Review Article

A literature review of the neurobiological and behavioral substrates of food addiction

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ABSTRACT

Background: Food addiction (FA) is a multifaceted behavioral disorder involving an uncontrollable craving for high-calorie, palatable foods. FA plays an important role in threatening public health. The purpose of this literature review is to systematically examine the multifaceted nature of FA by elucidating the neurobiological, psychological, and environmental factors involved.

Methods: This literature review adhered to PRISMA guidelines and searched international databases, including PubMed, Scopus, ScienceDirect, IranDoc, Civilica, and SID, from 2014 to July 2025. Fourteen high-quality studies that met the inclusion criteria were identified using relevant keywords.

Results: Three main drivers of key pathophysiological mechanisms in FA have been identified: hormonal dysregulation, impaired inhibitory control, and reward system dysregulation. Two emerging research areas have also been identified: genetic and epigenetic factors and dysregulation of the gut-brain axis. Factors influencing the development of FA include individual, sociocultural, neurobiological, and cognitive factors.

Conclusion: Managing FA requires an interdisciplinary approach targeting its behavioral and biological components. Effective prevention is a public health imperative that depends on a three-pronged strategy of public education, improved food policies, and healthier environments. This integrated approach, which spans subject treatment and population-level intervention, is essential to mitigating the incidence and societal burden of the disorder.

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Introduction

Addiction is a multifaceted neuropsychological disturbance characterized via considerable heterogeneity in its clinical presentation, attributable to the complex interplay of behavioral, biological, and environmental determinants. At a global level, it represents a significant public health burden, with substance use disorders (SUD) alone affecting approximately 243 million people [1-4]. The pathophysiology involves drug-induced neuro-adaptations, including repeated dopaminergic activation, which remodels synaptic and circuit-level architecture within limbic regions over time [5].

While traditionally associated with SUD, the addiction framework is increasingly applied to compulsive behaviors, containing food addiction (FA) [6]. FA is characterized by a persistent craving for and consumption of highly palatable, ultra-processed foods despite adverse consequences [7, 8]. A growing body of research identifies shared neurobiological mechanisms between FA and SUD, particularly regarding reward pathway dysregulation, behaviors such as loss of relapse and control, and ΔFosB expression [9, 10].

The addictive potential of processed foods is theorized to stem from their rapid absorption kinetics, high glycemic load, and enhanced sensory properties, which collectively potentiate their reward value [8]. FA is implicated in the etiology of obesity, with an estimated 24.9% of overweight or obese people exhibiting clinically significant FA signs [10]. Hence, FA is a distinct psychiatric construct, not synonymous with obesity or binge-eating disorder, and is linked to significant mental health and somatic comorbidities [11, 12].

Neurobiological evidence implicates dysregulation across many systems. Neuroimaging research reveal altered activation and connectivity in reward-related regions including insula, striatum, nucleus accumbens, orbitofrontal cortex, and homeostatic centers like the hypothalamus [13]. The underlying neurochemistry involves disruptions in mesolimbic dopamine, glutamatergic, endogenous opioid, and serotonin systems [14-16]. In addition, recent research highlights the role of the gut-brain axis, suggesting specific gut microbiota signatures, for example, alterations in *Blautia* species, might modulate reward processing and present therapeutic targets [17]. Genetic predispositions, such as polymorphisms in dopamine receptor genes, further contribute to people vulnerability [18].

Effective intervention requires a multidimensional approach. Early screening for FA and comorbid anxiety is critical for timely diagnosis [19, 20]. Interventions emphasizing self-control reinforcement and sustainable habit formation are foundational, with emerging mobile health (mHealth) technologies offering scalable platforms for behavior monitoring and staged withdrawal from problematic eating patterns [21, 22].

This article aims to provide a literature review of the multifaceted nature of FA and its intricate links with obesity. The literature review focuses on the neurobiological, psychological, and environmental factors linked to FA. Integrating nutritional neuroscience, behavioral addiction research, and public health perspectives, the review fills gaps in the current literature and establishes a foundation for future interdisciplinary studies on FA.

Materials and Methods

This literature review was conducted according to PRISMA guidelines. An extensive search of international databases (PubMed, Scopus, ScienceDirect, IranDoc, Civilica, Elmnet, SID, and Magiran) was performed for studies published between 2014 and July 2025. Keywords were extracted from the Medical Subject Headings (MeSH) database and supplemented with related terms addressing FA, obesity, dopamine, behavioral addiction, and microbiome.

The search yielded 112 records, of which 35 were duplicates. After removing the duplicates, 77 studies underwent title and abstract screening. Thirty-one articles were evaluated in full, and 14 met the inclusion criteria (Figure 1 and Table 1).

The final studies provided evidence-based insights into the neurobiological, psychological, and behavioral pathways linking FA to obesity (Figure 2).

Inclusion and quality criteria

We included clinical trial studies that specifically assessed FA, as well as studies that examined the behavioral, biological, or psychological aspects of FA and its association with obesity. These studies were published in journals as case reports, cross-sectional studies, or abstracts of congress proceedings. Studies with incomplete data, review articles, case reports, or without peer review were excluded.

Results and Discussion

This study on FA consistently links addictive-like eating behaviors to negative psychological, neurological, and metabolic outcomes (Figure 3).

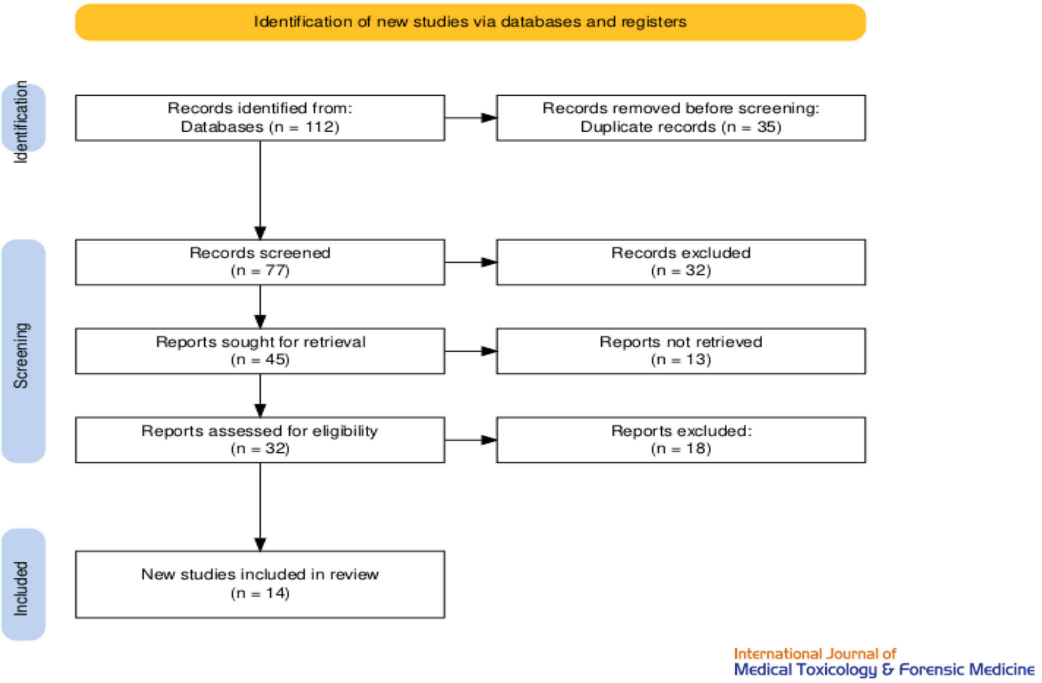

Figure 1. Flowchart of the Study Selection Process According to the PRISMA Guidelines.

Figure 2. Etiology of FA and Obesity.

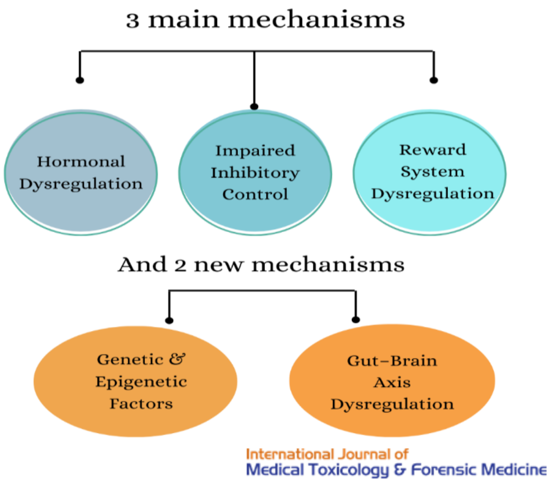

Figure 3. Key pathophysiological 5 mechanisms in FA.

Neurobiological mechanisms in FA

Neurobiological studies have demonstrated a decrease in the density and sensitivity of dopamine D2 receptors in key regions of the mesolimbic system. Highly processed foods activate brain reward pathways similarly to addictive substances, and the dopamine and opioid systems are involved in compulsive eating behaviors [23, 24]. Furthermore, the pathophysiology extends beyond central circuits to encompass bidirectional communication along the gut-brain axis. This axis operates through integrated metabolic, hormonal mechanisms, and neural. A critical component is the microbial secretion of neuroactive metabolites such as short-chain fatty acids and precursors for host neurotransmitter synthesis (e.g., GABA, serotonin), which directly modulate afferent signaling to reward and homeostatic brain centers, thereby regulating food cravings and hedonic tone. Empirical research suggests that targeted modulation of the intestinal microbiota, such as via the administration of specific probiotic formulations, can attenuate these signaling pathways and demonstrably diminish addictive-like feeding behaviors in preclinical studies [17]. In summary, a complex network of neural systems (including mesolimbic dopamine, endogenous opioids, glutamate, orexin, acetylcholine, and serotonin), the gut-brain axis, and hormonal axes (such as leptin) play a key role in the pathophysiology of FA by regulating reward mechanisms, appetite, and energy homeostasis. In other hand, research on FA should expand beyond neural circuit models and consider neuroadaptations, changes in gene expression, and intracellular signaling pathways underlying the transition from hedonic eating to compulsive consumption. For instance, chronic stress, a key risk factor, induces molecular alterations

Table 1. The summarized studies that reported FA.

No	Authors / Country / Year of Publication	Study Type	Population	Main Axis or Intervention	Findings
1	Pape et al. / Germany / 2025 [19]	RCT	Overweight/obese with FA (n=213)	Customized mHealth for Losing weight and psychological aspects of obesity	Weight loss and recovery eating behaviors in FA participants via mHealth
2	Saner et al. / USA / 2025 [29]	Cross-sectional	Binge-FA obese patients (n=37)	TOWARD principles: (e.g., text support, biofeedback, Therapeutic Carbohydrate Reduction)	FA symptoms ↓40.7%, binge eating ↓34.7%; weight improved, meds reduced, mental health boosted, fewer side effects.
3	Sukesh et al. / India / 2024 [30]	Cross-sectional	Adults (n=480)	FA relationship with obesity	Powerful FA-obesity link found.
4	Zielińska et al. / Poland / 2024 [11]	Cross-sectional observational	Overweight/obese adults (n=111)	FA links to physical/mental health, obesity Intensity, depression, inactive	22.7% FA prevalence (higher <50 y [28]ears); FA tied to higher BMI, fat, blood pressure, inactivity (>450 min/day linked to FA/depression); physical activity recommended
5	Samulėnaitė et al. / Lithuania / 2024 [17]	Comparative experimental (mice/humans)	(n=24 mice, 28 humans)	Gut-brain axis in FA: Blautia wexlerae & prebiotic trials	Blautia probiotic reduces FA via gut-brain axis
6	Krupa et al. / USA / 2024 [35]	Review	General population	Neurobiology and clinical characteristics of FA	Ultra-processed foods trigger reward system like drugs
7	Camacho-Barcia et al. / Spain / 2021 [28]	Longitudinal cohort	Overweight/obese adults with metabolic syndrome (n=342)	Metabolic and psychological, status in FA and the impact of weight loss	FA damage psychological/physical health and weight loss outcomes.
8	Mutlu / Ankara / 2021 [31]	Hospital-based study	Obese people (n=202)	FA prevalence in obesity	35% had FA; Rapid diagnosis key for management.
9	Koehler et al. / USA / 2021 [33]	RCT	Overweight/obese (n=83)	FA groups via Yale FA Scale; overweight start age	Groups classified by FA severity and overweight start age.
10	Ravichandran et al. / USA / 2021 [13]	Cross-sectional with resting-state fMRI	General population (n=150)	Brain reward network linkage changes linked to FA in obesity	Reward network disorders drive uncontrolled eating and FA, especially in women.
11	O'Brien et al. / Australia / 2020 [32]	Experimental	Students (n=1220)	FA model (neurobiological) vs. diet/exercise (willpower); control	FA model reduced weight stigma vs. diet/exercise model.
12	Cleo et al. / Australia / 2019 [22]	RCT	Overweight/obese (n=75)	Habit-based interventions for weight loss keeping	Reduced their total body weight by ≥5%
13	Vidmar et al./ USA / 2019 [21]	RCT	Adolescents with obesity (n=50)	Behavioral interventions: app-alone, app-coaching, in-person multidisciplinary	Effects of interventions on FA sign severity in adolescents
14	Raymond KL et al. / Australia / 2017 [12]	Cross-sectional	Type 2 diabetes patients (n=408)	Relationship between severity of processed FA and psychological distress	Depression, anxiety, and stress is higher in type 2 diabetes mellitus with severe PFA

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in the mesolimbic system that may lower the threshold for addictive-like eating by enhancing reward sensitivity and impairing prefrontal inhibitory control [25]. Recent epigenetic studies have identified microRNAs associated with increased vulnerability to FA in both human and animal models, demonstrating the role of post-transcriptional regulation in its pathophysiology [26]. These molecular insights are important because they substantiate FA as a disorder with a biological basis and provide novel biomarkers and potential targets for pharmacological intervention [27]. Consequently, future studies should prioritize high-throughput omics platforms to delineate these molecular determinants and their integration within established neural networks of FA.

Psychological and Individual Factors Influencing FA

Psychological factors, such as anxiety, depression, chronic stress, and poor emotion regulation, contribute to the onset and exacerbation of FA. Multiple cross-sectional and cohort studies have shown that the severity of FA is associated with increased psychological distress in individuals with type 2 diabetes and metabolic syndrome [28]. Functional magnetic resonance imaging (fMRI) studies have confirmed these findings and demonstrated alterations in brain reward network connectivity that may reveal the neural mechanisms of compulsive eating behaviors [13]. Emotional eating in women and poor inhibitory control in impulsive individuals can lead to uncontrollable overeating. Studies show that exposure to high-calorie foods decreases the behavioral control of individuals with FA, leading to intense cravings, loss of control over consumption, and repetitive behaviors that reinforce the cycle of addiction [11, 28].

Challenges and Therapeutic Approaches in treating FA

Promising results have been published in intervention studies. Mobile health (mHealth) interventions and app-based behavioral programs effectively reduce FA symptoms and promote weight loss [19, 21]. Habit-based interventions help maintain weight loss and improve quality of life [22]. The TOWARD metabolic program has demonstrated significant reductions in FA symptoms and binge eating, as well as improvements in metabolic health, without calorie restriction [29]. Psychological interventions, such as cognitive behavioral therapy, have been highly effective in reducing symptoms and improving eating behaviors. Dietary changes that encourage the consumption of less processed foods and regular physical activity play an important role in controlling obesity and reducing addiction symptoms. Early pharmacological research has also produced promising results in reducing cravings and regulating brain reward pathways. Prevention through public education and the creation of healthy environments are also critical components of managing FA. Results suggest that combining cognitive behavioral therapies with mindfulness-based interventions is essential for long-term recovery. Multimodal integrated models have been more successful in reducing relapse rates [11, 30, 31].

The Role of Environmental, Cultural, and Social Factors in the Spread of FA

Widespread access to energy-dense processed foods, pervasive advertising, and sedentary lifestyles contribute to overconsumption and obesity. Social factors, such as high job stress, low social support, and economic insecurity, can increase the risk of FA by reinforcing the use of food as a coping mechanism. Cultural shifts toward convenience and fast food significantly impact this process [32]. Hence, effective public health interventions must transcend the biomedical model of people responsibility. Policy initiatives should prioritize structural reforms, for example implementing fiscal measures (e.g., sugar sweetened beverage taxes), regulating the marketing of hyperpalatable foods to vulnerable populations such as children, deliver culturally sensitive nutritional education, and improving community food environments via access to healthy alternatives. Finally, public health and clinical practice should integrate personalized behavioral strategies, such as cognitive behavioral therapy (CBT) for stress management and habit reversal training that equip subjects to navigate this toxic environment, thereby mitigating the population-level spread of FA [31].

Prevalence of FA in the General Population and Among Individuals with Obesity

Studies using the Yale Obesity Scale 2.0 indicate that obesity prevalence is 5-10% in the general population and 15-25% in obese individuals. Using the Yale Food Addiction Scale (YFAS 2.0) for diagnosis has identified the degree of FA and revealed the diversity of individuals with the disorder [33]. Also, this questionnaire can be used to study FA in Persian non-clinical populations [34]. This disorder is approximately 25-30% more prevalent in women. These gender differences are influenced by biological, psychological, and sociocultural factors [35]. Existing diagnostic tools need to improve sensitivity and accuracy, particularly in clinical care. This study provides a comprehensive, multidimensional view of FA, offering a better understanding of this complex disorder. Studies confirm that FA exacerbates obesity and its comorbidities through intertwined psychological, neurological, and metabolic pathways.

Most studies focus on specific populations, such as obese, diabetic, or adolescent individuals. This limits the generalizability of the results. Further investigation of neurobiological mechanisms using larger, longitudinal designs is required. These limitations may affect the interpretation and applicability of the findings. To elucidate these mechanisms, future research integrating neuroimaging with behavioral and gut microbiome data is recommended. Interventional studies with longer follow-ups and more diverse populations are needed to confirm effective treatments. Clinical applications should consider personalized approaches based on biopsychosocial factors.

Conclusion

Multidimensional interventions, including CBT and mindfulness, as well as targeted therapies that affect the gut-brain axis and genetic-based approaches, have shown impressive results in controlling symptoms of FA. Effective treatment requires an interdisciplinary approach, while prevention necessitates public education and dietary policy reform. Future research could examine these approaches long-term effects on patients' quality of life.

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Ethical Approval

This article is a review with no human or animal sample.

Conflicts of Interest

The authors report there are no competing interests to declare.

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