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Research report

Food addiction as a causal model of obesity. Effects on stigma, blame, and perceived psychopathology

Janet D. Latner [a,*], Rebecca M. Puhl [b], Jessica M. Murakami [a], Kerry S. O'Brien [c]

[a] Psychology Department, University of Hawaii at Manoa, Honolulu, HI, USA
[b] The Rudd Center for Food Policy and Obesity, Yale University, New Haven, CT, USA
[c] Behavioural Studies, Monash University, Melbourne, Australia

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ABSTRACT

The present study examined the impact of the food-addiction model of obesity on weight stigma directed at obese people. Participants (n = 625) were randomly assigned to four experimental conditions. They were asked to read either a food-addiction explanatory model of obesity or a nonaddiction model, and subsequently read a vignette describing a target person who met the characteristics of one of these models and was either obese or of normal weight. Questionnaires assessed participants' stigmatization and blame of targets and their attribution of psychopathology toward targets. Additional questionnaires assessed stigma and blame directed toward obese people generally, and personal fear of fat. A manipulation check revealed that the food-addiction experimental condition did significantly increase belief in the food-addiction model. Significant main effects for addiction showed that the food-addiction model produced less stigma, less blame, and lower perceived psychopathology attributed to the target described in vignettes, regardless of the target's weight. The food-addiction model also produced less blame toward obese people in general and less fear of fat. The present findings suggest that presenting obesity as an addiction does not increase weight bias and could even be helpful in reducing the widespread prejudice against obese people.

Introduction

The stigmatization of obesity and discrimination against obese individuals is widespread and harmful (Puhl & Heuer, 2009). Different explanatory models of the causes of obesity have been shown to influence individuals' beliefs about obesity, and in some cases, to reduce or intensify weight bias toward obese persons. For example, a behavioral model of obesity (emphasizing contributing factors such as unhealthy eating or sedentary behavior) may increase levels of stigma toward obese people (O'Brien, Puhl, Latner, Mir, & Hunter, 2010; Teachman, Gapinski, Brownell, Rawlins, & Jeyaram, 2003), whereas physiological or genetic explanations of obesity have reduced stigma in some, though not all, studies (Crandall, 1994; O'Brien et al., 2010; Persky & Eccleston, 2011; Wiese, Wilson, Jones, & Neises, 1992).

One increasingly influential explanatory model of obesity is that overeating and obesity are caused by an addiction to food (Brownell & Gold, 2012; Davis & Carter, 2009; Gearhardt, Grilo, Dileone, Brownell, & Potenza, 2011; Volkow, Wang, & Baler, 2011; Volkow, Wang, Fowler, & Telang, 2008). This model, which likens food addiction to drug addiction in terms of its clinical profile and effects on the brain and behavior, has gained increasing research attention. In addition, although the U.S. media have largely portrayed overweight as due to personal behavior, with physiological/biological explanations in a smaller minority of reports (Saguy & Almeling, 2008), the addiction explanation of obesity is gaining traction within the popular culture, as evidenced by more recent media coverage (Cevallos, 2011; Huget, 2011). Given the developing state of the neuroscientific literature, the food-addiction model of obesity has also been critiqued as overly simplistic and dependent on superficial clinical overlap (Wilson, 2010; Ziauddeen, Farooqi, & Fletcher, 2012). Indeed, the food-addiction model has been extensively debated in multiple venues such as international conferences (European College of Neuropsychopharmacology, 2013), university campuses (Hellmich, 2007), and the international media (Brauser, 2013; Fleming, 2013).

Regardless of literature supporting or refuting the food-addiction model, however, little is known about how this explanatory model may impact public perceptions of obese people and the level of bias and stigma against them. Providing an explanation for obesity may be understood in the context of framing theory, which posits that an issue can be viewed from a variety of perspectives which can affect attitudes and behaviors. Framing in communication

* Corresponding author.
E-mail address: jlatner@hawaii.edu (J.D. Latner).

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provides meaning to events by organizing and shaping the interpretation of issues for the public (Chong & Druckman, 2007).

It is possible that providing an addiction-based explanation for obesity may either worsen or improve stigmatizing attitudes. Causal explanations that increase attributions of personal control may exacerbate stigma (DeJong, 1980; Puhl, Schwartz, & Brownell, 2005), and physiological explanations can alleviate stigma (Crandall, 1994; O'Brien et al., 2010; Persky & Eccleston, 2011; Wiese et al., 1992). Yet research also suggests that addictive behaviors such as smoking, alcohol use, and cocaine use are highly stigmatized, and even more stigmatized than obesity (DePierre, Puhl, & Luedicke, 2013; O'Brien, Latner, Carr, Misajon, Hunter, & Forrest, unpublished data; Phillips & Shaw, 2012). This may be due to the fact that everyone must eat food, whereas smoking and substance abuse require actively choosing and using the substance. One recent study that directly assessed public perceptions of food addiction found that labeling an individual as an "obese food addict" led to greater stigma than when the individual was labeled as either "obese" or "food addict" alone, suggesting an additive stigmatizing effect of the "obese food addict" label (DePierre et al., 2013). This study also found, surprisingly, that food addicts depicted as obese or of average weight were both stigmatized to a similar extent.

However, controlled studies are needed to compare the stigmatizing effects of presenting obesity as the result of an addiction to the stigmatizing effects of presenting obesity as a nonaddictive condition. Therefore, the present study explored the effect of learning about a food-addiction model of obesity on subsequent stigma toward obese individuals. In order to compare the addiction model of obesity to a contrasting nonaddiction model, background information and vignettes were presented about an obese individual or normal-weight individual who was either addicted to food or not addicted to food.

Material and methods

Participants

An online survey was hosted by Qualtrics.com, and participants were recruited from an online database (eLab) hosted by the Yale School of Management (http://elab.som.yale.edu). This web site draws from a sample of approximately 20,000 adults from across the United States who are recruited through advertisements on social networking websites. Registered participants in the panel are notified via e-mail when studies are posted, and they are invited to participate in any studies of their choosing. Participants were compensated with entry into a raffle to win a gift card. The study was approved by the University of Hawaii Institutional Review Board. Of the 665 individuals who began participation, 625 completed the survey and were retained for data analysis. Their mean age was 34.55 (14.65) years and mean BMI was 25.40 kg/m^2 (4.4% underweight [BMI < 18.5], 50.7% normal weight [BMI 18.5–24.9], 24.1% overweight [BMI 25–29.9], 15.6% obese [BMI ≥3 0]); 5.3% did not specify their weights or heights. Seventy-one percent of participants were White, 4.2% were African American, 16.7% were Asian or Pacific Islander, and 4.2% were Latino/a, 0.9% were American Indian or Alaskan Native, and 3.4% were of other ethnic identity; 61.8% were female, 36.9% were male, and 2.1% did not specify.

Procedures

Participants were randomized to read one paragraph of descriptive background information about body weight, followed by a one-paragraph vignette describing a food-addicted person or nonaddicted person, in a 2 (addiction *versus* nonaddiction) × 2 (obese *versus* normal weight) between-subjects design.

The model of food addiction framed by Gearhardt, Corbin, and Brownell (2009a, 2009b) formed the basis for the descriptive background information provided about addiction. In the food-addiction conditions, the background information included plain-language statements comparing the physiological process of food addiction to that of drug addiction and indicating that some people are addicted to food. Food-addicted individuals were described as having neural receptors in the brain similar to those activated by addictive drugs, leading the person to experience uncontrollable, compulsive food cravings whose intensity may overshadow the motivation to engage in other activities. The person was described as being compelled to seek and consume foods, especially high-fat, high-calorie foods, even in the face of negative consequences to health and quality of life and despite repeated attempts to stop overeating. Tolerance and withdrawal were described by indicating that increasing amounts of food are needed over time to satisfy the person's cravings and that the person feels physical and emotional withdrawal symptoms if the food is not consumed. The addicted individual was described as having experienced changes in brain structure and function as a result of the repeated effect of compulsive eating on brain neurotransmitter activity.

In the nonaddiction conditions, the descriptive background information was designed to contrast with the information provided in the addiction conditions. It contained no endorsement of neurophysiological changes or differences in brain activity, or of uncontrollable cravings, tolerance, or withdrawal. Because this study was not intended to compare a physiological explanation to a behavioral one, information about physiological maintaining factors of obesity was also presented in the nonaddiction model to balance the physiological factors described in the addiction model. Specifically, the description explained the role of genetic factors and homeostatic processes in influencing the development of body weight. The text also indicated that there is no evidence that people can be uncontrollably addicted to food, or that food acts like a drug with physiologically addictive properties, but that instead, some people consume an excess amount of calories due to high-fat, high-calorie food choices made repeatedly over time. It was indicated that when these individuals stop consuming these foods, they do not experience any physical and emotional withdrawal symptoms. The length of text was balanced across conditions (from 255 to 263 words), as shown in Appendix A.

In light of previous research suggesting that women are more vulnerable to weight stigmatization than men (Puhl, Andreyeva, & Brownell, 2008), vignettes about the specific target described a woman. In addition, the target ("Jennifer") was described as being 35 years old, matching the mean age of respondents typically recruited by eLab. After reading the information describing the nature and mechanisms of either a food addiction or a nonaddiction model, participants were provided with a vignette about a woman who was described as matching that description, with several of the key phrases of the background descriptions reiterated and applied specifically to "Jennifer." She was also described as having a height and weight equaling a body mass index (BMI; kg/m^2) of either 35.2 (obese) or 21.5 (normal weight; see Appendix A). After reading the background information and respective vignettes, participants were asked to respond to questionnaires asking their opinions about the target in the vignette specifically ("target-specific"), as well as a questionnaire about obesity stigma in general ("general obesity").

Measures

Universal measure of bias (UMB; target-specific)

The Universal Measure of Bias is a 20-item scale that allows the assessment of stigma directed at different targets (Latner, O'Brien, Durso, Brinkman, & MacDonald, 2008). The measure is designed to permit the insertion of a name or group into scale items while oth-