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Research report

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

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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

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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-

erwise retaining identical item wording and scoring, and it has been used in previous vignette-based experiments with specific target names inserted (e.g., Latner, Ebneter, & O'Brien, 2012). Each item is rated on a Likert scale (1 = strongly agree to 7 = strongly disagree). The UMB has been shown to have good convergent validity and internal consistency and a four-factor structure that includes: attraction, negative judgment, distance, and equal rights, as well as a total UMB score computed as the mean of all items. Higher scores indicate greater stigma (sample item: "People like Jennifer are attractive"). Cronbach's alpha in the present sample was .75.

Stigma questionnaire (target-specific)

This scale is a self-report stigma questionnaire that has been adapted as a composite measure by Ebneter, Latner, and O'Brien (2011) from previous measures (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000; Griffiths, Christensen, & Jorm, 2008; Stewart, Keel, & Schiavo, 2006) for use in assessing participant attitudes toward different targets. This composite measure has been shown to have good internal consistency and validity (Ebneter et al., 2011), and items are rated on a 5-point Likert scale from 1 = strongly agree to 5 = strongly disagree. Lower scores on this 18-item measure indicate more stigmatizing attitudes (sample item: "In your opinion, do you think Jennifer is less competent than peers?"). Cronbach's alpha in the present sample was .86.

Weight locus of control (WLOC; target-specific)

The WLOC is a reliable and valid 4-item measure of beliefs about internal and external controllability of weight status (Saltzer, 1982). Items are scored on a 6-point Likert scale from 1 = strongly disagree to 6 = strongly agree, and lower scores indicate a more internal (more blaming) weight locus of control orientation (sample item: "Whether Jennifer gains, loses, or maintains her weight is entirely up to her"). Cronbach's alpha in the present sample was .53; therefore, this scale was additionally analyzed as a 2-item version using only the two reverse-scored items, which had an improved Cronbach's alpha of .71.

Perceived psychopathology (target-specific)

Three items were included to assess participant perceptions of the level of psychopathology present in the target. Rated on a 4-point scale from 1 = strongly agree to 4 = strongly disagree, these items included "Jennifer is probably depressed," "Jennifer is a generally unhappy person," and "Jennifer has psychological problems." Thus, lower scores indicate greater psychopathology. The Cronbach's alpha for these items was .82.

Antifat attitudes (AFA; general obesity)

The 13-item AFA (Crandall, 1994) is a reliable and valid measure assessing attitudes toward obese persons in general (sample item: "I don't like fat people much"). Responses range from 0 = very strongly disagree to 9 = very strongly agree; higher scores indicate stronger antifat attitudes. The dislike, willpower, and fear of fat subscales assess obesity stigma, controllability beliefs, and concerns about personal weight gain, respectively. Cronbach's alpha in the present sample was .86.

Manipulation check

Three questions were included as a manipulation check to assess whether participants across conditions differed in their absorption of the key relevant information about food's addictive properties and food addiction's effects on body weight. These items were rated on a 6-point scale from 1 = strongly disagree to 6 = strongly agree ("Food has addictive properties, like a drug," "Body weight can result from being addicted to food," and "Obesity can result from being addicted to food."). These three items had a Cronbach's alpha of .86, and thus were combined for analysis into a single "addiction rating" score, with higher scores indicating greater addiction beliefs.

Statistical analyses

Preliminary analyses tested for differences between experimental groups in BMI and age. Dummy variables were assigned to each vignette group using a 2×2 classification: addiction model *versus* nonaddiction model and obese target *versus* normal-weight target. Subsequently, 2×2 ANOVAs were run on the following dependent variables: UMB total, attraction, distance, and negative judgment subscales, stigma questionnaire, WLOC, perceived psychopathology, and AFA dislike, willpower, and fear of fat subscales. ANOVA was also run on addiction rating scores as a manipulation check.

Results

No differences were found between randomized experimental groups for BMI or age of participants. For addiction rating scores, a main effect was found for addiction, consistent with the intent of the experimental design, with no main effect for weight and no interaction effect. Participants randomized to the addiction conditions had significantly higher addiction ratings scores than participants in the nonaddiction condition.

ANOVA revealed main effects for addiction on the UMB total, UMB equal rights, stigma questionnaire, WLOC (4-item version and 2-item version), perceived psychopathology, AFA willpower, and AFA fear of fat (Table 1). For all of these variables except equal rights, the addiction condition resulted in lower stigma. That is, a food-addiction model elicited judgments toward targets of lower overall negativity, lower stigma, lower blame, and lower perceived psychopathology compared with the nonaddiction model. Regarding views about obesity generally, the addiction model elicited lower controllability beliefs and less fear of fat. For UMB equal rights, participants were less likely to endorse that special effort should be made to ensure the rights of food-addicted individuals relative to the rights of nonaddicted individuals.

Main effects for weight emerged on the UMB attraction subscale, UMB equal rights, WLOC (4-item version and 2-item versions), perceived psychopathology, and AFA willpower. In all of these cases except equal rights, the obese-target condition elicited greater stigma than the normal weight target in both the addiction and nonaddiction conditions. Specifically, obese targets elicited ratings of less attraction, more blame, more psychopathology, and greater controllability beliefs about obesity generally. For UMB equal rights, participants were less likely to endorse that special effort should be made to ensure the rights of normal-weight individuals relative to obese individuals.

No interaction effects were found, except for the 2-item version of WLOC (not for the 4-item version). Obese food-addicted targets elicited greater blame for their weight than normal-weight food-addicted targets, whereas non-food-addicted targets elicited similarly high levels of blame regardless of their weight status.

Discussion

The present study examined the effects of the addiction model of obesity on weight stigma. As intended, the experimental manipulation check suggested that descriptive information about addiction was effective in influencing participants' belief in the addictive properties of food and about addiction as a reason for obesity and body weight. The experimental results indicated that providing an addiction-based etiological explanation of obesity may reduce weight bias relative to providing a non-addiction-based explanation. These results may be surprising in light of the severe prejudice shown against addictive behaviors, such as smoking, substance abuse, and

Table 1
Means (SD) of dependent measures and F values (effect sizes) for main effects of addiction, weight, and their interaction.

	Food addiction		Nonaddiction		F values (partial eta^2)		
	Obese	Normal weight	Obese	Normal weight	Addiction	Weight	Interaction
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)			
UMB							
Attraction	4.24^a (0.97)	3.66^b (0.67)	4.51^a (0.91)	3.66^b (0.75)	2.63 (.01)	76.15 (.16)**	2.79 (.01)
Negative-judgment	2.90 (1.18)	3.03 (1.03)	3.14 (1.25)	3.18 (1.07)	2.99 (.01)	.59 (.00)	.19 (.00)
Distance	2.98 (1.18)	2.98 (0.95)	3.25 (1.06)	2.99 (0.98)	1.72 (.00)	1.60 (.00)	1.54 (.00)
Equal rights	2.64^a (1.42)	3.19^{a,b} (1.45)	3.18^{a,b} (1.59)	3.70^b (1.31)	13.77 (.03)**	14.20 (.03)**	.01 (.00)
Total	3.19^a (0.91)	3.22^a (0.70)	3.52^b (0.93)	3.38^b (0.71)	9.36 (.02)**	.50 (.00)	1.02 (.00)
Stigma questionnaire	3.59^a (0.70)	3.72^a (0.59)	3.36^b (0.68)	3.33^b (0.46)	27.02 (.06)**	.72 (.00)	1.61 (.00)
WLOC (4-item version)	2.66^a (0.94)	3.34^{a,b} (0.77)	2.45^{a,b} (0.86)	2.98^b (0.64)	12.78 (.03)**	55.75 (.12)**	.88 (.00)
WLOC (2-item version)	2.66^a (1.29)	3.20^b (1.18)	2.31^c (1.01)	2.35^c (1.00)	29.28 (.07)**	6.89 (.02)**	5.18 (.01)*
Perceived psychopathology	3.42^a (1.65)	3.32^{a,b} (1.82)	3.50^{a,b} (1.72)	3.46^b (1.69)	32.26 (.07)**	28.14 (.07)**	3.23 (.01)
AFA							
Dislike	3.32 (1.82)	3.42 (1.65)	3.46 (1.69)	3.50 (1.72)	.41 (.00)	.17 (.00)	.03 (.00)
Willpower	5.91^a (1.81)	5.37^{a,b} (1.82)	6.41^{a,b} (1.49)	6.29^b (1.58)	18.08 (.04)**	3.91 (.01)*	1.65 (.00)
Fear of fat	5.60^a (2.43)	5.26^a (2.30)	5.95^b (2.40)	5.97^b (2.02)	5.34 (.01)*	.51 (.00)	.62 (.00)
Addiction rating	4.78^a (0.96)	4.71^a (0.98)	3.81^b (1.27)	4.08^b (1.15)	53.03 (.12)**	.81 (.00)	2.43 (.01)

Note: Higher scores indicate greater stigma for the UMB and AFA, and lower scores indicate greater stigma, blame, and psychopathology for the stigma questionnaire, WLOC, and perceived psychopathology, respectively. Partial eta^2 is a measure of effect size (.01 may be roughly interpreted as small, .06 as medium, and .14 as large effects).
* $p < .05$.
** $p < .01$. Within rows, mean scores with different superscript letters are significantly different ($p < 0.05$).

alcoholism (Bayer & Stuber, 2006; Keyes et al., 2010; Schomerus et al., 2011). However, presenting obese individuals as physically dependent on food, rather than as free agents of their own dietary decisions, reduced blame toward these specific individuals and, indeed, toward obese people in general. Controllability beliefs, and attributions of blame, are associated with negative beliefs about obese people and have been posited to underlie obesity stigma (Crandall, 1994; Puhl & Brownell, 2003). The interaction effect of addiction and target weight on one of the measures of blame (the 2-item WLOC) additionally suggests that blame was greater for the obese addicted target than the thinner addicted target, consistent with obesity stigma more generally.

We examined several forms of stigma in order to explore which facets of weight bias might be influenced by an addiction model. The food-addicted target was viewed less negatively overall compared with the nonaddicted target, as indicated by lower UMB total scores and lower stigma questionnaire scores. The addicted target was also viewed as being less at fault for her weight, or as having a more internal locus of control over weight compared with the nonaddicted target. Perhaps surprisingly, the food-addicted target was also viewed as having less psychopathology. Despite the fact that addictive disorders, but not obesity, are considered mental disorders in the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 2013), the general public may actually view an obese individual without a food addiction as more mentally impaired than an obese individual with a food addiction. This finding suggests that one form of prejudice against obese individuals is the perception that they are mentally unstable, and it may be that providing an addiction-based explanation for obesity alleviates this specific stereotype. Perhaps as a result of the perception of less psychological disability, participants placed less emphasis on the need to ensure the equal rights of food-addicted individuals. Finally, reading about addiction as an explanation for obesity also reduced the respondents' fear of personal fat/weight gain, and their belief that obesity in general is caused by a lack of willpower. An addiction explanation may have made obesity seem less of a personal threat; perhaps participants felt reassured that they did not experience food addiction and therefore were less at risk for obesity.

The implications of this study for public health messages are complex. On the one hand, it is possible that presenting obesity as an addictive disorder may somewhat help in alleviating public stigma associated with obesity. However, further research is needed to confirm this finding, especially given the high levels of stigma against other addictions found in previous studies (Bayer & Stuber, 2006; Keyes et al., 2010; Schomerus et al., 2011) and the additive stigmatizing effects previously found to be associated with a "food-addict" label when paired with obesity (DePierre et al., 2013). If the present findings are confirmed, it indicates that media messages presenting obesity as an addiction may not be harmful and could even be helpful in reducing the widespread prejudice against obese people. The present findings may also suggest that not describing obesity as personally controllable and due to poor control of eating and lack of exercise engenders less stigma than presenting obesity as beyond individual control. The addiction model may be a plausible and believable noncontrollable cause of obesity.

However, it is also important to note that this study (and other studies on stigma and prejudice) did not shed any light on the validity or clinical utility of the addiction model, which is still debated in the scientific literature (Wilson, 2010; Ziauddeen et al., 2012). In particular, the treatment implications of the addiction model have been questioned (Wilson, 2010). Further research is needed to resolve the ongoing debate about the food-addiction model, to determine whether food addiction is causally related to obesity, to explore whether it should be considered a psychiatric disorder in the DSM (e.g., Volkow & O'Brien, 2007), and to examine its treatment and public health implications (e.g., Gearhardt et al., 2011; Wilson, 2010).

The present study had several limitations. Despite the ability of the Internet and the present data collection procedures to recruit diverse samples (Gosling, Vazire, Srivastava, & John, 2004), further research with minority populations and international samples is needed to examine attitudes across cultures toward obesity and food addiction. In addition, the internal consistency of one of the measures used (4-item WLOC) was low in the present sample, and another measure (perceived psychopathology) was created for the present study, and prior evidence of its validity is lacking. Interestingly, not all of the measures of stigma in the present study showed greater bias against obese targets than lean targets; a greater number of main effects were found for addiction than for obesity. This finding, comparable to that of DePierre et al. (2013) who found similar stigmatization against lean and obese food-addicted targets, might have been due to the presentation of lean targets in this study as either food-addicted or as making poor food choices, possibly making the current study's lean targets seem more similar to obese targets than

in other studies of weight stigma. Finally, this study did not use figures to accompany vignettes in order to avoid the confounding effects of specifying the ethnicity of targets; additional future research using visual depictions of obese and normal-weight individuals is recommended.

Conclusion

Presenting a model of food addiction appeared to alleviate overall weight bias, reduce the blame directed at obese individuals for their actions and weight, reduce the perception that obese individuals are mentally impaired, and decrease the fear of personal weight gain. These results are potentially encouraging given the limited progress in research indicating that weight stigma can be successfully reduced (Daníelsdóttir, O'Brien, & Ciao, 2010), as they suggest that public representations of obesity as an addictive disorder do not increase weight bias and may even alleviate it. However, these findings should be interpreted and applied with caution. First, more research is needed to test the accuracy of the addiction model itself. Second, it is unknown what effect presenting obesity as a food addiction may have on obese individuals. For example, research is needed to examine whether it might affect or reduce their attempted behavioral changes to diet and exercise. Such results would need to be balanced against any potential decrease in stigma.

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Appendix A

Background information and vignette paragraphs shown to participants. Italicized text appeared only in the obese condition, and bracketed text appeared in only the normal weight condition.

Addiction conditions

Leading scientists have discovered that *obesity is often the result of* [certain individuals have a] physiological food addiction [whether or not they are overweight]. The physiological process of food addiction is similar to that of drug addiction. Some people are addicted to food. For them, food activates the same neural receptors in the brain that are activated by drugs such as cocaine. The person then experiences uncontrollable, compulsive food cravings. These cravings are so intense that they overshadow the motivation to engage in other activities. The person is 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. Despite

repeated attempts to stop overeating, food over-consumption is uncontrollable. Over time, more and more food is needed to satisfy the person's cravings, and the person feels physical and emotional withdrawal symptoms if the food is not consumed. This can lead to *obesity* [negative health consequences], particularly in individuals who are predisposed to changes in brain structure and function as a result of the repeated effect of compulsive eating on brain neurotransmitter activity.

Ashley is a 21 year old college student who fits the typical profile of someone who is addicted to food, *and she is obese* [even though she is normal weight]. She has frequent uncontrollable cravings that lead her to consume high-fat, high-calorie foods. These intense cravings are so powerful that they often interfere with other activities such as exercise. She weighs *205 pounds (93 kg)* [125 pounds (56.7 kg)] at her height of 5'4" (163 cm), *putting her into the obese weight category* [putting her into the normal weight category]. She has maintained a steady weight since reaching her full height.

Non-addiction conditions

Leading scientists have discovered that weight often results from a combination of genetic factors and deliberate lifestyle choices about food and physical activity. Maintaining weight requires a homeostasis, or balance, between the calories that the body takes in and those burned off by metabolic processes and physical activity. There is no evidence that people can be uncontrollably addicted to food, or that food acts like a drug with physiologically addictive properties. Instead, some people consume too many calories due to high-fat, high-calorie food choices made repeatedly over time. Similarly, some people engage in too little physical activity due to their activity choices. When they try to stop consuming these foods, these individuals are able to stop and do not experience any physical and emotional withdrawal symptoms. *Choosing too many high-fat, high-calorie foods, and choosing to do too little exercise, can lead to an imbalance in the individual's homeostasis (energy balance). This can lead to obesity, particularly in individuals with a low resting metabolic rate and a genetic predisposition towards obesity.* [Despite choosing too many high-fat, high-calorie foods, and choosing to do too little exercise, some individuals may still maintain homeostasis (energy balance). These individuals may remain at a normal weight, particularly if they have a high resting metabolic rate and do not have a genetic predisposition towards obesity.]

Ashley is a 21 year old college student who fits the typical profile of someone who makes unhealthy food choices, *and she is obese* [even though she is normal weight]. She makes frequent decisions to eat high-fat, high-calorie foods. She is in control of these choices. She also often chooses not to exercise, selecting instead to do other activities. She weighs *205 pounds (93 kg)* [125 pounds (56.7 kg)] at her height of 5'4" (163 cm), putting her into the obese weight category [putting her into the normal weight category]. She has maintained a steady weight since reaching her full height.