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Review

The impact of food addiction on the treatment of eating disorders and obesity: A systematic review

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ABSTRACT

Food addiction (FA) is a complex clinical condition that refers to addiction to highly palatable foods, represented by compulsive eating behavior and an incapacity to control food consumption, similar to other forms of addiction. This review examines the literature on FA and its impact on eating disorders and obesity. Using databases, such as PubMed, Cochrane Library and Google Scholar, recent studies were analyzed to show how FA may reduce treatment effectiveness, increase symptom severity, promote resistance to nutritional or pharmacological interventions, and elevate the risk of relapse.

The search strategy used the keywords 'food addiction', 'obesity', 'eating disorders', 'psychotherapy', and 'dietary therapy', limiting the reference period to studies published in the last five years. In reviewing the available articles, several nuances emerged that are fundamental to understanding FA, including neurobiological mechanisms, psychiatric comorbidities, environmental determinants, alterations in the gut microbiota, and the pervasive influence of ultra-processed foods. Taken together, the data indicate that FA not only intensifies symptom manifestation but also contributes to worse outcomes, with reduced compliance to standard treatments and an increased likelihood of relapse. These observations underscore the importance of recognizing FA as a critical component in clinical practice; neglecting its role and symptoms may compromise therapeutic efficacy. Further research is needed to establish integrative treatment models that include FA as a fundamental component of clinical patient care.

1. Introduction

Food addiction has attracted considerable attention. When it occurs in comorbidity with an eating disorder or clinical obesity, FA exacerbates symptoms and worsens prognosis [1,2].

Nosographic validation is still debated, as FA is characterized by a series of complex variables that make clinical and diagnostic definition difficult [3]. The interactions between biological and psychological mechanisms make it difficult to classify FA within the diagnostic framework of addictions. The growing availability and increasing consumption of ultra-processed foods (UPFs) and the role of gut microbiota (GM) alteration in regulating eating behavior and its response to such foods play a central role in the clinical picture of FA [4]. The significant overlap in symptoms with eating disorders and obesity contributes to the complexity of its diagnostic validation [4].

Characteristic symptoms of FA include: spasmodic and prolonged consumption, craving, loss of control, and numerous and frequent attempts to reduce consumption. These symptoms are shared with substance use disorders (SUD). In addition, continued consumption of UPFs, despite psychological and physical consequences, deterioration in social

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and occupational functioning, and the presence of tolerance and withdrawal symptoms (such as anxiety and irritability) are other characteristic symptoms of the clinical picture of FA [5].

Tests have been developed and validated to assess FA symptoms in children and adults, including the Yale Food Addiction Scale (YFAS) and its latest versions (mYFAS, YFAS 2.0, and YFAS-C) [6,7].

In order to better understand the etiopathogenesis of FA, it is necessary to distinguish between the concepts of hunger and food craving: even though they can coexist, they are distinct phenomena and cannot be superimposed [8]. Hunger is defined as the absence of satiety and can be alleviated by eating food. Food craving is defined as an excessive and uncontrollable need for a specific food, in which the senses and emotional factors play a central role [9]. Food craving involves cognitive responses, such as intrusive and recurring thoughts, physiological responses, such as salivation, and the urge to obtain that food, behavioral responses, such as actively seeking and consuming it, and emotional responses, such as irritability and anxiety [9].

Although a transient phenomenon, it shows stable individual differences in its frequency and intensity, sometimes referred to as 'characteristic food cravings' [9].

The clinical relevance of food craving and its associated behaviors becomes particularly evident when examining FA in eating disorders. FA is common in eating disorders, particularly binge-eating disorder and bulimia nervosa, conditions associated with greater clinical severity characterized by impulsivity, emotional dysregulation and frequent episodes of binge-eating [10].

Moreover, even in anorexia nervosa, especially in the binge-purge subtype, symptoms indicative of FA can be present, despite the overall restrictive eating patterns [11].

Due to the scarcity of focused clinical studies on FA, definitive therapeutic recommendations are currently lacking.

The overlap of FA symptoms with obesity and eating disorders poses a challenge for diagnosis, treatment, and clinical course. Establishing a specific symptom profile for FA is essential for improving therapeutic interventions and clinical outcomes [12].

Eating behavior has many nuances, and it is important not only what we eat, but also the timing of food intake, which can influence biological rhythms.

Alterations in the circadian system are associated with increased health risks and the pathogenesis of many diseases [13]. The circadian system regulates most of the body's physiological and behavioral processes, including eating behaviors [14]. It may be modulated by meal times, and eating late at night or at irregular times may affect metabolism and increase the risk of obesity [15,16]. The suprachiasmatic nuclei of the hypothalamus may act as a central clock in the circadian system, but there are also peripheral clocks in the body, liver, pancreas, and intestine [17]. The CLOCK, BMAL1, PER, and CRY genes may play an important role in peripheral clocks and coordinate metabolic processes in the body [18]. This dysregulation of eating behavior may lead to a misalignment between peripheral clocks and the central clock [18]. Genetic and environmental alterations of the circadian system may lead to increased vulnerability to addiction disorders [19]. Recent studies suggested that a disruption of the circadian system may lead to low mood, an increase in unhealthy food preferences and a higher risk of developing FA [20,21]. A recent study [22] suggested that in a sample of individuals residing in Arctic regions, excessive exposure to artificial light may lead to alterations in the circadian system and in the secretion of hormones that regulate sleep and appetite. These alterations have been associated with an increase in eating disorders and a higher risk of FA. This pilot study suggests that alterations in physiological rhythms contribute to an increased risk of eating disorder. In order to effectively treat FA, it is essential to take into account the circadian system and its effects on the body. In summary, meal timing may also be a key factor in FA. Eating at night or at irregular times may facilitate dysfunctional eating behaviors associated with addiction, reducing the effectiveness of clinical treatment and leading to a poorer prognosis.

The aim of this systematic review was to highlight FA as a distinct clinical entity, characterized by specific neurobiological mechanisms and behavioral patterns that allow it to be distinguished from other clinical conditions.

The identification of FA in obesity and eating disorders is necessary to make a correct diagnosis and expand more effective therapeutic interventions. Recent studies have found that a disruption of the circadian system may lead to low mood, higher dropout rates and ineffective psychotherapeutic and dietary treatment. This review emphasizes the need for a multidisciplinary approach with the aim of optimizing prognosis and clinical courses.

2. Materials and methods

The bibliographic search for the review was executed in August 2025 on several databases, specifically PubMed, Cochrane Library, and Google Scholar. The objective was to analyze studies published in the last five years (2021–2025). To maximize the sensitivity and specificity of the search, customized strings were constructed around predefined keywords, such as 'food addiction', 'obesity', 'eating disorders', 'psychotherapy', and 'diet therapy'. At each stage of the selection process, three reviewers independently evaluated the search results and reached a consensus on inclusion. The entire process was conducted in accordance with the recommendations of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) method [23]. The review was conducted and reported according to PRISMA 2020. We also used the REAPPRAISED checklist as a structured tool to identify and record potential publication-integrity red flags in the included literature, where applicable [24].

The reviewers independently screened the titles and abstracts of all identified articles to ensure that they met all of the following inclusion criteria: english language, analysis of food addictions and their treatment in eating disorders and obesity. Only articles available free of charge in full text were included. The flow chart for study selection is shown in Fig. 1.

This figure shows the study selection process, highlighting the number of articles identified, those excluded, and the final articles included in the review.

3. Results

The database search yielded 43 records. After removal of 3 duplicates, 40 records were independently screened by title and abstract, and 14 were excluded; 26 full-text articles were assessed for eligibility; 4 were excluded with reasons, resulting in 22 studies included in the review.

The included articles are:

Reviews: 15 articles

Scoping reviews: 1 article

Systematic reviews: 1 article

Systematic reviews and meta-analyses: 5 articles

From the research conducted, 4 reviews comprehensively examine the relationship between UPFs consumption and FA [25–28], 5 reviews highlighted the association between eating disorders, obesity, and FA [29–33], 4 reviews focus on the neurobiological mechanisms underlying FA and related eating behaviors [34–37], 2 reviews focused on analyzing the presence of FA in children and adolescents [38,39], 1 review explored the impact of impulsivity in FA [40] and 6 reviews focused on the treatment of FA [41–46] (Table 1).

4. Discussion

4.1. Definition and characteristics of food addiction

FA is increasingly recognized as a clinically relevant condition, supported by growing scientific evidence regarding its role in eating

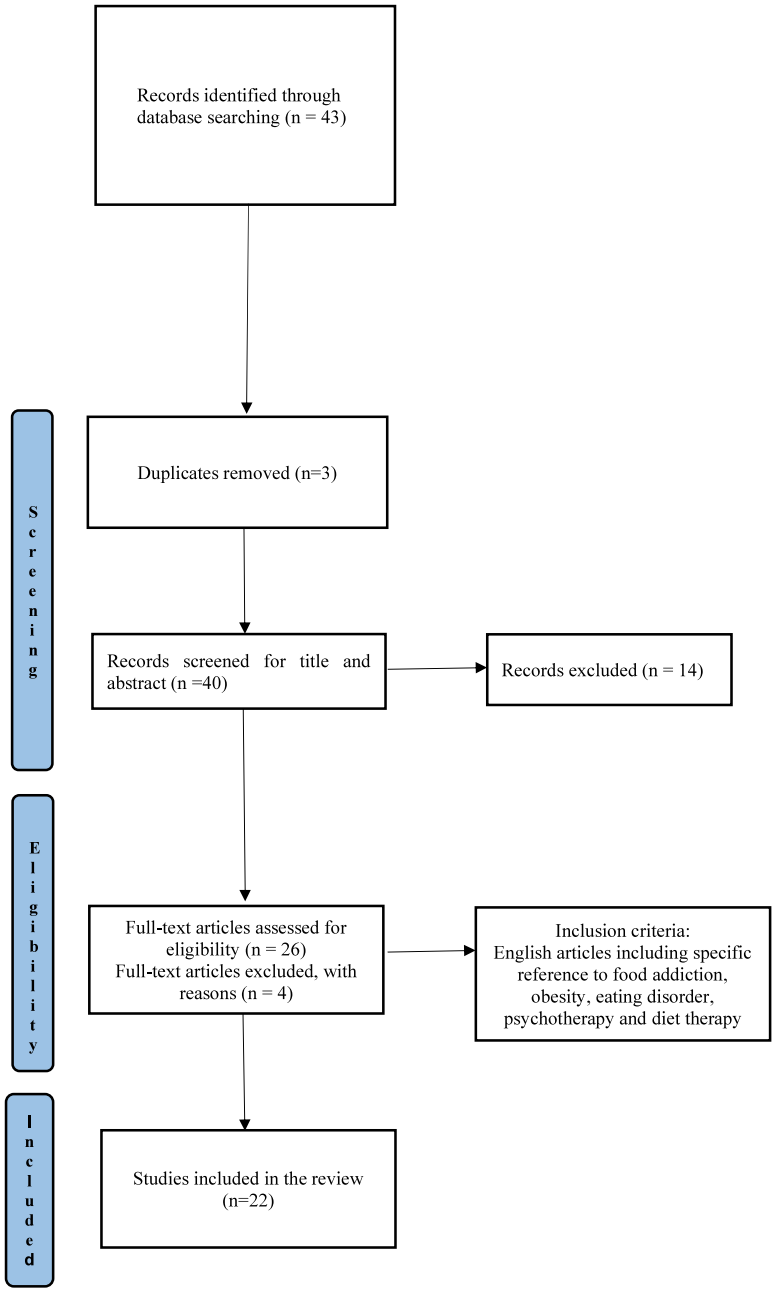

Fig. 1. Study selection process highlighting the number of articles identified, those excluded, and the final articles included in the review.

disorders and treatment approaches. FA encompasses features from several disorders: eating disorders (e.g., loss of control over food intake), SUDs (e.g., cravings and continued use despite harmful consequences), obsessive-compulsive traits (e.g., intrusive thoughts about food), and impulsivity. It is also associated with psychological (e.g., depression, low self-esteem), medical (e.g., diabetes, obesity, cardiovascular diseases), and social (e.g., weight-related stigma) issues [6].

Addiction involves a substance's capacity to alter the brain's reward system, triggering compulsive behaviors and specific neurobiological changes. UPFs share these effects, as their composition strongly stimulates reward pathways, driving cravings and loss of control similar to substance addiction [47]. The term "UPF" originates from the NOVA Food Classification System, the widely-known method used to categorize foods by processing level. UPFs are highly palatable, ready-to-eat, and typically inexpensive industrial products that accelerate poor health outcomes [48]. These include products marketed as healthy, such as vegan, light, organic, or gluten-free items and not only typical "junk foods" [48]. The mechanisms linking UPFs to health effects likely involve both their nutritional composition (generally high in energy, added sugars, saturated and trans fats, and sodium) and the consequences of processing. UPFs tend to be poorly balanced nutritionally, low in protein, fiber, and essential micronutrients, such as potassium, magnesium, phosphorus, zinc and vitamins (C, B12, D and niacin) [49].

Wiss and LaFata [25] highlight emerging evidence that links UPFs to depression and addiction-like eating behaviors, especially in binge-type eating disorders. They estimate that 14–20 % of the population may present symptoms of UPF addiction (UPFA), and argue that standard eating disorder treatments often overlook the unique metabolic and psychological effects of UPFs, neglecting their addictive properties [25]. They also highlight that UPF marketing disproportionately targets low-income and minority populations, compounding mental health risks. Consequently, they challenge the prevalent "all foods fit" model in eating disorder treatment and advocate for personalized interventions considering food processing and addictive potential, particularly in

Table 1
Summary of studies considered in the systematic review.

Authors	Title	Type of Article	Date	Findings
Wiss, DA; et al. [25]	Ultra-Processed Foods and Mental Health: Where Do Eating Disorders Fit into the Puzzle?	Review	2024	UPFs may exacerbate psychiatric symptoms, especially in major depressive disorder and eating disorders.
LaFata, EM; et al. [26]	Ultra-Processed Food Addiction: A Research Update	Review	2024	UPF addiction has prevalence rates, especially in the obese population. Further research into the treatment of UPF addiction would lead to better clinical outcomes.
Parnarouskis L. et al. [27]	Withdrawal: A key consideration in evaluating whether highly processed foods are addictive	Review	2022	The negative physiological and psychological symptoms of withdrawal occur in response to the reduction or cessation of substance use and plays a central role in addiction.UPF causes withdrawal-like symptoms in animals and humans but further research is needed to evaluate this clinical construct.
Santos, GCJ; et al. [28]	Dietary intake in children and adolescents with food addiction: A systematic review	Review	2024	During adolescence and childhood, FA is characterized by the consumption of UPFs and can contribute to increased obesity and metabolic disorders Nutritional strategies tailored to address addictive-like eating patterns are important for this population.
Di Giacomo, E; et al. [29]	Disentangling binge eating disorder and food addiction: a systematic review and meta-analysis	Systematic Review and Meta-Analysis	2022	FA is a distinct diagnosis from eating disorders and may have prognostic significance.
Ratković, D; et al. [30]	Comparison of binge-eating disorder and food addiction	Review	2023	BED and FA share many characteristics, but they also have distinctive traits, and correct differentiation increases effectiveness in treatment
Pereira, T: et al. [31]	Association between ultraprocessed foods consumption, eating disorders, food addiction and body image: a systematic review	Systematic review	2024	Body image distortion is an important risk factor in eating disorders, but no correlation with FA has been found.
Halbeisen G. et al. [32]	The prognostic role of food addiction for weight loss treatment outcomes in individuals with overweight and obesity: A systematic review and meta-analysis	Systematic Review and Meta-Analysis	2023	In the clinical population of obese and overweight patients failure to lose weight and compromised therapeutic adherence may be due to FA.
Jahrami, H; et al. [33]	A meta-analysis assessing reliability of the Yale Food Addiction Scale: Implications for compulsive eating and obesity	Review	2024	The YFAS is reliable in assessing FA in eating disorders and obesity.
Mestre-Bach, G; et al. [34]	Potential Biological Markers and Treatment Implications for Binge Eating Disorder and Behavioral Addictions	Review	2023	Alterations in ventral striatum activity could play in FA a decisive role like biomarkers for evaluating treatment outcomes.
Yu, Y; et al. [35]	Gut Hormones, Adipokines, and Pro- and Anti-inflammatory Cytokines/Markers in Loss of Control Eating: A Scoping Review	Scoping Review	2021	Altered levels of ghrelin, leptin and adiponectin may play a function in LOC eating and act as potential therapeutic targets, although further studies are needed to establish causal and treatment pathways.
Stuber, GD; et al. [36]	The neurobiology of overeating	Review	2025	Hyperpalatable foods are shown to induce synaptic plasticity in mesolimbic pathways, mirroring mechanisms seen in addiction. GLP-1 receptor agonists are identified as promising therapeutic agents because of their ability to reduce food intake and modulate neural activity related to reward.
Mehr, JB; et al. [37]	Sleep dysregulation in binge eating disorder and "food addiction": the orexin (hypocretin) system as a potential neurobiological link	Review	2021	Poor sleep quality may aggravate dysregulated eating behaviour. The orexins could play a role in FA, and their pharmacological treatment could be effective in treating eating symptoms.
Yekaninejad, MS; et al. [38]	Prevalence of food addiction in children and adolescents: A systematic review and meta-analysis	Systematic Review and Meta-Analysis	2021	During adolescence and childhood, FA was found to be more prevalent in obese individuals than in those of normal weight.
Bektaş, M; et al. [39]	The Effect of Food Addiction in Children on Obesity: A Systematic Review and Meta-Analysis Study	Systematic Review and Meta-Analysis	2021	FA has a major impact on childhood obesity.
Gaspar-Pérez, A; et al. [40]	Food Addiction and Impulsivity in Clinical Populations by Gender: a Systematic Review	Review	2023	FA is characterized by high impulsivity, with a tendency to seek immediate gratification through food. Revealed studies have gender differences in FA.
Silvestrini B, Silvestrini M [41]	Physiopathology and Treatment of Obesity and Overweight: A Proposal for a New Anorectic	Review	2024	Different treatment paths for the treatment of obesity and FA are described: diet therapy, bariatric surgery and pharmacological treatment. Expanding pharmacological treatment options for obesity offers new therapeutic possibilities to improve weight management and health outcomes.
Novelle, MG; et al. [42]	Decoding the Role of Gut-Microbiome in the Food Addiction Paradigm	Review	2021	The gut microbiome regulates eating behavior with the gut-brain axis. Interventions targeting could gut microbiota have potential therapeutic benefits in the treatment of FA.
Frank, J; et al. [43]	Brain–Gut–Microbiome Interactions and Intermittent Fasting in Obesity	Review	2021	Alterations in the gut microbiota lead to changes in eating behavior. Treatment of this system could be effective in the treatment of obesity.
Dyńka, D; et al. [44]	Ketogenic Diets for Body Weight Loss: A Comparison with Other Diets	Review	2025	Analysis of the effectiveness of ketogenic diets has been compared to other nutritional approaches for weight loss, metabolic improvement, and appetite regulation.
Ames G.E. et al. [45]	Behavioral Interventions to Attenuate Driven Overeating and Weight Regain After Bariatric Surgery	Review	2022	Eating behavior influenced by emotional dysregulation, reward-seeking and after bariatric surgery, deficits in executive functions are associated with an increased risk of regaining the weight initially lost due to the presence of FA and loss of control in eating.
Taba, JV; et al. [46]	The Development of Feeding and Eating Disorders after Bariatric Surgery: A Systematic Review and Meta-Analysis	Systematic Review and Meta-Analysis	2021	The prevalence of FA was found to be in patients after bariatric surgery, with evidence on risk factors and clinical implications.

Abbreviations: YFAS, Yale Food Addiction Scale BED, binge eating disorder; FA, food addiction; GLP-1, glucagon-like peptide 1; LOC, loss of control; UPF, ultra-processed foods.

binge-spectrum disorders [25].

A related study by LaFata et al. [26] estimates global UPFA prevalence at 14 % in adults and 15 % in adolescents, with higher rates in individuals with obesity (28 % in adults and 19 % in youth). Chronic UPF consumption has been linked to alterations in dopamine signaling, appetite — regulating hormones, and GM — factors associated with compulsive eating and increased obesity risk [26]. The authors also highlight potential withdrawal symptoms following UPF reduction, similar to those observed in SUDs. UPFA encompasses hallmark addiction features, including craving, tolerance, and persistent use despite negative consequences, while also manifesting in non-binge behaviors such as grazing. The addiction model better explains maladaptive eating patterns and underscores the need for targeted treatments, given the poor response of UPFA individuals to standard weight-loss programs [26].

Parnarouskis et al. [27] examined evidence suggesting that highly palatable (HP) foods may induce abstinence-like responses consistent with addiction models. In SUDs, such syndromes, characterized by physical, cognitive, and emotional symptoms, predict relapse and represent key therapeutic targets. Similarly, adverse reactions to UPF reduction may contribute to poor adherence and limited success of dietary interventions [27]. Both animal and human studies demonstrate behavioral and biological changes consistent with withdrawal following HP food reduction. Animal data suggest a time course similar to addictive substances, while human evidence relies mostly on retrospective self-reports, highlighting the need for prospective, controlled research [27].

Supporting this perspective, Jurema Santos et al. [28] found that children and adolescents with FA exhibit distinct dietary patterns characterized by high UPF consumption, reinforcing the potential link between FA and excessive UPF intake from early life stages.

4.2. Implications of food addiction for obesity and eating disorder

FA is becoming increasingly relevant in eating disorders and obesity, a subpopulation that could benefit from targeted therapeutic strategies. Rather than being subsumed under pre-existing disorders, FA appears to represent a distinct clinical entity with its own unique characteristics, distinct from pre-existing disorders. Therefore, it is crucial that FA be properly recognized and systematically studied to support the development of personalized treatments capable of effectively addressing its symptoms and improving clinical outcomes.

FA can be considered a transnosographic construct observed across various psychopathological conditions, including eating and nutrition disorders [50], and obesity [51].

Identifying FA is important, as it shows significant diagnostic overlaps with other eating and nutrition disorders, as well as obesity [6].

Jahrami et al. [33] analyzed the role of FA in eating disorders and obesity, with a particular focus on the validity and reliability of the YFAS and its variants (YFAS 2.0, mYFAS, YFAS-C). These tools showed high consistency and robustness in detecting FA, suggesting their reliability as screening tools.

Di Giacomo et al. [29] emphasized that FA can be a negative prognostic factor in eating disorders and often occurs in conjunction with binge eating disorder (BED) and bulimia nervosa. FA and BED share some symptoms, such as loss of control, persistence in engaging in the eating behavior even in the presence of negative repercussions, and frequent unsuccessful attempts to reduce consumption, which can complicate differential diagnosis. Nonetheless, FA is more strongly characterized by addiction-like mechanisms, whereas BED is more firmly situated within the spectrum of eating disorders, highlighting the need for precise clinical assessment and individualized interventions [29]. FA also overlaps with bulimia nervosa, sharing patterns of compulsivity, impaired reward processing, and reduced inhibitory control, resembling substance use disorders [29]. In anorexia nervosa, restrictive eating may take on addictive characteristics, with individuals developing tolerance to the psychological and physiological effects of fasting, and experiencing withdrawal-like symptoms upon refeeding [29]. FA symptoms have been found to be more prevalent in anorexia nervosa the binge-purge subtype (AN-BP) than in the restrictive subtype (AN-R), possibly due to the presence of binge-eating behaviors, which mirror those observed in FA [10]. Similarly, Ratković et al. [30] reported overlapping features between BED and FA, including eating when not hungry, emotional instability, and loss of control. However, they also underscore the distinctive addiction-related features of FA — such as craving, tolerance, withdrawal, and low guilt — which differentiate it from BED. Recognizing these subtle differences is critical for tailoring treatment strategies. Cognitive-behavioral therapy (CBT), while effective for BED, may require adaptation to address the addiction components of FA [30].

Pereira et al. [31] sought to investigate a possible correlation between the consumption of ultra-processed foods (UPFs), eating disorders, FA and body image. No relevant associations emerged among the variables considered in the systematic review. Significant gaps in the literature have been identified. Although some associations between these variables have been found, the studies considered have methodological limitations. More in-depth research is needed to understand the causal relationships between the variables [31].

There is a bidirectional relationship between eating disorders and circadian rhythm disruption 16[16]. The circadian system may influence eating behavior through the interaction of the suprachiasmatic nuclei of the hypothalamus, which receives signals from light (day/-night), with food chronobiology [16]. Several peripheral biological clocks in the body can coordinate with the central clock and determine the times of day when it is most efficient to eat [52]. Eating at irregular times can lead to circadian desynchronization and metabolic alterations, increasing the risk of obesity, diabetes, and eating disorder [52,53]. Food quality and quantity can influence circadian system functioning, especially peripheral clocks. Ultra-processed foods (UPFs) and calorie restriction may affect the gene expression of circadian rhythms, causing changes in metabolic and hormonal rhythms [53]. Circadian desynchronization may have significant effects on mental health and can be considered a risk factor for psychiatric disorders, such as eating ones, major depressive, bipolar, anxiety, addiction, psychotic and sleep disorders [54]. Binge eating and night eating disorder are characterized by daytime and night-time loss of control overeating, that may lead to alterations in the circadian system and metabolism, creating a vicious cycle in which circadian alteration may lead an exacerbation of loss of control overeating [55]. Eating behavior may be regulated by the metabolic system, the circadian and the reward system, and a misalignment of these three systems may lead to a dysregulation of eating behavior, greater vulnerability to consuming ultra-processed foods and engaging in addiction-like eating behaviors [56]. Understanding the link among circadian rhythms, eating disorders and food addiction is essential in order to tailor nutritional and psychotherapeutic interventions, optimising diet therapy, improving sleep and synchronising the use of psychotherapeutic strategies with circadian rhythms.

Obesity is a chronic condition, and multiple factors can influence its development. FA can also be observed in a specific phenotype of overweight and obese patients characterized by impairment in cognitive functioning and difficulty in impulse control, psychopathological symptoms, a less regular diet, and frequent episodes of emotional eating [7].

Halbeisen et al. [32] aimed to investigate whether FA could compromise the effectiveness of weight loss treatments by reducing adherence to interventions and promoting weight regain. The severity of

FA symptoms predicts less weight loss among patients, and the impact of FA appears to vary among ethnic groups due to lack of access to healthy foods [32]. Although FA has been widely studied, the role of cultural factors and individual characteristics in influencing treatment outcomes remains unclear. In summary, further research is required to understand the prognostic role of FA in eating disorders and obesity in order to improve treatment strategies [32].

FA can coexist with a variety of dysfunctional eating behaviors, reflecting its complexity: it is not limited to a single behavioral pattern, but manifests through multiple forms of pathological eating behavior. FA may represent a clinical feature that exacerbates difficulties in regulating eating in a healthy way and worsens the clinical picture, complicating symptom management. Therefore, an accurate assessment of FA is essential to plan targeted and comprehensive interventions in order to optimize clinical outcomes.

4.3. Neurobiological foundations and therapeutic perspectives based on neurobiology

Food consumption and energy expenditure are regulated by complex and distributed neural networks involving thousands of genes and several key brain regions, including the hippocampus, insula, amygdala, striatum, and orbitofrontal cortex [57]. These regions are central to the learning and processing of food-related rewards, as well as to the allocation of attention and effort toward their acquisition. The mesolimbic reward system facilitates the physiological and cognitive encoding of reward, promoting behaviors aimed at securing pleasurable stimuli, thereby promoting goal-directed behaviors aimed at securing pleasurable experiences [57]. Within this system, hypothalamic neurons, particularly in the arcuate nucleus, play a crucial role in controlling food intake. Melanin-concentrating hormone (MCH) and Orexin, hypothalamic neuropeptides, induce both food intake and reward-driven processes. Moreover, other mediators within the arcuate nucleus, including neuropeptide Y (NPY), alpha-melanocyte-stimulating hormone, agouti-related protein (AgRP), and γ-aminobutyric acid (GABA), contribute to energy homeostasis [57]. Peripheral signals are combined into this central system through the gut–brain axis, where insulin, leptin, ghrelin and peptide YY (PYY) convey information about hunger, satiety, and energy status [57]. Ghrelin, secreted by the stomach, increases during fasting and promotes both hunger and food-seeking behavior. In contrast, leptin, secreted by adipose tissue, signals energy sufficiency by inhibiting orexigenic neurons (NPY/AgRP) and stimulating anorexigenic ones, thereby contributing to long-term energy balance. Importantly, ghrelin stimulates dopaminergic pathways, while leptin and insulin exert inhibitory effects [57]. These circuits are relevant in both SUDs and behavioral addictions, where altered brain functioning and dysregulated reward processing have been observed. Similar changes are evident in overeating and obesity, particularly in relation to the motivational properties of food. Mestre-Bach et al. [34] highlighted the role of the ventral striatum (VS) in behavioral addictions, such as internet gaming disorder, gambling disorder, and FA or BED. Highly palatable foods activate reward-related neural pathways comparable to those engaged by addictive substances, especially in individuals with BED. The VS, as a hub of reward processing, is increasingly being studied as a potential biomarker, particularly in relation to pharmacological and psychotherapeutic interventions [34]. Biomarker studies by Yu et al. [35] reported that individuals with BED and LOC eating exhibit distinct profiles, including lower fasting levels of acyl ghrelin and adiponectin, and higher levels of high-sensitivity C-reactive protein (hsCRP), leptin, and erythrocyte sedimentation rate. Altered postprandial responses are also observed, with attenuated ghrelin and cholecystokinin (CCK) activity and amplified PYY secretion [35]. Notably, GLP-1 receptor agonists have demonstrated therapeutic effects reducing binge-eating behaviors and modulating reward-related activity in ventral tegmental area and nucleus accumbens, thereby helping to restore balance between homeostatic and hedonic drives [35]. Expanding on this, Stuber et al. [36] emphasized the dual mechanism of GLP-1 analogs, which not only suppress appetite but also influence neural circuits involved in reward. These agents thus represent a promising therapeutic avenue for treating overeating driven by hedonic dysregulation.

Furthermore, Mehr et al. [37] suggested that BED may reflect a compulsive phenotype, driven by the addictive potential of certain foods. Their findings underscore the parallels with SUDs, including LOC, persistent attempts to abstain, and shared craving-related neurobiology. Crucially, they also draw attention to the often-overlooked role of sleep disturbances, proposing that chronic consumption of palatable foods may hyperactivate the orexin system, thereby disrupting sleep–wake cycles and perpetuating binge-eating episodes [37].

Although overeating and FA exhibit several neurobiological similarities with traditional addictions, they should not be considered synonymous. Rather, they exist along a continuum of reward system dysregulation, in which both neural and hormonal imbalances contribute to maladaptive eating behaviors [36].

4.4. Integrative therapeutic approaches in the management of food addiction

FA is a complex disorder involving biological, genetic, psychological, and social factors, and therefore requires an integrated treatment approach within a biopsychosocial model, combining psychotherapy, pharmacotherapy, and diet therapy.

FA is not limited to a specific population and can occur in children and adolescents as well as in adults, often in conjunction with problems such as overweight and obesity. Yekaninejad et al. [38] described how the prevalence of FA is also significantly high in children — adolescents with higher estimates among those with overweight/obesity with an estimate that is comparable to that of adults. Young people may share comparable risks for developing FA as adults and specific attention to this group is essential to prevent or treat this clinical condition in this at-risk population. Bektaş et al. [39] showed that FA in children is significantly associated with increased obesity, confirming that excessive consumption of highly palatable and processed foods contributes to compulsive eating behaviors and a higher risk of being overweight.

Gaspar et al. [40] described how impulsivity plays a crucial role in the development and maintenance of FA. Impulsivity is defined as the tendency to seek immediate rewards without considering the long-term consequences, and the impulsive trait in FA is articulated by a tendency to seek immediate gratification through UPFs without thinking about the negative effects. A significant association has been found in clinical samples between impulsivity and FA. Some studies suggest that impulsivity may manifest differently between men and women, but gender differences in this relationship are still being studied and need further investigation. Impulsivity difficulties favor excessive intake of UPF, as the inability to control impulses can lead to immediate unhealthy food choices, such as the consumption of highly palatable foods [40]. Understanding these relationships could improve personalized therapeutic treatments, allowing for the development of targeted interventions that address both the psychological and behavioral aspects of impulsivity, as well as those related to UPF selection. This would allow more effective interventions, improving the regulation of eating behavior and supporting more functional management.

Pharmacological strategies have also shown promise. GLP-1 receptor agonists, such as liraglutide and semaglutide, have demonstrated efficacy in reducing appetite and modulating the brain's reward system, thereby attenuating cravings for hyper-palatable foods, as suggested by Silvestrini et al. [41].

These drugs not only improve satiety but also directly influence neural circuits associated with motivation and pleasure, providing a dual therapeutic action. Another proposed pharmacological innovation is the combined use of glucose and tryptophan administered sublingually. This formulation may facilitate rapid crossing of the blood–brain barrier, influencing central hunger and reward circuits. However,

scientific evidence is limited and further scientific studies are needed [41].

Glucose serves as an energy substrate for the brain, while tryptophan, a precursor of serotonin, helps reduce food-related compulsive behaviors and promotes homeostatic regulation of appetite. In addition to these options, lipase inhibitors (e.g., orlistat) and other neuromodulators are being studied for their potential in treating FA. However, their side effects and variability in individual responses remain significant limitations [41].

Chronotherapy is a therapeutic strategy that optimizes the efficacy of drug treatments by taking into consideration the body's circadian rhythm [58]. The efficacy of drug treatment and the incidence of adverse effects may be influenced by the time of day the treatment is administered in relation to the body's circadian rhythms [58]. Chronotherapy applied to pharmacokinetics and pharmacodynamics may maximize the effectiveness of treatment and reduce adverse effects [58].

The circadian system may modulate drug absorption, distribution, metabolism, and elimination, as well as the body's response to drugs [59]. Hepatic and extrahepatic enzymes and the gut microbiota are biotransformation pathways that may determine how long a drug remains in an organism and influence biological response [60]. These pathways may be regulated by the circadian system, and the hour at which a drug may be taken is critical to its effectiveness [60]. The timing of drug intake may influence therapeutic efficacy and drug toxicity [61]. In addition to choosing the drug, it may necessary to carefully select when to administer it in order to minimize side effects [61]. Organ systems responsible for drug metabolism, such as the liver and kidneys, may be influenced by the circadian system and may metabolize a drug faster or slower depending on the time of administration [58]. These fluctuations may make timing of intake crucial to maximize efficacy and reduce side effects [58]. A healthy circadian rhythm is linked to good metabolic [53] and hormonal health [62]. The quality and quantity of food consumed, exposure to sunlight, physical activity and meal times may have a significant impact on the circadian system [53]. Chrononutrition may have an important role on health, and a programmed diet may have positive effects on metabolic health [53].

Tryptophan may be a key role in the connection between nutrition and the circadian system [63]. Tryptophan is an essential amino acid present in many foods and is a precursor of melatonin and serotonin The availability and timing of tryptophan intake can significantly impact mood, the circadian system, and appetite regulation, and has significant implications for weight management, eating disorders, and FA [63]. Tryptophan metabolism is not static and may be influenced by the day/night cycle and light [64]. Serotonin and melatonin, derived from tryptophan, may play different roles and act at different times of the day in regulating physiological processes [64]. Serotonin functions during wakefulness, while melatonin is synthesized at night and plays a key role in the sleep-wake cycle [64]. Tryptophan metabolites may play a key role in regulating the circadian functioning of the central clock in the suprachiasmatic nucleus and the peripheral clock in the liver [64]. Consuming tryptophan at specific times of day may have many positive effects on sustaining circadian rhythms [64]. Knowledge of these mechanisms may be important in understanding eating disorders and FA.

Behavioral therapies remain a cornerstone in FA management. Acceptance and Commitment Therapy (ACT) focus on accepting negative emotions without resorting to food as a compensatory mechanism, whereas Cognitive Behavioral Therapy (CBT) aims to strengthen inhibitory control and restructure maladaptive eating patterns. Techniques such as mindful eating and craving-management strategies (e.g., urge surfing) have also proven useful in enhancing self-regulation and reducing impulsive consumption [45].

Dietary approaches can contribute significantly to addressing FA by modulating the gut-brain axis and improving metabolic and neurobiological responses. As pointed out by Novelle et al. [42], the gut microbiota plays a central role in regulating appetite and reward mechanisms. Alterations in microbial composition induced by diets with a predominance of ultra-processed foods can disrupt the production of metabolites involved in satiety and reinforcement pathways, thereby supporting addiction-like behaviors. Conversely, targeted dietary approaches, such as eating plans designed to be high in prebiotic fiber, promote the production of short-chain fatty acids (SCFAs), such as propionate and butyrate, that not only regulate satiety hormones, such as GLP-1 and PYY, but also influence dopaminergic neurotransmission, reducing dysfunctional eating behaviors and compulsive food cravings [42]. The available results indicate that the well-being of the gut microbiota is an important therapeutic target for nutritional approaches in FA. In this regard, some dietary approaches that differ from the more conventional Mediterranean diet have been studied as possible dietary strategies [42]. For example, intermittent fasting (IF) has been highlighted as a nutritional strategy capable of influencing the brain-gut-microbiome interaction [42]. Frank et al. [43] confirmed that fasting programs can contribute to the alignment of the circadian rhythm, attenuating systemic inflammation and promoting changes in the gut microbial communities, thus improving metabolic flexibility and appetite regulation. In addition to these metabolic outcomes, IF is believed to act on neural circuits involved in reward processing through signals derived from the gut microbiota, which could mitigate cravings and compulsive eating behaviors [43]. These mechanisms suggest that IF may serve as an additional option for FA, simultaneously acting on the homeostatic and hedonic pathways of food intake [43].

Ketogenic diets have also recently received attention as a possible intervention for weight reduction while also controlling symptoms of compulsive eating. Dyńka et al. [44] compared ketogenic diets with other nutritional approaches, demonstrating that a diet low in carbohydrates, or sometimes zero carbohydrates, and high in fat can be effective in promoting weight loss and improving metabolic parameters, stabilizing appetite regulation. These effects appear particularly relevant in FA, as the total exclusion of carbohydrate-rich and hyper-palatable foods may reduce exposure to triggers of compulsive cravings and loss of control. However, uncertainties remain regarding the long-term adherence and safety of the dietary plan that disrupts metabolic circuits in favor of fats, indicating that ketogenic diets should be considered within a broader and more personalized management framework for FA [44].

Bariatric surgery is another important intervention. As noted by Ames et al. [45], procedures such as Roux-en-Y gastric bypass and sleeve gastrectomy can also act as modulators of compulsive eating behaviors through hormonal and gut microbiota-mediated changes. However, not all patients experience remission of FA. Taba et al. [46] identified FA among the eating disorders that can develop or persist after bariatric surgery, along with compulsive and nighttime eating disorders. Although the demonstrated results have methodological limitations, these data demonstrate the importance of structured assessment tools and multidisciplinary management strategies for addressing FA in post-surgical populations.

In summary, FA management benefits from a comprehensive, multidisciplinary approach, integrating dietary, pharmacological, and behavioral strategies tailored to individual needs. Early detection and personalized care are essential to improve long-term outcomes and prevent the escalation of compulsive eating behaviors.

5. Conclusions

In conclusion, FA is increasingly identified as a relevant but often overlooked factor in the manifestation, maintenance, and severity of both eating disorders and obesity. As a transdiagnostic element, FA not only acts as a predisposing factor but also maintains and exacerbates the severity of clinical symptoms, thus creating a vicious cycle that complicates therapeutic efforts. Despite the absence of formal recognition in DSM-5 and the lack of unified treatment protocols, a growing body of evidence highlights the clinical relevance of FA. Neurobiological and

psychological studies, particularly those examining the impact of UPF consumption, demonstrate striking parallels between FA and SUD, both of which are driven by a dysregulated reward circuit and impaired self-regulation [65].

The circadian system regulates the sleep-wake cycle and may influence eating behavior [66]. Chronotype, individual's predisposition to be more active at a specific time of day, may modulate the sleep-wake rhythm and food intake [58,66]. Poor circadian hygiene, such as disruption of the sleep-wake rhythm, irregular meal times and inadequate exposure to natural light, may be considered an important factor in maintaining FA and lead to alterations in the circadian system, the metabolic and endocrine systems, increasing the risk of diabetes and obesity [62]. There are various strategies for improving circadian health and increased exposure to daylight, evening melatonin administration, and daytime exercise can lead to better sleep quality, better cardiometabolic and neurological health [13]. Apart from food quantity and quality, the time of food intake may be a risk factor for an individual's overall health [67]. Eating at night and between meals are examples of poor circadian hygiene and synchronizing the timing of food intake with the sleep-wake cycle may optimize cardiometabolic health and may be an effective strategy for preventing disease [67].

Chronopharmacology, a branch of pharmacology that focuses on regulating the timing of drug administration to improve clinical outcomes with fewer side effects, may be a promising opportunity to improve clinical outcomes in multiple diseases [58,68]. Chronopharmacology may be a promising area of clinical interest for personalized medicine and optimization of the treatment of FA should take chronopharmacology into account.

In the treatment of FA, understanding and considering the circadian system and related neurotransmitters may represent an optimization of symptom outcomes and a reduction in the side effects of drugs.

Personalized interventions that integrate medical, nutritional, and psychotherapeutic strategies are essential for interrupting compulsive eating cycles and improving long-term outcomes. It is important to emphasize that such approaches should also consider the broader psychosocial context that contributes to the increased development of FA.

Future studies should prioritize identifying specific neurobiological and behavioral mechanisms underlying FA, with the goal of developing and validating targeted assessment tools and evidence-based treatments. This includes evaluating emerging pharmacological agents, refining psychotherapeutic models, and exploring the role of dietary interventions.

In summary, although research on FA is still in its infancy, a growing body of evidence highlights its relevance in clinical recognition and management, especially in eating disorders and obesity. Moving forward, the establishment of standardized diagnostic frameworks and personalized treatment approaches will be essential to effectively address the rising public health impact of addictive eating behaviors.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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CRediT authorship contribution statement

Anna Laura Amato: Writing – original draft, Formal analysis, Data curation, Conceptualization. **Paola Gualtieri:** Writing – review & editing, Writing – original draft, Conceptualization. **Michela Cirillo:** Writing – original draft, Formal analysis, Data curation. **Giada La Placa:** Writing – original draft, Formal analysis, Data curation. **Giulia Frank:** Writing – original draft, Data curation. **Rossella Cianci:** Writing – review & editing, Writing – original draft. **Laura Di Renzo:** Writing – review & editing, Conceptualization.

Declaration of competing interest

There are no conflicts of interest.

References

[1] S. Maqsood, F. Ahmed, M.T. Arshad, A. Ikram, M.A. Abdullahi, Comparative analysis of food addiction and obesity: a critical review, Food Sci. Nutr. 13 (2025) e70799, https://doi.org/10.1002/fsn3.70799.
[2] C. Hauck, B. Cook, T. Ellrott, Food addiction, eating addiction and eating disorders, Proc. Nutr. Soc. 79 (2020) 103–112, https://doi.org/10.1017/S0029665119001162.
[3] J. Oliveira, G.C. Bestetti, I.D.C. Stelmo, L. Antunes, P.R. Vilella, Questioning the validity of food addiction: a critical review, Front. Behav. Neurosci. 19 (2025) 1562185, https://doi.org/10.3389/fnbeh.2025.1562185.
[4] D.A. Wiss, N. Avena, M. Gold, Food addiction and psychosocial adversity: biological embedding, contextual factors, and public health implications, Nutrients 12 (2020) 3521, https://doi.org/10.3390/nu12113521.
[5] H. Krupa, A.N. Gearhardt, A. Lewandowski, N.M. Avena, Food addiction, Brain Sci. 14 (2024) 952, https://doi.org/10.3390/brainsci14100952.
[6] O. Vasiliu, Current status of evidence for a new diagnosis: food addiction-a literature review, Front. Psychiatry 12 (2022) 824936, https://doi.org/10.3389/fpsyt.2021.824936.
[7] J. Oliveira, M.S. Colombarolli, T.A. Cordás, Prevalence and correlates of food addiction: systematic review of studies with the YFAS 2.0, Obes. Res. Clin. Pract. 15 (2021) 191–204, https://doi.org/10.1016/j.orcp.2021.03.014.
[8] P.J. Stover, M.S. Field, M.L. Andermann, R.L. Bailey, R.L. Batterham, E. Cauffman, G. Frühbeck, P.O. Iversen, P. Starke-Reed, S.M. Sternson, S. Vinoy, A.V. Witte, C. S. Zuker, B. Angelin, Neurobiology of eating behavior, nutrition, and health, J. Intern. Med. 294 (2023) 582–604, https://doi.org/10.1111/joim.13699.
[9] A. Meule, Twenty years of the food cravings questionnaires: a comprehensive review, Curr. Addict. Rep. 7 (2020) 30–43, https://doi.org/10.1007/s40429-020-00294-z.
[10] D. Wiss, T. Brewerton, Separating the signal from the noise: how psychiatric diagnoses can help discern food addiction from dietary restraint, Nutrients 12 (2020) 2937, https://doi.org/10.3390/nu12102937.
[11] I. Sanchez, I. Lucas, L. Munguía, L. Camacho-Barcia, M. Giménez, J. Sánchez-González, R. Granero, N. Solé-Morata, A.N. Gearhardt, C. Diéguez, S. Jiménez-Murcia, F. Fernández-Aranda, Food addiction in anorexia nervosa: implications for the understanding of crossover diagnosis, Eur. Eat. Disord. Rev. 30 (2022) 278–288, https://doi.org/10.1002/erv.2897.
[12] M. Fontanari, A.L. Amato, M. Dri, F. Nicoletti, A. Moia, A. De Lorenzo, Food addiction: umbrella review della letteratura più recente, Nóos 27 (2021) 37–54, https://doi.org/10.1722/3653.36343.
[13] D. Gubin, D. Weinert, O. Stefani, K. Otsuka, M. Borisenkov, G. Cornelissen, Wearables in chronomedicine and interpretation of circadian health, Diagnostics 15 (2025) 327, https://doi.org/10.3390/diagnostics15030327.
[14] Y.-I. Kim, E. Kim, Y. Lee, J. Park, Role of late-night eating in circadian disruption and depression: a review of emotional health impacts, Phys. Act. Nutr. 29 (2025) 018–024, https://doi.org/10.20463/pan.2025.0003.
[15] L.C. Ruddick-Collins, P.J. Morgan, A.M. Johnstone, Mealtime: a circadian disruptor and determinant of energy balance? J. Neuroendocrinol. 32 (2020) e12886 https://doi.org/10.1111/jne.12886.
[16] A. Kandeğer, Ü. Eğilmez, Y. Selvi, Feeding and eating disorders in the context of circadian rhythms, Alpha Psychiatry 22 (2021) 278–284, https://doi.org/10.1530/alphapsychiatry.2021.21151.
[17] D.J. Stenvers, C.F. Jonkers, E. Fliers, P.H.L.T. Bisschop, A. Kalsbeek, Nutrition and the circadian timing system, Prog. Brain Res. 199 (2012) 359–376, https://doi.org/10.1016/B978-0-444-59427-3.00020-4. Elsevier.
[18] G.D.M. Potter, J.E. Cade, P.J. Grant, L.J. Hardie, Nutrition and the circadian system, Br. J. Nutr. 116 (2016) 434–442, https://doi.org/10.1017/S0007114516002117.
[19] R.W. Logan, W.P. Williams, C.A. McClung, Circadian rhythms and addiction: mechanistic insights and future directions, Behav. Neurosci. 128 (2014) 387–412, https://doi.org/10.1037/a0036268.
[20] M.F. Borisenkov, S.V. Popov, T.A. Tserne, L.A. Bakutova, A.A. Pecherkina, O. I. Dorogina, E.A. Martinson, V.I. Vetosheva, D.G. Gubin, S.V. Solovieva, E. F. Turovinina, E.E. Symanyuk, Food addiction and symptoms of depression among inhabitants of the European North of Russia: associations with sleep characteristics and photoperiod, Eur. Eat. Disord. Rev. 28 (2020) 332–342, https://doi.org/10.1002/erv.2728.
[21] M.F. Borisenkov, T.A. Tserne, L.A. Bakutova, D.G. Gubin, Food addiction and emotional eating are associated with intradaily rest–activity rhythm variability, Eat. Weight Disord. - Stud. Anorex. Bulim. Obes. 27 (2022) 3309–3316, https://doi.org/10.1007/s40519-022-01461-z.
[22] M. Borisenkov, D. Gubin, A. Markov, A. Shigabaeva, M. Mezhakova, K. Voronin, Circadian factors of food addiction in Arctic residents: a pilot study, Biol. Rhythm Res. 57 (2026) 30–43, https://doi.org/10.1080/09291016.2025.2530501.
[23] M.J. Page, J.E. McKenzie, P.M. Bossuyt, I. Boutron, T.C. Hoffmann, C.D. Mulrow, L. Shamseer, J.M. Tetzlaff, E.A. Akl, S.E. Brennan, R. Chou, J. Glanville, J.

M. Grimshaw, A. Hróbjartsson, M.M. Lalu, T. Li, E.W. Loder, E. Mayo-Wilson, S. McDonald, L.A. McGuinness, L.A. Stewart, J. Thomas, A.C. Tricco, V.A. Welch, P. Whiting, D. Moher, The PRISMA 2020 statement: an updated guideline for reporting systematic reviews, BMJ (2021) n71, https://doi.org/10.1136/bmj.n71.
[24] A. Grey, M.J. Bolland, A. Avenell, A.A. Klein, C.K. Gunsalus, Check for publication integrity before misconduct, Nature 577 (2020) 167–169, https://doi.org/10.1038/d41586-019-03959-6.
[25] D.A. Wiss, E.M. LaFata, Ultra-processed foods and mental health: where do eating disorders fit into the puzzle? Nutrients 16 (2024) 1955, https://doi.org/10.3390/nu16121955.
[26] E.M. LaFata, K.C. Allison, J. Audrain-McGovern, E.M. Forman, Ultra-processed food addiction: a research update, Curr. Obes. Rep. 13 (2024) 214–223, https://doi.org/10.1007/s13679-024-00569-w.
[27] L. Parnarouskis, A.M. Leventhal, S.G. Ferguson, A.N. Gearhardt, Withdrawal: a key consideration in evaluating whether highly processed foods are addictive, Obes. Rev. 23 (2022) e13507, https://doi.org/10.1111/obr.13507.
[28] G.C. Jurema Santos, M.S. De Sousa Fernandes, P.G. Carniel, A. Da Silva Garcêz, C. Góis Leandro, R. Canuto, Dietary intake in children and adolescents with food addiction: a systematic review, Addict. Behav. Rep. 19 (2024) 100531, https://doi.org/10.1016/j.abrep.2024.100531.
[29] E. di Giacomo, F. Aliberti, F. Pescatore, M. Santorelli, R. Pessina, V. Placenti, F. Colmegna, M. Clerici, Disentangling binge eating disorder and food addiction: a systematic review and meta-analysis, Eat. Weight Disord. 27 (2022) 1963–1970, https://doi.org/10.1007/s40519-021-01354-7.
[30] D. Ratković, V. Knežević, A. Dickov, E. Fedrigolli, M. Čomić, Comparison of binge-eating disorder and food addiction, J. Int. Med. Res. 51 (2023) 03000605231171016, https://doi.org/10.1177/03000605231171016.
[31] T. Pereira, M.C. Mocellin, C. Curioni, Association between ultraprocessed foods consumption, eating disorders, food addiction and body image: a systematic review, BMJ Open 14 (2024) e091223, https://doi.org/10.1136/bmjopen-2024-091223.
[32] G. Halbeisen, M. Pahlenkemper, L. Sabel, C. Richardson, Z. Agüera, F. Fernandez-Aranda, G. Paslakis, The prognostic role of food addiction for weight loss treatment outcomes in individuals with overweight and obesity: a systematic review and meta-analysis, Obes. Rev. 26 (2025) e13851, https://doi.org/10.1111/obr.13851.
[33] H. Jahrami, W. Husain, K. Trabelsi, A. Ammar, S.R. Pandi-Perumal, Z. Saif, M. N. Potenza, C. Lin, A.H. Pakpour, A meta-analysis assessing reliability of the Yale Food Addiction Scale: implications for compulsive eating and obesity, Obes. Rev. 26 (2025) e13881, https://doi.org/10.1111/obr.13881.
[34] G. Mestre-Bach, M.N. Potenza, Potential biological markers and treatment implications for binge eating disorder and behavioral addictions, Nutrients 15 (2023) 827, https://doi.org/10.3390/nu15040827.
[35] Y. Yu, I.D. Fernandez, Y. Meng, W. Zhao, S.W. Groth, Gut hormones, adipokines, and pro- and anti-inflammatory cytokines/markers in loss of control eating: a scoping review, Appetite 166 (2021) 105442, https://doi.org/10.1016/j.appet.2021.105442.
[36] G.D. Stuber, V.M. Schwitzgebel, C. Lüscher, The neurobiology of overeating, Neuron 113 (2025) 1680–1693, https://doi.org/10.1016/j.neuron.2025.03.010.
[37] J.B. Mehr, D. Mitchison, H.E. Bowrey, M.H. James, Sleep dysregulation in binge eating disorder and "food addiction": the orexin (hypocretin) system as a potential neurobiological link, Neuropsychopharmacology 46 (2021) 2051–2061, https://doi.org/10.1038/s41386-021-01052-z.
[38] M.S. Yekaninejad, N. Badrooj, F. Vosoughi, C.-Y. Lin, M.N. Potenza, A.H. Pakpour, Prevalence of food addiction in children and adolescents: a systematic review and meta-analysis, Obes. Rev. 22 (2021) e13183, https://doi.org/10.1111/obr.13183.
[39] M. Bektaş, D. Demir, Ş. Demir, İ. Bektaş, The effect of food addiction in children on obesity: a systematic review and meta-analysis study, J. Pediatr. Res. 8 (2021) 414–423, https://doi.org/10.4274/jpr.galenos.2021.29963.
[40] A. Gaspar-Pérez, G. Paslakis, M. Rosinska, L. Munguía, S. Jiménez-Murcia, A. Szilágyi, R. Miranda-Olivos, L. Camacho-Barcia, C.B. Reque, B. Pászthy, F. Fernández-Aranda, Food addiction and impulsivity in clinical populations by gender: a systematic review, Curr. Addict. Rep. 10 (2023) 793–816, https://doi.org/10.1007/s40429-023-00514-2.
[41] B. Silvestrini, M. Silvestrini, Physiopathology and treatment of obesity and overweight: a proposal for a new anorectic, J. Obes. 2024 (2024) 9587300, https://doi.org/10.1155/2024/9587300.
[42] M.G. Novelle, Decoding the role of gut-microbiome in the food addiction paradigm, Int. J. Environ. Res. Public Health 18 (2021) 6825, https://doi.org/10.3390/ijerph18136825.
[43] J. Frank, A. Gupta, V. Osadchiy, E.A. Mayer, Brain–gut–microbiome interactions and intermittent fasting in obesity, Nutrients 13 (2021) 584, https://doi.org/10.3390/nu13020584.
[44] D. Dyńka, Ł. Rodzeń, M. Rodzeń, A. Pacholak-Klimas, G. Ede, S. Sethi, D. Łojko, K. Bartoń, K. Berry, A. Deptuła, Ż. Grzywacz, P. Martin, J. Unwin, D. Unwin, Ketogenic diets for body weight loss: a comparison with other diets, Nutrients 17 (2025) 965, https://doi.org/10.3390/nu17060965.
[45] G.E. Ames, A.M. Koball, M.M. Clark, Behavioral interventions to attenuate driven overeating and weight regain after bariatric surgery, Front. Endocrinol. 13 (2022) 934680, https://doi.org/10.3389/fendo.2022.934680.
[46] J. Taba, M. Suzuki, F. Nascimento, L. Iuamoto, W. Hsing, L. Pipek, L. Carneiro-D'Albuquerque, A. Meyer, W. Andraus, The development of feeding and eating disorders after bariatric surgery: a systematic review and meta-analysis, Nutrients 13 (2021) 2396, https://doi.org/10.3390/nu13072396.
[47] A.N. Gearhardt, J. Hebebrand, The concept of "food addiction" helps inform the understanding of overeating and obesity: yes, Am. J. Clin. Nutr. 113 (2021) 263–267, https://doi.org/10.1093/ajcn/nqaa343.
[48] F. Capozzi, F. Magkos, F. Fava, G.P. Milani, C. Agostoni, A. Astrup, I.S. Saguy, A multidisciplinary perspective of ultra-processed foods and associated food processing technologies: a view of the sustainable road ahead, Nutrients 13 (2021) 3948, https://doi.org/10.3390/nu13113948.
[49] M. Tristan Asensi, A. Napoletano, F. Sofi, M. Dinu, Low-grade inflammation and ultra-processed foods consumption: a review, Nutrients 15 (2023) 1546, https://doi.org/10.3390/nu15061546.
[50] A. Piccinni, R. Bucchi, C. Fini, F. Vanelli, M. Mauri, T. Stallone, E.D. Cavallo, C. Claudio, Food addiction and psychiatric comorbidities: a review of current evidence, Eat. Weight Disord. - Stud. Anorex. Bulim. Obes. 26 (2021) 1049–1056, https://doi.org/10.1007/s40519-020-01021-3.
[51] R. De Almeida, G. Kamath, P. Cabandugama, Food addiction in application to obesity management, Mo Med. 119 (2022) 372–378.
[52] E. Challet, The circadian regulation of food intake, Nat. Rev. Endocrinol. 15 (2019) 393–405, https://doi.org/10.1038/s41574-019-0210-x.
[53] E. Grosjean, V. Simonneaux, E. Challet, Reciprocal interactions between circadian clocks, food intake, and energy metabolism, Biology 12 (2023) 539, https://doi.org/10.3390/biology12040539.
[54] L. Kivelä, M.R. Papadopoulos, N. Antypa, Chronotype and psychiatric disorders, Curr. Sleep Med. Rep. 4 (2018) 94–103, https://doi.org/10.1007/s40675-018-0113-8.
[55] S.A. Plano, S. Soneira, C. Tortello, D.A. Golombek, Is the binge-eating disorder a circadian disorder? Front. Nutr. 9 (2022) 964491 https://doi.org/10.3389/fnut.2022.964491.
[56] J. Mendoza, Food intake and addictive-like eating behaviors: time to think about the circadian clock(s), Neurosci. Biobehav. Rev. 106 (2019) 122–132, https://doi.org/10.1016/j.neubiorev.2018.07.003.
[57] A. Jais, J.C. Brüning, Arcuate nucleus-dependent regulation of metabolism—pathways to obesity and diabetes mellitus, Endocr. Rev. 43 (2021) 314–328, https://doi.org/10.1210/endrev/bnab025.
[58] C.-I. Colita, D.M. Hermann, M. Filfan, D. Colita, T.R. Doepnner, O. Tica, D. Glavan, A. Popa-Wagner, Optimizing chronotherapy in psychiatric care: the impact of circadian rhythms on medication timing and efficacy, Clocks Sleep 6 (2024) 635–655, https://doi.org/10.3390/clockssleep6040043.
[59] A. Okyar, D. Ozturk Civelek, Y.K. Akyel, S. Surme, Z. Pala Kara, İ.H Kavakli, The role of the circadian timing system on drug metabolism and detoxification: an update, Expert Opin. Drug Metab. Toxicol. 20 (2024) 503–517, https://doi.org/10.1080/17425255.2024.2356167.
[60] L. Cheruzel, U.A. Argikar, P. Brunetti, M. Chatzopoulou, S. Cho, R.D. Crouch, D. Dhaware, K.M. Johnson, V. Kramlinger, J. Liu, B. Ma, H. Maw, K. Rehm, H. K. Seneviratne, L.W.T. Tang, G. Zhong, S.C. Khojasteh, Biotransformation research advances –2024 year in review, Drug Metab. Rev. 57 (2025) 432–466, https://doi.org/10.1080/03602532.2025.2538676.
[61] R. Dallmann, A. Okyar, F. Lévi, Dosing-time makes the poison: circadian regulation and pharmacotherapy, Trends Mol. Med. 22 (2016) 430–445, https://doi.org/10.1016/j.molmed.2016.03.004.
[62] O.H. Meléndez-Fernández, J.A. Liu, R.J. Nelson, Circadian rhythms disrupted by light at night and mistimed food intake alter hormonal rhythms and metabolism, Int. J. Mol. Sci. 24 (2023) 3392, https://doi.org/10.3390/ijms24043392.
[63] J. Hu, H. Ye, M. Wang, H. Yang, M. Wu, J. Cao, C. Sun, Y. Wang, The effects of food on circadian rhythm: a comprehensive review, eFood 6 (2025) e70092, https://doi.org/10.1002/efd2.70092.
[64] P. Petrus, M. Cervantes, M. Samad, T. Sato, A. Chao, S. Sato, K.B. Koronowski, G. Park, Y. Alam, N. Mejhert, M.M. Seldin, J.M. Monroy Kuhn, K.A. Dyar, D. Lutter, P. Baldi, P. Kaiser, C. Jang, P. Sassone-Corsi, Tryptophan metabolism is a physiological integrator regulating circadian rhythms, Mol. Metab. 64 (2022) 101556, https://doi.org/10.1016/j.molmet.2022.101556.
[65] Z. Guleken, T. Uzbay, Neurobiological and neuropharmacological aspects of food addiction, Neurosci. Biobehav. Rev. 139 (2022) 104760, https://doi.org/10.1016/j.neubiorev.2022.104760.
[66] B. Başar Gökcen, From eveningness to food addiction: exploring the roles of night eating syndrome and mindful eating, J. Eat. Disord. 13 (2025) 234, https://doi.org/10.1186/s40337-025-01421-9.
[67] A.W. McHill, M.P. Butler, Eating around the clock: circadian rhythms of Eating and metabolism, Annu. Rev. Nutr. 44 (2024) 25–50, https://doi.org/10.1146/annurev-nutr-062122-014528.
[68] M. Kaşkal, M. Sevim, G. Ülker, C. Keleş, B.T. Bebitoğlu, The clinical impact of chronopharmacology on current medicine, Naunyn Schmiedebergs Arch. Pharmacol. 398 (2025) 6179–6191, https://doi.org/10.1007/s00210-025-03788-7.