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Review Article

The Intersection of Obesity and Behavioral Addictions: A Comprehensive Narrative Review of Neurobiological Mechanisms and Clinical Implications

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Abstract

Objectives: Obesity is a pervasive global health challenge with a complex etiology. Recent conceptualizations propose that certain eating behaviors, particularly towards ultra-processed foods, mirror the patterns seen in substance use disorders, giving rise to the "food addiction" model. This narrative review aims to synthesize contemporary evidence on the neurobiological and psychological overlap between obesity and behavioral addictions, and to discuss diagnostic tools and treatment modalities informed by this model.

Methods: A narrative review of literature published between 2018 and 2025 was conducted, focusing on studies elucidating the reward pathway dysfunctions, psychological correlates, and advancements in treating addiction-like eating behaviors.

Results: Evidence indicates that hyperpalatable foods significantly alter the brain's reward circuitry, notably through dopamine release and receptor downregulation in the mesolimbic pathway, paralleling mechanisms in substance addiction. Neuroimaging studies consistently show altered prefrontal-striatal connectivity, contributing to diminished inhibitory control and heightened cue reactivity. Psychologically, traits such as impulsivity and stress-mediated emotional eating are strongly associated with compulsive consumption patterns. The Yale Food Addiction Scale (YFAS) has emerged as a key tool for identifying individuals who may exhibit these addictive-like eating behaviors. Pharmacological interventions like GLP-1 receptor agonists and naltrexone/bupropion, alongside cognitive-behavioral therapies and neuromodulation techniques, show promising efficacy by targeting these underlying addictive mechanisms.

Conclusion: The behavioral addiction model provides a valuable framework for understanding and treating a distinct subgroup of obesity characterized by compulsive overeating. Integrating addiction-based approaches into obesity management, including novel pharmacotherapies, psychotherapy, and public health policies regulating ultra-processed foods, offers a promising multidisciplinary strategy for improving outcomes in treatment-resistant cases.

Keywords: Behavioral addiction, Dopamine, Food addiction, Obesity, Reward system, Yale food addiction scale

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INTRODUCTION

Obesity represents one of the most significant public health crises of the 21st century, with over 650 million adults affected worldwide [1]. Traditionally attributed to a simple imbalance between energy intake and expenditure, the persistence and complexity of obesity have prompted explorations into more nuanced etiological models. Among these, the concept of "food addiction" has gained substantial traction, proposing that certain foods, particularly those engineered to be hyperpalatable (high in refined carbohydrates, fats, and salts), can trigger addictive-like neurobiological and behavioral responses [2,3].

This model draws direct parallels to substance use disorders, suggesting that the chronic consumption of these foods can hijack the brain's natural reward pathways, leading to a loss of control, overeating, withdrawal symptoms, and continued use despite negative consequences. This article provides a comprehensive narrative review of the current evidence linking obesity and behavioral addictions. It examines the shared neurobiological substrates, explores key psychological factors, evaluates diagnostic criteria such as the Yale Food Addiction Scale (YFAS), and discusses emerging treatment strategies derived from addiction medicine. The ultimate goal is to elucidate how adopting an addiction framework can enhance our understanding and management of compulsive eating behaviors in obesity. The review is structured as follows: first, we explore the neurobiological underpinnings of addictive eating; second, we discuss the psychological and behavioral dimensions; third, we evaluate diagnostic considerations with a focus on the YFAS; fourth, we review evidence-based interventions; and finally, we discuss future directions and conclusions.

NEUROBIOLOGICAL UNDERPINNINGS OF ADDICTIVE EATING

The core of the food addiction hypothesis lies in the shared neurobiology between compulsive overeating and substance addiction, primarily centered on the brain's reward system.

Dopaminergic Pathways and Reward Deficiency

The consumption of hyperpalatable foods leads to a surge of dopamine in the nucleus accumbens, a key region in the brain's reward circuit [4]. This acute reward response is similar to that induced by addictive drugs. However, with chronic overconsumption, neuroadaptations occur. There is a downregulation of dopamine D2 receptors, leading to a blunted reward response [5]. This state of "reward deficiency" drives individuals to consume larger quantities of palatable food to achieve the same level of satisfaction, perpetuating a cycle of compulsive overeating. This process shares similarities with the Incentive Sensitization Theory of addiction, which posits that repeated exposure to a reward (e.g., drugs, hyperpalatable food) sensitizes the mesolimbic dopamine system to the reward's incentive salience, making cues associated with it powerful triggers for craving and compulsive behavior, even if the pleasure derived from consumption diminishes [6].

Neuroimaging Evidence of Altered Circuitry

Functional magnetic resonance imaging (fMRI) studies provide compelling visual evidence for this model. It is important to note that while fMRI is a powerful and widely used tool in scientific research for elucidating brain function and connectivity in obesity and addiction, its application remains primarily experimental and has not yet been integrated into routine evidence-based clinical practice for diagnosing or treating these conditions. Nonetheless, it holds significant promise for future clinical translation, such as in identifying neural biomarkers for treatment selection [7,8]. Individuals with obesity often show:

- **Hyperactivation** of reward-related regions (e.g., the striatum) in response to food cues [7].
- **Hypoactivation** of prefrontal cortical regions, including the dorsolateral prefrontal cortex (dlPFC) and orbitofrontal cortex (OFC), which are critical for executive function, decision-making, and inhibitory control [7].
- Recent advances further demonstrate **altered functional connectivity** between the dlPFC and the striatum. This disrupted communication weakens top-down control over motivated behaviors, making

it increasingly difficult to resist powerful food cues and contributing to compulsive intake patterns [9].

The Role of Sensory Cues: From Olfaction and Vision to Appetite

The brain's reward system is profoundly influenced by external sensory cues, which can powerfully drive appetite and compulsive seeking behavior.

Neurological Pathways from Olfaction to Appetite

Olfaction is intimately linked to food reward and appetite regulation. Odorants bind to receptors in the olfactory epithelium, sending signals directly to the olfactory bulb. This information is then relayed to key brain regions involved in reward and feeding behavior, such as the orbitofrontal cortex (OFC), amygdala, and piriform cortex, which have dense projections to the nucleus accumbens and hypothalamus [10,11]. Food odors can trigger anticipatory dopamine release in the striatum, enhancing the desire for food and initiating food-seeking behavior. In individuals with compulsive eating patterns, this olfactory-appetite circuit may be hyper-responsive, making smells from the environment (e.g., a bakery) potent triggers for cravings and relapse, even in the absence of hunger [12].

The Impact of Visual Stimuli on Appetite

In the modern digital era, visual cues are omnipresent drivers of eating behavior. The constant exposure to images and advertisements of highly palatable, often ultra-processed foods on television, social media, and digital marketing platforms can significantly promote appetite and craving [13]. Neuroimaging studies show that viewing images of palatable food activates the same reward regions (e.g., striatum, OFC) as actual food consumption [14]. This visual food cue reactivity is often heightened in obesity and is associated with increased calorie intake. Conversely, exposure to images of healthy foods or public health campaigns promoting nutritious choices can, in some contexts, temporarily inhibit appetite for less healthy options, highlighting the potential for visual media to be harnessed for positive behavioral change [15].

PSYCHOLOGICAL AND BEHAVIORAL DIMENSIONS

The neurobiological vulnerabilities are often compounded by specific psychological traits and are influenced by broader environmental factors.

Impulsivity and Disinhibition

Elevated trait impulsivity is a robust predictor of binge eating behaviors and poorer long-term outcomes in weight management programs [16,17]. Impulsivity facilitates the initial reward-seeking behavior and undermines the self-regulatory capacity needed to maintain dietary changes.

Emotional Eating and Stress

Stress activates the hypothalamic-pituitary-adrenal (HPA) axis, releasing cortisol, which can increase appetite and drive a preference for energy-dense, palatable foods [18]. For many, eating becomes a primary coping mechanism to alleviate negative affect, reinforcing the addictive cycle.

Cue Reactivity and Conditioning

Environmental cues—from food advertisements to the smell of a bakery—can become powerful triggers through classical conditioning. These cues elicit anticipatory dopamine release, creating intense cravings and motivating seeking behavior, even in the absence of physiological hunger [13]. The pervasiveness of these cues in our "obesogenic" environment, often targeted by sophisticated marketing strategies, particularly in socioeconomically disadvantaged areas, significantly exacerbates the risk of developing and maintaining compulsive eating patterns [19].

Genetic and Epigenetic Factors

Individual susceptibility to obesity and addictive-like eating is also influenced by genetic and epigenetic factors. Genome-wide association studies have identified numerous genetic variants associated with obesity risk, including those in genes related to dopaminergic signaling (e.g., DRD2, ANKK1), leptin signaling (LEP, LEPR), and fat mass and obesity-associated genes (e.g., FTO) [20]. These genetic predispositions can influence reward sensitivity, impulse control, and metabolic

efficiency. Furthermore, epigenetic modifications—changes in gene expression without altering the DNA sequence—induced by factors such as early life stress, diet, and environmental toxins can permanently alter the regulation of genes involved in stress response, reward, and appetite, potentially increasing the risk for compulsive overeating later in life [21].

DIAGNOSTIC CONSIDERATIONS: THE YALE FOOD ADDICTION SCALE (YFAS)

Operationalizing "food addiction" for clinical and research purposes has been primarily achieved through the Yale Food Addiction Scale (YFAS) [22]. Modeled on the DSM-5 criteria for substance use disorders, the YFAS assesses symptoms such as:

- Consuming larger amounts or for longer periods than intended.
- Persistent desire or unsuccessful efforts to cut down.
- Significant time spent obtaining or consuming food.
- Craving or strong urge to eat.
- Recurrent use resulting in failure to fulfill role obligations.
- Continued use despite social or interpersonal problems.
- Giving up important social, occupational, or recreational activities.
- Recurrent use in physically hazardous situations.
- Use despite knowledge of persistent physical or psychological problems.
- Tolerance (need for markedly increased amount to achieve desired effect).
- Withdrawal (characteristic withdrawal syndrome or substance taken to relieve withdrawal).

While the construct's validity is debated, with critics arguing it may pathologize normal behavior and that it shows significant diagnostic overlap with conditions like Binge Eating Disorder(BED) [23], the YFAS has proven particularly useful in identifying a distinct subgroup of individuals with obesity who exhibit more severe, compulsive eating patterns and may be less responsive to standard weight-loss interventions [24].

A key limitation of the YFAS is its reliance on self-report, which is susceptible to recall bias and social desirability effects, and its inability to directly measure the underlying neurobiological processes of addiction.

EVIDENCE-BASED INTERVENTIONS INFORMED BY ADDICTION SCIENCE

The reconceptualization of certain obesity cases as a behavioral addiction opens the door for novel treatment approaches.

Pharmacotherapy

Medications that target neurotransmitter systems involved in reward and craving are showing promise:

- **Naltrexone/Bupropion:** This combination acts on the opioid and dopaminergic/noradrenergic systems, respectively, to reduce food cravings and reward value [25].
- **GLP-1 Receptor Agonists (e.g., Semaglutide, Tirzepatide):** Originally for diabetes, these drugs cause significant weight loss by promoting satiety, reducing gastric emptying, and, crucially, **diminishing the reward salience of palatable foods**, thereby reducing cravings and compulsive food-seeking behaviors [26].
- **Emerging Agents:** Novel triple agonists (targeting GLP-1, GIP, and glucagon receptors) demonstrate even greater efficacy and are under active investigation [27].

Cognitive-Behavioral Therapy (CBT)

CBT, particularly enhanced protocols (CBT-E), is effective in addressing the maladaptive thoughts and behaviors that sustain binge eating. It helps patients identify triggers, develop alternative coping strategies for emotional distress, and challenge cognitive distortions (e.g., "all-or-nothing" thinking about food) [28,29].

Neuromodulation

Non-invasive brain stimulation techniques are being explored to rectify the neural imbalances identified in obesity.

- **Repetitive Transcranial Magnetic Stimulation (rTMS):** By applying magnetic pulses to stimulate the hypoactive dorsolateral prefrontal cortex

(dlPFC), rTMS can enhance inhibitory control and reduce cravings and binge-eating frequency [8,30].

DISCUSSION AND FUTURE DIRECTIONS

The addiction model of obesity is not a one-size-fits-all explanation but offers a crucial lens for a significant patient subgroup. Future research must focus on:

- **Precision Medicine:** Identifying genetic and neuroimaging biomarkers (e.g., variants in the FTO or DRD2 genes; specific fMRI connectivity patterns [9,20]) that predispose individuals to reward-based eating to allow for targeted interventions.
- **The Gut-Brain Axis:** Investigating how the gut microbiota influences craving and eating behavior through communication with the brain presents a novel therapeutic frontier. For instance, specific microbial metabolites can modulate dopamine and serotonin production, influence gut permeability, and send vagal signals to the brain, thereby affecting mood, reward, and food choices. Interventions such as prebiotics, probiotics, and fecal microbiota transplantation are being explored.
- **Public Health Policy:** The evidence supports population-level strategies to reduce the environmental drivers of addictive eating. Real-world examples include taxes on sugar-sweetened beverages (implemented in over 50 countries), restrictions on marketing of ultra-processed foods to children (as in Chile and the UK), and mandates for front-of-package warning labels [19]. These policies aim to create healthier food environments and reduce cue reactivity at a societal level.

CONCLUSION

A substantial body of neurobiological, psychological, and clinical evidence supports the overlap between obesity and behavioral addictions. For individuals exhibiting compulsive, addiction-like eating patterns, standard lifestyle interventions often fall short. Integrating principles from addiction science—through pharmacological agents that blunt cravings, psychotherapies that rebuild self-regulation, neuromodulation that restores cortical control, and policies that create healthier food environments—provides a more comprehensive and practical framework for addressing this complex and treatment-resistant aspect of obesity. A multidisciplinary approach is paramount for improving outcomes in this population.

Article Information Form

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