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A narrative review of potential treatment strategies for food addiction

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Abstract

The concept of food addiction (FA) remains controversial with research being in the nascent stages; FA like any addiction can have a devastating impact on the lives of those afflicted. There exists a clinical need for treatment strategies for those affected. This article reviews potential treatment strategies for FA. The treatment strategies target four core behaviours of the addiction phenotype specifically craving through the opioid system, impulsivity as a personality trait, compulsivity through the serotonergic system and lastly motivation through the dopaminergic system. A range of pharmacological and psychological interventions are reviewed. Future research should seek to test and validate the proposed clinical treatment strategies.

Keywords

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A NARRATIVE REVIEW OF POTENTIAL TREATMENT STRATEGIES FOR FOOD ADDICTION

ABSTRACT

The concept of food addiction (FA) remains controversial with research being in the nascent stages, FA like any addiction can have a devastating impact upon the lives of those afflicted. There exists a clinical need for treatment strategies for those affected. This article reviews potential treatment strategies for FA. The treatment strategies target four core behaviours of the addiction phenotype specifically craving through the opioid system, impulsivity as a personality trait, compulsivity through the serotonergic system and lastly motivation through the dopaminergic system. A range of pharmacological and psychological interventions are reviewed. Future research should seek to test and validate the proposed clinical treatment strategies.

INTRODUCTION

The notion of food addiction (FA) or the potential addictive properties of certain foods for instance chocolate has long been considered in the general population and the popular media [1]. In the last decade the phenomenon of FA has received growing academic and empirical interest [2, 3]. This interest has seen an increased amount of research in the area along with the development and validation of a specific scale to measure the phenomenon based upon the substance dependence criteria in the DSM-IV [4]; the Yale Food Addiction Scale (YFAS) [5].

Although interest in the phenomenon of FA has increased the notion of FA remains controversial [6]. At the most fundamental level the concept of FA suffers from a lack of conceptual clarity with researchers failing to explicitly define the concept [7]. FA is commonly paralleled to that of drug addiction which is an addiction to an exogenous substance. As such if this is the case the implicated substance/s or in this case nutrients or food additives are yet to be determined [8].

However, many researchers broadly implicate highly processed foods which contain high combinations of sugar, salt and/or fat as being responsible for FA [8-10]. With the terms palatable and hyperpalatable food commonly being utilised to describe these foods [11].

While some suggest that sugar may be addictive in humans having found that rats under certain conditions display addictive-like eating behaviour towards sucrose [12, 13]. Others contend that there is no evidence that sugar is addictive in humans [14]. Alternatively Cocores and Gold [15] maintain that salt in food has an addictive potential as it produces a hedonic feeling of reward as it acts like an opiate agonist in the brain.

Moreover others assert that it is not hyperpalatable food *per se* that promotes addictive like eating, it is the schedule under which it is consumed; that is a restrictive and subsequent binge consumption paradigm that results in promoting addictive like eating behaviours [16–18]. Thus although the concept of FA is commonly described as being akin to drug addiction in practice it is treated by researchers as laying anywhere on a continuum between an exogenous and endogenous addiction. Furthermore it is unclear whether it is an addiction in the sense of drug addiction (exogenous), a behavioural addiction or even an eating disorder [19].

Recently Hebebrand and colleagues [19] concluded that although research pertaining to FA is still in the early stages there is enough empirical evidence in both animals and humans to support the idea that a phenomenon of addictive like eating exists. However they contend that the use of the term FA is misleading and the phenomenon would be better understood by the term eating addiction or even as an addictive eating disorder [19].

Thus the phenomenon in accordance with Hebebrand and colleagues [19] is more closely aligned to a behavioural addiction. However as eating is necessary for survival and as such fulfils a homeostatic drive this potentiates the opportunity for endogenous compounds to have a role in addictive like eating behaviour. Albayrak, Wolfe and Hebebrand [20] contend that endogenous chemicals may be the link between drug and behavioural addictions.

In addition to the shortcomings of the FA concept pertaining to the definition and aetiology outlined above, others have highlighted that the concept is impeded by its own measurement [11]. That is the construct of FA according to some has become synonymous with the measure that was developed to measure it, the YFAS which it is also based upon.

That is some ascertain the explanation for the existence of the FA concept is circular and research needs to move beyond the bounds of the YFAS [11].

Further the YFAS has been criticised as being a measure of eating behaviour as oppose to a measure of FA [19]. Moreover some have ascertained that the YFAS is not accounting for any additional variance above and beyond that captured by measures of binge eating behaviour [11]. Others such as Ziauddeen, Farooqi and Fletcher [6] have emphasised the inconsistencies between FA, overweight, obesity and binge eating also suggesting that binge eating may better account for the phenomenon. Highlighting the common misconception of treating overweight and obesity as a homogenous disorder when overweight and obesity are truly heterogeneous in nature [6]. Furthermore it has even been suggested that FA is a sub-type of binge eating disorder (BED) and that bulimia nervosa is an extreme form of BED [11].

Nonetheless interest in the phenomenon continues to grow; with interest further heightened since the inclusion of the first behavioural addiction; gambling disorder in the DSM-5 [21]. As traditionally the term addiction has been reserved to describe dependence upon certain drugs of abuse. That is the term addiction has historically been aligned with addiction to exogenous substances. This reconceptualization of addiction potentiates the opportunity for other addictions including behavioural addictions to be considered and included in future editions of the DSM. Thus FA could possibly be added to the DSM in the future.

In accordance with the changes related to addiction in the DSM-5 recently Gearhardt and colleagues [22] released an updated version of the YFAS; the YFAS 2.0. Although the authors reported that the scale seems to have sound psychometric properties the scale being newly

developed requires further comprehensive testing and validation before definitive conclusions can be drawn.

In parallel to these developments the overweight and obesity epidemic has continued to rise [23]. Recently Hendrie and colleagues' [24] calculated projected estimates of the average body mass index (BMI) by 2019 for both Australian males and females with the averages being 28.0kg/m^2 and 27.6kg/m^2 respectively. Further the prevalence of obesity is expected to rise 6 to 7 percentage points by 2019 with 28.7% of males and 29.2% of females anticipated to be obese [24].

Thus although the phenomenon of FA is controversial; research remains in the nascent phase and as such in the context of the worldwide growing obesity epidemic further work is required. Therefore this article seeks to review potential strategies for the treatment of FA based upon treating core addiction phenotypic behaviours in the context of addictive like eating.

This will allow the exploration of the concept of FA beyond the bounds dictated by the YFAS which is one of the issues with the phenomenon that was outlined above. It will also facilitate a greater understanding of the concept in relation to disorders associated with binge eating; thus allowing identification of potential differences and similarities between the concepts. Moreover and most fundamentally this interventionist paradigm offers utility for the treatment of addictive like eating behaviour which clearly exists regardless of the designation granted and as such has the potential to assist with combatting a portion of the overweight and obesity epidemic.

The Addiction Phenotype:

Addiction is a debilitating neuropsychiatric disorder that is commonly identified through behavioural symptoms [25]. The concept of addiction represents a synthesis of dependence and compulsion. Thus four key addiction behaviours commonly associated with the addiction phenotype have been selected as the focus for the development of clinical guidelines for the treatment of food addiction. These four core behaviours are *craving*, *impulsivity*, *compulsivity* and *motivation*.

The use of phenotypic behaviours associated with addiction as oppose to clinical criteria facilitates the understanding of the FA phenomenon beyond the bounds dictated by the YFAS as mentioned above. As well as overcoming a common issue associated with addiction criteria; the use of consequences as the basis of the criteria [26]. Below each of the four core addiction behaviours and their proposed treatment targets are discussed.

Behaviour: Craving

Craving is a hallmark feature of addiction with individuals' with drug addiction commonly reporting very strong cravings for the substance they are addicted too [27, 28]. The concept of craving has been prominent in various models of addiction specifically conditioning-based, cognitive, psychobiological and motivational models of addiction for decades [28]. The constant cravings can impede the individual's attempts to give up the substance leading to repeated failures and relapses [27, 28]. In regards to food; human weight cycling is indicative of repeated attempts and failures related to controlling weight and the intake of certain foods [27].

Food cravings have been found to be stronger in individuals with BED in comparison to individuals without BED and with similar BMIs [27]. Further research has also indicated

differences between obese individuals' with and without BED in regards to genotype, specifically; significant differences in frequencies of opiod and dopamine polymorphisms have been found between the two groups [29]. Clark [30] ascertains that both binge eating and obesity predispose individuals to have a strong bias towards immediate gratification; as such driving craving and consummatory seeking behaviours. In regards to food and overeating these existing biases are further heightened by the obesigenic environment [30].

The opioid system has direct involvement in both drug and FA [18]. The opioid system plays a central role in the rewarding properties of hedonically pleasing stimuli [18]. This reinforcement stems directly from the activation of the opioid system as well as the indirect activation of the mesolimbic dopamine pathway [18]. Thus the opioid and dopamine systems work collectively in reinforcing hedonically pleasing stimuli [18].

The opioid system is known to be directly involved in consummatory aspects of behaviour such as hedonic eating or the intake of drugs of abuse [31]. Recent research has pointed to the role of the opiate system in anticipatory aspects of ingestive behaviour as well. That is the opiate system appears to moderate the influence of food-related stimuli and the motivation for food seeking behaviour [31]. In the context of individuals with BED and / or obesity such individuals have been found to be more sensitive to environmental food associated cues thus driving the craving (anticipatory) and subsequent reward seeking behaviour (consummatory) [31].

Treatment Target: Opiate system

In order to assist individuals in dealing with the strong cravings associated with addiction a number of pharmaceutical treatments have been developed; these treatments target the opiod system[18, 31]. In relation to addictive like eating these treatments have been trialled with patients with BED and / or obesity.

Randomised trials have investigated the use of a number of anti-craving and anti-addiction medications in the treatment of BED [32]. The results have been mixed however the use of the opioid antagonist intranasal spray Naloxone has shown some promise with a significant reduction in the time spent binge eating for the group using the Naloxone spray in comparison to those using the placebo spray [32].

Recently Mason and colleagues [33] trialled the use of naltrexone; an anti-addiction medication commonly utilised in opiod and alcohol addiction with a group of obese women to negate cravings associated with addictive like eating. The results indicated that reported craving intensity did not differentiate between the naltrexone and placebo conditions. However there was some evidence that the opioidergic blockade decreased the association between the reward driven eating and daily food craving intensity [33].

In addition, as overcoming an addiction requires an individual to be able to cope with negative emotions and cravings; it has been suggested that treatments that target emotion regulation and distress tolerance are pivotal to the treatment of both drug addiction and BED [27]. Thus the psychological treatments of motivational interviewing and CBT are utilised to assist abstinence motivation, emotion regulation and the development of specific coping strategies [34]. These techniques can be utilised independently or in collaboration with a pharmaceutical treatment.

Behaviour: Impulsivity

Impulsivity is a personality trait that is associated with multiple psychiatric disorders [35]. Impulsivity is defined as "acting on the spur of the moment without thinking, planning, or considering the consequences" [36]. The trait of impulsivity is a known risk factor for the development of multiple addictions [6, 36, 37].

Impulsive intake of a substance in this context food; is associated with a loss of control [34]. Moreover the propensity to act impulsively in relation to food has been implicated as a potential factor contributing to obesity [38]. A recent review found increased impulsivity in relation to reward sensitivity in obese individuals that was further heightened in individuals with BED [38].

Studies in obese subjects with and without BED have indicated deficits in decisional impulsivity specifically in regards to delay discounting and risk taking in the context of ambiguity indicating a potential deficit as a function of obesity [37]. Further this deficit appears to be mediated by the level of obesity and the context of the food cues [37].

Motor impulsivity (motor response inhibition or waiting impulsivity) seems to be more impaired in obese subjects with BED than obese non-BED controls in relation to food related stimuli but not neutral stimuli [37]. More widespread impairments generally apparent in other addictions in relation to impulsivity were not evident in obesity and obesity coupled with BED [37].

Treatment Target: Trait of Impulsiveness

Impulsivity appears to be a predisposing trait to addiction with the personality trait initially driving the behaviour and the start of the addiction [39, 40]. Impulsivity can also be a

consequence of addiction with chronic substance use leading to further elevated levels of impulsivity, recent research also indicates a similar relationship between impulsivity and behavioural addictions [41]. Moreover impulsivity is also related to treatment outcomes and time to relapse [42, 43] Thus the primary treatment target is the trait of impulsivity.

Psychological treatments such as CBT and group therapy aimed at increasing mindfulness and awareness, along with the acceptance and tolerance of emotions are known to assist with negating impulsivity in substance use disorders [42]. Specifically mindfulness assists as awareness is focused upon the present moment and environment which helps to circumvent automated and impulsive behaviours [42]. Further treatments that foster problem-solving and decision making skills are also beneficial [27].

Moreover treatments that specifically focus upon cue and reward sensitivity in addiction are also effective in assisting impulse control [44]. A recent review of both human and animal studies indicated increased cue reactivity in a subset of individuals as being the impetus for impulsive behaviour [44]. Therefore treatments focused upon teaching individuals with addiction to avoid attending too or extinguishing the salience of cues has proven efficacious [44].

In addition to psychological therapies there are pharmaceutical treatments that assist with controlling impulsivity. In relation to FA, lisdexamfetamine dimesylate has proven effective in the treatment of BED [45]. Lisdexamfetamine dimesylate is the only drug with FDA approval to treat BED [45]. A recent study by McElroy and colleagues [45] found that lisdexamfetamine dimesylate reduced impulsivity, binge eating severity as well as total binge eating days in patients with BED.

Behaviour: Compulsivity

Compulsivity and impulsivity are related constructs that are thought to involve corresponding processes and the same underlying neural substrates [37]. Models of addiction posit that impulsivity and compulsivity lie at opposing ends of a continuum; that is impulsivity drives the initial process leading to addiction where upon compulsive behaviours take over and the behaviour is continued regardless of the negative consequences [37]. Compulsivity is associated with repeating a specific behaviour regardless of the negative consequences [37]. Research has shown that subjects with BED display impairments across multiple domains of compulsivity in comparison to obese controls [37]. However the level of these deficits appears to be a function of BMI with subjects with and without BED and higher BMIs displaying similar impairments [37].

Kenny [46] after completing a review of the common neural mechanisms that underlie both the over consumption of hyperpalatable food and drugs of abuse concluded that there is enough evidence to view obesity as a 'compulsive consummatory behaviour' akin to drug addiction. As such obesity and binge eating in particular can be understood as a compulsive behaviour.

Another disorder that manifests compulsive behaviour is obsessive compulsive disorder (OCD) with dysfunction in the serotonergic system thought to be central to the disorder [47]. Therefore treatments aimed at the behaviour of compulsivity should target the serotonergic system.

Treatment Target: Serotonin system

The ideology of compulsive eating and a loss of control in regards to food consumption is central to the phenomenon of FA. Thus as noted above the serotonergic system is a common treatment target in another disorder that involves compulsivity; OCD and selective serotonin reuptake inhibitors (SSRIs) have also been utilised in the treatment of BED. Recently Berkman and colleagues [48] conducted a systematic review and meta-analysis of the management of BED. They concluded that second generation antidepressants are superior to placebo in reducing eating related obsessions and compulsions and reducing binge eating episodes. Further they also reduced depressive symptomology a common comorbid condition with BED [48]. SSRIs have also shown some effectiveness in treating binge eating and purging in bulimia nervosa [32].

Although not targeting the serotonergic system recently Hurley and colleagues [49] found that N-acetylcysteine decreased binge eating in the animal model. N-acetylcysteine is a prodrug that acts upon the glutamate and dopamine neurotransmission [50]. Although data is limited N-acetylcysteine has also shown promise in the treatment of addictions including gambling disorder as well as OCD; reducing compulsive behaviours in these disorders [50].

In addition to pharmaceutical treatments psychotherapeutic interventions are also recommended. In regards to compulsive eating similarly to the psychological treatments for other core addiction behaviours stimulus control and effective coping strategies are implicated [27]. Such as avoiding hyperpalatable foods that could lead to compulsive overeating and finding alternatives for pleasure and reward [27].

Behaviour: Motivation

As oppose to drugs of abuse the degree to which food is rewarding is moderated by homeostatic mechanisms driven by the current level of hunger of the individual [34]. Thus food can be consumed for energetic reasons as well as the foods own reinforcing (rewarding) properties [34]. Studies pertaining to the neural peptides leptin, ghrelin and orexin support a close link between the homeostatic and motivational systems [34]. Studies have indicated that during periods of hunger the motivational system directs attention towards food and food related stimuli [34].

The mesolimbic dopamine system has a known role in reward processing, reward reinforcement and attending to motivationally salient stimuli [18]. In addiction, drugs of abuse directly impact upon the mesolimbic dopaminergic system with drugs of abuse and associated stimuli elevating dopamine levels in the striatum. In FA hyperpalatable foods and food associated cues have a similar effect on dopamine levels in the striatum [18]. Thus the mesolimbic dopaminergic system plays a pivotal role in motivation.

Treatment Target: Dopamine system

Drugs of abuse are known to impact upon striatal dopamine receptor availability and striatal function, recently Gearhardt and colleagues [9] reported that obesity in humans is also associated with a reduction in striatal dopamine receptor availability and striatal dysfunction. Further research preceding addiction has also shown that individuals vulnerable to developing an addiction have a pre-existing decrease in striatal dopamine D2 receptor availability [18]. This renders individuals as being more predisposed to the rewarding properties of desirable stimuli and as such the potentially rewarding stimuli is more salient [18].

Grosshans, Loeber and Kiefer [34] report that there is evidence that pharmacotherapy developed for the treatment of addiction specifically relapse prevention in addiction is also effective in the treatment of obesity. These treatments all target the dopaminergic mesolimbic system [34].

Recent years have seen the development of psychological treatment options for addiction such as motivational interviewing and cognitive behavioural therapy (CBT) to bolster abstinence motivation and assist with strategies to alleviate the possibility of relapse [34]. These treatments target the cognitive focus upon the rewarding stimuli either directly or indirectly through the addition of an alternative reinforcer; hence these therapeutic options target the mesolimbic reward system [34]. Further CBT and interpersonal therapy although efficacious in the short and long-term reduction of binge eating in BED are not associated with clinically significant weight loss rather they alleviate the potential to gain further weight through continued binge eating [51].

Conclusion:

This article sought to review potential strategies for the treatment of FA based upon four core behaviours of the addiction phenotype. To our knowledge this is the first review of clinical treatment strategies directly aimed at targeting FA. The reviewed treatments target addictive like eating through core behaviours synonymous with addiction generally. Namely; targeting craving through the opiod system, targeting impulsivity as a personality trait, targeting compulsivity through the serotonergic system and lastly targeting motivation through the dopaminergic system.

It is thought that utilising these treatment strategies should assist with alleviating addictive like eating behaviours. It is intended that these treatments would be utilised to provide individualised treatment. That is targeting addictive like eating through the most prominent and worrying behaviour or behaviours for each individual. Therefore treatment could be targeted at a single behavioural domain or at multiple domains. Further treatment could be solely pharmacological or psychological, or both could be utilised in combination to target a single or multiple behaviours.

Although the notion of individualised treatment was a primary tenet underlying this review of potential treatment strategies, the strategies espoused are sufficiently general so they are applicable to the broader population struggling with addictive like eating. With the inclusion of the four general addiction specific behaviours being broad enough to facilitate wide-ranging treatment of addictive like eating in the larger population as well as diverse population segments. The development of these potential treatment strategies although not yet validated offer both an opportunity to view the phenomenon of FA from an alternate perspective beyond that delineated in YFAS as well as offering a potential approach to the treatment of addictive like eating.

Research pertaining to FA in general is in the nascent stage and as such further work is needed in order to better understand the phenomenon. Specifically further research should focus upon the measurement, course and treatment of FA, as well as the most appropriate designation for the construct. In addition better methods for the assessment of psychological, behavioural and psychosocial variables that are related to FA are required.

Specifically in relation to the espoused treatment strategies extensive testing and validation is required to elucidate whether they are effective in negating FA behaviours in the general

affected population as well as in diverse population segments. Further research should also seek to ascertain the most effective methods and modes of assessment to delineate the specific behaviour or behaviours to be targeted which would inform individualised treatment.

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