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One size does not fit all: Understanding the five stages of ultra-processed food addiction

Author:
Vera I. Tarman[1]

Affiliation:
[1]Department of Family Medicine, Faculty of Medicine, University of Toronto, Toronto, Canada

Corresponding author:
Vera Tarman,
veratarman@gmail.com

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In recent years, research has demonstrated that ultra-processed foods are highly addictive. However, there has been limited discussion on the clinical syndrome of food addiction, and how exposure to addictive foods can result in a chronic progressive syndrome, which can be further understood in five distinct stages. Understanding these stages sheds light on why diagnosis and successful treatment may vary among individuals and why a one-size-fits-all approach is ineffective.

Keywords: addiction; mental health; obesity; low carb; metabolic health.

Introduction

There is growing recognition that hyperpalatable foods high in sugar, fat and sodium can produce addictive processes in the brain, such as drugs of abuse. Animal models and human neuroimaging studies have repeatedly shown that foods rich in sugar and fat activate the brain reward circuitry in ways comparable to those of addictive substances.[1,2,3] The behavioural profile of continued compulsive overconsumption despite negative consequences, withdrawal symptoms and failed attempts to cut back intake further support the concept of 'food addiction'.[4,5] While the idea of food addiction has garnered interest in recent years, the notion of a progressive food addiction syndrome with distinct stages remains less examined[6] and is even discouraged in some clinical settings. Here, I propose a stage-based model of ultra-processed food addiction that integrates evidence on neurobiology, hormonal factors and addiction criteria that mark its development from mild to severe. I discuss the implications of this framework for diagnosis and tailored treatment approaches targeted at the stage of progression.

This article draws upon evidence suggesting that excessive consumption of hyperpalatable foods can lead to addictive-like changes in the brain's reward system, specifically focusing on the dopaminergic system, which changes over time. According to the neuroadaptive addiction model,[7] it is hypothesised that a food addiction syndrome develops and progresses through distinct stages of increasing physical and psychological dependence, like alcohol and cocaine addiction.

Five stages have been proposed: (1) pre-addiction, (2) early addiction, (3) mid-addiction, (4) late-stage and (5) end-stage food addiction.[8] It is argued that the diagnosis and treatment of food addiction should be tailored to these stages, considering the varying levels of neural dysfunction and symptom severity along the continuum of this chronic disorder. Thus, it is evident that a single dietary intervention cannot be universally effective for individuals with food addiction. Instead, each intervention must be customised to address a specific stage of addiction.

Hormonal and neurobiological changes when eating processed food

Hormonal and neurobiological changes occur when individuals consume processed foods, especially highly palatable foods that are rich in sugar, fat and salt. These changes can contribute to the development of addictive behaviours and, ultimately, addiction.

It is well established that derangement of hormones can contribute to this process. Ghrelin is responsible for regulating hunger. Elevated ghrelin levels can drive hunger and increase food intake. On the other hand, leptin is responsible for signalling satiety and inhibiting food intake. Consumption of highly palatable foods, particularly those high in sugar, fat and salt, has been shown to disrupt the balance of these hormones; for example, it can increase ghrelin levels. As a result, individuals may experience increased cravings and overeating as signals for hunger and

fullness become dysregulated. Additionally, consumption of processed foods has been hypothesised to lead to leptin resistance, where the body no longer responds to satiety signals.[9] Disruptions in the normal interplay between ghrelin and leptin can result in overeating and continued consumption of these foods.

Insulin, another hormone involved in regulating blood sugar levels, also plays a role in food overconsumption. Insulin helps the body to convert glucose into energy and promotes satiety. However, the prolonged consumption of ultra-processed foods can lead to the development of insulin resistance. This means that insulin no longer effectively suppresses appetite, leading to increased hunger and cravings for highly refined carbohydrates. It also causes peripheral hormonal changes, including reduced postprandial release of satiety peptides such as peptide tyrosine-tyrosine (PYY) and Glucagon-like peptide 1 (GLP-1).[10]

Thus, these hormonal changes can contribute to an overeating profile that resembles food addiction. Although they may not yet indicate the criteria for full blown addiction, they are laying the groundwork for hedonic dysregulation to occur. Dopamine is recruited whenever hunger occurs, whether it is triggered by ghrelin or insulin, and this leads the individual to seek food. Leptin, on the other hand, plays a role in suppressing dopamine, preventing the person from anticipating food.

Furthermore, consuming processed foods that are high in sugar and fat can trigger neurobiological changes in the reward circuitry of the brain.[11,12] The key brain regions implicated include the striatum, prefrontal cortex, amygdala and insula.[13] Neurotransmitters and neuropeptides include dopamine, endogenous opioids and endocannabinoids. These brain systems enhance the incentive salience of food cues, motivate feeding behaviour and mediate the feelings of pleasure and reward. Several studies have provided evidence of the addictive properties of highly processed foods, showing that highly palatable foods can lead to cravings, compulsive overeating and changes in brain chemistry, such as those observed with drug addiction[10]. Although the hormonal model of aberrant eating, discussed above, can explain some addiction-like behaviours associated with processed food consumption, understanding neurobiological changes helps to further understand how the development of an actual food addiction syndrome occurs, complete with the progressive stages of reward dysfunction, impulsivity and emotional dysregulation.

Addiction: A chronic progressive condition

Addictive disorders are chronic conditions that worsen over time, a phenomenon known as the allostatic model of addiction.[7] This model proposes that the brain's reward system undergoes neuroadaptations from repeated overstimulation, leading to progressively greater loss of control over substance use. As dependence intensifies, escalating consumption is required to achieve the desired effects, owing to the development of tolerance.

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), published by the American Psychiatric Association,[14] includes 11 criteria for the diagnosis of substance abuse, of which an addict must fulfil at least two criteria. The criteria are:

1. Using larger amounts of substance for longer than intended.
2. Craving for substances.
3. The substance has been used for a significant amount of time.
4. Repeated attempts to quit and/or control substance use.
5. Social or interpersonal problems that are related to substance use.
6. Major roles are neglected when using a substance.
7. Activities are discontinued owing to overuse of the substance.
8. Hazardous conditions arise from the continued use of these substances.
9. Physical and psychological problems develop because of use of substance
10. Tolerance develops because of overuse of the substance.
11. Withdrawal occurs when a substance is stopped.

Addiction is a gradual, progressive disorder that develops over time. As more criteria are met with repeated overstimulation of the brain's reward system, a higher level of severity and dysfunction in addiction occurs, from mild (1–3) to moderate (4–5) to severe (6–11).

The progression of an ultra-processed food addiction syndrome: A neurobiological perspective

Although sugar or ultra-processed food addiction is not classified as a substance use disorder in the DSM-5, it is likely that similar neural adaptations occur in food addiction, as with other drugs. Clinicians typically find that sugar and ultra-processed food addiction meet five of the criteria (1, 2, 4, 9 and 10) established by the DSM-5 for substance abuse diagnosis.

Studies have shown that continued overconsumption of hyperpalatable foods leads to gradual downward shifts in dopamine signalling as well as dysfunction in prefrontal cognitive control circuits and stress pathways. Animal research indicates that so-called 'food addiction' behaviours can emerge and slowly intensify over time, rather than being present from the initial exposure. These findings are in accordance with the allostatic model of addiction as a chronic, progressive disease.[7]

Building on the allostatic model of addiction, Davis, a researcher studying eating disorders, proposed an addiction neuroadaptation model to understand the development and progression of aberrant eating. Convinced by her inability to

explain the overeating she witnessed presumably because of an eating disorder, Davis posited that dopamine, a neurotransmitter associated with reward and pleasure, plays a pivotal role in the development and maintenance of aberrant eating behaviours.[15] She suggested that repeated consumption of hyperpalatable foods, such as those high in sugar and processed ingredients, leads to dopamine neuroadaptation, wherein the brain's reward system becomes desensitised, requiring larger amounts of food to achieve the same level of pleasure. This gradual neuroadaptation (essentially, the allostatic attempt to limit reward) contributes to the progression of food addiction.

According to Davis, the first stage of food addiction is characterised by binge eating episodes, during which individuals lose control over their food consumption. The release of dopamine during these episodes reinforces the behaviour, leading to a cycle of reward-seeking and overeating. As addiction progresses, individuals may develop tolerance for the rewarding effects of food, requiring larger quantities or more intense flavours to achieve the same level of satisfaction. Over time, compulsive eating is essential for avoiding withdrawal and maintaining normal functioning. Eventually, normal functioning is lost, and the individual continues to compulsively eat despite many negative consequences. At this point, the individual fulfils all the DSM-5 criteria for addiction.

The development and progression of a food addiction syndrome

Based on the allostatic framework and Davis' application of it to compulsive overeating, it is proposed that food addiction progresses through stages of increasing physical and psychological dependence on hyperpalatable foods, into a continuum of symptomatology that can be called food addiction syndrome. Werdell,[8] a pioneer clinician in the field, conceptualised specific stages of food addiction that reflect this progression of symptoms and proposed five stages: the pre-addiction stage, characterised by occasional overconsumption of sugar and refined carbohydrates; the early addiction stage, where excessive overeating occurs without consistent control over intake; the middle addiction stage, signifying compulsive binge eating with withdrawal symptoms when particular foods are stopped; late-stage addiction, characterised by total loss of control despite negative consequences and finally, end-stage food addiction, in which tolerance, withdrawal and negative consequences are prominent features. I propose that Werdell's clinical understanding of the stages of food addiction is grounded in the allostatic neuroadaptive processes of the brain and can offer a helpful framework for comprehending the evolution and discrete phases of food addiction syndrome and, ultimately, its treatment.

Pre-addiction stage

In the pre-addiction stage, consumption of sugar and fat leads to increased dopamine signalling and opioid release in reward regions, such as the nucleus accumbens and the ventral tegmental area. This enhances the reward value and motivational salience of the food cues.[14] Over time, mild downregulation of D2 dopamine receptors may begin, indicating early neuroadaptive changes. Thus, individuals may engage in occasional episodes of overeating hyperpalatable foods without experiencing significant negative consequences or a loss of control.

Early addiction stage

In the early phase of addiction, there is a further decrease in D2 receptors, reduced dopamine communication and decreased responsiveness to rewards. Additionally, there may be slight difficulties in prefrontal regulation of food cues and eating behaviours.[11] Thus, this stage is marked by the onset of excessive eating, strong desires and lack of consistent control over the consumption of highly tempting foods. There may even be slight signs of withdrawal when the intake of certain foods is limited. At this stage, it may be challenging to differentiate between a developing food addiction fueled by hedonic impulses and the hormonal imbalances of ghrelin and insulin previously discussed, where the neurochemical changes of addiction may not yet be present. It is highly probable that both factors contribute to this early stage. Also, the situation may be mistaken for the onset of an eating disorder. Diagnosis is particularly challenging at this stage.

Middle-stage addiction

The mid-level stage of addiction involves a significant decrease in striatal D2 receptors and dopamine release as well as potential reductions in opioid signalling. Deficits in prefrontal inhibitory control are accompanied by hormonal changes such as decreased release of satiety peptides and increased resistance to leptin and insulin.[10] This results in frequent episodes of binge eating, intense cravings and signs of compulsive eating behaviour. Abstaining from specific foods leads to more pronounced withdrawal symptoms. In the middle and later stages, the manifestations of food addiction become more evident and are less likely to be mistaken for an eating disorder or hormonally imbalanced eating.

Late-stage addiction

In late-stage addiction, dopamine and opioid signalling are further depleted, and pronounced downregulation of D2 receptors heightens cravings.[10] Prefrontal executive dysfunction is more severe, marked by impulsivity and a dominant focus on food rewards over other goals. Metabolic disturbances are more significant and withdrawal symptoms become more intense. Thus, bingeing escalates, control of overeating is largely lost and tolerance develops, as indicated by increased intake to achieve the desired effects. It is at this point that jobs are lost, relationships are strained and health issues (mood, obesity and other metabolic irregularities) become prominent.

End-stage addiction

As the food addiction syndrome progresses to more severe stages, brain reward deficits are pronounced by the end stage, with extremely low dopamine signalling and D2 receptors, as well as possible opioid depletion. Metabolic derangements are significant and executive function is grossly impaired.[16] Food consumption occurs compulsively despite minimal pleasure or rewards and is characterised by a complete loss of control, overeating and an inability to abstain from binge food without severe withdrawal. Death inevitably follows as the outcome of extensive metabolic disease although it is often not recognised as being related to food addiction.

Treatment implications based on stages of a food addiction syndrome

The proposed stages of a food addiction syndrome have critical implications in treatment.

To begin with, it is crucial to recognise that the individual has a food addiction, as opposed to an eating disorder, such as binge eating disorder or bulimia nervosa. The proposed treatments of limiting specific foods could potentially endanger the client who is seeking to limit foods because of a pathology related to the eating disorder. Therefore, the accuracy of the diagnosis is of utmost importance and significant research is being done to find tools that can ensure the correct diagnosis.[3,17]

Once the diagnosis of food addiction has been ascertained, it is essential to assess the stage of addiction the individual is experiencing to provide the most appropriate course of action. In the early stages of food addiction, interventions aimed at increasing inhibitory control and addressing hormonal imbalances may be effective and sufficient. These interventions may include cognitive-behavioural therapy to improve impulse control and self-regulation and counseling on mindful eating and nutritional education. Dietary changes that address hormonal dysregulation and promote satiety might also be beneficial. For example, a low-carbohydrate food and/or keto plan that expressly avoids refined carbohydrates may prevent the progression of neuroadaptation and further development of addiction.

More intensive interventions may be necessary in the middle to later stages of food addiction. Strict abstinence from trigger foods (typically sugar and flour) is the primary goal, as individuals have lost control of their consumption. These can include not only refined carbohydrates but also other trigger foods, such as cheese, nuts and sweeteners, depending on the individual's prior overexposure to these rewarding foods. These foods should be eliminated from an individual's diet, as even small amounts can trigger cravings and lead to binge episodes.

The standard American diet varies significantly from these modifications, so at this point, individuals may need extensive nutritional support to follow these guidelines. Furthermore, addiction's progressive nature makes these change increasingly challenging, and additional personal support may be necessary. Substantial lifestyle adjustments of this magnitude often necessitate substantial support, such as individual and group counselling, to achieve lasting success.

In severe stages of food addiction, yet more drastic measures may be required to correct neurobiological imbalances. Late-stage food addiction may require treatment, such as medications for cravings (such as Naltrexone and/or Bupropion or a GLP-1AR) and bariatric surgery to restrict food intake and its metabolism. Residential programmes and long-term 12-step groups such as Overeater's Anonymous or outpatient community groups are encouraged to maintain behavioural changes.[8]

Overall, management of food addiction should be customised to address a particular phase of the syndrome. This understanding of the progression of addiction through stages emphasises the significance of accurately assessing the severity of food addiction to determine suitable interventions for each person. An incorrect diagnosis of the stage of illness may lead to ineffective or harmful treatment. For instance, imposing strict abstinence from problematic food on individuals in the pre-addiction stage may be unnecessarily restrictive and discourage the patient from seeking further help. However, outpatient nutrition counseling and modest reduction or avoidance of refined carbohydrates or other trigger foods would likely not suffice for those in advanced stages of addiction. It could lead to further addiction, much like an alcoholic is unable to tolerate a moderate drinking plan for a very long time. As well, in order to overcome mid- to end-stage food addiction and maintain stability in recovery, one cannot fail to address the common challenges that typically arise with any addictive disorder, such as poor self-esteem, feelings of shame and social isolation. Aligning the treatment intensity with the progression of food addiction severity is crucial.

Conclusions

In conclusion, understanding the progressive nature of the ultra-processed food addiction syndrome is crucial for healthcare professionals to provide effective diagnosis and treatment. By recognising the stages of the ultra-processed food addiction syndrome and tailoring interventions accordingly, we can address the unique neuroadaptations and challenges faced by individuals at each stage of the illness. However, further clinical research is needed to validate the proposed model and to identify the most effective interventions for food addiction at its various stages. By continuing to explore this complex disorder, we can pave the way for improved outcomes and a better quality of life for those affected by ultra-processed food addiction.

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Author's contributions

V.I.T. is the sole author of this article and was responsible for the literature review, conceptualised the central argument, wrote the article and then reviewed it for consistency and clarity of mission.

Ethical considerations

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Data availability

All of the research cited in my literature review can be found on publicly available data sets, namely Amazon.com for books and sites such as PubMed (nih.gov).

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