

## Obesity Facts

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Obesity Facts

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## **Research Article**

### **Correlates of Food Addiction and Eating Behaviours in Patients with Morbid Obesity**

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**Short Title:** Correlates of Food Addiction and Eating Behaviours

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#### **Abstract**

**Introduction:** Food Addiction (FA) is a promising construct regarding the multifactorial aetiology of obesity and the search for therapeutic approaches. However, there is an ongoing debate regarding the overlap/differentiation with eating disorders and the classification as a substance- or behaviour-related addiction. Energy-dense foods, especially those combining carbohydrates and fat, are associated with addictive eating and suspected of playing a role in the genesis of FA. This study aims to further understand the clinical significance of FA and to identify possible therapeutic targets. A special focus is set on potentially addictive foods (combination of carbohydrates and fat).

**Methods:** Based on the Yale Food Addiction Scale 2.0 a cohort of 112 German adults with morbid obesity was divided into two sub-samples (patients with and without FA), which were examined for differences in the variables listed below.

**Results:** The prevalence of FA was 25%. Patients meeting criteria for FA showed higher degrees of hunger, emotional, binge and night eating than patients without FA. In addition, hunger and disinhibition were found to be significant predictors of FA. FA was not associated with sex, age, BMI, cognitive restraint, rigid and flexible control, prevalence of substance use, age of onset of obesity,

stress level, level of social support, reduction of BMI during a weight loss programme or programme withdrawal rate. There was no significant difference in the consumption of foods rich in both carbohydrates and fat, nor of fat or carbohydrates alone.

**Conclusion:** FA can be considered as a sub-phenotype of obesity, occurring in approximately 25% of obesity cases. Dysfunctional emotional coping mechanisms associated with low distress tolerance showed to be significantly related to FA and should be targeted therapeutically. Behavioural interventions should include a bio-psycho-social model. Binge eating episodes were found to be characteristic for FA and the already stated overlap between FA and binge eating behaviour can be confirmed. The results do not support a decisive difference due to a substance-related component of FA. Despite this, the existence of FA as a distinct entity cannot be excluded, as not all patients with FA exhibit binges.

## Introduction

Obesity is a growing public health problem. Over the last 30 years obesity prevalence has nearly doubled worldwide [1]. In Germany almost 24% of the population meet the criteria for obesity [2]. Beside negative impact on both physical and mental health of persons concerned [2], unsatisfactory attempts in prevention and therapy are also a financial burden on the public health system [3]. The origins of obesity are multifactorial. An obesogenic environment (increased availability of energy-dense food, larger portion size) as well as decreased physical activity (sedentary lifestyle) are essential contributors [4].

The idea of a Food Addiction (FA) was already introduced in 1956 by Theron Randolph and has become an emerging field of study in recent years [5]. A number of studies suggest that in some cases overeating might result from an eating-related addiction similar to drug addiction [4, 6]. Neuro-imaging studies indicate that FA is associated with neural alterations in impulse control [7], dysfunctional reward-related neural activity [8] and down-regulation in the dopamine system [4, 9], as seen in drug addiction. There is also a significant overlap with eating disorders especially binge eating disorder and with behaviour-related addictions.

The construct of FA was created by transferring the diagnostic criteria for substance-related and addictive disorders (SRAD) of the DSM (Diagnostic and Statistical Manual of Mental Disorders) [10] to addictive-like eating. Nonetheless, FA is not a clinical diagnosis, neither in the Diagnostic and Statistical Manual of Mental Disorders version 5 (DSM-5) nor the International Classification of Disease (ICD-11) [11], but remains for now a scientific construct of interest [8].

The Yale Food Addiction Scale 2.0 (YFAS 2.0) is a validated self-report questionnaire asking these DSM-5 criteria in order to measure addictive-like eating behaviour and to 'diagnose' FA [12]. The average prevalence of FA is 10% in healthy populations [11] and is higher in populations with obesity (25% on average [11]) and especially among individuals with eating disorders such as binge eating (70 to 90% [13]) and bulimia nervosa (95%) [6, 11].

While there is increasing research on the topic [5, 14], the construct of FA is not fully understood and remains a controversial issue [14]. Despite the above mentioned overlap of FA and binge eating, these two eating (disorders) traits are assumed to be different clinical phenomena [15]. The precise overlapping and distinctive features between FA and established eating disorders, especially binge eating, are not clear and warrant further examination [14].

Moreover, there is an ongoing debate on whether FA should be assigned to the substance-related or behaviour-related addictions [14]. On the one hand, studies posit a neurobiological vulnerability of certain individuals who appear to be susceptible to coping with negative emotions by using food [6]. In this sense, 'eating addiction' should be considered as a more appropriate term. On the other hand, animal models suggest that specific highly processed foods, which are similar to drugs of abuse with respect to their pharmacokinetics and their triggered neural responses, hold an addictive potential [6, 8]. Experimental studies mostly in animals have shown that energy-dense foods, especially those high in sugar and fat, have similar biochemical effects as drugs (increase of dopamine, unnaturally high

levels of reward) and are associated with addictive eating behaviour [4, 16–18]. There are, however, some points of criticism about analogising certain foods with drugs of abuse. Some point out that food and calories, in contrast to drugs, are necessary for survival [3, 8]. Moreover, foods are never pure substances but mixtures of different chemical substances [19]. Pure sugar and pure fat typically do not cause addictive eating behaviour. But a mixture of both in a palatable food matrix might do in vulnerable patients. There is a clear research gap in the context of addictive eating concerning the differentiation between foods containing carbohydrates, fat and foods that combine the two [4]. Some studies have examined the association of FA and problematic food categories, however, to our knowledge, these have done so solely in healthy populations, not in a sample with morbid obesity [4, 17, 20].

### **Objectives**

This study aims to further scrutinise the construct of FA. Correlates proposed in previous studies were tested to contribute to a better understanding and definition of FA. Given that not every individual affected by obesity fulfils the YFAS criteria for FA, a distinction between obesity with and without FA is of crucial interest. This study compares groups with morbid obesity with and without FA with respect to sex, age, BMI, onset of obesity in the course of life, eating behaviours (hunger, disinhibition, cognitive restraint, rigid and flexible control), dysfunctional eating behaviours (binge eating, emotional eating, night eating), prevalence of substance use, stress level, level of social support, reduction of Body Mass Index (BMI) during a weight loss programme and programme withdrawal rate. In addition, the study focuses on differences in problematic food categories (foods containing carbohydrates, foods containing fat, foods containing both), to address the above-mentioned research gap. It was hypothesised that the current failure of obesity treatment might be partly due to the lack of treatment of FA [11]. In this regard, it is of essential interest to further understand the clinical significance of FA and to identify possible therapeutic targets.

### **Hypotheses**

Based on previous research in a sample with morbid obesity the prevalence of FA was assumed to be 25% [11]. As earlier studies suggest, it was hypothesised that the group with FA is characterised by more females [14, 21], lower age [5, 22], higher BMI [14, 23–25], an earlier onset of obesity in the course of life, a higher grade of hunger [14], disinhibition [14] and rigid control [19] of eating behaviour, however a lower grade of cognitive restraint and flexible control, higher scores of binge eating behaviour [14, 23–27], emotional eating [26–28] and night eating [14, 29], more distinct problematic consumption of, in particular, foods that combine fat and carbohydrates, higher prevalence of other substance use [25], higher stress level [30], less social support, less BMI reduction during the weight loss programme [27] and a higher programme withdrawal rate.

## **Materials and Methods**

### **Study Sample**

A total of 112 patients were included into the study. They were recruited from the Adiposity Centre of the university hospital in Göttingen, Germany, during a two year period. All participants were included in a one-year conservative weight reduction programme. As part of the programme's primary assessment participants completed three self-report questionnaires: The German version of the Yale Food Addiction Scale 2.0 (YFAS 2.0) [23], the 'Fragebogen zum Essverhalten' (FEV, German version of the 'Three Factor Eating Questionnaire' (TFEQ)) [31, 32] and the 'Fragebogen zu Körpergewicht und Lebensstil' (Questionnaire on Body Weight and Lifestyle), based on the English-language "Weight and Lifestyle Inventory" [33]. None of the patients declined to participate in the study.

### **Food Addiction Assessment**

The YFAS 2.0 [12] is a validated, self-report questionnaire which allows the identification of participants exhibiting addiction-like eating behaviour. For this the diagnostic criteria for substance-related and addictive disorders (SRAD) of the DSM-5 (Diagnostic and Statistical Manual of Mental

Disorders) [10] have been transferred to addictive-like eating. In 35 questions with a scale, ranging from 'never' to 'every day', [23] the YFAS measures these eleven criteria (loss of control, unsuccessful cut-down, time spent, activities given up, aversive consequences, tolerance, withdrawal symptoms, interpersonal problems, impaired daily functioning, dangerous situations, craving) [10] as well as clinical impairment. The 'diagnosis' of FA can be given if a person meets two or more criteria and is clinically significantly impaired [22]. Because FA is not registered as a SRAD in the DSM-5, the term 'diagnosis' cannot be seen from a clinical point of view, only from a scientific one. It simply indicates the fulfilment of the YFAS 2.0 criteria for FA. The YFAS allowed dividing the sample into two sub-samples: Participants with FA and participants without FA. These sub-samples were subsequently examined for differences concerning several variables.

### **Eating Behaviours**

Different eating behaviours were measured with the extended FEV (Fragebogen zum Essverhalten) [31], the German version of the 'Three Factor Eating Questionnaire' (TFEQ) [32]. A total of 88 questions with mostly dichotomous answering options (yes or no) cover the following five dimensions of eating behaviour: *Disinhibition*, *hunger* and *cognitive restraint* (i.e. the degree of deliberate control of eating behaviour) with the subcategories *flexible control* and *rigid control* [34].

### **Binge Eating**

One question of the FEV extension relates to episodes of ravenous appetite. If the question is answered in the affirmative, the respondent is requested to answer seven more sub-questions concerning the characteristics of binge-eating according to the diagnostic criteria for binge eating disorder of the DSM-5 (a. eating considerably huge amounts, b. eating more quickly than usual, c. eating until a feeling of unpleasant fullness, d. eating alone out of embarrassment about the amount, e. lack of sufficient control about what and how much food, f. eating despite lack of physical hunger, g. feeling of guilt, depression or disgust by oneself) [10]. Depending on how many of these seven items applied to the respondent, a binge eating score from zero to seven was set. It should be noted that the available data did not allow a diagnosis of clinically confirmed binge eating disorder; they only provided indications of binge eating behaviour.

### **Night Eating**

Patients were classified with night eating behaviour according to two questions of the FEV reflecting the description of night eating syndrome in the DSM-5 [10]. One question asks about becoming hungry in the late evening or nighttime. Another question of the FEV extension is split into three sub-questions: a. bad sleep after an extensive dinner, b. loss of appetite and skip of breakfast on the following day, c. frequent nocturnal rising in order to eat. Only if all three sub-questions were answered with 'yes', this question was classified affirmatively. To attribute night eating behaviour to a subject, at least one of the two questions had to be answered in the affirmative.

### **Emotional Eating**

To measure the degree of emotional eating behaviour, a score ranging from zero to five was created out of five relevant questions (two of YFAS, three of FEV). One question of the FEV enquires the habit of eating out of fear or stress. Another question of the FEV is about eating too much out of grief. The third question of the FEV relates to the symptom of comforting with food in case of loneliness. For each question answered affirmatively one point was given. One question of the YFAS enquires the consumption of certain food out of emotional problems due to the lack of these foods. Another question of the YFAS is about the decrease of negative emotions such as sadness or an increase in well-being by eating more. As the YFAS has an eight-point Likert-scale, the numeric value was divided by eight (scale from 1/8 to 8/8) in order to achieve a weighting equal to the FEV (0 or 1). For this reason, the created emotional eating score ranges from zero to five and can also contain decimals.

### **Problematic Food Categories**

The sub-samples (patients with FA/without FA) were also compared concerning the type of food they consume in situations of engorgement. One question in the 'Questionnaire on Body Weight and Lifestyle' requests the participants to note down their preferred foods in situations of over-eating in a

free text field. The listed food items were classified into three categories: (a) Foods containing mainly carbohydrates (e.g. pasta, bread, potatoes, lemonade), (b) foods containing mainly fat (e.g. meat, sausage, nuts, butter) and (c) foods containing both carbohydrates and fat (e.g. pizza, chips, crisps, cake, cookies, ice cream). Participants could be assigned to either one, two or all three categories, or, in case of an uncompleted free text field, to none of them.

#### **Other variables**

Body height and weight were measured in the clinic at the beginning of the programme and BMI was calculated. The 'Questionnaire on Body Weight and Lifestyle' recorded information on age, gender, age at onset of obesity in the course of life, stress level of the last six weeks (on a scale from zero to three), degree of social support of personally important persons (scale from zero to six), BMI reduction during the programme and prevalence of other substance use (alcohol, smoking, other substances). Programme withdrawal rate was operationalised by either official written notice of termination or a break-off of weekly measurements and documentation of body weight during the respective programme year. For an overview of all score ranges and its meanings, see Table 1 and its notes.

#### **Statistical Analysis**

Statistical analyses were performed with SPSS (Statistical Package for Social Science Software, IBM SPSS Statistics) versions 25 and 26 [35]. T-tests examined the differences between the sub-samples with and without FA in metric variables (binge eating score, emotional eating score, the five FEV dimensions of eating behaviour, BMI, age, age at onset of obesity, stress level, degree of social support, weight loss during the programme) after normal distribution has been tested graphically using Q-Q plots. Chi-square tests examined differences in categorical variables (prevalence of night eating, problematic consumption of the three food categories, sex, prevalence of other substance use, programme withdrawal rate). P-values less than 0.05 were considered as statistically significant. To counteract alpha error cumulation p-values were adjusted using Bonferroni correction. In addition, multiple linear regression analysis tested possible determinants of FA. To further differentiate FA from binge eating, in a second step the collective was divided into two new subgroups (patients with exclusively FA without binge eating versus patients with exclusively binge eating without FA) and these were compared as described above. Tables and figures were created in the spreadsheet Microsoft Excel version 16.

### **Results**

#### **Food Addiction and Sex, Age and BMI**

Prevalence of FA in this sample of patients with morbid obesity was 25%. Table 1 shows the mean values of all analysed variables, including the differentiation in patients with and without FA, as well as the results of the inferential statistics. No significant differences were found for sex, age and BMI.

#### **Tab. 1**

#### **Food Addiction and FEV Eating Behaviours**

Regarding the FEV dimensions of eating behaviour significant differences between patients with and without FA were found for hunger with a large effect size ( $p < .001^{***}$ ,  $d = -0.99$ ). Disinhibition, cognitive restraint, rigid and flexible control showed no significant differences. A multiple linear regression analysis was conducted with the variables disinhibition, hunger, cognitive control, age and BMI. The  $R^2$  for the overall model was .318 (adjusted  $R^2 = .27$ ), indicative for a high goodness-of-fit according to Cohen (1988),  $F(5, 71) = 6.635$ ,  $p < .001$ . The variables disinhibition and hunger were able to predict the prevalence of FA statistically significantly (disinhibition: regression coefficient  $b = .034$ ,  $p = .006$ ; hunger:  $b = .055$ ,  $p < .001$ ). Age proved to be a trend level significant predictor ( $b = -.007$ ,  $p = .058$ ).

#### **Food Addiction and Other Eating Disorders**

Emotional eating score, binge eating score and night eating prevalence was significantly higher in patients with FA than in patients without FA (emotional eating score:  $p = .02^*$ ,  $d = -0.77$ ; binge eating score:  $p = .006^{**}$ ,  $d = -1.11$ ; night eating prevalence:  $p = .006^{**}$ ,  $d = 0.00$ ). These findings are demonstrated in figure 1. Figure 2 illustrates the overlap between FA and binge eating respectively night eating. No relevant differences were found when comparing the subgroups 'only FA without binge eating' and 'only binge eating without FA'. Only the extent of social support was significantly more pronounced in the group 'binge eating only' ( $p < .001^{**}$ ,  $d = -1.11$ ). The descriptive and analytical statistical key figures can be seen in Table S2 (supplementary material).

**Fig. 1**

**Fig. 2**

### **Food Addiction and Problematic Food Categories**

Patients with FA descriptively consumed more foods rich in both carbohydrates and fat than patients without FA, although not statistically significant and with a small effect size. There were no differences respective of foods containing carbohydrates or fat alone. The differences between the sub-samples with and without FA concerning all three food categories are illustrated in figure 3.

**Fig. 3**

### **Food Addiction and Further Tested Variables**

Stress level, social support, age at onset of obesity, BMI reduction during the weight loss programme, programme withdrawal rate and prevalence of other substance use did not differ significantly between the two sub-samples. No substances other than alcohol and smoking were reported.

## **Discussion**

### **Food Addiction and Sex, Age, BMI, Onset of Obesity and Substance Use Prevalence**

One quarter of the sample met the YFAS 2.0 criteria for FA. This is consistent with other studies involving patients with pronounced obesity [11, 19]. The high prevalence among this sample of subjects with morbid obesity in contrast to lower prevalences in samples of healthy subjects indicates a considerable relevance of FA for the phenotype of some cases of obesity, but not all. However, three quarter of the obesity cases in this sample remain not associated with FA. In conclusion, for the majority of obesity cases FA can apparently not serve as an aetiologic construct [36].

#### **Sex**

No statistically significant sex differences were observed between patients with and without FA, though descriptively, men had a higher prevalence than women. This is unexpected as not in line with most previous studies, which concordantly found women exhibiting a higher prevalence of FA than men [14, 17, 21, 22, 26] or no sex difference at all [4]. However, the majority of these studies were conducted with normal-weight populations. A single study of individuals with obesity was found, it showed a descriptively higher prevalence in men [37].

#### **Age**

According to previous studies on populations with both normal-weight and obesity, it would have been expected that the group with FA would show a lower age [5, 22, 37]. However, our study results do not support this hypothesis, as the descriptive differences were not significant and of small effect size.

#### **BMI**

The sample exhibited a mean BMI of  $45.2 \text{ kg/m}^2$ , corresponding to obesity class 3 (according WHO classification) [38]. No significant differences in BMI were observed between the sub-samples with

and without FA, endorsing previous studies conducted in populations with obesity [23, 39, 40]. The lack of difference can be explained by the selection of the sample: All patients were participants of a weight reduction programme and were accordingly affected by pronounced obesity.

#### **Substance Use**

The total prevalence of substance use (alcohol and/or nicotine) was 52%, without a significant difference between the subgroups with and without FA. It is important to note that the questionnaire data used is not suitable for diagnosing a substance use disorder (SUD) according to DSM-criteria. The prevalence of SUD according to DMS-criteria is significantly lower in comparable populations of patients with morbid obesity (0.6-1.7% current SUD, 1-36% lifetime SUD) [41].

#### **Food Addiction and FEV Eating Behaviours**

The current study observed significantly more distinct feelings of hunger, with a large effect size, among patients with FA in contrast to those without, supporting previous studies in however normal-weight populations [14, 21]. Moreover, hunger and disinhibition proved to be significant predictors for FA. In samples with obesity, previous studies found correspondingly higher levels of impulsivity [23, 40]. The other FEV dimensions 'cognitive restraint' and its sub-categories 'rigid control' and 'flexible control' showed no significant differences. On the one hand, this is at odds with another study on a sample with obesity, which found an association between binge eating and cognitive restraint [42]. On the other hand, previous studies on normal-weight populations have found the same lack of effect [21, 43]. High grades of disinhibition and hunger as decisive elements of impulsivity [44] are suggestive of dysfunctional behavioural regulation mechanisms. Patients with FA seem to be more susceptible to disruptions in their planned eating, whether from external stimuli or internal, such as perceived hunger. This might contribute essentially to the binge eating behaviour seen in FA. As previously stated, impulsivity is associated with binge eating disorder [45]. Previous studies have observed a relationship between low distress tolerance and both disinhibition and emotional eating [46]. In combination with the present findings this suggests that dysfunctional emotional coping mechanisms associated with low distress tolerance could be a mediator between FA and disinhibition respectively hunger.

#### **Food Addiction and Other Eating Disorders**

##### **Emotional Eating**

Patients with FA exhibited significantly higher emotional eating scores than patients without FA, with medium effect size, in line with previous studies on populations with obesity [27, 47]. These findings suggest that unpleasant feelings, such as fear, anger, sorrow, loneliness, sadness and helplessness, can trigger uncontrolled excessive intake of food, as a means to dampen unpleasant emotional states. Given that not all individuals use food to regulate their emotions, the prerequisite for such behaviour might be disturbed emotional coping mechanisms. A previous study observed a greater emotional reactivity and a stronger tendency toward 'self-soothing' with food in patients with FA, in contrast to patients without [28]. As disturbances in emotional coping can obviously contribute decisively to the development of addictive-like eating behaviour, FA appears to hold features of behavioural addiction.

##### **Binge Eating**

The study at hand revealed significantly higher scores for binge eating behaviour in patients with FA in contrast to those without FA, with a large effect, in accordance with many previous studies on patients with obesity [23, 27, 48, 49]. Consequently, the uncontrolled consumption of excessively high amounts of food in a short time might be a decisive characteristic of not only binge eating disorder but of FA as well. As previously stated, there is great 'overlap between the symptoms assessed by the YFAS 2.0 and the clinical features of BED' [50], leading to difficulties in the differentiation of those two eating (disorders) phenomena. This is also confirmed by our data: Significant differences between the subgroups with and without FA (emotional eating, night eating, hunger) are not confirmed in the comparison of FA and binge eating. Accordingly, these variables must not be interpreted as characteristics of FA alone but characterise binge eating as well. Despite this, binge eating and FA cannot be regarded the same as is shown by the wide range of the binge



eating score in the sub-sample with FA. There were as well some patients fulfilling the FA criteria while exhibiting very low binge eating scores. This is in line with previous research concluding that BED and FA might be considered as distinct clinical phenomena [15]. Regarding the lack of effect when comparing FA and binge eating, the relatively small sample size and composition of the collective in this study should also be considered.

Significantly higher scores of binge eating behaviour in patients with FA lead to the hypothesis that FA might be characterised by eating to excess in a short period of time rather than perpetual food intake, so called grazing [15]. The high levels of disinhibition and hunger as decisive elements of impulsivity [44] may contribute significantly to the development of binge eating episodes. It would be of crucial interest to differentiate the manner of binges in FA and binge eating disorder [14]. This merits scrutiny in future research.

### **Night Eating**

The sub-sample with FA exhibited a significantly higher prevalence of night eating than the sub-sample without FA, supporting previous studies on populations with obesity [47, 48], albeit with a small effect. Consequently, it can be postulated that FA might be associated with difficulties in the maintenance of a circadian eating rhythm.

### **Food Addiction and Problematic Food Categories**

As previous studies, however in normal-weight populations, suggested [17, 51], patients with FA were assumed to have significantly more problems with foods containing both carbohydrates and fat than patients without FA. However, this effect was only observed descriptively, without statistical significance and with small effect size. The lack of a relevant difference in our study could be related to the small size of our sample and its composition. The aforementioned association of addictive eating behaviour and energy-dense food, as e.g. the combination of fat and carbohydrates, remains to be clarified in further studies. Foods containing only carbohydrates or fat showed no relevant differences. These findings are consistent with studies (on normal-weight individuals), which stated no association between FA and foods containing only carbohydrates [20].

### **Limitations**

Generalisability of the findings is limited due to the small number of subjects in relation to the large number of variables tested. The results should be reviewed in future studies.

Concerning binge eating this study is limited as the available data does not allow a full diagnosis of a clinically approved disorder according to DSM-criteria, but merely indicate binge eating behaviour. The results are also limited with regard to emotional eating, as no validated construct was available to capture emotional eating. Instead, five questions from two questionnaires were combined into the emotional eating score, so that the two questionnaires may be confounded. The reliability of all three sum scores (binge eating, emotional eating and night eating) could not be tested using Cronbach's alpha or did not yield a satisfactory result, which is why the significance of the results of is limited. Future research should verify the results with validated tools.

Another limitation of this study is the self-report of the examined data on problematic foods and the consequent difficulty to classify the listed food items of the questionnaire's free text field. For this reason, the distinction between the food categories might be inaccurate, which could be relevant regarding the lack of significance of our results and should be further investigated in future studies. Moreover, it would be of interest to examine in which context the problematic foods are consumed (in the context of a meal, a snack, a binge eating episode), which was not captured by the present study.

### **Conclusion**

The questionnaire-based trait FA can be considered as a sub-phenotype of obesity, occurring in approximately one quarter of obesity cases. Dysfunctional emotional coping mechanisms associated with low distress tolerance were found to be significantly related to FA and should be suggested as

therapeutic targets. Behavioural interventions should include a bio-psycho-social model [52] and address social and cultural aspects that may promote pathological eating behaviour (media, beauty ideals, affiliation, exclusion).

In addition, binge eating episodes were found to be characteristic for FA. The results point to the already stated overlap between FA and binge eating disorder. However, the existence of FA as a distinct (entity) phenomenon should not be excluded, as not all patients with FA exhibit binges. Further research needs to work out whether FA needs to be seen as a separate disorder or as a symptom/variant of existing eating disorders. The same applies to the question of a substance-related or behavioural component of FA.

#### **Statement of Ethics**

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1964 and its later amendments. This study protocol was reviewed and approved by the Ethics Commission of the University Medical Centre Göttingen, approval number 6/22/18. Written informed consent was obtained from participants prior to the study.

#### **Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

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#### **Author Contributions**

Pia Schankweiler, Thomas Ellrott and Carolin Hauck/Cirkel designed the study. Pia Schankweiler played the leading role in analysing the data. Pia Schankweiler, Thomas Ellrott, Carolin Hauck/Cirkel and Dirk Raddatz played an important role in interpreting the results. Pia Schankweiler drafted the manuscript. All authors revised and approved the final version.

#### **Data Availability Statement**

All data generated or analysed during this study are included in this article and its supplementary material files. Further enquiries can be directed to the corresponding author.

## References

### Literature Cited

1. Baxter J, Armijo PR, Flores L, Krause C, Samreen S, Tanner T. Updates on Monogenic Obesity in a Multifactorial Disease. *Obes Surg* 2019.
2. Kim TJ, Makowski AC, dem Kneesebeck O von. Obesity stigma in Germany and the United States - Results of population surveys. *PLoS One* 2019; 14(8):e0221214.
3. Long CG, Blundell JE, Finlayson G. A Systematic Review of the Application And Correlates of YFAS-Diagnosed 'Food Addiction' in Humans: Are Eating-Related 'Addictions' a Cause for Concern or Empty Concepts? *Obes Facts* 2015; 8(6):386–401.
4. Schulte EM, Avena NM, Gearhardt AN. Which foods may be addictive? The roles of processing, fat content, and glycemic load. *PLoS One* 2015; 10(2):e0117959.
5. Meule A. Back by Popular Demand: A Narrative Review on the History of Food Addiction Research. *Yale J Biol Med* 2015; 88(3):295–302.
6. Fernandez-Aranda F, Karwautz A, Treasure J. Food addiction: A transdiagnostic construct of increasing interest. *Eur Eat Disord Rev* 2018; 26(6):536–40.
7. Ding Y, Ji G, Li G, Zhang W, Hu Y, Liu L et al. Altered Interactions Among Resting-State Networks in Individuals with Obesity. *Obesity (Silver Spring)* 2020; 28(3):601–8.
8. Cassin SE, Buchman DZ, Leung SE, Kantarovich K, Hawa A, Carter A et al. Ethical, Stigma, and Policy Implications of Food Addiction: A Scoping Review. *Nutrients* 2019; 11(4).
9. Frank J, Gupta A, Osadchiv V, Mayer EA. Brain-Gut-Microbiome Interactions and Intermittent Fasting in Obesity. *Nutrients* 2021; 13(2).
10. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. 5th ed. Washington D.C.: American Psychiatric Association; 2013.
11. Constant A, Moirand R, Thibault R, Val-Laillet D. Meeting of Minds around Food Addiction: Insights from Addiction Medicine, Nutrition, Psychology, and Neurosciences. *Nutrients* 2020; 12(11).
12. Gearhardt AN, Corbin WR, Brownell KD. Development of the Yale Food Addiction Scale Version 2.0. *Psychol Addict Behav* 2016; 30(1):113–21.
13. Romero X, Agüera Z, Granero R, Sánchez I, Riesco N, Jiménez-Murcia S et al. Is food addiction a predictor of treatment outcome among patients with eating disorder? *Eur Eat Disord Rev* 2019; 27(6):700–11.
14. Hauck C, Cook B, Ellrott T. Food addiction, eating addiction and eating disorders. *Proc Nutr Soc* 2020; 79(1):103–12.
15. Davis C. A commentary on the associations among 'food addiction', binge eating disorder, and obesity: Overlapping conditions with idiosyncratic clinical features. *Appetite* 2016; 115:3–8.
16. Lindgren E, Gray K, Miller G, Tyler R, Wiers CE, Volkow ND et al. Food addiction: A common neurobiological mechanism with drug abuse. *Front Biosci (Landmark Ed)* 2018; 23:811–36.
17. Ayaz A, Nergiz-Unal R, Dedebayraktar D, Akyol A, Pekcan AG, Besler HT et al. How does food addiction influence dietary intake profile? *PLoS One* 2018; 13(4):e0195541.
18. Gearhardt AN, Hebebrand J. The concept of "food addiction" helps inform the understanding of overeating and obesity: YES. *Am J Clin Nutr* 2021; 113(2):263–7.
19. Hauck C, Weiß A, Ellrott T. „Food Addiction“, gezügeltes Essverhalten, mentaler Gesundheitsstatus und Heißhungersymptome bei morbidem Adipositas. *Adipositas - Ursachen, Folgeerkrankungen, Therapie* 2016; 10(04):215–20.
20. Lemeshow AR, Rimm EB, Hasin DS, Gearhardt AN, Flint AJ, Field AE et al. Food and beverage consumption and food addiction among women in the Nurses' Health Studies. *Appetite* 2018; 121:186–97.
21. Albayrak O, Focker M, Kliewer J, Esber S, Peters T, Zwaan M de et al. Eating-related Psychopathology and Food Addiction in Adolescent Psychiatric Inpatients. *Eur Eat Disord Rev* 2017; 25(3):214–20.
22. Hauck C, Weiss A, Schulte EM, Meule A, Ellrott T. Prevalence of 'Food Addiction' as Measured with the Yale Food Addiction Scale 2.0 in a Representative German Sample and Its Association with Sex, Age and Weight Categories. *Obes Facts* 2017; 10(1):12–24.
23. Meule A, Müller A, Gearhardt AN, Blechert J. German version of the Yale Food Addiction Scale 2.0: Prevalence and correlates of 'food addiction' in students and obese individuals. *Appetite* 2017; 115:54–61.

24. Penzenstadler L, Soares C, Karila L, Khazaal Y. Systematic Review of Food Addiction as Measured with the Yale Food Addiction Scale: Implications for the Food Addiction Construct. *Curr Neuropharmacol* 2019; 17(6):526–38.
25. Horsager C, Færk E, Lauritsen MB, Østergaard SD. Validation of the Yale Food Addiction Scale 2.0 and estimation of the population prevalence of food addiction. *Clin Nutr* 2020; 39(9):2917–28.
26. Brunault P, Courtois R, Gearhardt AN, Gaillard P, Journiac K, Cathelain S et al. Validation of the French Version of the DSM-5 Yale Food Addiction Scale in a Nonclinical Sample. *Can J Psychiatry* 2017; 62(3):199–210.
27. Burmeister JM, Hinman N, Koball A, Hoffmann DA, Carels RA. Food addiction in adults seeking weight loss treatment. Implications for psychosocial health and weight loss. *Appetite* 2013; 60(1):103–10.
28. 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(3):711–7.
29. Nolan LJ, Geliebter A. Validation of the Night Eating Diagnostic Questionnaire (NEDQ) and its relationship with depression, sleep quality, "food addiction", and body mass index. *Appetite* 2017; 111:86–95.
30. Lin Y-S, Tung Y-T, Yen Y-C, Chien Y-W. Food Addiction Mediates the Relationship between Perceived Stress and Body Mass Index in Taiwan Young Adults. *Nutrients* 2020; 12(7).
31. Pudel V WJ. Fragebogen zum Essverhalten (FEV): Handanweisung; 1989.
32. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J Psychosom Res* 1985; 29(1):71–83.
33. Wadden TA, Foster GD. Weight and Lifestyle Inventory (WALI). *Obesity (Silver Spring)* 2006; 14 Suppl 2:99S-118S.
34. Westenhoefer J, Stunkard AJ, Pudel V. Validation of the flexible and rigid control dimensions of dietary restraint. *Int J Eat Disord* 1999; 26(1):53–64.
35. IBM SPSS Statistics for Windows. Version Version 25.0., 26.0. Armonk, NY; Released 2017.
36. Chao AM, Shaw JA, Pearl RL, Alamuddin N, Hopkins CM, Bakizada ZM et al. Prevalence and psychosocial correlates of food addiction in persons with obesity seeking weight reduction. *Compr Psychiatry* 2017; 73:97–104.
37. Guerrero Pérez F, Sánchez-González J, Sánchez I, Jiménez-Murcia S, Granero R, Simó-Servat A et al. Food addiction and preoperative weight loss achievement in patients seeking bariatric surgery. *Eur Eat Disord Rev* 2018; 26(6):645–56.
38. World Health Organisation. Global Strategy on Diet, Physical Activity and Health; 2019. Available from: URL: [https://www.who.int/dietphysicalactivity/childhood\\_what/en/](https://www.who.int/dietphysicalactivity/childhood_what/en/).
39. Eichen DM, Lent MR, Goldbacher E, Foster GD. Exploration of "food addiction" in overweight and obese treatment-seeking adults. *Appetite* 2013; 67:22–4.
40. Meule A, Hermann T, Kübler A. Food addiction in overweight and obese adolescents seeking weight-loss treatment. *Eur Eat Disord Rev* 2015; 23(3):193–8.
41. Mitchell JE, Selzer F, Kalarchian MA, Devlin MJ, Strain GW, Elder KA et al. Psychopathology before surgery in the longitudinal assessment of bariatric surgery-3 (LABS-3) psychosocial study. *Surg Obes Relat Dis* 2012; 8(5):533–41.
42. Brytek-Matera A, Obeid S, Akel M, Hallit S. How Does Food Addiction Relate to Obesity? Patterns of Psychological Distress, Eating Behaviors and Physical Activity in a Sample of Lebanese Adults: The MATEO Study. *Int J Environ Res Public Health* 2021; 18(20).
43. Gearhardt AN, White MA, Potenza MN. Binge eating disorder and food addiction. *Curr Drug Abuse Rev* 2011; 4(3):201–7.
44. Lyke JA, Spinella M. Associations among aspects of impulsivity and eating factors in a nonclinical sample. *Int J Eat Disord* 2004; 36(2):229–33.
45. Leehr EJ, Schag K, Bruckmann C, Plewnia C, Zipfel S, Nieratschker V et al. A Putative Association of COMT Val(108/158)Met with Impulsivity in Binge Eating Disorder. *Eur Eat Disord Rev* 2016; 24(2):169–73.
46. Kozak AT, Davis J, Brown R, Grabowski M. Are overeating and food addiction related to distress tolerance? An examination of residents with obesity from a U.S. metropolitan area. *Obes Res Clin Pract* 2017; 11(3):287–98.
47. Masheb RM, Ruser CB, Min KM, Bullock AJ, Dorflinger LM. Does food addiction contribute to excess weight among clinic patients seeking weight reduction? Examination of the Modified Yale Food Addiction Survey. *Compr Psychiatry* 2018; 84:1–6.

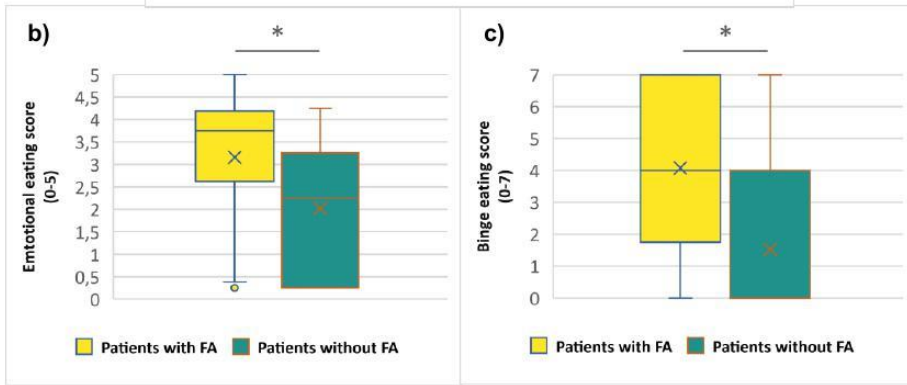
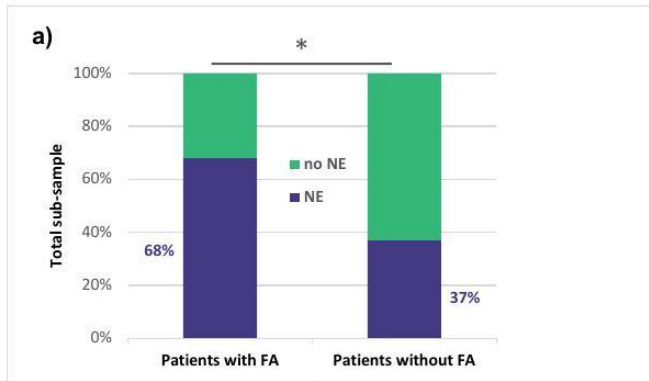
48. Koball AM, Clark MM, Collazo-Clavell M, Kellogg T, Ames G, Ebbert J et al. The relationship among food addiction, negative mood, and eating-disordered behaviors in patients seeking to have bariatric surgery. *Surg Obes Relat Dis* 2016; 12(1):165–70.
49. Ceccarini M, Manzoni GM, Castelnovo G, Molinari E. An Evaluation of the Italian Version of the Yale Food Addiction Scale in Obese Adult Inpatients Engaged in a 1-Month-Weight-Loss Treatment. *J Med Food* 2015; 18(11):1281–7.
50. Carter JC, van Wijk M, Rowsell M. Symptoms of 'food addiction' in binge eating disorder using the Yale Food Addiction Scale version 2.0. *Appetite* 2019; 133:362–9.
51. Pedram P, Sun G. Hormonal and dietary characteristics in obese human subjects with and without food addiction. *Nutrients* 2014; 7(1):223–38.
52. Engel GL. The need for a new medical model: a challenge for biomedicine. *Science* 1977; 196(4286):129–36.

### **Figure Legend**

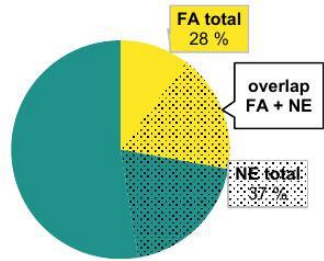
Fig 1. *Comparison of Patients With and Without Food Addiction (FA) With Regard to a) Night Eating, b) Emotional Eating and c) Binge Eating*

Fig 2. *Overlap of Food Addiction (FA) and Night Eating (NE) Respectively Binge Eating Behaviour (BE) in a Patient Population with Morbid Obesity*

Fig 3. *Comparison of Patients With and Without Food Addiction (FA) With Regard to Problematic Food Categories*

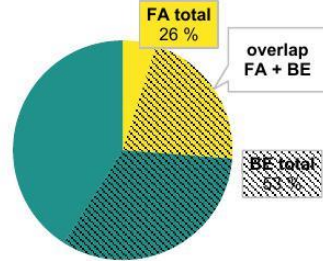


a)

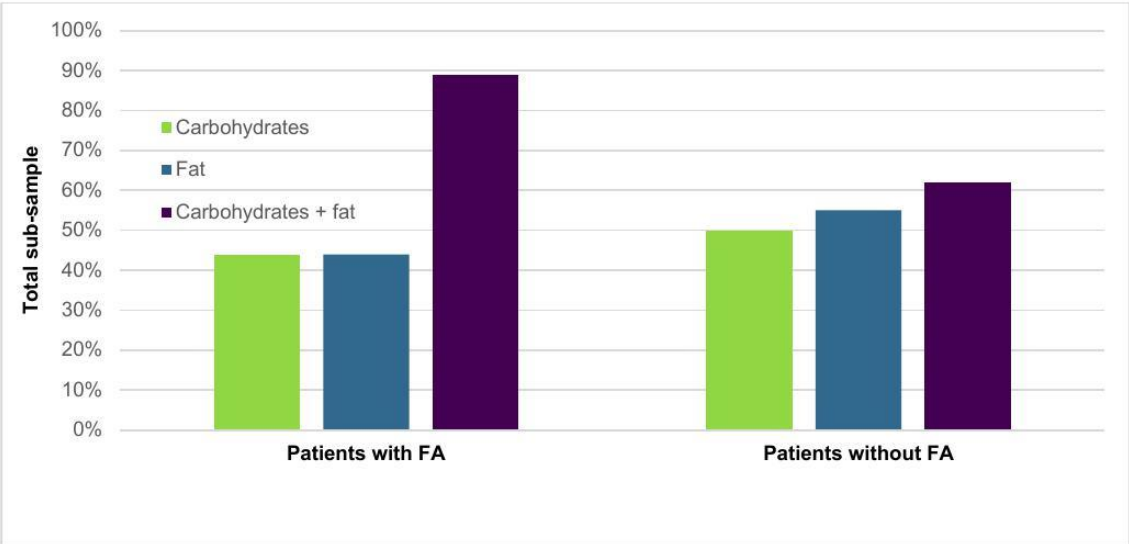


■ only FA (Food Addiction) ■ overlap FA + NE  
■ only NE (Night Eating) ■ neither FA nor NE

b)



■ only FA (Food Addiction) ■ overlap FA + BE  
■ only BE (Binge Eating) ■ neither FA nor BE





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**Tab. 1**  
*Variables Analysed of the Total Sample and in Comparison of the Sub-samples With and Without Food Addiction (FA)*

		<b>Total</b>	<b>Patients with FA</b>	<b>Patients without FA</b>	
<b>Metric variables</b> (scale/unit)	<b>n</b>	<b>M (SD; Min - Max)</b>	<b>M (SD; Min - Max)</b>	<b>M (SD; Min - Max)</b>	<b>Results t-test</b>
<b>Age</b> (in years)	n = 108	45.1 (13.83; 19 - 74)	40.6 (12.45; 23 - 74)	46.7 (14.00; 19 - 74)	t (49.67) = 2.17, d = 0.45
<b>BMI</b> (kg/m <sup>2</sup> )	n = 108	45.2 (7.60; 30.9 - 68.2)	46.6 (6.37; 36.4 - 61.0)	44.7 (7.91; 30.9 - 68.2)	t (48.45) = -1.23, d = 0.25
<b>Emotional eating score</b> (0 - 5)	n = 101	2.3 (1.54); 0.25 - 5)	3.2 (1.46; 0.25 - 5)	2.0 (1.47; 0.25 - 4.25)	t (41.24) = -3.37, d = -0.77 *
<b>Binge eating score</b> (0 - 7)	n = 104	2.1 (2.54; 0 - 7)	4.1 (2.65; 0 - 7)	1.5 (2.17; 0 - 7)	t (36.84) = -4.43, d = -1.11 **
<b>Dimensions of FEV</b>					
Disinhibition (0 - 16)	n = 97	8.3 (3.56; 1 - 16)	9.9 (3.90; 1 - 15)	7.8 (3.35; 2 - 16)	t (43.86) = -2.41, d = -0.61
Hunger (0 - 14)	n = 98	7.8 (3.48; 0 - 14)	10.2 (2.30; 6 - 14)	7.0 (3.46; 0 - 14)	t (62.82) = -5.15, d = -0.99 ***
Cognitive restraint (0 - 21)	n = 83	6.9 (3.58; 0 - 13)	7.0 (3.29; 2 - 13)	6.8 (3.7; 0 - 13)	t (44.79) = -0.20, d = -0.05
Rigid control (0 - 16)	n = 89	6.6 (2.33; 1 - 11)	6.9 (2.39; 1 - 11)	6.5 (2.31; 1 - 11)	t (87.00) = -0.84, d = -0.20
Flexible control (0 - 12)	n = 96	2.2 (2.27; 0 - 10)	1.8 (1.88; 0 - 6)	2.4 (2.39; 0 - 10)	t (53.21) = 1.35, d = 0.28
<b>Age at onset of obesity</b> (years)	n = 84	26.6 (14.15; 3 - 70)	20.4 (11.75; 3 - 54)	28.9 (14.36; 6 - 70)	t (48.16) = 2.78, d = 0.62
<b>Stress level</b> (1 - 3)	n = 99	2.3 (0.61; 1 - 3)	2.4 (0.57; 1 - 3)	2.3 (0.63; 1 - 3)	t (45.51) = -0.56, d = -0.12
<b>Social support</b> (0 - 6)	n = 73	4.3 (1.53; 2 - 6)	4.6 (1.50; 3 - 6)	4.2 (1.54; 2 - 6)	t (24.55) = 0.93, d = -0.26
<b>BMI reduction</b> (kg/m <sup>2</sup> )	n = 90	9.5 (5.19; -0.5 - 27.0)	9.8 (5.00; 0.6 - 18.9)	9.4 (5.28; -0.5 - 27.0)	t (32.18) = 0.29, d = -0.07
<b>Categorical variables</b>					
	<b>n</b>	<b>number of patients (percentage of total)</b>	<b>number of patients (percentage of probands with FA)</b>	<b>number of patients (percentage of probands without FA)</b>	<b>Results x<sup>2</sup>-Test</b>
<b>Number of subjects</b>		112 (101 %) <sup>1</sup>	27 (25 %)	83 (76 %)	
<b>Sex</b> female	n = 108	62 (57 %)	13 (21 %)	49 (79 %)	x <sup>2</sup> (1, N = 108) = 1.26, φ = 0.26
male	n = 108	46 (43 %)	14 (30 %)	32 (70 %)	
<b>Night eating prevalence</b>	n = 95	96 (38 %)	36 (68 %)	60 (37 %)	x <sup>2</sup> (1, N = 95) = 13.07, φ = 0.00 **
<b>Problems with foods</b>					
Carbohydrates	n = 60	29 (48 %)	8 (44 %)	21 (50 %)	x <sup>2</sup> (1, N = 95) = 00.16, φ = 0.69
Fat	n = 60	31 (52 %)	8 (44 %)	23 (55 %)	x <sup>2</sup> (1, N = 60) = 00.54, φ = 0.46
Carbohydrates + fat	n = 60	42 (70 %)	16 (89 %)	26 (62 %)	x <sup>2</sup> (1, N = 60) = 04.37, φ = 0.04
<b>Prevalence of substance use</b>	n = 99	51 (52%)	10 (43 %)	41 (54 %)	x <sup>2</sup> (1, N = 99) = 00.78, φ = 0.38
<b>Withdrawal rate</b>	n = 109	19 (17 %)	6 (23 %)	13 (16 %)	x <sup>2</sup> (1, N = 109) = 00.76, φ = 0.39

Note 1: <sup>1</sup> rounding error

Note 2: \*p<0.05; \*\*p<0.01; \*\*\*p<0.001