13 (.04)	-.26	.002	-.05 (.03)	-.14	.07
Urgency ^d	.57 (.20)	.20	.004	.09 (.12)	.05	.46
Premeditation (lack of)						
Age	.01 (.01)	.07	.44	.05 (.01)	.45	<.001
Income	-.14 (.04)	-.28	.001	-.05 (.03)	-.14	.05
Premeditation	.27 (.22)	.09	.21	-.09 (.13)	-.04	.52
Perseverance (lack of)						
Age	.01 (.01)	.05	.65	.05 (.01)	.46	<.001
Income	-.13 (.04)	-.26	.002	-.05 (.03)	-.14	.07
Perseverance	.45 (.23)	.14	.049	.10 (.14)	.05	.47
Sensation Seeking						
Age	.00 (.01)	-.01	.96	.05 (.01)	.41	<.001
Income	-.13 (.04)	-.27	.001	-.05 (.03)	-.13	.07
Sensation Seeking	-.32 (.18)	-.14	.07	-.23 (.11)	-.15	.03
<i>Impulsive Action</i>						
Commission Errors						
Age	.01 (.01)	.05	.52	.05 (.01)	.46	<.001
Income	-.14 (.04)	-.28	.001	-.05 (.03)	-.14	.06
Commission Errors	.05 (.54)	.01	.93	.00 (.34)	.00	1.00
Omission Errors						
Age	.01 (.01)	.05	.56	.05 (.01)	.44	<.001
Income	-.13 (.04)	-.27	.001	-.04 (.03)	-.12	.09

	Food Addiction			Obesity ^{a,b}		
	B (SE)	β	p	B (SE)	β	p
Omission Errors	.76 (1.37)	.04	.58	1.97 (.83)	.15	.02
Go Trial Reaction Time						
Age	.01 (.01)	.05	.53	.04 (.01)	.39	<.001
Income	-.14 (.04)	-.28	.001	-.04 (.02)	-.13	.08
Go Trial RT	.00 (.00)	.00	.98	.00 (.00)	.24	<.001
				<i>Impulsive Choice</i>		
Delay Discounting ^a						
Age	.01 (.01)	.06	.48	.05 (.01)	.46	<.001
Income	-.11 (.04)	-.24	.004	-.05 (.03)	-.14	.07
Delay Discounting ^a	.23 (.10)	.16	.03	.03 (.07)	.03	.68

Notes: B = unstandardized coefficient; SE = standard error; β = standardized coefficient; p values <.05 are considered significant.

^aA composite variable was used (Obesity = BMI, percent adiposity, and waist, hip, and neck circumferences; Urgency = UPPS-P Negative Urgency and Positive Urgency scale scores; Delay Discounting = the four individual delay discounting indices).

N = 181;

^bn = 177.

Table 6

Combined Hierarchical Regression Predicting Food Addiction from the Urgency and Delay Discounting Composites

Predictors	R^2	B (SE)	β	p
Step 1	.10 ***			
Age		.01 (.01)	.05	.53
Income		-.14 (.04)	-.28	.001
Step 2	.07 *			
Age		.01 (.01)	.05	.55
Income		-.11 (.04)	-.23	.007
Urgency ^a		.29 (.10)	.20	.004
Delay Discounting ^a		.23 (.10)	.16	.03

Notes. Z-scores for the Urgency and delay discounting composites were used for this analysis. B = unstandardized coefficient; SE = standard error; β = standardized coefficient; p values <.05 are considered significant.

^aA composite variable was used (Urgency = UPPS-P Negative Urgency and Positive Urgency scale scores; Delay Discounting = the four individual delay discounting indices).

* $p < .05$,

*** $p < .005$,

$N = 181$.

Mediation Models Results

Table 7

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:					
Urgency ^a → Food Addiction (YFAS) → Obesity ^a	b(YX) .09				
	b(MX) .57**				
	b(YM.X) .15**	.32***	.08	.02	.19
	b(YX.M) .01				
	Age .05***				
	Income -.03				
Model 2:					
Delay Discounting ^a → Food Addiction (YFAS) → Obesity ^a	b(YX) .03				
	b(MX) .26*				
	b(YM.X) .15**	.32***	.04	.01	.09
	b(YX.M) -.01				
	Age .05***				
	Income -.03				

Note: N = 177. Number of bootstrapped resamples = 5000. X = independent variable; M = mediator; Y = dependent variable; YX = direct effect of X on Y; MX = direct effect of X on M; YM.X = direct effect adjusting for the mediator; YM.X = indirect (mediating) effect; BC = bias-corrected; CI = confidence interval; YFAS = Yale Food Addiction Scale total symptom count.

^a A composite variable was used (Obesity = BMI, percent adiposity, and waist, hip, and neck circumferences; Urgency = UPPS-P Negative Urgency and Positive Urgency scale scores; Delay Discounting = the four individual delay discounting indices).

* P<.05;

** P<.01;

*** P<.001.