Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity
https://doi.org/10.1007/s40519-020-01021-3

REVIEW

Food addiction and psychiatric comorbidities: a review of current evidence

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Received: 12 June 2020 / Accepted: 12 September 2020
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Abstract

Background Food addiction (FA) is characterised by the consumption of appetible foods and by addictive psychological and behavioural symptoms such as cravings, tolerance, limited control of substance intake and withdrawal symptoms. Despite previous research on FA has been hindered by the lack of a formal definition for this condition, recent global trends have stirred the interest of the scientific community towards a proper classification and construct of FA. More specifically, recent studies have pointed towards shared defective neurobiological mechanisms as well as frequent comorbidities between FA, eating disorders, mood disorders, anxiety disorders and substance-related and addictive disorders.

Objective In this review, we will provide an overview of the complex symptomatology of food addiction evaluating its relationship with mood disorders, anxiety disorders, eating disorders and substance-related and addictive disorders.

Methods We wrote a systematic review and followed a PRISMA methods.

Results Patients with FA and substance use disorders show similar risk factors, neurobiological and hormonal correlates, personality traits and symptom profiles. The presence of FA appears to be directly proportional to the burden of symptoms of affective disorder. The comorbidity between FA and other eating disorders is associated with worse clinical conditions and symptoms.

Conclusion FA should be considered a sort of transnosological construct existing in different psychopathological domains that have similarities with substance-related, affective, and eating disorders. Furthermore, FA seems to be likely an important factor related to several psychopathological dimensions, but further studies are needed to clarify this view.

Level of evidence Level V, review article.

Keywords Food addiction · Mood disorders · Psychiatric comorbidities · Eating disorders · Substance use disorders · Psychopathological dimension

Introduction of food addiction

The conceptualization that foods, particularly highly palatable ones, may be the subject of addiction has been intensely debated since 1956, when the concept of food addiction (FA) was first proposed as "a common pattern of symptoms descriptively similar to those of other addictive processes" [1]. Recent global trends, such as the increase

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This article is part of the Topical Collection on Food and Addiction.

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Published online: 23 September 2020

in prevalence of obesity, the changing food environment, and the doubling of mental health conditions, have stirred the interest of the scientific community towards a proper classification and construct of FA. Most importantly, evidence of shared defective neurobiological mechanisms, as well as frequent comorbidities between FA, eating disorders (ED), mood disorders and substance-related and addictive disorders, could point not only to a redefinition of FA as a proper mental disorder, but also to a revision of the dichotomy between "substance-based" and behavioural addictions [2]. FA is characterised by addictive psychological and behavioural symptoms similar to those displayed in typical substance-related disorders. These include the urge to consume a said substance of abuse and relative tolerance and "withdrawal-like" physiological effects if food intake is ceased or reduced [3, 4].

Research on FA is hindered by the lack of a formal definition for this condition, as distinct from other behavioural addictions or ED [5].

FA is assessed by Yale Food Addiction Scale (YFAS) questionnaire, a 25-item self-report assessing addictive eating behaviours over the last 12 months [6]. The YFAS was first developed by Gearhardt et al. [7] basing it on the Diagnostic Statistical Manual of Mental Disorders Text Revision (DSM-IV-TR) diagnosis framework for opioid abuse as well as self-reports assessing interpersonal addictions. With the publication of the DSM-5 in 2013, the YFAS tool was revised (YFAS 2.0) to reflect the change in addiction classification, substance abuse and substance dependence [6]. YFAS 2.0 assesses seven symptoms of addiction as applied to food: decreased control over intake, persistent desire or frequent ineffective attempts to stop, withdrawal, resistance, consumption of large quantities of food or for a prolonged period of time, spending a lot of time obtaining food, giving up to significant activities and clinically relevant disability. A categorical diagnostic cut-off is met when three symptoms, together with a clinically significant impairment or distress linked to eating, are present. In 2014, a short version of YFAS known as mYFAS was developed with only nine items [8], while the Yale Food Addiction Scale for Children (YFAS-C) developed to assess the role that food addiction plays in children [9]. The YFAS represents the only available tool to measure addictive feeding behaviour and has been translated and validated in several European countries including Italy.

As 7 years have passed since the publication of the DSM-5, we believe that a revision of behavioural addictions, namely FA, is needed. The purpose of the current review is to provide a novel input for research and debate on whether FA has the properties of a clinical disorder with specific neurobiological correlates. This paper offers a valid background overview of the recent literature on FA and its correlation with eating, affective and substance-related and addictive disorders where we outline both psychometric and neurobiological evidence.

Methods

Data collection, review, reporting, and discussion were conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) Statement [10, 11].

The literature search was carried out in PubMed and PsychINFO databases using varying combinations of keywords: "food addiction" AND (mood disorders OR addictive OR anxiety OR depression OR bipolar OR eating disorder OR bulimia nervosa OR anorexia nervosa OR binge eating disorder).

Inclusion criteria for our research included: articles published in English from January 2018 onwards; studies carried out in clinical sample of patients of age 13 years old onwards; reliable diagnosis of psychiatric disorders according to structured interviews and standardized criteria. Among the inclusion criteria, particular attention was given to the use of any YFAS scale for the assessment of FA including YFAS, YFAS 2.0 and m-YFAS scale in each language (Fig. 1).

Finally, because not all overweight/obese individuals or individuals with ED report addiction-like symptoms related to food, studies which describe food addiction exclusively by diagnosis of weight, BMI or ED were excluded [5].

No articles met the search criteria in PsychINFO.

Articles were included if their stated purpose were to test food addiction. We included studies with healthy participants or affected by psychiatric disorders or obesity.

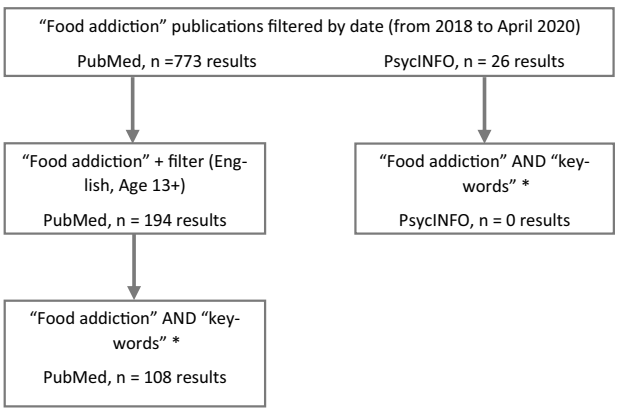

Fig. 1 Research flow diagram provided in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. *"food addiction" AND (mood disorders OR addictive OR anxiety OR depression OR bipolar OR eating disorders OR bulimia nervosa OR anorexia nervosa OR binge eating disorder). To see the articles included and excluded view the appendix

Articles selected from PubMed and PsychINFO were reviewed first by title, then abstract, and finally full article for relevancy and eligibility using the inclusion criteria described above. A flowchart for study inclusion is depicted in Fig. 1. The search was last updated in April 2020.

The data extraction form used for this study was modelled after forms used in similar systematic reviews [12].

Food addiction and addictive disorders

Substance dependence is broadly described as a chronically relapsing condition characterised by (1) desire to seek and consume the drug; (2) lack of control in reducing drug intake; and (3) the appearance of negative emotional state (e.g., dysphoria, anxiety and irritability) indicating a motivational withdrawal syndrome when exposure to the substance is stopped. Although food is not regarded as a substance of abuse, the diagnosis and construct of FA were first developed from the similarities observed between substance dependence and excessive overeating [13].

On a psychological level, individuals diagnosed with FA have been reported to display personality traits correlated with well-recognized addictive disorders such as higher alexithymia, higher neuroticism, lower conscientiousness, and extraversion than patients without FA [14]. Most importantly, FA is associated with increased impulsivity [4], similarly to individuals affected with substance-use disorders (SUDs). Similarly, women with SUD or FA display comparable post-traumatic stress disorder (PTSD) symptoms, depression profiles and risk factors, with the exception of trauma experiences [15]. The lack of control over one's emotions correlates with impaired decision-making which is further strongly correlated with the development of addiction [16].

Behaviourally, one hallmark of FA is the continual consumption of food throughout the day in the form of grazing, a behaviour commonly defined as the repetitive consumption of small amounts of food over an extended period of time and, importantly, the inability to restrain from repeated snacking despite the intention to stop [17]. The intense desire to consume food appears to be driven by the natural reward of highly palatable foods rich in calories, sugar and salt. Together, grazing and reward-based eating echo the hallmarks of common substance use disorders. A similar phenomenon can be observed for craving and withdrawal, two common behaviours displayed in substance abuse and FA [18]. Withdrawal symptoms are evaluated through the highly processed food withdrawal scale (ProWS) developed by Schulte et al. The ProWS demonstrated convergent validity with addictive-like eating, BMI, and weight cycling and discriminant validity with dietary restraint [19].

According to Ivezaj et al., potential treatments for FA could reflect an addiction framework involving harm reduction or abstinence-based models versus traditional cognitive behavioural therapy used to treat disordered eating. Moreover, a positive effect of physical activity has been observed as a possible compensatory strategy for addictive overeating. Low physical activity levels could in fact lead to increased food cravings and disinhibition, which may contribute towards the addictive process. Similarly, physical activity may have the potential to serve as a treatment for conventional SUDs (e.g., alcohol, nicotine, drugs) by potentially reducing cravings, withdrawal symptoms, anxiety and depression, and increasing abstinence [20]. A further technique to soothe food craving is a hypocaloric diet restriction, as observed by Chao et al. [21]. Restricting food intake causes recurrent thoughts and preoccupations about eating in a similar manner to substance use.

The similarities between FA and common SUDs have been also highlighted in shared neurobiological mechanisms. Food-addicted individuals demonstrate greatest cravings for carbohydrates and fats [17, 22]. These foods are notoriously known for their involvement with the reward circuitry of the dopaminergic system, equally activated by substances of abuse. More specifically, a study conducted by Contreras-Rogriduez et al. (2018) revealed that higher FA symptoms are associated with significant differences in the connectivity of the ventral caudate-hippocampus between fasted and fed conditions [17]. This brain region is responsible for the coding of reward-related food memories, increasing reactivity to reward prediction signals and contributing to the detection of novel and aversive food signals. Therefore, the hyper-connectivity in the VC—hippocampal network may indicate an enhanced reactivity of food reward-related circuitry among people with FA characteristics when fasting [23]. A further brain network that plays a pivotal role in the promotion of motivational behavioural responses, and has been implicated in addiction disorders, involves the nucleus accumbens (NAcc), amygdala and the anterior insula (aINS) circuit. More specifically, a study conducted by Osadchiy et al. (2018) demonstrated a positive relationship between functional and anatomical connectivity of the amygdala–NAcc circuit and the consumption of hedonic foods via the brain–gut microbiome axis [24]. On the other hand, the study conducted by Beyer et al. (2019) found that FA symptoms do not account for the bulk of the differences in the structural brain changes. Still, FA symptoms may explain additional variation in the reward network's orbitofrontal cortex. Longitudinal studies conducting anatomical and functional MRIs may further disrupt the neural mechanisms of addictive eating behaviours [25].

FA has been shown to have a hormonal dimension. More specifically, ghrelin and leptin have been identified as significant players in the regulation of FA symptomatology [26,

27]. Leptin is an adipocyte-secreted hormone and produced at levels proportional to the amount of adipose tissue. Leptin signal's long-term energy storage and its release are modified by acute changes in caloric intake [28]. Ghrelin is a hormone and a key regulator of nutrient sensing, meal initiation and appetite. Ghrelin signalling has been increasingly recognized as a significant regulator of obesity, insulin resistance and diabetes [29]. Ghrelin interacts with the dopaminergic reward circuitry and increased levels of such hormone may lead to a net increase in food craving [26].

Leptin modulates the function of mesolimbic dopamine neurons and plays a role in anticipatory food behaviours. Leptin levels are strongly correlated with food-related addictive behaviours and are typically considered a hallmark of obesity. In the context of FA, leptin is hypothesized to have a possible neuroendocrine effect for addictive behaviour. According to this hypothesis, low levels of leptin would induce a feeling of hunger and a desire to feed. Peters et al. (2018) observed that FA and normal body weight adolescents show decreased serum leptin rates [27].

Deficiencies in decision-making ability and attention control have also been highlighted in individuals affected by FA. Interestingly, these impairments have often been identified in addictive and behavioural disorders. More specifically, individuals who met the requirements of YFAS were shown to commit more omissions on the continuous performance test (CPT) suggesting that an increased difficulty in focus attention is present [30].

Comorbidity with affective disorders

Food intake plays a pivotal role in the regulation of mood and emotional state.

In the context of FA, multiple studies have highlighted substantial diagnostic overlap or comorbidity between addictive-like food intake and mood and anxiety disorders [31] including depressive symptomatology, bipolar spectrum disorder, as well as exposure to early life psychological and sexual abuse and a lower overall quality of life [18, 32].

In obese patients, where FA is significantly expressed, a strong correlation exists between the condition and higher prevalence of many psychological disorders including present and past mood disorders [26, 34, 35], anxiety disorders and suicidality risk [36].

Similar results have been obtained for the adolescent population where, even in its early stages, higher levels of FA symptoms are directly proportional to a stronger tendency to experience negative affect, such as low self-esteem, depression and anxiety [27, 37, 38]

Higher levels of depression and anxiety have also been shown in individuals diagnosed with BED with at least moderate 'food addiction' symptoms [23, 26, 36].

Interestingly, these findings may indicate that the presence of FA can lead to a more disturbed BED presentation characterised by greater psychopathology distress [16, 40].

Irrational beliefs (IB) are significantly correlated with FA along with depression and trait anxiety. IB are habitual affect-eliciting thought patterns originating from a core process of perfectionism or absolutist thinking and are known to be a primary source of psychopathologies, including food disorders and addictive behaviour. However, while higher trait anxiety, depression and emotional eating are strongly predicted by IB, only emotional eating has been shown to regulate the impact of IB on FA [41].

Increased consumption of highly palatable foods and associated disordered eating habits may be adopted to decrease psychological discomfort or dampen physiological stress responses, thereby serving as a possible coping mechanism for stress or low mood [14, 42].

This hypothesis is reinforced by the positive correlations between, emotional eating and food addiction behaviours and mild to severe depressive symptoms [15] and higher BMI [13, 43].

The unadjusted association between negative affectivity and weight does not support the hypothesis that obesity is a necessary consequence of depression, but rather that FA acts as a mediator between psychological distress and BMI in both sexes. Moreover, the closer association between FA and depression than between FA and BMI indicates that depression is a contributing factor to the development of FA in teenagers and precedes obesity development [26, 44].

The heightened susceptibility to stress in adolescents with depression, might hinder the sensitivity of the brain reward system, which plays a pivotal role in the development of FA [45]

The psychological underpinnings of comfort eating in depression are also related to physiological pathways. Mills et al. [14] observed the role of satiety hormones ghrelin and leptin in relation to emotional eating and FA. More precisely, the absence of satiety behaviours in association with depressogenic increased eating could reflect a leptin resistance, which could consequently represent a potential therapeutic target for problematic eating and weight gain risk in individuals with major depressive disorder (MDD), particularly females. FA is positively correlated with urinary cortisol which has been previously correlated with greater hedonic and less conscientious eating in obese women [23, 27].

Food addiction and eating disorders

Feeding and ED are characterized by "a persistent disturbance of eating or eating related behaviour" causing significant impairment in physical health or psychosocial functioning, as stated in DSM-5 (DSM-5, APA, 2013). Binge eating

disorder (BED) is more often linked to FA. Several studies showed a higher prevalence of FA in BED patients versus participants without any history of EDs and in particular FA. Carter and colleagues analysed a sample in which 92% of BED participants met criteria for at least mild FA compared to the 6% of the control group. Moreover, in the same study, matching criteria for moderate/severe FA were linked to higher eating pathology and higher levels of general psychopathology [39]. More specifically, FA symptoms were found as predictors of a more severe clinical presentation of BED and the number of displayed YFAS symptoms predicted binge frequency. These findings could denote a global representation of the condition not only related to western culture considering that a survey conducted on Indian residents found similar results [23]. An analysis on a sample of 969 adolescents using YFAS and Binge eating scale (BES) confirmed precedent data, showing a link between binge eating behaviour and YFAS score and confirming the same FA prevalence in a younger cohort [37]. The relationship between these two conditions should be investigated in depth considering that some data showed FA symptoms score alone explained more unique variance in psychological distress and functioning impairment in individuals with BED symptomatology than other well-characterized BED features [16]. Data in veterans seeking weight loss confirmed this aspect. In that sample, FA accounted for 15% of the BMI variance contributing three times more than general eating pathology to weight gain [43].

Not all evidences highlighted overlapping features, considering hormonal patterns. Ghrelin levels were associated with FA positive score while BED was associated with nesfatin-1 [26]. On the other hand, important evidences regarding close similarities between eating disorders and FA came from obese patients seeking bariatric surgery. Higher emotional eating and higher external eating were associated with FA severity suggesting a role of these dimensions as preludes of FA itself [36]. 18% of a sample of patients who underwent sleeve gastrectomy and who met YFAS criteria and for loss of control eating (LOC) emerged as a more severe subgroup with elevated eating disorder psychopathology and problematic eating behaviours [18].

As underlined by the reported studies, dimensions and sub-threshold symptoms, not only full-blown conditions, could be of great importance in understanding the process from normal to pathological eating. Some findings revealed that binge eating and addictive tendencies towards food are not necessarily synonymous. Other aspects could account for overeating in FA like reward driven eating and compulsive grazing as assessed in a Canadian and Australian sample [4].

In this line, dissatisfaction of body image, an eating disorder inventory (EDI) subscale, showed a strong correlation with YFAS symptoms in Hungarian children and could be seen as a predictor of addictive-like behaviours that could lead to obesity [46].

FA could also act as a predictor of treatment outcome. Romero et colleagues (2019) suggested that FA was associated with poor outcome in BED patients receiving cognitive behavioural therapy [47].

Discussion

In this review, we investigated current literature relative to the correlations of FA with substance-related, addictive-like, affective and ED. The initial search yielded a total of 773 publications on FA using PubMed and 26 publications using PsychINFO. A total of 38 publications were identified, while the others were excluded as not meeting the pre-specified selection criteria of our review.

FA and substance use disorders

The term FA combines the concepts of "substance-based" and behavioural addiction, but the available evidence for a substance-based FA is poor, and systematic clinical and translational studies are still meagre. Some findings support FA as a construct consistent with a kind of substance use disorder diagnosis: certain foods, demonstrate the greatest addictive potential [12].

Moreover, FA and SUD seem to share similar psychopathological mechanisms: the chronic condition, the desire to seek and consume the food or the substance, the feeling of lack of control, and the subsequent negative emotional state indicating a motivational withdrawal syndrome when exposure to the substances is stopped. Patients with FA and SUD show similar risk factors, neurobiological and hormonal correlates, personality traits and symptom profiles [48].

FA and affective disorders

Several studies have highlighted the effects of the comorbidity between FA and mood or anxiety disorders, including depressive symptomatology and bipolar spectrum disorder. The presence of FA appears to be directly proportional to the burden of symptoms of affective disorder. In special populations (e.g. obese patients, adolescents), there is a strong correlation between FA and higher prevalence of mood disorders, anxiety disorders and suicidality risk.

FA and eating disorders

Based on a recent review, there is evidence that FA may represent a distinct phenomenon from established ED such as bulimia nervosa or binge eating disorder [49].

To evaluate the contribution of FA to ED, it becomes essential to have valid and reliable instruments to assess FA behaviours. The YFAS scale appears as a valid tool to identify individuals showing symptoms of "dependence" on certain foods. Studies using the YFAS reported that patients scoring high on this scale showed more frequent binge eating episodes [4]. It seems clear that the comorbidity between FA and other ED is associated with worse clinical conditions and symptoms [50].

Conclusions

To our knowledge, the concept of FA is still a controversial topic: although not formally recognized by the DSM-5, it has been well described in literature. Similarly, it is still a matter of debate whether FA is a categorical disorder [51] or a psychopathological dimension.

Although FA could be considered a peculiar pattern of eating behaviour that can be diagnosed using categorical criteria, it might be also interpreted as a comorbid dimension with anxiety or mood disorders. Nonetheless, FA appears to share neurobiological mechanisms with SUD. In particular, studies on animal model identified similar tolerance/withdrawal patterns in response to palatable food [12]. These patterns remain undetected in humans because of the numerous and overlapping neurobiological pathways of addiction that make their neurobiological analysis extremely challenging. However, the presence of signs and symptoms compatible with SUD suggest that these mechanisms could be present. Tolerance and withdrawal in response to food intake received poor interest; therefore, further studies on this topic are necessary, especially considering evidences already present on addictive effects of sugar.

Otherwise, FA could be considered a sort of transnosological construct existing in different psychopathological domains, overlapping with substance-related, affective and eating disorders. Anyway, FA seems to be play a pivotal role in several psychopathological dimensions.

What is already know on this subject?

Based on the current literature, available data about neurobiological similarities between FA, SUD, affective disorders, and ED are still scarce. Efforts to identify more specific diagnostic criteria are clearly needed. A better understanding of the relationship between FA and other psychiatric disorders, such as SUD, and mood or anxiety disorders, would allow a more appropriate treatment.

What does this study add?

The debate on the independent diagnosis of FA within psychiatric disorders remains open. FA could represent a regulatory mechanism in affective disorders, a kind of SUD, as well as a distinct pattern of ED. Understanding the relationship between FA and psychopathological dimensions, seems to be informative in identifying the mechanisms underlying the development, maintenance, prevention and treatment of psychiatric disorders that might underlie FA behaviours.

Acknowledgements Prof. Piccinni planned the paper. Drs. Bucchi and Drs. Fini selected and reviewed the available literature. All authors wrote the paper that was revised and approved for publication by Dr. Cargioli.

Compliance with ethical standards

Conflict of interest The authors have declared that no competing interest exists.

Ethical approval This article does not contain any studies with human participants or animal performed by any of the authors.

Informed consent For this type of study formal consent is not required.

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