



The association of “food addiction” with disordered eating and body mass index



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ABSTRACT

Introduction: The contribution of an addictive process to elevated body mass index (BMI) and disordered eating is an area of growing interest. Yet, little is known about how “food addiction” may be related to disordered eating and obesity. The ability of addictive-like eating to account for eating pathology not captured by traditional eating disorders is unknown. No prior research has examined the association of “food addiction” with bulimia nervosa (BN). Finally, little is understood about the association of “food addiction” with patterns of dieting and weight gain. The current study was conducted to address these gaps in the literature.

Material and methods: Participants (N = 815) were recruited from online advertisements nationwide and completed measures related to “food addiction”, BMI, weight history, and disordered eating.

Results: Addictive-like eating was associated with elevated current and lifetime highest BMI, weight cycling, and eating pathology. The prevalence of “food addiction” was higher in participants with BN than in those with binge eating disorder (BED). “Food addiction” continued to be related to clinically relevant variables, especially elevated BMI, even when participants did not meet criteria for BED or BN. The co-occurrence of “food addiction” with eating disorders appears to be associated with a more severe variant of eating pathology.

Discussion: An addictive-type response to highly palatable food may be contributing to eating-related problems, including obesity and eating disorders. BN relative to BED appears to be more strongly associated with “food addiction.” Additionally, the concept of “food addiction” appears to capture clinically relevant information in participants who do not meet criteria for either BN or BED. Further examination of “food addiction” may be important in understanding the mechanisms underlying certain types of problematic eating behavior.

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1. Introduction

Evidence is growing that an addictive process may play a role in certain types of eating behavior. Theories of “food addiction” suggest that certain highly processed foods may have an addictive potential and that some types of obesity and disordered eating may be the result of an addictive response to these foods (Gearhardt, Davis, Kuschner, & Brownell, 2011; Gold, Frost-Pineda, & Jacobs, 2003). If an addictive mechanism contributes to eating patterns marked by compulsive overconsumption, this may lead to the development of novel eating-focused treatment approaches (e.g., addiction pharmacology, harm reduction). Behavioral (e.g., withdrawal, tolerance, bingeing) and biological (e.g., dopaminergic downgrading, opioid release) indicators of addiction in animals consuming highly palatable foods or ingredients in these foods (e.g., fat, sugar) have been found (Avena, Rada, & Hoebel, 2008;

Johnson & Kenny, 2010). Neurobiological studies have identified shared neural underpinnings associated with obesity and substance dependence, such as increased activation in motivation-related regions in response to cues and diminished activation in reward regions in response to consumption (Volkow, Wang, Tomasi, & Baler, 2013). The development of the Yale Food Addiction Scale (YFAS) (Gearhardt, Corbin, & Brownell, 2009) has provided a tool to assess the diagnostic symptoms of substance dependence in relation to eating behavior. Elevated YFAS scores have been linked with patterns of neural activation associated with addictive behaviors (Gearhardt, Yokum et al., 2011), a higher likelihood of a composite index of elevated dopamine signaling (Davis et al., 2013) and a greater severity of disordered eating (Davis, 2013a; Gearhardt, White, Masheb, & Grilo, 2013; Gearhardt et al., 2012). Despite increased interest in “food addiction,” a number of areas require further research, such as further examination of the relationship of addictive-like eating with eating disorders and obesity.

Theoretically, addiction and traditional eating disorder perspectives have different explanatory mechanisms about loss of control over eating. The “food addiction” perspective highlights the addictive potential

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of the highly processed food and suggests that these foods may have the ability to “hijack” the reward system in at-risk individuals (Gearhardt, Davis et al., 2011; Gold et al., 2003). In contrast, traditional eating disorder approaches highlight rigid dietary restraint, as well as shape and weight concern as causal mechanisms (Fairburn, Cooper, Shafran, & Wilson, 2008; Polivy & Herman, 2002). Yet, overlap is also significant across these theoretical approaches, with impulsivity, reward dysfunction, and emotion dysregulation proposed as important contributors to eating psychopathology from both addiction and traditional eating disorder perspectives (Davis, Strachan, & Berkson, 2004; Dawe & Loxton, 2004; Fischer, Anderson, & Smith, 2004). The diagnostic criteria for binge eating disorder (BED) and substance dependence also share a number of characteristics, like diminished control over consumption and continued use despite negative consequences (Gold et al., 2003). This overlap has led to questions about the discriminant validity of “food addiction” from BED. In other words, the “food addiction” construct may be capturing variability already accounted for by BED. Prior research with obese individuals with BED found that YFAS “food addiction” and a diagnosis of BED did not completely overlap, with around fifty percent of obese patients diagnosed with BED meeting the “food addiction” threshold (Gearhardt, White et al., 2013; Gearhardt et al., 2012). In clinical BED samples, elevated YFAS scores were related to more frequent binge eating episodes, elevated emotion dysregulation, and increased eating pathology (Gearhardt, White et al., 2013; Gearhardt et al., 2012). Thus, “food addiction” may be associated with more severe pathology in the context of BED (Davis, 2013b). Further, Umberg, Shader, Hsu, and Greenblatt (2012) proposed that bulimia nervosa (BN) may be more likely than BED to be associated with “food addiction,” since the binge/restrict pattern central to this disorder may increase the likelihood of affective and biological changes implicated in addictive disorders. To date, no studies have examined the construct of “food addiction” in a sample of participants with BN.

Further, there is no previous research on the association of “food addiction” and disordered eating in a sample that contains not only individuals with clinically relevant eating disorders, but also eating pathology that may not be captured by traditional eating disorder diagnoses – (e.g., subthreshold eating pathology or problematic eating not associated with binge eating). Understanding the ability of “food addiction” to account for problematic patterns of eating that are not captured by eating disorder categories may be particularly relevant given the high rates of Eating Disorder Not Otherwise Specified (EDNOS) diagnoses (Machado, Machado, Gonçalves, & Hoek, 2007). Although individuals with EDNOS diagnoses are considered to have a level of psychopathology that necessitates treatment (Ricca et al., 2001; Turner & Bryant-Waugh, 2004), the mechanisms underlying these unspecified eating patterns are not well understood, which limits the development of more targeted treatment approaches. Therefore, the capability of “food addiction” to provide relevant information outside of formal eating disorder diagnoses (e.g., BED, BN) speaks to the validity of the construct and may provide clinically useful information.

The association of “food addiction” with obesity is also relatively unknown. Elevated levels of addictive-like eating behavior hypothetically would be related to compulsive overconsumption of highly palatable (and calorie-dense) foods. This relationship should result in a greater risk of obesity. A recent study found that the risk of “food addiction” increased with obesity status and the severity of addictive-like eating was positively related to measures of adiposity (e.g., body fat, BMI) (Pedram et al., 2013). However, little is known about how “food addiction” may be related to specific patterns of weight history, such as weight cycling (e.g., repeated periods of losing and regaining weight), age at onset of dieting and weight gain, and current dieting behavior, which are known to influence adiposity, BMI, and problematic eating (Enriquez, Duncan, & Schur, 2013; Fairburn et al., 1998; Foster, Wadden, Kendall, Stunkard, & Vogt, 1996). Further, no prior research has examined whether the association of addictive-like eating with elevated BMI is confined to individuals with traditional eating disorders who are at

increased risk for obesity (i.e., those with BED). Thus, it is possible that “food addiction” is only related to obesity among individuals who also have BED.

In the current study, we aim to address a number of gaps in the current literature on “food addiction” regarding disordered eating and obesity. We examine the association of “food addiction” with BED, BN, and BMI in a large non-clinical sample. This sample provides an opportunity to evaluate the potential link between “food addiction” and BN for the first time, as well as to investigate the ability of “food addiction” to account for eating pathology not captured by other eating disorders. Additionally, we will examine the association between addictive-like eating and body weight, including current BMI, highest lifetime BMI, dieting status and frequency of weight cycling. We will also identify whether “food addiction” is associated with elevated BMI outside of the context of eating disorders associated with increased adiposity (e.g., BED).

2. Materials and methods

2.1. Participants

Participants were drawn from a sample of 1141 community volunteers who responded to an online advertisement for a research study about “eating habits,” “health behaviors,” “dieting,” or “weight control.” Eight hundred and fifteen participants in the full sample completed the YFAS and were included in the sample for the current study. The study recruited participants through Craigslist classifieds postings in different United States cities (e.g. New York, Los Angeles, Washington DC) and required participants to be 18 years or older. Participants were on average 33 years old (range 18–73). The sample was 11.9% male ($n = 97$) and 88.1% female ($n = 717$) and one participant did not report gender. The racial/ethnic distribution for the study sample was: 79.1% Caucasian, 6.0% Hispanic, 5.7% African American, 5.5% Asian, and 3.7% reporting “other.” One participant failed to report race/ethnicity. The participants’ body weight ranged from underweight to severely obese (BMI range 14.60–69.23) with the average BMI in the overweight category ($M = 28.70$, $SD = 8.77$).

2.2. Procedures

Participants were required to provide informed consent prior to completing the survey, but no personal identifying information was collected. The study was approved by the Yale Institutional Review Board. All survey measures were hosted on SurveyMonkey (<http://www.surveymonkey.com>), a research-based data gathering website that uses a secure 128-bit encryption. Participants were offered a 1 in 20 chance to win a \$50 gift certificate in exchange for participation.

2.3. Assessments and measures

Participants provided basic demographic information and completed a battery of self-report measures. Self-reported height and weight were used to compute participant BMI (kg/m^2).

The *Eating Disorder Examination Questionnaire (EDE-Q)* (Fairburn & Beglin, 1994) is the self-report version of the Eating Disorder Examination interview (Fairburn & Cooper, 1993) and assesses eating disorders and their features. The EDE-Q assesses the frequency of different over-eating behaviors over the previous 28 days, such as objective binge eating episodes (OBEs; eating unusually large amounts of food while experiencing a subjective sense of loss of control), subjective binge eating episodes (SBEs; loss of control over eating but not eating an objectively large amount of food) and purging behaviors (e.g., self-induced vomiting, laxative misuse, diuretic misuse). The EDE-Q also contains four subscales that assess levels of dietary restraint, eating concern, shape concern, and weight concern and generates a global score. The EDE-Q has extensive psychometric support for use in disordered eating groups (Grilo, Masheb, Lozano-Blanco, & Barry, 2004; Grilo, Masheb, &

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