RESEARCH ARTICLE

Food Addiction and Bulimia Nervosa

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Abstract

In individuals with obesity and binge eating disorder (BED), eating patterns can show addictive qualities, with similarities to substance use disorders on behavioural and neurobiological levels. Bulimia nervosa (BN) has received less attention in this regard, despite their regular binge eating symptoms. The *Yale Food Addiction Scale* (YFAS) was developed according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, diagnostic criteria for substance use disorders, and food addiction can be diagnosed when at least three addiction symptoms are endorsed and a clinically significant impairment or distress is present. Although the prevalence of food addiction diagnoses is increased in individuals with obesity and BED, recent studies which used the YFAS showed that there are also individuals with normal weight who can be classified as being 'food addicted'. Based on self-reported eating disorder symptoms, women with current ($n = 26$) or remitted ($n = 20$) BN, and a control group of women matched for age and body mass index ($n = 63$) completed the YFAS and other measures. Results revealed that all patients with current BN received a food addiction diagnosis according to the YFAS while only six (30%) women with remitted BN did. None of the women in the control group received a food addiction diagnosis. Results provide support for the notion that BN can be described as addiction-like eating behaviour and suggest that food addiction most likely improves when BN symptoms remit.

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Keywords

food addiction; Yale Food Addiction Scale; bulimia nervosa; binge eating; substance dependence

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Introduction

In recent years, a food addiction model of overeating and obesity has gained more and more popularity (Gearhardt, Davis, Kuschner, & Brownell, 2011). Specifically, there is a plethora of theoretical articles discussing the parallels between substance use disorders (SUDs) and obesity or binge eating disorder (BED; e.g. Barry, Clarke, & Petry, 2009; Corsica & Pelchat, 2010; Davis & Carter, 2009; Gearhardt, Corbin, & Brownell, 2009a; Pelchat, 2009). Part of this debate concerns the question whether high-calorie foods or specific ingredients (e.g. sugar) have an addictive potential comparable with drugs of abuse (Avena, Rada, & Hoebel, 2008; Gearhardt et al., 2011; Thornley, McRobbie, Eyles, Walker, & Simmons, 2008).

Nevertheless, food addiction remains a highly controversial and heavily debated issue (Avena, Gearhardt, Gold, Wang, & Potenza, 2012; Meule & Kübler, 2012; Ziauddeen, Farooqi, & Fletcher, 2012a, 2012b; Ziauddeen & Fletcher, 2013). For example, although neuroimaging studies suggest that obesity and binge eating are associated with alterations in dopaminergic signalling and with food-cue elicited hyperactivation of reward-related brain areas, which are comparable with processes seen in drug users (e.g. Volkow, Wang, Fowler, & Telang, 2008), opponents of the food addiction concept note that there are also substantial differences in the neural effects of food and drugs (e.g. Benton, 2010) and that neuroimaging research on brain responses to food and food-related stimuli in individuals with obesity or BED 'is most remarkable for its variability and inconsistency' (Ziauddeen, Farooqi, & Fletcher, 2012b, p. 283). Another major point of discussion is that researchers do not have a unique definition of food addiction or disagree about the exact food addiction equivalents of SUD symptoms (Meule & Kübler, 2012; Ziauddeen et al., 2012b). For example, evidence for some addiction symptoms in relation to food (e.g. development of tolerance or withdrawal symptoms) is restricted to animal models, which have been criticised for being inappropriate when drawing inferences for human eating behaviour (Rippe, in press). Others have argued that—even when the food addiction model is restricted to individuals with binge eating—some substance dependence criteria are only partially adaptable to food/eating-related behaviours and that the core psychopathology of eating disorders is substantially different from substance dependence, for example, because of the crucial involvement of weight and shape concerns (Wilson, 2010; Ziauddeen et al., 2012b). In support of the food addiction concept, others have countered these criticisms by arguing that heterogeneity of brain imaging results regarding food addiction might be due to study differences in stimulus materials,

stimulus presentation and studied populations rather than to conceptual problems or absence of a neural basis for food addiction (Avena et al., 2012). Similarly, the paradigms used in existing animal models may indeed be comparable with the eating behaviour of individuals with eating disorders, and substance dependence criteria can be translated quite well to eating, depending on their definitions and phrasing (Meule, 2014; Meule & Kübler, 2012).

An important step towards overcoming some of these issues was the development of the *Yale Food Addiction Scale* (YFAS; Gearhardt, Corbin, & Brownell, 2009b). This 25-item instrument measures the presence of food addiction symptoms based on the seven substance dependence criteria in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. In addition, two items assess clinically significant impairment or distress, which result from overeating. When both clinically significant impairment and distress are present *and* at least three of the seven criteria are met, then food addiction can be 'diagnosed'. Prevalence rates for food addiction according to the YFAS range between 5–10% in non-clinical samples (Flint et al., 2014; Gearhardt et al., 2009b; Mason, Flint, Field, Austin, & Rich-Edwards, 2013; Meule, Vögele, & Kübler, 2012; Pedram et al., 2013), 15–25% in obese samples (Burmeister, Hinman, Koball, Hoffmann, & Carels, 2013; Davis et al., 2011; Davis et al., 2013; Eichen, Lent, Goldbacher, & Foster, 2013; Lent, Eichen, Goldbacher, Wadden, & Foster, 2014) and 40–60% in obese individuals with BED or morbidly obese bariatric patients (Gearhardt, White, Masheb, & Grilo, 2013; Gearhardt et al., 2012; Meule, Heckel, & Kübler, 2012).

Thus, food addiction symptomatology can only be observed in a subset of obese individuals and appears to be rather associated with binge eating than obesity *per se* (Davis, 2013). For instance, a recent study showed that obese individuals receiving a food addiction diagnosis could be differentiated from non-addicted obese individuals by a dopaminergic multilocus genetic profile as well as features of eating behaviour such as food cravings and binge eating (Davis et al., 2013). Moreover, a substantial proportion of non-obese individuals also receives a food addiction diagnosis (Meule, 2012; Meule & Gearhardt, in press), but mere absence of obesity is a poor description of this population, and the role of eating disorders, which do not necessarily go along with obesity such as bulimia nervosa (BN), is largely unclear.

Although an addiction model of BN has sometimes been discussed in theoretical articles (Umberg, Shader, Hsu, & Greenblatt, 2012; Vandereycken, 1990), only few studies have examined BN from an addiction perspective. Most of those studies compared scores on personality questionnaires between individuals with BN and individuals with substance dependence in an attempt to identify an 'addictive personality' among those with BN (de Silva & Eysenck, 1987; Feldman & Eysenck, 1986; Hatsukami, Owen, Pyle, & Mitchell, 1982; Kagan & Albertson, 1986). Only one study investigated if eating behaviour in BN can actually be described as an addictive behaviour (Speranza et al., 2012). Results showed that individuals with BN met Goodman's addictive disorder criteria (Goodman, 1990) in the same proportion as drug-addicted individuals. A shortcoming of using these criteria, however, is that they did not specify the problematic behaviour. That is, it may refer to eating behaviour as well as compensatory behaviours such as vomiting or exercising.

To date, no study has examined food addiction as measured with the YFAS in individuals with BN. In the current study, we expected that a large proportion of participants reporting bulimic behaviours would be diagnosed with food addiction, comparable with prevalence rates found in individuals with BED (see preceding text). Food addiction diagnoses were expected to be less frequently found in individuals reporting to be remitted from BN and absent in a control group of women matched for age and body mass index (BMI). The remission group was included to learn about the stability of food addiction in BN. If related to the full-blown symptoms of BN, the remitted group should show lower levels of food addiction. If stable and independent of bulimic symptoms, then the remitted group should show similarly high degrees of food addiction as the BN group. Furthermore, we descriptively evaluated endorsement rates of each food addiction symptom as these may differ between obese and non-obese samples (Meule, Heckel, et al., 2012).

In addition to these main research questions, we explored the following correlates of addictive-like eating. First, similar to previous findings, scores on the YFAS (i.e. the dichotomous diagnostic score and the continuous symptom count) were expected to be positively related to eating disorder pathology, restrained, emotional, external eating, and depressive symptoms (Gearhardt et al., 2009b; Gearhardt et al., 2013; Gearhardt et al., 2012; Meule, Heckel, Jurowich, Vögele, & Kübler, in press; Meule, Vögele, et al., 2012). Second, as bulimic symptomatology is positively related to impulsivity and borderline personality disorder (BPD; Waxman, 2009) and YFAS scores have been associated with higher impulsivity (e.g. Davis et al., 2011; Meule, et al., in press), we expected that scores on the YFAS would also be positively related to BPD symptomatology. Third, we explored whether YFAS scores are associated with participant characteristics such as age, years of education and BMI.

Materials and methods

Participants and procedure

This study was reviewed and approved by the ethical committee of the University of Salzburg, Austria. Women with a current diagnosis or a history of BN were recruited via outpatient clinics and counselling centres specialised on eating disorders in southern Germany. Participants signed written informed consent.

Women who reported to have a current diagnosis of BN ($n = 34$) were excluded if they were currently underweight (BMI $< 17.5\,kg/m^2$), scored below the cut-off of the *Eating Disorder Examination Questionnaire* (EDE-Q) of 2.3 (Mond, Hay, Rodgers, Owen, & Beumont, 2004)[1] or reported less than eight binge or less than eight purging episodes within the past 28 days on the EDE-Q. As a result, eight participants were excluded, leaving a final sample size of $n = 26$ for the current BN group.

Women who reported to have a history of BN ($n = 26$) were excluded if they were currently underweight (BMI $< 17.5\,kg/m^2$),

[1]Note that we used this cut-off only for this group to ensure that individuals in the BN group met full criteria for BN. We did not use it for the other two groups as the aim here was to ensure that individuals did not exhibit bulimic symptomatology, but it is still possible to have high EDE-Q total scores in the absence of an eating disorder.

engaged in regular binge eating (more than 4 binge days within the past 28 days) or reported at least one binge episode *and* one purging episode within the past 28 days on the EDE-Q. As a result, six participants were excluded, leaving a final sample size of $n = 20$ for the remitted BN group. All participants in the remitted BN group reported that their last week with at least two binge/purge episodes was at least 3 months ago ($M = 21.70$ months, $SD = 24.97$).

A control group of women without eating disorders were recruited via a mailing list of the University of Ulm, Germany, and completed the questionnaires online ($n = 70$). Participants in this group were excluded if they were currently underweight (BMI $< 17.5\,kg/m^2$), engaged in regular binge eating (more than 4 binge days within the past 28 days) or reported at least one binge episode *and* one purging episode within the past 28 days on the EDE-Q. As a result, seven participants were excluded leaving a final sample size of $n = 63$ for the control group.

Measures

Yale Food Addiction Scale

The German version of the 25-item YFAS (Meule, Vögele, et al., 2012) was used for the assessment of food addiction. A symptom count ranging between zero and seven food addiction symptoms can be calculated as a continuous score. Food addiction diagnoses can be calculated as a dichotomous score when at least three symptoms are met and a clinically significant impairment or distress is present. As the YFAS includes different response categories (for detailed scoring instructions, please see Meule & Gearhardt, in press), internal consistency is calculated after relevant items have been dichotomized, and thus, Kuder-Richardson's alpha coefficient is used, which was $\alpha = .94$ in the current study.

Eating Disorder Examination Questionnaire

The German version of the 28-item EDE-Q (Hilbert & Tuschen-Caffier, 2006) was used for the assessment of eating disorder pathology within the past 28 days. Twenty-two of these items measure eating disorder symptomatology on the subscales restraint, eating concern, weight concern and shape concern. Internal consistency was Cronbach's $\alpha = .97$ for the total scale and ranged between $\alpha = .90$ and .92 for the subscales. The six remaining items assess binge eating and compensatory behaviours. Of these, only the two items assessing days with objective binge episodes (i.e. consumption of large amounts of food *and* a feeling of loss of control) and the number of times of self-induced vomiting were used in the current study.

Dutch Eating Behaviour Questionnaire

Twenty items of the German version of the Dutch Eating Behaviour Questionnaire (Grunert, 1989) were used for the assessment of emotional and external eating behaviour. The items of the restraint subscale were not administered as restraint was already covered by the EDE-Q. Internal consistencies were Cronbach's $\alpha = .95$ (emotional eating) and $\alpha = .88$ (external eating) in the current study.

Borderline Symptom List short version

The 23-item short version of the German Borderline Symptom List (Bohus et al., 2009) was used for the assessment of BPD symptomatology within the past 7 days. Internal consistency was Cronbach's $\alpha = .96$ in the current study.

Brief Symptom Inventory

The 18-item short version of the German Brief Symptom Inventory (Spitzer et al., 2011) was used for the assessment of symptoms of somatization, depressiveness and anxiety within the past 7 days. Only its total score, the so-called *Global Severity Index*, was used in the current study, and internal consistency was Cronbach's $\alpha = .92$.

Centre for Epidemiologic Studies Depression Scale short version

The 15-item short version of the German version of the Centre for Epidemiologic Studies Depression Scale (Hautzinger, Bailer, Hofmeister, & Keller, 2012) was used for the assessment of depressive symptoms in the past 7 days. Internal consistency was Cronbach's $\alpha = .93$ in the current study.

Sociodemographic and anthropometric information

Participants reported their sex (male/female), age (years), height (cm), weight (kg), nationality (Austrian/German/other) and years of education (including school, job training and university/college years). Individuals with a history of BN additionally reported the time since their last regular binge/purge episodes (i.e. at least twice a week) in months.

Data analyses

Group differences on continuous variables were tested with analyses of variance and followed up with Scheffé tests. Group differences regarding the frequency of YFAS diagnoses were tested with χ^2-tests. To elucidate associations between study variables and the YFAS, the diagnostic score of the YFAS was used across all groups, and participants receiving a food addiction diagnosis (food addiction group) were compared with those not receiving a food addiction diagnosis (no food addiction group) with unpaired *t*-tests. Furthermore, relationships between the YFAS symptom count and study variables were examined with Pearson correlations across all participants. Endorsement rates (in %) of each food addiction symptom are presented descriptively for the food addiction and the no food addiction group.

Results

Group differences

Groups did not differ in age or years of education (Table 1). Most participants had normal weight, except that one participant in the BN group and five participants in the control group were overweight (BMI $> 24.99\,kg/m^2$). However, groups did not differ in BMI (Table 1). The proportion of food addiction diagnoses significantly differed between groups ($\chi^2_{(2)} = 88.75$, $p < .001$). All individuals with current BN, six individuals with remitted BN, and none of the women in the control group received a food addiction diagnosis (Figure 1; all groups differed significantly from each other: all $\chi^2_{(1)}$s 20.37, $p < .001$). Likewise, the current BN group reported more food addiction symptoms than the remitted BN group, which in turn reported more food addiction symptoms than the control group (see Table 1 for means, standard deviations and test statistics).

Table 1 Descriptive and test statistics of continuous study variables

$N = 109$	Current BN ($n = 26$)		Remitted BN ($n = 20$)		Control group ($n = 63$)		ANOVA		
	M	*SD*	*M*	*SD*	*M*	*SD*	$F_{(2,106)}$	*p*	η_p^2
Age (years)	25.23	5.82	25.55	3.72	23.57	4.20	2.09	ns	.04
Education (years)	14.19	2.12	15.05	2.69	15.29	3.37	1.23	ns	.02
Body mass index (kg/m^2)	20.92	1.92	21.92	1.50	21.84	2.65	1.61	ns	.03
YFAS symptom count	6.27^a	1.04	3.95^b	1.79	0.86^c	0.90	221.73	<.001	.81
EDE-Q—total	4.21^a	0.81	2.13^b	1.00	0.82^c	0.80	151.77	<.001	.74
EDE-Q—restraint	3.99^a	1.23	1.83^b	1.15	0.72^c	1.01	83.38	<.001	.61
EDE-Q—eating concern	4.39^a	1.12	1.58^b	0.87	0.28^c	0.45	283.80	<.001	.84
EDE-Q—weight concern	4.06^a	1.26	2.40^b	1.25	0.93^c	1.04	72.38	<.001	.58
EDE-Q—shape concern	4.40^a	0.99	2.70^b	1.18	1.33^c	1.18	68.53	<.001	.56
EDE-Q—binge days	32.54^a	21.13	1.25^b	1.68	0.10^b	0.53	96.70	<.001	.65
EDE-Q—self-induced vomiting	35.38^a	23.58	0.10^b	0.31	0.06^b	0.35	94.11	<.001	.64
DEBQ—external eating	35.42^a	7.97	32.00^{a,b}	6.55	30.02^b	7.15	5.14	.007	.09
DEBQ—emotional eating	37.46^a	7.27	27.45^b	6.22	19.56^c	7.56	57.00	<.001	.52
BSL-23	1.61^a	0.78	0.61^b	0.47	0.37^b	0.56	39.37	<.001	.43
BSI-18—total	1.15^a	0.56	0.46^b	0.45	0.31^b	0.46	27.87	<.001	.35
CES-D—short form	18.96^a	6.59	12.05^b	6.57	7.38^c	6.48	29.31	<.001	.36

Notes: BN, bulimia nervosa; YFAS, Yale Food Addiction Scale; EDE-Q, Eating Disorder Examination Questionnaire; DEBQ, Dutch Eating Behaviour Questionnaire; BSL-23, Borderline Symptom List; BSI-18, Brief Symptom Inventory; CES-D, Centre for Epidemiologic Studies Depression Scale.
Group means with different superscripts are significantly different from each other based on Scheffé tests (all *p*'s < .03).

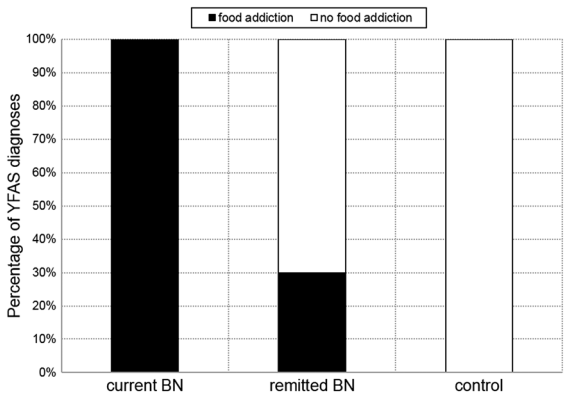

Figure 1. Percentage of diagnoses according to the Yale Food Addiction Scale (YFAS) in individuals with a current diagnosis of bulimia nervosa (BN) and remitted BN and in the control group

Similar group differences were found for the EDE-Q total score and all of its subscales, emotional eating and depressive symptoms. However, the remitted BN group did not differ from the control group with regard to binge eating and purging frequency (confirming our group assignment), external eating, BPD symptomatology and the Global Severity Index (Table 1).

Associations with the Yale Food Addiction Scale

The food addiction group had fewer years of education and a marginally lower BMI than the no food addiction group. Age did not differ between groups. The food addiction group had higher eating disorder and general psychopathology than the no food addiction group on all questionnaire measures (Table 2).

Mimicking those results, the YFAS symptom count was positively correlated with all measures of eating disorder and general psychopathology and marginally negatively correlated with BMI. Food addiction symptoms were uncorrelated with age or years of education (Table 2). The most often endorsed food addiction symptom was a *persistent desire or unsuccessful effort to cut down or control eating*, while the least often endorsed symptom was *tolerance* (Table 3).

Discussion

The current study aimed to investigate the concept of food addiction and its correlates in women with bulimic symptomatology. All participants currently exhibiting bulimic behaviours and 30% of those with a history of BN could be classified as 'food addicted' according to the YFAS while none of the women in the control group received such a diagnosis. This finding is in line with the study by Speranza *et al.* (2012) as it clearly demonstrates that eating behaviour in individuals with BN is highly comparable with substance use in SUDs and that bulimic behaviour may itself be described as an addicted behaviour. These findings based on self-reported behaviour are complemented by recent examinations of neurobiological processes that show similarities between BN and substance dependence as well (Hadad & Knackstedt, 2014).

Notably, prevalence of YFAS diagnoses was higher in the current sample of individuals with BN as compared with samples of individuals with BED, in which about 50% received a diagnosis (Meule & Gearhardt, in press). This finding is in line with the fact that the binge episodes differ between individuals with BN and BED in various aspects. For example, those with BN consume more carbohydrates/sugar, experience eating as less pleasurable and have more negative food-related, eating-related and stress-related

Table 2 Comparisons between participants with and without an YFAS diagnosis and correlations with the YFAS symptom count

$N = 109$	Food addiction ($n = 32$)		No food addiction ($n = 77$)		*t*-test		Food addiction symptoms	
	M	*SD*	*M*	*SD*	$t_{(107)}$	*p*	*r*	*p*
Age (years)	25.31	5.49	23.92	4.16	1.44	ns	.13	ns
Education (years)	13.97	2.12	15.40	3.22	2.32	.02	−.18	ns
Body mass index (kg/m^2)	21.02	1.90	21.89	2.45	1.79	.08	−.19	.05
EDE-Q—total	3.85	1.15	1.04	0.95	13.17	<.001	.82	<.001
EDE-Q—restraint	3.61	1.51	0.92	1.08	10.57	<.001	.73	<.001
EDE-Q—eating concern	3.94	1.42	0.49	0.70	16.95	<.001	.86	<.001
EDE-Q—weight concern	3.78	1.37	1.18	1.21	9.83	<.001	.73	<.001
EDE-Q—shape concern	4.07	1.29	1.58	1.27	9.31	<.001	.74	<.001
EDE-Q—binge days	26.91	22.42	0.21	0.77	10.50	<.001	.65	<.001
EDE-Q—self-induced vomiting	28.78	25.36	0.06	0.34	10.00	<.001	.64	<.001
DEBQ—external eating	34.94	7.97	30.31	6.94	3.03	.003	.34	<.001
DEBQ—emotional eating	35.78	7.71	20.91	7.92	9.00	<.001	.75	<.001
BSL-23	1.46	0.79	0.40	0.55	8.05	<.001	.63	<.001
BSI-18—total	1.06	0.59	0.32	0.45	7.09	<.001	.58	<.001
CES-D—short form	18.38	6.89	7.94	6.36	7.62	<.001	.60	<.001

Notes: YFAS, Yale Food Addiction Scale; EDE-Q, Eating Disorder Examination Questionnaire; DEBQ, Dutch Eating Behaviour Questionnaire; BSL-23, Borderline Symptom List; BSI-18, Brief Symptom Inventory; CES-D, Centre for Epidemiologic Studies Depression Scale.

Table 3 Endorsement rates of Yale Food Addiction Scale symptoms

$N = 109$	Food addiction ($n = 32$)		No food addiction ($n = 77$)	
	n	%	*n*	%
Persistent desire or unsuccessful effort to cut down or control eating	32	100	42	54.5
Giving up social, occupational, or recreational activities	31	96.9	10	13.0
Withdrawal symptoms	31	96.9	14	18.2
Spending much time obtaining food or eating or recover from its effects	28	87.5	8	10.4
Continued eating despite physical or psychological problems	27	84.4	9	11.7
Consumption of large amounts or over a longer period than intended	26	81.3	10	13.0
Tolerance	21	65.6	7	9.1

cognitions during binge episodes than those with BED (Fitzgibbon & Blackman, 2000; Hilbert & Tuschen-Caffier, 2007; Mitchell et al., 1999). The possibility exists that the higher general psychopathology of individuals with BN contaminated YFAS scores, that is, more severe YFAS scores may be due to more severe psychopathology overall. However, a recent study among overweight individuals found that the relationship between YFAS scores and higher psychopathology was fully mediated by binge eating severity, suggesting that the association between food addiction and psychopathology can indeed be explained by disordered eating behaviour (Imperatori et al., in press).

It should be noted, however, that the core psychopathology of BN also includes an overvaluation of body weight and shape that drives dysfunctional dieting, resulting in binge eating and dysfunctional compensatory behaviours (Wilson, 2010). These aspects are absent in SUDs, suggesting that, besides many similarities, there are also some fundamental differences between BN and drug addiction. In the current study, not only binge eating but also weight and shape concern were highly correlated with the number of food addiction symptoms. Thus, future research on food addiction may examine the role of such aspects in more detail beyond focusing on binge eating.

Previous studies on food addiction in non-obese individuals found that between 5% and 10% fulfil food addiction criteria according to the YFAS (Meule, 2011, 2012). However, this research did not assess BN symptomatology. The high percentage of food addiction in BN suggests that some of these 'healthy participants' might actually be those with BN or with subclinical bulimic symptoms. Note that none of our healthy, non-BN or non-BN-history individuals fulfilled the YFAS diagnostic criteria. This supports the idea that non-bulimic, non-obese individuals rarely qualify for a food addiction diagnosis. However, future studies with large samples of normal-weight individuals who are free of bulimic symptoms are needed to elucidate if YFAS food addiction status is exclusively met by participants with BN or if those with BN only account for a subset of normal-weight individuals receiving a food addiction diagnosis. Examination of relationships with BMI revealed that there was a small, negative association between food addiction symptomatology and body mass, which contrasts findings of a positive relationship in samples with a wide range in BMI (Flint et al., 2014; Pedram et al., 2013). Bulimic individuals may not gain weight because of compensatory behaviours despite engaging in addiction-like eating. To conclude, results of the current study suggest that bulimic symptomatology is strongly associated with addiction-like eating as measured with the YFAS and that it contributes to an attenuation of the positive relationship between food addiction symptomatology and BMI.

Replicating prior studies in which the YFAS was used, food addiction symptomatology was related to higher eating pathology and depressive symptoms (Gearhardt et al., 2009b; Gearhardt et al., 2012; Gearhardt et al., 2013; Meule, Vögele, et al., 2012). In addition, the present study is the first to show that food addiction also goes along with BPD symptomatology. As impulsivity is

a core feature of BPD (i.e. it is even included in the diagnostic criteria; American Psychiatric Association, 2013; Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001), this finding mirrors findings of a positive correlation between impulsivity scales and the YFAS (Davis et al., 2011; Meule, 2013; Meule, Lutz, Vögele, & Kübler, 2012; Murphy, Stojek, & MacKillop, 2014; Meule et al., in revision). Future studies may investigate if impulsivity mediates the relationship between BPD and food addiction symptomatology.

When examining the most often endorsed food addiction symptoms, differences could be observed to previous studies. In line with other studies, the most often endorsed symptom was *a persistent desire or unsuccessful efforts to cut down or control eating* (Flint et al., 2014; Gearhardt, Corbin, & Brownell, 2008). Yet, two other most often endorsed symptoms were *giving up important activities* and *withdrawal symptoms* (Table 3), which are less frequently endorsed in samples with obese individuals (Eichen et al., 2013; Gearhardt et al., 2012; Gearhardt et al., 2013; Meule, Heckel, et al., 2012; Meule, Hermann, & Kübler, 2013). Contrarily, the least often endorsed symptom in the present study was *tolerance*, which is one of the most often endorsed symptoms in obese individuals (Eichen et al., 2013; Gearhardt et al., 2012; Gearhardt et al., 2013; Meule, Heckel, et al., 2012; Meule, Hermann, & Kübler, 2013). A possible explanation for this may simply be an effect of age: samples of obese individuals usually have a higher age as compared with our sample of young adults, and development of tolerance may primarily occur after several years of addiction-like eating. Future studies may directly compare endorsement rates of food addiction symptoms between different age and weight classes to reveal if there are actually systematic and stable differences between normal-weight and obese individuals who receive a food addiction diagnosis.

One potential clinical implication of the present finding of addiction-like characteristics in individuals with BN would be to adapt SUD interventions for BN. This might include psychoeducational modules on addiction or stimulus control procedures that reduce exposure to 'addictive' foods (in contrast to current practice in BN psychotherapy). For example, case reports exist, which showed that providing an addiction framework motivated change in patients with BN and their families (Slive & Young, 1986). Some self-help groups encourage to avoid certain 'addictive' foods (e.g. Russel-Mayhew, von Ranson, & Masson, 2010), which is usually considered contraindicated in the treatment of BN (Wilson, 2010). Techniques such as motivational interviewing or acceptance-oriented imagery to cope with urges may be adapted from SUD treatments to enhance motivation to change and dealing with food cravings (Davis & Carter, 2014). Finally, pharmacotherapy targeting opioid and dopaminergic neurotransmitter systems have been shown to be effective in SUD treatments and may also be helpful in reducing binge eating (Davis & Carter, 2014; Hadad & Knackstedt, 2014). For example, a recent animal study showed that an aldehyde dehydrogenase inhibitor, which has been shown to reduce alcohol and cocaine intake in rats, selectively attenuated binge eating of palatable foods and dopamine release in sugar-bingeing rats (Bocarsly et al., 2014).

A major limitation of the current study was that all information was assessed via self-report. For example, it is known that self-reported height and weight is biassed such that height usually is overestimated and weight is underestimated. However, it has also been found that although such discrepancies exist, reports are usually sufficiently accurate (Bowman & DeLucia, 1992; Pursey, Burrows, Stanwell, & Collins, 2014). Moreover, scores on the EDE-Q highly correlate with those obtained through the EDE-Interview, particularly the frequency of objective binge episodes and self-induced vomiting (Hilbert, Tuschen-Caffier, Karwautz, Niederhofer, & Munsch, 2007). Thus, we would argue that although we relied on self-report, our classification in diagnostic groups was valid. Yet, future studies are needed in which bulimic symptomatology is examined via diagnostic interviews. Finally, although each food addiction symptom as measured with the YFAS was endorsed by a subset of participants, support for the existence of some of those symptoms, for example, tolerance and withdrawal, by other measures is restricted to animal studies (Meule & Kübler, 2012). Thus, sophisticated experimental or field studies in humans are necessary to validate the YFAS food addiction assessment. For example, elaborated longitudinal studies in which food intake is objectively measured are needed to further support the existence of these symptoms in humans. Other studies may make use of *Ecological Momentary Assessment* in order to reveal if individuals with an YFAS diagnosis do in actuality spend more *time in activities necessary to obtain food, eat, and recover from overeating* as compared with individuals not receiving an YFAS diagnosis. Such studies may provide less biassed support for the existence of food addiction symptoms in individuals with BN and humans in general.

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