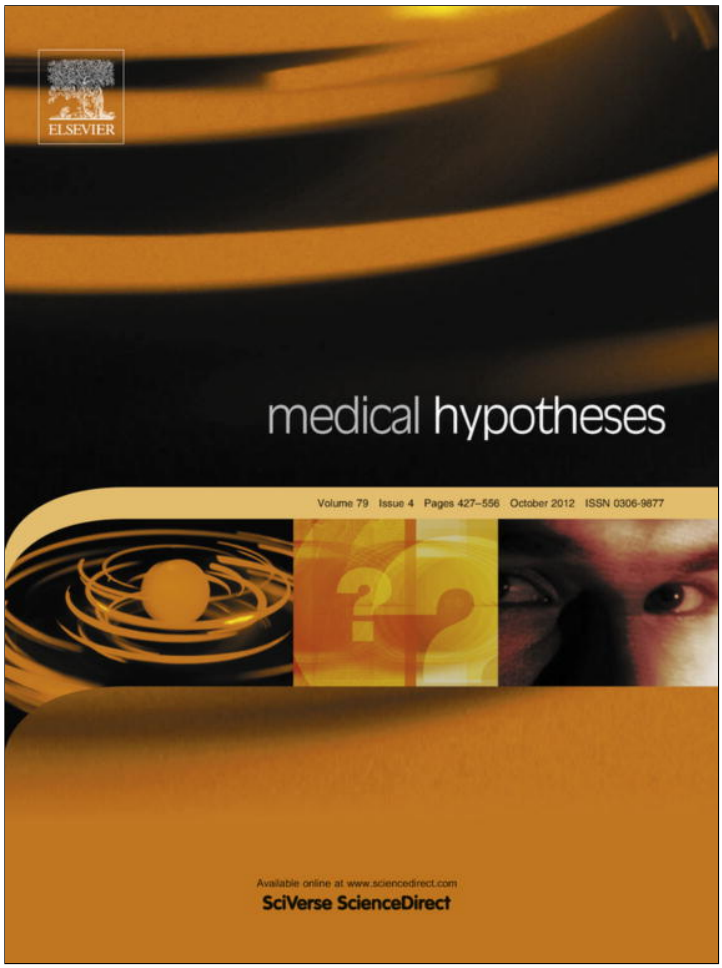

Medical Hypotheses 79 (2012) 508–511

Contents lists available at SciVerse ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

Food addiction and body-mass-index: A non-linear relationship

Adrian Meule *

Department of Psychology I, University of Würzburg, Marcusstr. 9-11, 97070 Würzburg, Germany

ARTICLE INFO

Article history:
Received 28 May 2012
Accepted 6 July 2012

ABSTRACT

Excessive food consumption has been recognized to show similarities with substance dependence. Subsequently, it has been proposed that food addiction might contribute to the obesity epidemic. Recent studies using questionnaires for the assessment of food addiction have found statistically significant, but negligible positive correlations with body-mass-index (BMI). Moreover, group comparisons between food-addicted and non-addicted individuals in normal-weight or obese samples did not show differences in BMI. However, the prevalence of food addiction diagnoses is remarkably increased in obese individuals. In the current article, it is suggested that there might be a cubic relationship between food addiction and BMI. Food addiction symptomatology may remain stable in the under- and normal-weight range, increase in the overweight- and obese range, and level off at severe obesity. Empirical data in support of this view are presented.

Introduction

Obesity is a condition of excessive fat accumulation in adipose tissue to the extent that health may be impaired [1]. Body-mass-index (BMI), which is defined as weight in kilograms divided by the square of height in meters (kg/m^2), is commonly used to classify underweight, normal-weight, overweight, and obesity in adults, although it does not distinguish between weight associated with muscles and weight associated with fat. According to the World Health Organization (WHO), obesity is classified as a BMI > 29.99 kg/m^2 and individuals with a BMI > 34.99 kg/m^2 are described as severely obese [1]. Obesity is a worldwide health and economic burden [2]. In the US population, for example, some 30% of adults are currently obese [3,4]. While the prevalence of obesity seems to stabilize, the prevalence of severe obesity continues to rise [3–6].

In the light of this obesity epidemic, a food addiction model of obesity and overeating is gaining more and more popularity in recent years [7–11]. This food addiction model proposes that there are striking similarities – both neurobiological and behavioral – between obesity (or overeating) and substance dependence and suggests that *hyperpalatable* foods could have an addiction potential [9]. One of the most highlighted arguments is that DSM-IV substance dependence criteria also apply to overeating, e.g. when they are modified with references to binge eating [12]. Another line of argument refers to a possible *reward deficiency syndrome* reflected by a downregulation of striatal D2-receptor availability and concurrent hypersensitivity to palatable food-cues in obese individuals [13].

There have also been critical evaluations of the food addiction model [14,15]. For instance, definitions and interpretations of food addiction symptoms, i.e. the eating-related equivalents to substance dependence criteria, are unclear and inconsistent [15,16]. Moreover, the concept of food addiction lacks a clear addictive agent. Animal studies suggest that high-sugar foods in particular are associated with addiction-like eating behaviors [17,18]. Accordingly, it has been suggested that foods with a high glycemic index may have an addictive potential in humans [19]. However, refined foods [20] or those high in salt [21] have also been discussed to be associated with food addiction. Finally, it has been proposed that the combination of excessive amounts of sugar, fat and sodium may make foods *hyperpalatable* and, therefore, may have an addictive potential [22]. While most of the food addiction literature has focused on obesity or binge eating disorder (BED, see below), it has also been recognized that only a subset of obese individuals could be described as food addicted and that symptoms of food addiction are also present in under- or normal-weight individuals [15,23–25], those with bulimia nervosa (BN) in particular [26–28].

Few questionnaires have been provided that assess addicted eating behavior. Merlo et al. [29] used the *Eating Behaviors Questionnaire* (EBQ) which was specifically created for their study in a sample of overweight children. Questions include the "3 C's" of addiction (compulsive use, attempts to cut down, continued use despite consequences) and one question directly asks "Do you think you are addicted to food?" [29]. A recent study used Eysenck's *Addiction Scale* (AS) [30] for the assessment of general addictive personality and some custom-made questions for the

* Corresponding author. Tel.: +49 931 31 808 34; fax: +49 931 31 824 24.
E-mail address: adrian.meule@uni-wuerzburg.de

0306-9877/$ - see front matter © 2012 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.mehy.2012.07.005

assessment of food addiction symptoms [31]. The most commonly used measure for identifying food addiction symptomatology is the *Yale Food Addiction Scale* (YFAS) [32]. With the YFAS, a food addiction symptom count can be calculated which corresponds to the criteria for substance dependence of the DSM-IV-TR [33]. Furthermore, two items assess a clinically significant impairment or distress from eating. Food addiction can be "diagnosed" when three symptoms and a clinically significant impairment or distress are present [32].

Food addiction and BMI

Some studies investigated the relationship between food addiction symptomatology and BMI in samples with a wide body mass range. For instance, a small positive correlation ($r = .17$) between YFAS symptoms and BMI was found in a predominantly normal-weight sample in Germany [34]. Prevalence of food addiction diagnoses differed between weight classes such that obese participants had higher prevalence (37.5%) than overweight (14.0%) or normal-weight (6.3%) participants [34]. Contrarily, food addicted participants did not differ in BMI from non-addicted participants in a comparable sample [35]. Moreover, no correlation between the YFAS and BMI was reported in a similar sample in the US that had a smaller sample size [32]. In a sample of 39 young healthy women ranging from lean to obese, there was no association between the YFAS and BMI [36]. The authors speculated that some individuals may experience compulsive eating behavior but engage in compensatory behaviors to maintain a lower weight or that lean participants who endorse FA are at risk for future weight gain [36].

There are also some studies in which food addiction was assessed in samples restricted to overweight or obese individuals. There was a small positive correlation ($r = .31$) between scores on the EBQ and BMI in a study with overweight children [29]. Contrarily, the AS did not correlate with BMI in obese adults seeking bariatric surgery [31]. Likewise, food addicted individuals (as measured with the YFAS) did not differ in BMI from non-addicted individuals in two obese samples [37,38].

To sum up, studies that calculated correlations between BMI and food addiction symptoms or compared groups of food addicted vs. non-addicted individuals for BMI did not yield a clear positive relationship between BMI and food addiction.

Binge eating and BMI

Research on food addiction is in its infancy and, therefore, only few data are available on food addiction symptomatology and BMI. A field of research where there are plenty of studies and which is closely related to food addiction, is binge eating and BED. Research criteria for BED include recurrent episodes of binge eating, i.e. eating large amounts of food in a discrete period of time with a sense of lack of control over eating, which occur at least 2 days a week for 6 months and are associated with marked distress [33]. Diagnostic criteria for BED will be included in the fifth revision of the DSM [39]. Although food addiction and BED have multiple similarities, the two constructs do not entirely overlap [40]. For example, only 56.8% of obese patients with BED could be classified as food addicted using the YFAS [41]. Vice versa, 72.2% of obese individuals who received an YFAS diagnosis were also diagnosed with BED [37].

Without any doubt, BED is associated with overweight and obesity [42–44]. Positive, but rather small relationships have been found between BMI and binge eating severity in samples with diverse body weight [45–47]. Higher binge eating frequency could be found in obese as compared to non-obese women [48]. This positive association between BMI and binge eating seems to be vanished within obese samples. No differences in BMI can be found when obese binge eaters are compared with obese non-binge eaters [49–53]. In one study, even lower BMI was found in obese binge eaters compared to obese non-binge eaters which, however, could be due to the recruitment procedure [54,55]. In samples with severely obese individuals seeking bariatric surgery, no differences were found in BMI between patients with full BED and less regular binge eating [56] or between groups which were classified as non-binge eaters, once weekly binge eaters, and twice weekly binge eaters [57].

To conclude, there seems to be only a small positive relationship between binge eating severity and BMI in samples with a wide body mass range and there is no such relationship within obese samples.

A proposed non-linear relationship

At first glance, results about the relationship between BMI and food addiction (or binge eating, respectively) seem inconsistent. While many studies show a positive association between variables, comparing groups with and without food addiction or BED usually do not find differences in BMI. Interestingly, researchers usually assume linear relationships between variables and only few studies have examined non-linear relationships between BMI and eating-related constructs (e.g. reward sensitivity) [58–60].

The lack of differences in BMI between food-addicted and non-addicted individuals could be explained by the fact that groups were either compared within samples of primarily normal-weight participants [35] or within samples of morbidly obese patients [37,38]. However, positive correlations between food addiction and BMI could be primarily due to increases in food addiction symptomatology in overweight and moderate obese individuals. This view would therefore assume a cubic relationship between food addiction symptomatology and BMI. Specifically, there may be a stable degree of food addiction symptomatology in the under- and normal-weight range and within morbidly obese samples. Increases in food addiction symptomatology might be particularly pronounced in overweight and moderate obesity.

Fig. 1 displays aggregated data from three studies in which the YFAS was used and BMI was assessed ($N = 1499$) [34,35,38]. Participants were classified in weight categories according to standard guidelines [1]. The relationship between weight categories and both food addiction symptoms and food addiction diagnoses could be mapped with a third order polynomial function (Fig. 1). This function explained 95% (symptoms) and 89% (diagnoses) of the variance, respectively (which was higher than plotting a quadratic (symptoms: $R^2 = .87$; diagnoses: $R^2 = .83$) or linear (symptoms: $R^2 = .85$; diagnoses: $R^2 = .73$) function). As can been seen in Table 1, food addiction symptomatology did not differ between underweight and normal-weight individuals, but increased from normal-weight to overweight individuals. Furthermore, food addiction symptomatology did not differ between overweight individuals and those with moderate obesity, but between individuals with moderate obesity and severe obesity (Table 1). Finally, no differences were found between obesity classes II and III (Table 1).

Conclusions and caveats

Several limitations have to be considered when assuming this non-linear relationship between BMI and food addiction. First, although classification in weight categories using BMI are usually based on standard guidelines provided by the WHO [1], those categories are not based on scientific evidence but are rather arbitrary ranges. Second, BMI is not an optimal measure of adiposity as it is based solely on weight and height and does not exactly represent

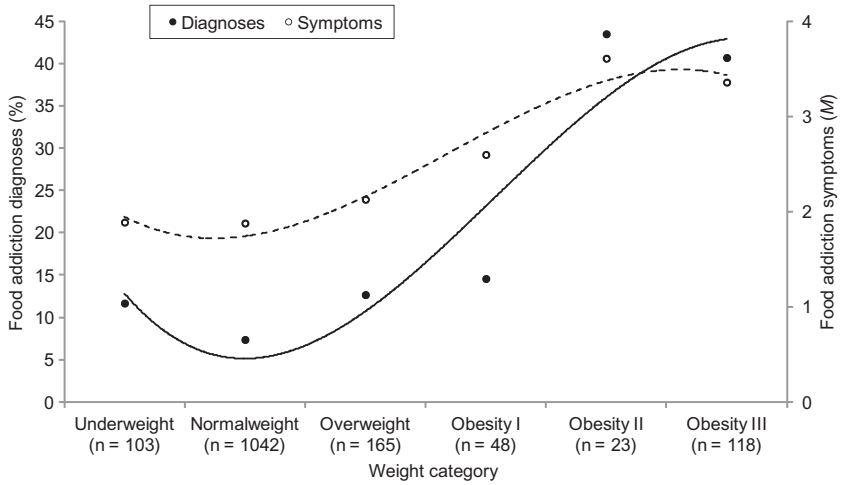

Fig. 1. Food addiction diagnoses and food addiction symptoms as measured with the Yale Food Addiction Scale as a function of weight category (classification based on [1]). Curved lines represent third order polynomial trend lines (diagnoses = continuous line (R^2 = .89); symptoms = dashed line (R^2 = .95)).

Table 1
Food addiction diagnoses and food addiction symptoms as measured with the Yale Food Addiction Scale as a function of weight category (classification based on [1]).

	Underweight (n = 103)		Normal-weight (n = 1042)		Overweight (n = 165)		Obesity I (n = 48)		Obesity II (n = 23)		Obesity III (n = 118)
Diagnoses (%)	11.7	=	7.4[a]	<	12.7[a]	=	14.6[b]	<	43.5[b]	=	40.7
Symptoms (M(SD))	1.9 (1.5)	=	1.9 (1.4)[c]	<	2.1 (1.4)[c]	=	2.6 (1.7)[d]	<	3.6 (1.5)[d]	=	3.4 (1.8)

[a] $\chi^2_{(1)}$ = 5.4, p = .02.
[b] $\chi^2_{(1)}$ = 7.1, p = .008.
[c] U = 75069.0, p = .005.
[d] U = 340.0, p = .008.

the distribution between lean mass and adipose tissue. Accordingly, discrepancies could emerge as a result of which measure is used. For instance, Timmerman and Stevenson [47] found a small positive association between binge eating severity and BMI, but not with body fat percentage. Third, the empirical data presented in this article have to be interpreted with caution because data were aggregated across different studies. This procedure, of course, lacks validity from a methodological point of view because it is possible that some individuals participated in more than one of those studies.

If there is a cubic relationship between food addiction and BMI, the question remains why is this so? It could be speculated that if food addicted individuals in under-to-normal-weight ranges mainly represent those with BN or anorexia nervosa subtypes with binge/purge behaviors, then BMI would be kept in those ranges through compensatory behaviors (i.e. purging or intermittent dietary restriction) despite the presence of addicted eating behavior or binge eating, respectively.

In the case of the second plateau in severely obese individuals, reaching physical limits might constrain a further increase in BMI despite addicted eating patterns. However, psychological factors might also reach a vertex where further increases are not possible. For example, Abilés et al. [61] found increased reports of food cravings in participants with moderate obesity as compared to normal-weight controls, but no differences were observed between severely obese patients (obesity type III and IV). Another explanation could be that within the population of obese individuals, those with food addiction differ from their non-addicted counterparts in several aspects of eating behavior, but not in total food intake. For example, a recent laboratory study found that obese binge eaters exhibited a faster eating rate and ingested larger spoonfuls as compared to obese non-binge eaters, but did not differ in the total amount of energy consumed [52].

Finally, a further factor moderating the relationship between BMI and food addiction could be the intake of alcoholic beverages. Studies investigating the relationship between alcohol use and BMI yielded inconsistent findings [62]. Yet, some studies point out an inverse association between alcohol use and BMI which could be explained by food and alcohol competing for the same brain reward sites [63–65]. In line with this idea, participants who were diagnosed with food addiction reported less alcohol use than their non-addicted counterparts in a sample of severely obese individuals seeking bariatric surgery [66]. Hence, although food addicted individuals might eat more than non-addicted obese individuals, the latter ones might get additional calories from alcoholic beverages which contribute to equal BMI in both groups.

In conclusion, a cubic relationship between BMI and food addiction symptomatology was proposed in the current article indicating an increase in food addiction symptomatology in overweight and obese individuals but stable levels in under-/normal-weight and severely obese individuals. This relationship provides an explanation for the fact that there is a small positive relationship between food addiction (or binge eating) and BMI, but no differences can be found when only normal-weight or severely obese samples are investigated.

Conflict of interest statement

None declared.

Acknowledgements

The author is supported by a grant of the research training group 1253/2 which is supported by the German Research Foundation (DFG) by federal and Länder funds. DFG had no role in the

study design, collection, analysis or interpretation of the data, writing the manuscript, or the decision to submit the paper for publication.

References

[1] World Health Organization. Obesity: preventing and managing the global epidemic. Geneva: World Health Organization; 2000.
[2] Wang YC, McPherson K, Marsh T, Gortmaker SL, Brown M. Health and economic burden of the projected obesity trends in the USA and the UK. Lancet 2011;378:815–25.
[3] Bessesen DH. Update on obesity. J Clin Endocr Metab 2008;93:2027–34.
[4] Yanovski SZ, Yanovski JA. Obesity prevalence in the United States – up, down, or sideways? New Engl J Med 2011;364:987–9.
[5] Finucane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet 2011;377:557–67.
[6] Rokholm B, Baker JL, Sorensen TIA. The levelling off of the obesity epidemic since the year 1999 – a review of evidence and perspectives. Obes Rev 2010;11:835–46.
[7] Barry D, Clarke M, Petry NM. Obesity and its relationship to addictions: Is overeating a form of addictive behavior? Am J Addict 2009;18:439–51.
[8] Davis C, Carter JC. Compulsive overeating as an addiction disorder. A review of theory and evidence. Appetite 2009;53:1–8.
[9] Gearhardt AN, Davis C, Kuschner R, Brownell KD. The addiction potential of hyperpalatable foods. Curr Drug Abuse Rev 2011;4:140–5.
[10] Grosshans M, Loeber S, Kiefer F. Implications from addiction research towards the understanding and treatment of obesity. Addict Biol 2011;16:189–98.
[11] Pelchat ML. Food addiction in humans. J Nutr 2009;139:620–2.
[12] Cassin SE, von Ranson KM. Is binge eating experienced as an addiction? Appetite 2007;49:687–90.
[13] Kenny PJ. Reward mechanisms in obesity: new insights and future directions. Neuron 2011;69:664–79.
[14] Wilson GT. Eating disorders, obesity and addiction. Eur Eat Disord Rev 2010;18:341–51.
[15] Ziauddeen H, Farooqi IS, Fletcher PC. Obesity and the brain: how convincing is the addiction model? Nat Rev Neurosci 2012;13:279–86.
[16] Meule A, Kübler A. The translation of substance dependence criteria to food-related behaviors: different views and interpretations. Front Psychiat 2012;3:1–2.
[17] Avena NM, Rada P, Hoebel BG. Evidence for sugar addiction: behavioral and neurochemical effects of intermittent, excessive sugar intake. Neurosci Biobehav Rev 2008;32:20–39.
[18] Avena NM, Rada P, Hoebel BG. Sugar and fat bingeing have notable differences in addictive-like behavior. J Nutr 2009;139:623–8.
[19] Thornley S, McRobbie H, Eyles H, Walker N, Simmons G. The obesity epidemic: Is glycemic index the key to unlocking a hidden addiction? Med Hypotheses 2008;71:709–14.
[20] Ifland JR, Preuss HG, Marcus MT, et al. Refined food addiction: a classic substance use disorder. Med Hypotheses 2009;72:518–26.
[21] Cocores JA, Gold MS. The salted food addiction hypothesis may explain overeating and the obesity epidemic. Med Hypotheses 2009;73:892–9.
[22] Gearhardt AN, Grilo CM, DiLeone RJ, Brownell KD, Potenza MN. Can food be addictive? Public health and policy implications. Addiction 2011;106:1208–12.
[23] Blundell JE, Finlayson G. Food addiction not helpful: the hedonic component – implicit wanting – is important. Addiction 2011;106:1216–8.
[24] Meule A. How prevalent is "food addiction"? Front Psychiat 2011;2:1–4.
[25] Rogers PJ. Obesity – Is food addiction to blame? Addiction 2011;106:1213–4.
[26] Broft AI, Berner LA, Martinez D, Walsh BT. Bulimia nervosa and evidence for striatal dopamine dysregulation: a conceptual review. Physiol Behav 2011;104:122–7.
[27] Speranza M, Revah-Levy A, Giquel L, et al. An investigation of Goodman's addictive disorder criteria in eating disorders. Eur Eat Disord Rev 2012;20:182–9.
[28] Umberg EN, Shader RI, Hsu LK, Greenblatt DJ. From disordered eating to addiction: the "food drug" in bulimia nervosa. J Clin Psychopharm 2012;32:376–89.
[29] Merlo LJ, Klingman C, Malasanos TH, Silverstein JH. Exploration of food addiction in pediatric patients: a preliminary investigation. J Addict Med 2009;3:26–32.
[30] Eysenck HJ. Addiction, personality and motivation. Hum Psychopharm Clin 1997;12:S79–87.
[31] Lent MR, Swencionis C. Addictive personality and maladaptive eating behaviors in adults seeking bariatric surgery. Eat Behav 2012;13:67–70.
[32] Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale food addiction scale. Appetite 2009;52:430–6.
[33] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. Washington, DC: American Psychiatric Association; 1994.
[34] Meule A, Vögele C, Kübler A. German translation and validation of the Yale Food Addiction Scale. Diagnostica 2012;58:115–26.
[35] Meule A, Kübler A. Food cravings in food addiction: the distinct role of positive reinforcement. Eat Behav 2012;13:252–5.
[36] Gearhardt AN, Yokum S, Orr PT, Stice E, Corbin WR, Brownell KD. Neural correlates of food addiction. Arch Gen Psychiat 2011;68:808–16.
[37] Davis C, Curtis C, Levitan RD, Carter JC, Kaplan AS, Kennedy JL. Evidence that 'food addiction' is a valid phenotype of obesity. Appetite 2011;57:711–7.
[38] Meule A, Heckel D, Kübler A. Factor structure and item analysis of the Yale Food Addiction Scale in obese candidates for bariatric surgery. Eur Eat Disord Rev, in press.
[39] Wonderlich SA, Gordon KH, Mitchell JE, Crosby RD, Engel SG. The validity and clinical utility of binge eating disorder. Int J Eat Disord 2009;42:687–705.
[40] Gearhardt AN, White MA, Potenza MN. Binge eating disorder and food addiction. Curr Drug Abuse Rev 2011;4:201–7.
[41] Gearhardt AN, White MA, Masheb RM, Morgan PT, Crosby RD, Grilo CM. An examination of the food addiction construct in obese patients with binge eating disorder. Int J Eat Disord 2012;45:657–63.
[42] Hudson JI, Hiripi E, Pope HG, Kessler RC. The prevalence and correlates of eating disorders in the national comorbidity survey replication. Biol Psychiat 2007;61:348–58.
[43] Striegel-Moore RH, Franko DL. Epidemiology of binge eating disorder. Int J Eat Disord 2003;34:S19–29.
[44] Wilfley DE, Wilson GT, Agras WS. The clinical significance of binge eating disorder. Int J Eat Disord 2003;34:S96–S106.
[45] Picot AK, Lilenfeld LRR. The relationship among binge severity, personality psychopathology, and body mass index. Int J Eat Disord 2003;34:98–107.
[46] Reichborn-Kjennerud T, Bulik CM, Sullivan PF, Tambs K, Harris JR. Psychiatric and medical symptoms in binge eating in the absence of compensatory behaviors. Obes Res 2004;12:1445–54.
[47] Timmerman GM, Stevenson JS. The relationship between binge eating severity and body fat in nonpurge binge eating women. Res Nurs Health 1996;19:389–96.
[48] Darby A, Hay P, Mond J, Rodgers B, Owen C. Disordered eating behaviours and cognitions in young women with obesity: relationship with psychological status. Int J Obes 2007;31:876–82.
[49] Guss JL, Kissileff HR, Devlin MJ, Zimmerli E, Walsh BT. Binge size increases with body mass index in women with binge-eating disorder. Obes Res 2002;10:1021–9.
[50] Hsu LKG, Mulliken B, McDonagh B, et al. Binge eating disorder in extreme obesity. Int J Obes 2002;26:1398–403.
[51] Müller A, Claes L, Mitchell JE, Fischer J, Horbach T, de Zwaan M. Binge eating and temperament in morbidly obese prebariatric surgery patients. Eur Eat Disord Rev 2012;20:e91–5.
[52] Schulz S, Laessle RG. Stress-induced laboratory eating behavior in obese women with binge eating disorder. Appetite 2012;58:457–61.
[53] Wang GJ, Geliebter A, Volkow ND, et al. Enhanced striatal dopamine release during food stimulation in binge eating disorder. Obesity 2011;19:1601–8.
[54] Davis C, Patte K, Curtis C, Reid C. Immediate pleasures and future consequences. A neuropsychological study of binge eating and obesity. Appetite 2010;54:208–13.
[55] Davis CA, Levitan RD, Reid C, et al. Dopamine for "wanting" and opioids for "liking": a comparison of obese adults with and without binge eating. Obesity 2009;17:1220–5.
[56] Powers PS, Perez A, Boyd F, Rosemurgy A. Eating pathology before and after bariatric surgery: a prospective study. Int J Eat Disord 1999;25:293–300.
[57] Elder KA, Grilo CM, Masheb RM, Rothschild BS, Burke-Martindale CH, Brody ML. Comparison of two self-report instruments for assessing binge eating in bariatric surgery candidates. Behav Res Ther 2006;44:545–60.
[58] Davis C, Fox J. Sensitivity to reward and body mass index (BMI): evidence for a non-linear relationship. Appetite 2008;50:43–9.
[59] Davis C, Strachan S, Berkson M. Sensitivity to reward: implications for overeating and overweight. Appetite 2004;42:131–8.
[60] Verbeken S, Braet C, Lammertyn J, Goosens L, Moens E. How is reward sensitivity related to bodyweight in children? Appetite 2012;58:478–83.
[61] Abilés V, Rodríguez-Ruiz S, Abilés J, et al. Psychological characteristics of morbidly obese candidates for bariatric surgery. Obes Surg 2010;20:161–7.
[62] Sayon-Orea C, Martinez-Gonzalez MA, Bes-Rastrollo M. Alcohol consumption and body weight: a systematic review. Nutr Rev 2011;69:419–31.
[63] Gearhardt AN, Corbin WR. Body mass index and alcohol consumption: family history of alcoholism as a moderator. Psychol Addict Behav 2009;23:216–25.
[64] Gearhardt AN, Harrison ELR, McKee SA. Does co-morbid depression alter the inverse relationship between obesity and substance use disorders? Drug Alcohol Depen 2012;124:185–8.
[65] Kleiner KD, Gold MS, Frost-Pineda K, Lenz-Brunsman B, Perri MG, Jacobs WS. Body mass index and alcohol use. J Addict Dis 2004;23:105–18.
[66] Meule A, Heckel D, Vögele C, Kübler A. Food addiction in obese candidates for bariatric surgery. In: Vögele C, editor, 30. Symposium clinical psychology and psychotherapy of the DGPs section clinical psychology and psychotherapy. Luxembourg: University of Luxembourg; 2012. p. 61.