



Research report

Preliminary validation of the Yale Food Addiction Scale

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ABSTRACT

Previous research has found similarities between addiction to psychoactive substances and excessive food consumption. Further exploration is needed to evaluate the concept of “food addiction,” as there is currently a lack of psychometrically validated measurement tools in this area. The current study represents a preliminary exploration of the Yale Food Addiction Scale (YFAS), designed to identify those exhibiting signs of addiction towards certain types of foods (e.g., high fat and high sugar). Survey data were collected from 353 respondents from a stratified random sample of young adults. In addition to the YFAS, the survey assessed eating pathology, alcohol consumption and other health behaviors. The YFAS exhibited adequate internal reliability, and showed good convergent validity with measures of similar constructs and good discriminant validity relative to related but dissimilar constructs. Additionally, the YFAS predicted binge-eating behavior above and beyond existing measures of eating pathology, demonstrating incremental validity. The YFAS is a sound tool for identifying eating patterns that are similar to behaviors seen in classic areas of addiction. Further evaluation of the scale is needed, especially due to a low response rate of 24.5% and a non-clinical sample, but confirmation of the reliability and validity of the scale has the potential to facilitate empirical research on the concept of “food addiction”.

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Obesity continues to grow as a major health risk to not only the American public, but globally (Mokdad, Marks, Stroup, & Gerberding, 2004) and the sustainability of weight loss with modern treatments remains a challenge. Widespread prevention efforts have had limited success in the long term. Society’s strong motivation to lose weight combined with the tremendous amount of energy and resources spent on the “obesity epidemic” suggests that the problem of obesity is not driven by a lack of motivation or effort. Evocative research from the addiction and nutrition fields has recently uncovered similarities in patterns of food intake and consumption of drugs of abuse. These findings have led to the controversial theory that some foods, or things added to foods, may trigger an addictive process. This may help more fully explain the difficulty people experience in adhering to healthier food choices.

Most of the evidence for food’s addictive properties lies in the biological realm. There is now considerable evidence that food and drugs of abuse exploit similar pathways in the brain, namely the dopamine and opiate systems (Hoebel, Rada, Mark, & Pothos, 1999; Nieto, Wilson, Cupo, Roques, & Noble, 2002). Although dopamine release is not equivocal to addictive properties, dopamine has been associated with the perceived value of reward of both food and psychoactive substances. The more rewarding the food or drug is evaluated to be, the greater the release of extracellular dopamine in

the nucleus accumbens (Volkow et al., 2002). Further, lesions of the dopaminergic system or pharmacological blockade of dopamine receptors reduce the reward value of both sugar rich foods and drugs of abuse (Avena & Hoebel, 2003; Colantuoni et al., 2001, 2002). Positron emission tomographic (PET) imaging studies have also shown that both obese individuals and drug dependent individuals have significantly lower dopamine receptor levels (Wang et al., 2001). Similarly, the opiate system has been implicated in both drug and food consumption. Consumption of both alcohol and food, especially high fat sweets, can cause endogenous opiates to be released in the brain (Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1995), and opiate blockers such as naloxone can reduce the reinforcement value and craving for alcohol in dependent participants (O’Malley, Krishnan-Sarin, Farren, Sinja, & Kreek, 2002). Naloxone also reduces consumption and preference for sweet high fat foods in both normal weight and obese binge eaters (Drewnowski et al., 1995). Although there are many biological similarities between food and drug consumption, there are also important differences. Pleasurable food activates the brain through fast sensory signals and through slow ingestion processes, such as increasing glucose in the brain. In contrast, drugs activate the same reward system through direct pharmacological effects (Volkow & Wang, 2005). Regardless of these differences, the similar neurobiological processes that result from food and drug activation provide support for the concept of food addiction.

Animal research has also linked sugar consumption with behavioral indicators of dependence (Avena & Hoebel, 2003; Avena,

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Long, & Hoebel, 2005; Colantuoni et al., 2002; Gosnell, 2005; Hoebel et al., 1999; Rada, Avena, & Hoebel, 2005). Rats maintained on an intermittent diet of sucrose solution and chow show behavioral and neurochemical changes similar to rats dependent on drugs (Hoebel et al., 1999). Rats on daily intermittent sucrose slowly increased their sugar consumption from 37 to 112 ml per day, possibly reflecting a tolerance effect (Rada et al., 2005). Removal of sugar from dependent animals results in a drop in body temperature and behavioral changes associated with withdrawal, such as anxious and agitated actions (Colantuoni et al., 2001). Finally, locomotor cross-sensitization with amphetamines and cocaine and an increased motivational tendency towards ethanol occurs in sugar-binging rats (Avena & Hoebel, 2003; Gosnell, 2005).

Although the biological support for “food addiction” is impressive, there have been few rigorous explorations of the behavioral indicators of dependence in humans. Initial evidence has come from observations of similarities between substance dependence and eating behavior. Gold, Frost-Pineda, and Jacobs (2003) found that the majority of the substance dependence diagnostic criteria are similar to the criteria necessary to meet a diagnosis of binge eating disorder, such as a loss of control over consumption and an inability to successfully stop or cut down on consumption despite an expressed desire to do so. Others note that, similar to individuals with substance dependence, some obese individuals continue to eat unhealthy foods even in the face of severe negative consequences, such as diabetes, heart disease, and stigmatization (Volkow & O'Brien, 2007). There is also some evidence for development of tolerance to food, as suggested by the changing patterns of binge eating in those struggling with bulimia nervosa. As illness duration grows longer, the frequency of binges, the amount of food consumed, the length of the episode, and the feeling of being out of control all increase (Brown, Spanos, Devlin, & Walsh, 2007). Although little empirical work has been done on the existence of withdrawal from certain types of food, one case study documented the re-emergence of anxiety and panic symptoms when a patient went on a low-carbohydrate diet, the Atkins diet (Ehrenreich, 2006). Atkins himself warned dieters that they may experience “fatigue, faintness, palpitations, headaches, and cold sweats” when reducing carbohydrates, but did not offer data to support this claim (Atkins, 2002.)

Despite limited research on the topic, reference to “food addicts” has started to appear in the literature. One study found that “chocolate addicts” had physical, behavioral, and emotional responses to chocolate that were similar to drug addicts' responses to drug cues (Tuomisto, Lappalainen, Heterington, Morris, & Tuomisto, 1997). Although, these findings are intriguing, self-identification was used to classify the group of “chocolate addicts.” Self-identification may be especially problematic, as those who are dependent often lack insight into the existence or extent of their problems (Farid, Clark, & Williams, 1998). The concept of food addiction has also made it into popular culture through weight loss books, groups, and programs that are based upon principles of addiction. The development of validated tools to precisely assess the construct of food addiction appears to be the next logical step. Our aim is to help fill this gap by developing a tool to operationalize “food addiction,” to establish its convergent, discriminant, and incremental validity, and to begin the examination of how some eating behavior might map onto the diagnostic criteria for substance dependence.

Method

Participants

A total of 1440 participants were randomly selected from the undergraduate population of a private university in the northeast.

Participants were stratified by gender and year in school to provide a generally representative sample. Three hundred and fifty-three participants initiated the survey, for a response rate of 24.5%. Of the 353 who initiated the study, 233 (66.0%) completed relevant measures for this study. The sample of participants included in the data analyses had an average age of 20.11 (S.D. = 1.38); 72.5% were Caucasian, 18.5% were Asian-American, and 9.0% were African American. Women (64.2%) had a higher response rate than men (35.8%) overall and were more likely to complete all measures. The average self-reported body mass index (BMI) of the participants was 22.58 (S.D. = 3.18) with the majority of participants falling within a normal weight range (73.5%). Few participants were obese (2.7%) or underweight (4.7%), although a significant percentage of participants did have a BMI that placed them into the overweight category (18.7%).

Measures

Binge Eating Scale (BES, Gormally, Black, Daston, & Rardin, 1982). The BES is a 16-item questionnaire designed to describe both behavioral manifestations (e.g., eating large amounts of food) and feelings/cognitions surrounding a binge episode (e.g., guilt, fear of being unable to stop eating). The BES is successful in differentiating between those with no, moderate, and severe binge eating tendencies (Gormally et al., 1982). Internal reliability in the current sample was excellent (Cronbach's $\alpha = .93$).

BIS/BAS Reactivity (BIS/BAS, Carver & White, 1994). The BIS/BAS is a measure used to assess Gray's Behavioral Inhibition (BIS) and Behavioral Approach Systems (BAS; Carver & White, 1994). The instrument is a 20-item self-report measure from which one can derive a global BIS score and three BAS related subscale scores: Drive (DRV), Fun Seeking (FS), and Reward Responsiveness (RR). In the current sample, the BIS scale demonstrated an internal reliability of .78 and the BAS scales had an average internal reliability of .71.

Eating Troubles Module (EAT-26, Garner, Olmsted, Bohr, & Garfinkle, 1982). The EAT-26 is based on the Eating Attitudes Test and is a widely used standardized measure of the extent of symptoms and concerns characteristic of eating disorders. Scores equal to or greater than 20 are indicative of high eating disorder risk. The EAT-26 exhibited an internal reliability of .91.

Emotional Eating Scale (EES; Arnow, Kenardy, & Agras, 1995). The EES is used to assess overeating initiated by emotional stimuli. The 25-item adjective checklist asks respondents to rate, on a 5-point Likert scale, the degree to which each mood state generates a desire to undereat (moderately or greatly), has no effect on eating, or generates a desire to overeat (moderately or greatly). The scale has good construct, discriminant, and criterion validity (Arnow et al., 1995), as well as an internal reliability of .95 in the current sample.

Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989). The RAPI is a 23-item scale used to assess adolescent and young adult problem drinking in a unidimensional and brief fashion. The RAPI had an internal reliability of .88 in the current sample.

Daily Drinking Questionnaire (DDQ; Collins, Parks, & Marlatt, 1985). We used a revised version of the DDQ that asked separate questions about frequency (drinking days per week) and quantity (number of drinks per drinking day) to better differentiate these aspects of drinking behavior. The DDQ has demonstrated convergent validity with other college student drinking measures, ranging from .50 to .60 (Baer, Stacy, & Latimer, 1991; Collins et al., 1985; Corbin, Morean, & Benedict, 2008; Larimer, Irvine, Kilmer, & Marlatt, 1997) and had an internal reliability of .83 in the current study.

Table 1
DSM-IV-TR substance dependence criteria.

(1) Substance taken in larger amount and for longer period than intended
(2) Persistent desire or repeated unsuccessful attempt to quit
(3) Much time/activity to obtain, use, recover
(4) Important social, occupational, or recreational activities given up or reduced
(5) Use continues despite knowledge of adverse consequences (e.g., failure to fulfill role obligation, use when physically hazardous)
(6) Tolerance (marked increase in amount; marked decrease in effect)
(7) Characteristic withdrawal symptoms; substance taken to relieve withdrawal

Procedures

Measurement development. The content of the Yale Food Addiction Scale (YFAS) is composed of questions based upon substance dependence criteria in the DSM-IV-TR (American Psychiatric Association [DSM-IV-TR], 2000) and scales used to assess behavioral addictions, such as gambling, exercise, and sex, including the South Oaks Gambling Screen (Lesieur & Blume, 1987), the Exercise Dependence Scale (Hausenblas & Downs, 2002), and the Carnes' Sexual Addiction Screening Tool (Carnes, 1989). Questions were adapted to assess the full range of diagnostic criteria related to the consumption of high fat and high sugar foods.

The original pool of items was developed by the authors prior to review by experts in the addiction, obesity and eating pathology fields. The experts were asked to review item content and question wording and to indicate any criteria that they believed were not adequately assessed. In addition, clinical patients in treatment for binge eating reviewed the scale for relevance and clarity. After making revisions based on feedback from both experts and patients, two to four questions were selected for each of the DSM-IV-TR criteria for substance dependence (see Table 1). The same groups of experts then reviewed the revised version of the scale to identify any problems related to content and scoring. In addition to the items related to each of the seven dependence criteria, clinical significance was assessed with two questions assessing the extent to which eating behavior causes significant impairment or leads to significant distress for the individual (see Table 2).

Various scoring options, including dichotomous, frequency, and Likert scale options were considered. A combination of dichotomous and frequency scoring were deemed to be the most appropriate to capture the diagnostic criteria. Frequency scoring was used to assess behaviors that could plausibly occur occasionally in non-problem eaters (i.e.—criteria associated with excess consumption, emotional eating, dieting). Dichotomous scoring was used for questions that were considered more severe, and thus would likely indicate eating problems (i.e.—continuing to consume foods in a certain way in the face of emotional or physical problems). Instructions for completing the measure made reference specifically to high fat and high sugar foods. These food types are highly preferred by individuals with eating problems and are most often reported in food binges (Allison & Timmerman, 2007; Drenowski, 1995; Drenowski, Kurth, Holden-Wiltse, & Saari, 1992; Kales, 1990).

The scale was distributed as part of a larger health behaviors survey that gathered information about alcohol and drug use, eating behavior, partner violence, and gambling from participants 18 years and older at a private college in the northeast. Participation in the survey was voluntary and informed consent was obtained from all qualifying participants. Information was collected through a web-based survey program, Survey Monkey. Questionnaires could be completed at the participants' discretion at any Internet-wired computer. All individuals who completed the consent form were entered into a lottery for gift certificates worth \$50 with odds of winning at approximately 1 in 40. One thousand four hundred and forty participants were randomly selected from the undergraduate directory to provide a representative sample of

the campus and were contacted via e-mail asking them to participate in a 30–50-min survey on a secure web server. Due to survey programming, participants were only able to complete the survey once. All questions required a response to move on to the next page of the survey and each question included a response option of “I choose not to answer” with the exception of the question regarding participant age (an inclusion criteria). Participants could discontinue the survey at any time without losing eligibility for the prize drawing.

Data management

All relevant scales were examined for missing values. Although 233 participants completed all relevant measures, many participants had missing values for one or more of the items on these measures. In particular, 16 participants missed at least one item on the BES and 49 participants missed at least one item on the EES. To maintain as many participants as possible for data analyses, the sum of all available scores on the BES scale was calculated for all participants who completed at least 14 of 16 (87.5%) of the scale questions. The sum was then divided by the number of completed items to yield a mean BES score. A similar procedure was used for the EES, with mean scores calculated for all participants who completed at least 21 of 25 (84%) of the scale questions. Due to remaining missing values, sample sizes for data analyses ranged from 221 to 187. Prior to data analyses, distributions of all summary scores were examined for normality and outliers. No outliers were identified for removal, but the count version of the YFAS, the RAPI, the EAT-26, and the EES all exhibited moderately positively skewed distributions. Analyses were conducted with both the original data and with log-transformed data (Tabachnick & Fidell, 2005). The results were equivalent in both cases, thus the original form was kept for ease of interpretation.

Data analytic plan

Initially, diagnostic thresholds for the continuous items on the YFAS were determined by examining the distributions and scatter plots in relation to established measures of eating pathology. Next, the factor structure of the individual items and the seven dichotomous diagnostic criteria was examined using exploratory factor analysis. Internal reliability (α) of the resulting factors was assessed. Analyses were then conducted to establish the convergent, discriminant, and incremental validity of the scale. Convergent validity was assessed by examining correlations between the YFAS and other well established predictors of eating pathology. Discriminant validity was assessed by examining correlations between the YFAS and well-validated measures of alcohol use, alcohol-related problems, and impulsivity. Although comorbidity of alcohol use disorders and certain eating disorders is relatively common (Dansky, Brewerton, & Kilpatrick, 2000) and impulsivity has been found to correlate positively with certain eating disorders (Fahy & Eisler, 1993), only modest correlations were expected between YFAS scores and these related but distinct constructs. Incremental validity was investigated using multiple regression. The YFAS and other established measures of eating pathology were entered simultaneously as predictors of scores on the BES, to determine if the YFAS accounted for unique variability in eating-related problems.

Results

Establishing thresholds for diagnostic criteria on the YFAS

For the continuously scored items on the YFAS, rates of endorsement at different cut-offs were examined to identify possible

Table 2

Sample questions from the Yale Food Addiction Scale.

IN THE PAST 12 MONTHS:				
1) I find that when I start eating certain foods, I end up eating much more than I had planned.				
Never	Once a month or less	Two to four times a month	Two to three times per week	Four or more times per week or daily
2) Not eating certain types of food or cutting down on certain types of food is something I worry about.				
Never	Once a month or less	Two to four times a month	Two to three times per week	Four or more times per week or daily
3) I spend a lot of time feeling sluggish or lethargic from overeating.				
Never	Once a month or less	Two to four times a month	Two to three times per week	Four or more times per week or daily
4) There have been times when I consumed certain foods so often or in such large quantities that I spent time dealing with negative feelings from overeating instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy.				
Never	Once a month or less	Two to four times a month	Two to three times per week	Four or more times per week or daily
5) I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems.				
Yes			No	
6) Over time, I have found that I need to eat more and more to get the feeling I want, such as reduced negative emotions or increased pleasure.				
Yes			No	
7) I have had withdrawal symptoms when I cut down or stopped eating certain foods. (Please do NOT include withdrawal symptoms caused by cutting down on caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.) For example: Developing physical symptoms, feeling agitated, or feeling anxious				
Never	Once a month or less	Two to four times a month	Two to three times per week	Four or more times per week or daily
8) My behavior with respect to food and eating causes significant distress.				
Never	Once a month or less	Two to four times a month	Two to three times per week	Four or more times per week or daily
9) I experience significant problems in my ability to function effectively (daily routine, job/school, social activities, family activities, health difficulties) because of food and eating.				
Never	Once a month or less	Two to four times a month	Two to three times per week	Four or more times per week or daily

Note: For the complete scale and scoring instructions please contact the corresponding author.

cut-offs that would not over or under identify participants with eating-related problems. Based on the extant literature for both substance use disorders and eating disorders, rates between 5 and 20% were considered optimal for diagnostic criteria (American Psychiatric Association [DSM-IV-TR], 2000). Scatter plots were then created to explore the relation between scores on YFAS items and increased risk for eating pathology (BES, EAT-26, EES, and BMI). The goal was to identify cut-offs that were most clearly associated with increased risk for eating pathology. The combination of these methods converged to identify the most appropriate cut-off values for creating dichotomous variables. Using the dichotomous items, each of the seven diagnostic criteria was considered to have been met if one or more item representing the criteria was endorsed. Two different summary scores were created based on the diagnostic criteria; a dichotomous diagnosis (yes/no) and a symptom count (0–7). For the diagnostic version, which resembles a diagnosis of substance dependence, criteria were considered met if participants endorsed three or more criteria as well as at least one of the two clinical significance items (impairment or distress). The symptom count score was a simple sum of the seven diagnostic criteria. The median number of criteria met for this sample was 1 and 11.4% of participants in the sample met criteria for “food dependence,” which is similar to the percent of participants who met suggested clinical cut-offs on the BES (13.2%) and the EAT-26 (13.4%).

Factor structure and reliability

An exploratory factor analysis for dichotomous data was conducted using the Mplus statistical package (Muthén & Muthén, 1998–2004) to explore the number of underlying factors based on the original 21 items included in the YFAS (not including the clinical significance questions). One item did not correlate strongly with the remaining items on the scale with a low factor loading of .33 and was therefore removed from the analysis. This item (failed attempts to cut down on consumption of high fat/high sugar foods) was endorsed by many participants (41.6%), which may have contributed to the low correlation with other items that were infrequently endorsed. Factor analysis with the remaining items identified four factors based on eigenvalues (11.12, 1.99, 1.46, 1.07). Although four factors were identified based on eigenvalues greater than one, plotting of the factors suggested a single factor structure. All items had factor loadings for the single factor of .50 or higher, resulting in good internal reliability (Kuder–Richardson $\alpha = .86$). A parallel factor analysis was conducted for the seven dichotomous diagnostic criteria and again a single factor structure was identified. All criteria had factor loadings for the single factor of .69 or higher. Internal reliability for the single factor was adequate (Kuder–Richardson $\alpha = .75$), for such a brief measure.

Convergent, discriminant, and incremental validity

Convergent validity of the scale was established by examining correlations between scores on the YFAS and other measures relevant to eating behavior (emotional eating and eating troubles scores). Correlations with both the count measure and the

Table 3
Convergent validity.

	EAT-26	EES
Symptom count	.46**	.51**
Diagnosis	.61**	.46**

Note: FAS, Food Addiction Scale; EAT-26, Eating Attitudes Test-26; EES, Emotional Eating Scale.

** Correlation is significant at the .01 level (one-tailed).

Table 4
Discriminant validity.

	DDQ frequency	DDQ quantity	RAPI	BAS total	BIS total
Symptom count	-.063	-.032	.170 [†]	.115	.348**
Diagnosis	.042	.072	.158 [†]	.128	.248**

Note: FAS, Food Addiction Scale; DDQ, Daily Drinking Questionnaire; RAPI, Rutgers Alcohol Problem Index; BAS, Behavioral Activation System; BIS, Behavioral Inhibition System.

[†] Correlation is significant at the .05 level (two-tailed).

** Correlation is significant at the .01 level (one-tailed).

diagnostic measure of the YFAS were statistically significant and ranged from .46 to .61 (see Table 3). Discriminant validity was assessed by evaluating correlations between YFAS scores and scores on measures of related but independent constructs (alcohol use and related problems and impulsivity). No significant correlations were observed between YFAS scores (diagnostic or count) and a measure of alcohol consumption. Small but statistically significant correlations of .16 and .17 were observed between YFAS scores (diagnostic and count) and alcohol problems. A small but significant correlation was also observed between the BIS (behavioral inhibition scores) and both the YFAS count scores (.35) and the YFAS diagnostic scores (.25), but BAS (behavioral activation) scores were not significantly correlated with YFAS scores (see Table 4).

Incremental validity was assessed using hierarchical multiple regression. YFAS scores were entered along with other measures of eating pathology (emotional eating and eating troubles) as predictors of binge eating pathology (BES scores). Problem eating attitudes (EAT-26) and emotional eating (EES) were entered in step one of the regression model with YFAS scores entered in block 2. The EAT 26, $t = 6.98$, $\beta = .37$, $p < .01$, and EES, $t = 9.77$, $\beta = .53$, $p < .001$, were both significant predictors of the continuous binge eating measure, accounting for 49.9% of the variance. After controlling for variance accounted for at step one of the model, the symptom count version of the YFAS was a significant predictor in step two of the model, $t = 9.05$, $\beta = .48$, $p < .001$, accounting for an additional 14.8% of unique variance in binge eating scores.

Similar results were found for the diagnostic version of the scale. Eating pathology, $t = 7.15$, $\beta = .38$, $p < .001$, and emotional eating scores, $t = 9.64$, $\beta = .51$, $p < .001$, were significant predictors of binge eating, accounting for 47.4% of the variance in step one of the regression model. After controlling for variance accounted for in step one, the diagnostic version of the YFAS was a significant predictor at step two of the model, $t = 5.05$, $\beta = .33$, $p < .001$, accounting for 5.8% of the unique variance.

Discussion

The goal of the current study was to develop a psychometrically sound tool for use in future food addiction studies and to determine the extent to which this new measure accounts for unique variability in eating-related problems. Factor analysis identified a single factor structure for the YFAS and Cronbach's α indicated adequate reliability of the single factor scale. Both the symptom count and diagnostic version of the YFAS had high convergent validity with other eating pathology measures, and discriminant validity with alcohol and behavioral inhibition and activation measures. Finally, both versions of the YFAS demonstrated incremental validity, accounting for unique variability in binge eating behavior. Thus, preliminary analyses of the YFAS suggest that it may be a useful tool to identify those with addictive tendencies toward food.

Although this study represents only a preliminary validation of the YFAS, this is a critical step to advancing the literature on the topic of food addiction. The exploration of “food addiction” in

humans is in its nascent stages. Future projects that hope to explore the concept of “food addiction” will benefit from having a more sound assessment tool. For example, the YFAS could be used in behavioral, psychophysiological, weight loss, dietary, and neuroimaging studies to further explore whether food addiction is a valid and useful concept. The use of a validated tool may increase confidence in the methodological rigor of such studies, thus contributing to the “food addiction” literature. It will be especially important to distinguish between those who simply indulge in unhealthy foods and those who have truly lost control over their eating behavior.

Specifying whether food can trigger an addictive process has potentially important implications. Empirically supported treatments for at least some individuals with pathological eating issues might be designed to account for such a process. This may be especially relevant as “food addiction” treatments with no empirical support are already being marketed to and relied upon by the public. Further, the constant advertising and ubiquitous nature of unhealthy foods may be taking advantage of cue-triggered relapse to derail public health interventions designed to decrease obesity. Unlike drugs, the importance of sensory processes in food consumption (Volkow & Wang, 2005) may result in food advertising having an increased tendency to trigger overconsumption. In addition, there could be social and even legal implications for the heavy marketing of certain foods to children, the promotion of such foods in schools, etc., but first there must be robust science showing that food can be addictive.

Although the current study has potentially important implications for both treatment and prevention, the results must be interpreted in light of several limitations. First, the scale was validated in a population of college students, making it important to examine validity in other populations. In addition, the response rate of 24.5%, leaves open the possibility that the sample is not representative of the original population from which it was drawn. However, in using a random sample of students, the study sample is likely to be more representative than if a convenience sample (e.g., subject pool) were used, despite the relatively low response rate. Nonetheless, external validity needs further attention now that the psychometric properties of the YFAS have been examined in a college sample. In particular, the current sample had few obese participants, limiting our ability to draw conclusions about the existence of an addictive process within this population. Additionally, BMI was not directly measured, which increases the possibility that body weight was underreported and height was overreported (Larson, Ouwens, Engels, Eisinga, & van Strien, 2008). Future studies hoping to examine food addiction and BMI would benefit from direct measurement of both height and weight. In terms of further validation of the scale, the cross-sectional study design did not permit test–retest validity to be established or allow us to conduct a confirmatory factor analysis, and the absence of dietary intake data did not allow us to examine the effects of certain macronutrients on compulsive eating behavior.

Despite the limitations of the current study, the results add important behavioral support to the literature on food and addiction and offer the initial validation of a food addiction scale. Future studies in this area may benefit from the use of such a measure to identify participants who are most likely to exhibit symptoms of dependence to certain types of food.

References

- Allison, S., & Timmerman, G. M. (2007). Anatomy of a binge: food environment and characteristics of a nonpurge binge episodes. *Eating Behaviors*, 8, 31–38.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. text revision). Washington, DC.
- Arnow, B., Kenardy, J., & Agras, W. S. (1995). The Emotional Eating Scale: the development of a measure to assess coping with a negative affect by eating. *International Journal of Eating Disorders*, 18, 79–90.
- Atkins, R. C. (2002). *Dr. Atkins' new diet revolution* (2002 edition). North Yorkshire: Guill.
- Avena, N. M., & Hoebel, B. G. (2003). A diet promoting sugar dependency causes behavioral cross-sensitization to a low dose of amphetamine. *Neuroscience*, 122, 17–20.
- Avena, N. M., Long, K. A., & Hoebel, B. G. (2005). Sugar-dependent rats show enhanced responding for sugar after abstinence: evidence of sugar deprivation effect. *Physiology & Behavior*, 84, 359–362.
- Baer, J. S., Stacy, A., & Larimer, M. (1991). Biases in the perception of drinking norms among college students. *Journal of studies on alcohol*, 52, 580–586.
- Brown, A. J., Spanos, A., Devlin, M. J., & Walsh, B. T. (2007, May). Bulimia nervosa as an addiction: evidence for a tolerance effect in patterns of binge eating over the course of illness. *Poster presented at the international conference of eating disorders*.
- Carnes, P. J. (1989). *Contrary to love*. Center City: Hazelden Publishing and Education.
- Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. *Journal of Personality and Social Psychology*, 67, 319–333.
- Colantuoni, C., Schwenker, J., McCarthy, P., Rada, P., Ladenheim, B., Cadet, J.-L., et al. (2001). Excessive sugar intake alters binding to dopamine and mu-opioid receptors in the brain. *Neuroreport*, 12, 3549–3552.
- Colantuoni, P., Rada, P., McCarthy, C., Patten, C., Avena, N. M., Chadeayne, A., et al. (2002). Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. *Obesity Research*, 10, 478–488.
- Collins, R. L., Parks, G. A., & Marlatt, G. A. (1985). Social determinants of alcohol consumption: the effects of social interaction and model status on the self-administration of alcohol. *Journal of Consulting and Clinical Psychology*, 53, 189–200.
- Corbin, W. R., Morean, M. E., & Benedict, D. (2008). The Positive Drinking Consequences Questionnaire (PDCQ): validation of a new assessment tool. *Addictive Behaviors*, 33, 54–68.
- Dansky, B. S., Brewerton, T. D., & Kilpatrick, D. G. (2000). Comorbidity of bulimia nervosa and alcohol use disorders. *International Journal of Eating Disorders*, 27, 180–190.
- Drewnowski, A. (1995). Metabolic determinants of binge eating. *Addictive Behaviors*, 20(6), 733–745.
- Drewnowski, A., Krahn, D. D., Demitrack, M. A., Nairn, K., & Gosnell, B. A. (1995). Naloxone, an opiate blocker, reduces the consumption of sweet high-fat foods in obese and lean female binge eaters. *American Journal of Clinical Nutrition*, 61, 1206–1212.
- Drewnowski, A., Kurth, C., Holden-Wiltse, J., & Saari, J. (1992). Food preferences in human obesity: carbohydrates versus fats. *Appetite*, 18, 207–221.
- Ehrenreich, M. J. (2006). A case of the re-emergence of panic and anxiety symptoms after initiation of a high-protein, very low carbohydrate diet. *Psychosomatics*, 47, 178–179.
- Fahy, T., & Eisler, I. (1993). Impulsivity and eating disorders. *The British Journal of Psychiatry*, 162, 193–197.
- Farid, B., Clark, M., & Williams, R. (1998). Health locus of control in problem drinkers with and without liver disease. *Alcohol and Alcoholism*, 33, 184–187.
- Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological Medicine*, 12, 871–878.
- Gold, M. S., Frost-Pineda, K., & Jacobs, W. S. (2003). Overeating, binge eating, and eating disorders as addictions. *Psychiatric Annals*, 33(2), 117–122.
- Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese person. *Addictive Behaviors*, 7, 47–55.
- Gosnell, B. (2005). Sucrose intake enhances behavioral sensitization produced by cocaine. *Brain Research*, 1031, 194–201.
- Hausenblas, H. A., & Downs, D. S. (2002). How much is too much? The development and validation of the exercise dependence scale. *Psychology and Health*, 17, 387–404.
- Hoebel, B., Rada, P. V., Mark, G. P., & Pothos, E. (1999). Neural systems for reinforcement and inhibition of behavior: relevance to eating, addiction and depression. *Well-being: Foundations of Hedonic Psychology*, 560–574.
- Kales, E. (1990). Macronutrient analysis of binge eating in bulimia. *Physiology and Behavior*, 48, 837–840.
- Larimer, M. E., Irvine, D. L., Kilmer, J. R., & Marlatt, G. A. (1997). College drinking and the Greek system: examining the role of perceived norms for high-risk behavior. *Journal of College Student Development*, 38(6), 587–598.
- Larson, J. K., Ouwens, M., Engels, R. C. M. E., Eisinga, R., & van Strien, T. (2008). Validity of self-reported weight and height and predictors of weight bias in female college students. *Appetite*, 50, 386–389.
- Lesieur, H. R., & Blume, S. B. (1987). The South Oaks Gambling Screen (SOGS): a new instrument for the identification of pathological gambling. *American Journal of Psychiatry*, 144, 1184–1188.
- Mokdad, A. H., Marks, J. S., Stroup, M. F., & Gerberding, J. L. (2004). Actual causes of death in the United States. *Journal of the American Medical Association*, 291(10), 1238–1245.
- Muthén, B., & Muthén, L. (1998–2004). *MPlus manual* (3rd ed.). LA: Author.
- Nieto, M., Wilson, J., Cupo, A., Roques, B. P., & Noble, F. (2002). Chronic morphine treatment modulates the extracellular levels of endogenous enkephalins in rat brain structures involved in opiate dependence: a microdialysis study. *Journal of Neuroscience*, 22, 1034–1041.
- O'Malley, S. S., Krishnan-Sarin, S., Farren, C., Sinja, R., & Kreek, M. (2002). Naltrexone decreases craving and alcohol self-administration in alcohol-dependent subjects and activates the hypothalamo–pituitary–adrenocortical axis. *Psychopharmacology*, 160, 19–29.
- Rada, P., Avena, N. M., & Hoebel, B. G. (2005). Daily bingeing on sugar repeatedly releases dopamine in the accumbens shell. *Neuroscience*, 134, 737–744.
- Tabachnick, B. G., & Fidell, L. S. (2005). *Using multivariate statistics* (5th ed.). Boston: Allyn & Bacon.

- Tuomisto, T., Lappalainen, R., Heterington, M., Morris, M. F., & Tuomisto, M. T. (1997). Affective, physiological and overt behavioral responses to chocolate in self-identified 'chocolate addicts'. *International Journal of Psychophysiology*, 25, 38–39.
- Volkow, N. D., & O'Brien, C. P. (2007). Issues for DSM-V: should obesity be included as a brain disorder? *American Journal of Psychiatry*, 164, 708–710.
- Volkow, N. D., Wang, G. J., Fowler, J. S., Logan, J., Jayne, M., Franceschi, D., et al. (2002). "Nonhedonic" food motivation in humans involves dopamine in the dorsal striatum and methylphenidate amplifies this effect. *Synapse*, 44, 175–180.
- Volkow, N. D., & Wang, R. A. (2005). How can drug addiction help us understand obesity. *Lancet*, 357, 354–357.
- Wang, G.-J., Volkow, N. D., Logan, J., Pappas, N., Wong, C., Zhu, W., et al. (2001). Brain dopamine and obesity. *Lancet*, 357, 354–357.
- White, H. R., & Labouvie, E. W. (1989). Toward the assessment of adolescent problem drinking. *Journal of Studies on Alcohol*, 50, 30–37.