

Addictive Properties of Ultra-processed Foods? A Review of the Evidence for Hyper-palatable Formulations and Eating Behavior

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Abstract

The pervasive consumption of ultra-processed foods (UPFs) and their potential addictive properties have emerged as a critical public health challenge, necessitating a comprehensive synthesis of the evidence. This review aims to consolidate the growing body of research linking hyper-palatable UPF formulations to addictive-like eating behaviors, thereby addressing a significant gap in understanding their impact on individual and societal health. This review examines the neurobiological basis of hedonic eating, including the activation of reward pathways akin to substance use disorders, and outlines standardized definitions for identifying hyper-palatable food formulations. It explores the behavioral and psychological dimensions of UPF consumption, such as links to stress, mental health, and food addiction. Epidemiological evidence connecting UPF intake to chronic diseases like obesity, metabolic syndrome, and inflammatory conditions is summarized, alongside clinical correlates that highlight overlaps between UPF addiction and eating disorders. Key intervention studies assessing the effects of UPF diets on energy intake, weight gain, and body composition are analyzed. The development and utility of assessment tools for UPF addiction and withdrawal are discussed. Finally, the environmental, societal, and policy influences that drive UPF consumption and complicate public health responses are reviewed. Future research should prioritize longitudinal and mechanistic studies to clarify the causal pathways through which UPFs exert addictive and metabolic effects. There is also a pressing need to develop and evaluate integrated policy, clinical, and public health strategies aimed at mitigating the individual and societal burdens associated with UPF consumption.

Keywords: Addiction, Chronic disease, Eating behavior, Hyper-palatable food, Neurobiology, Public health, Ultra-processed food, Withdrawal

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Citation: Vasudevan V, Durga PSSL, Kapa VK, Sriram T (2026) Addictive Properties of Ultra-processed Foods? A Review of the Evidence for Hyper-palatable Formulations and Eating Behavior. *Nanotechnol Nanomater Res*, Volume 7:2. 132. DOI: <https://doi.org/10.47275/2692-885X-132>

Received: June 04, 2026; **Accepted:** August 17, 2026; **Published:** August 24, 2026

Introduction

The addictive properties of UPFs have garnered increasing scientific attention, with a growing body of evidence suggesting that these foods may possess characteristics akin to substances of abuse [1-10]. This review overviews the current understanding of the neurobiological, behavioral, and societal factors that underpin the potential for UPFs to induce addictive-like eating behaviors, emphasizing the role of hyper-palatable formulations and their impact on eating behavior.

Central to this discourse is the neurobiological basis of hedonic eating, which involves brain systems that regulate reward and motivation. Wilcox [11] highlights that food intake, especially in relation to highly palatable foods (HPFs)—those rich in sugar, fat, and ultra-processed ingredients—activates neural pathways similar to those engaged by addictive substances. Neuroimaging and animal studies reveal that HPFs can induce changes in brain chemistry, paralleling the neuroadaptations observed in substance use disorders. These

alterations include increased dopamine release in reward circuits, which reinforces consumption and may lead to compulsive eating behaviors. Further supporting this perspective, Gearhardt and Schulte [12] argue that ingredients in UPFs possess reinforcing potential that can drive compulsive consumption, akin to addictive drugs. They propose that the criteria used to define addiction—such as compulsive use, psychoactive effects, and reinforcement—are applicable to HPFs, suggesting that these foods meet established scientific benchmarks for addiction. Similarly, Gearhardt and DiFeliceantonio [13] extend this argument by proposing that highly processed foods can be considered addictive substances based on criteria derived from tobacco addiction, emphasizing their capacity to cause controlled or compulsive use, mood-altering effects, and reinforcement of behavior.

The concept of hyper-palatable formulations is central to understanding the addictive potential of UPFs. Contreras-Rodriguez et al. [14] describe UPFs as formulations of ingredients designed through industrial processes to maximize palatability, often resulting



in automatic recruitment of brain regions associated with food choice and consumption. This automatic activation may impair inhibitory control, particularly in the prefrontal cortex, thereby fostering compulsive eating patterns. Kelly et al. [15] further emphasize that UPFs, especially those categorized as NOVA 4, constitute a significant proportion of caloric intake in Western diets, which underscores their pervasive influence on eating behavior. The behavioral and psychological dimensions of UPF addiction are also well-documented. Ho and Verdejo-Garcia [16] adopt a multilevel framework to explore how neurocognitive systems, individual mindsets, and contextual factors interact to promote addictive eating behaviors. They highlight that the environment, including marketing and easy availability of UPFs, can reinforce compulsive consumption. Vasiliu [17] adds that the prevalence of food addiction, as measured by tools like the Yale Food Addiction Scale, is notably high—up to 20% in the general population, indicating that a significant subset of individuals exhibits behaviors consistent with addiction.

The mechanistic insights into how UPFs influence brain function extend to the role of neuropeptides and neurotransmitter systems. Mehr et al. [18] focus on the orexin (hypocretin) system, which is implicated in sleep regulation and reward processing, suggesting that dysregulation in this system may link binge eating disorder and food addiction. This neurobiological overlap underscores the complex interplay between sleep, reward, and compulsive eating, further supporting the addictive potential of UPFs. Epidemiological evidence links UPF consumption with adverse health outcomes, including obesity, metabolic syndrome, and inflammatory diseases. Srour et al. [19] review data indicating that high intake of UPFs correlates with increased risk of chronic diseases, partly through mechanisms involving the gut microbiota and systemic inflammation. Forde [20] critiques the mechanistic explanations for these associations, emphasizing that the sustained effects of UPF diets on energy intake and weight gain are well-documented, although the precise pathways remain to be fully elucidated.

The clinical implications of recognizing UPFs as addictive substances are profound. Ifland and Brewerton [21] argue that the phenomenology and pathophysiology of UPF addiction overlap significantly with binge-type eating disorders, suggesting that treatment approaches from addiction medicine—such as harm reduction or abstinence—may be applicable. Similarly, the development of tools like the modified Highly Processed Food Withdrawal Scale (mProWS) by Hu et al. [22] indicates that withdrawal symptoms from UPF reduction are measurable and may influence treatment strategies. Policy and public health considerations are also prominent. Hebebrand et al. [23] describe efforts to reach a consensus among international experts on the addictive potential of UPFs, emphasizing the need for regulatory measures akin to those used for addictive substances. The potential for UPFs to impact mental health, including associations with depression and eating disorders, further underscores the importance of addressing their pervasive presence in the food environment [24].

In summary, the evidence from neuroscientific, behavioral, epidemiological, and clinical studies converges on the notion that UPFs, particularly HPFs, possess addictive properties that influence eating behavior. These foods activate reward pathways, reinforce consumption, and may lead to compulsive eating patterns similar to substance use disorders. Recognizing the addictive potential of UPFs has significant implications for treatment, policy, and public health strategies aimed at mitigating their impact on individual health and societal well-being. Continued research is essential to fully elucidate the mechanisms involved and to develop effective interventions to address this modern dietary challenge.

Defining and Identifying Hyper-palatable Food Formulations

The concept of HPFs has garnered increasing attention within nutritional science and food industry research, primarily due to their potential role in promoting overconsumption and contributing to obesity and related health issues [25-33]. Despite the widespread recognition of HPFs, there remains a lack of consensus regarding their precise definition and the criteria used to identify such foods. The existing literature reflects a multifaceted approach, incorporating nutrient composition, processing techniques, and sensory attributes to characterize hyper-palatable formulations.

One of the foundational efforts in establishing a standardized definition of HPFs was undertaken by Fazzino et al. [34], who developed a quantitative framework to identify HPFs based on nutrient combinations that enhance palatability. This approach emphasizes specific thresholds for nutrients such as fat, sugar, and sodium, which, when combined in certain proportions, significantly increase the food's appeal. The standardized definition has been instrumental in enabling researchers to systematically classify foods as hyper-palatable, facilitating comparisons across studies and food products [35]. Building upon this, recent studies have explored the role of food formulation and processing in contributing to hyper-palatability. For instance, the work by Ghosh et al. [36] highlights technological advancements in formulation and processing that enable the development of high-concentration antibody drug products, which, although focused on pharmaceuticals, underscores the importance of formulation techniques in achieving specific product characteristics. Similarly, in the context of foods, sophisticated processing methods and attractive packaging are used to enhance sensory appeal, which may contribute to hyper-palatable qualities [37]. These insights suggest that formulation and processing are independent but interconnected factors influencing hyper-palatability.

The identification of HPFs also involves analyzing ingredient lists and nutrient profiles. Examination of UPFs reveals that many are formulated with nutrient levels exceeding established thresholds for palatability [38, 39]. Such foods often contain high levels of fats, sugars, and sodium, which are deliberately manipulated to maximize consumer appeal. The review by Jun et al. [38] emphasizes that UPFs are characterized not only by their processing extent but also by their nutrient composition, which often surpasses the thresholds associated with hyper-palatability. Furthermore, the literature discusses the importance of sensory and packaging attributes in defining HPFs. The use of sophisticated and attractive packaging, along with formulation strategies that enhance taste and texture, plays a significant role in making foods more appealing and potentially hyper-palatable [37]. This aligns with the notion that hyper-palatable foods are designed to be highly attractive, often employing sensory cues that stimulate the reward pathways in the brain, thereby promoting overconsumption [34].

The debate around terminology and classification is also prominent. Some researchers advocate for clear distinctions between terms such as highly processed foods, UPFs, and HPFs, emphasizing that each term captures different aspects of food processing and formulation [40, 41]. For example, while UPFs are defined primarily by their industrial processing level, HPFs are characterized by their nutrient composition and sensory attributes that induce palatability. Clarifying these distinctions is crucial for developing effective regulatory and public health strategies. Recent studies have also explored the potential



utility of warning labels to inform consumers about hyper-palatable formulations. The proposal is that identifying and labeling HPFs could help consumers make healthier choices and encourage manufacturers to reformulate products to reduce hyper-palatability [42]. This approach underscores the importance of a clear, operational definition of hyper-palatable foods to support policy interventions.

In summary, the current literature underscores that defining and identifying HPFs involves a combination of nutrient thresholds, processing techniques, sensory attributes, and packaging features. The development of standardized, quantitative definitions—such as those proposed by Fazzino et al. [34] has been pivotal in advancing research and policy discussions. However, the complexity of food formulation and the multifaceted nature of palatability suggest that a comprehensive approach, integrating nutrient composition, processing methods, and sensory appeal, is essential for accurately characterizing hyper-palatable formulations. Continued research is needed to refine these definitions and develop practical tools for industry regulation and public health initiatives.

Behavioral and Psychological Dimensions of UPF Consumption

The consumption of UPFs has garnered increasing attention not only for its physical health implications but also for its complex behavioral and psychological dimensions [43-50]. The literature indicates that UPF intake is intricately linked with various mental health outcomes, psychological stress, eating behaviors, and neurobiological mechanisms, highlighting a multifaceted relationship that warrants comprehensive exploration (Table 1).

One of the prominent psychological dimensions associated with UPF consumption is perceived stress. Cortes et al. [51] conducted a cross-sectional study in Brazil to examine the association between UPF intake and perceived stress levels among working-class young adults. Their findings suggest that higher consumption of UPFs correlates with elevated perceived stress, implying that psychological stress may influence dietary choices or that UPF consumption could exacerbate stress perceptions. This bidirectional relationship

underscores the importance of considering psychological stress as both a consequence and a potential driver of unhealthy eating behaviors. Further emphasizing the behavioral aspect, Teo et al. [52] investigated how texture-based differences in eating rate influence energy intake for minimally processed versus ultra-processed meals. Their research indicates that the texture and consequent eating rate of UPFs can lead to increased energy intake, which may contribute to overeating and subsequent weight gain. This mechanistic insight suggests that the sensory and textural properties of UPFs can modulate eating behaviors, potentially reinforcing consumption patterns associated with psychological reward and habit formation.

The psychological impact of UPF consumption extends to mental health conditions such as anxiety and depression. Coletro et al. [53] explored the association between symptoms of anxiety and depression and food consumption during the COVID-19 pandemic in Brazil. Their results demonstrated that higher intake of UPFs was linked with increased symptoms of anxiety and depression, indicating that dietary patterns characterized by UPFs may be associated with adverse mental health outcomes during stressful periods. Similarly, Lane et al. [54] examined the relationship between UPF consumption and psychological distress in adults over a 15-year follow-up in Australia. Their findings revealed that elevated UPF intake was associated with higher levels of psychological distress, which can be considered an indicator of depression, further supporting the link between unhealthy dietary patterns and mental health deterioration. Adolescents and children are also vulnerable to the psychological effects of UPF consumption. Lane et al. [55] studied adolescent girls in Iran and found that high UPF intake was associated with reduced quality of life, which encompasses psychological well-being. Silva et al. [56] analyzed Brazilian adolescents and identified factors associated with UPF consumption, including behavioral and socioeconomic variables, suggesting that early-life dietary habits may have long-term psychological implications. The systematic review by Santos et al. [57] further consolidates evidence that children and adolescents with food addiction exhibit higher consumption of sugary snacks, chips, and other UPFs, linking these dietary patterns to potential addictive behaviors and associated psychopathologies.

Table 1: A thematic overview of behavioral and psychological research on UPFs.

Dimension	Key constructs	Representative findings from literature	Implications
Psychological stress and mood	Perceived stress, anxiety, depression, psychological distress	- Positive association between UPF intake and perceived stress [51]. - UPF consumption linked to higher anxiety/depression symptoms, especially during COVID-19 [53]. - Longitudinal data show UPF intake associated with increased psychological distress [54].	Suggests a bidirectional relationship: stress may drive UPF intake, and UPFs may worsen mental health.
Eating behavior and microstructure	Eating rate, energy intake, texture, compulsivity	- UPF texture leads to faster eating rate and greater energy intake vs minimally processed meals [52]. - UPFs are designed for hyper-palatability, which may override satiety signals and promote rapid, compulsive consumption.	Highlights how food engineering influences eating pace and volume, contributing to overconsumption.
Food addiction and reward	Addictive-like eating, reward pathway activation, craving, loss of control	- UPF consumption meets criteria for addiction [13]. - Neurobiological models show UPFs trigger dopamine release and compulsive use patterns akin to substances [58]. - Withdrawal symptoms upon UPF reduction are measurable (mProWS).	Supports framing UPF overconsumption through an addiction lens, with implications for treatment (e.g., abstinence protocols).
Mental health and quality of life	Depression, anxiety, well-being, quality of life	- Higher UPF intake correlates with reduced quality of life in adolescents [54]. - Metabolomic signatures link UPF intake to incident mental disorders [59]. - UPF addiction prevalence is elevated in individuals with poor mental health.	UPFs may adversely affect mental health through biological (e.g., inflammation) and behavioral pathways.
Socioemotional and contextual factors	Social isolation, adverse childhood experiences, emotional eating, environmental cues	- UPF addiction is more likely in individuals reporting social isolation. - Adverse childhood experiences increase risk for UPF addiction and related psychopathology [24]. - Marketing and easy availability reinforce compulsive consumption [16].	Emphasizes the role of trauma, environment, and social determinants in UPF-related behaviors.
Developmental and lifespan considerations	Adolescence, aging, intergenerational patterns	- Adolescents with high UPF intake show poorer psychological outcomes. - UPF addiction is prevalent in older adults, especially women, and linked to poor physical/mental health. - Maternal UPF consumption may increase offspring obesity risk.	Points to vulnerable periods and the potential for long-term, cross-generational health impacts.



The concept of food addiction and its neurobiological underpinnings is gaining prominence in understanding the behavioral dimensions of UPF consumption. Araç [58] discusses how certain eating behaviors towards UPFs mirror substance use disorders, proposing a 'food addiction' model that involves neurobiological mechanisms similar to those seen in behavioral addictions. This perspective suggests that UPFs may trigger reward pathways in the brain, fostering compulsive consumption patterns that resemble addictive behaviors, which can complicate efforts to modify dietary habits. The influence of UPFs on mental health is further supported by studies examining biological markers. Yuan et al. [59] identified a circulating metabolomic signature associated with UPF intake and found that this signature correlates with incident mental disorders, indicating a biological pathway linking UPF consumption to psychological conditions. This metabolomic approach provides evidence that UPFs may influence mental health through biochemical mechanisms, possibly involving neuroinflammatory or neurochemical alterations.

Behavioral responses to UPFs are also modulated by individual psychological states such as anxiety. Neves et al. [60] demonstrated that anxiety symptoms influence food consumption differently depending on nutritional status during the COVID-19 pandemic. Their longitudinal study suggests that anxiety may lead to increased UPF intake, especially among individuals with excess weight, highlighting the role of emotional states in shaping dietary behaviors. Similarly, Zheng et al. [61] found that psychological symptoms in Chinese adolescents are associated with UPF consumption and exercise duration, indicating that mental health status can influence dietary choices across different populations. The social and environmental context also plays a role in shaping the behavioral and psychological dimensions of UPF consumption. Jesus et al. [62] examined Brazilian education workers and identified factors associated with UPF intake, including psychological and social variables, emphasizing that workplace and social environments can influence dietary behaviors linked to mental health. Finally, the broader implications of UPF consumption on mental health and behavior are discussed in reviews by Wiss and LaFata [24] and Prescott et al. [63], which synthesize evidence on how UPFs may affect mental health through biological, psychological, and social pathways. These reviews highlight the complex interplay between UPF consumption, neurobiological mechanisms, and behavioral addictions, suggesting that addressing UPF intake requires an integrated approach that considers psychological vulnerabilities, neurobiological responses, and social determinants.

In summary, the literature underscores that the behavioral and psychological dimensions of UPF consumption are multifaceted, involving stress, addictive behaviors, neurobiological mechanisms, and emotional states. The evidence suggests that UPFs are not merely dietary choices but are embedded within complex psychological frameworks that influence and are influenced by mental health conditions. Understanding these dimensions is crucial for developing effective interventions aimed at reducing UPF consumption and mitigating its adverse psychological and behavioral consequences.

Epidemiological Evidence: UPFs and Chronic Disease Risk

The epidemiological evidence linking UPFs to chronic disease risk has garnered increasing attention in recent years, reflecting a global shift in dietary patterns characterized by the proliferation of HPF products. Multiple studies and reviews have examined the associations between UPF consumption and various health outcomes, emphasizing

the potential role of UPFs as contributors to the rising prevalence of chronic diseases such as non-alcoholic fatty liver disease (NAFLD), metabolic syndrome, cardiovascular diseases, inflammatory bowel diseases (IBD), and certain cancers.

A significant body of evidence suggests that high intake of UPFs is associated with an increased risk of NAFLD. Zhang et al. [64] conducted a prospective cohort study involving over 16,000 participants, aiming to elucidate the relationship between UPF consumption and NAFLD risk. Their findings indicated a positive association, highlighting UPFs as potential dietary contributors to liver fat accumulation and related metabolic disturbances. Complementing this, Henney et al. [65] systematically reviewed the literature and confirmed that UPF intake is linked to NAFLD development, with evidence quality assessed through standardized tools supporting this association. Similarly, Grinshpan et al. [66] conducted a systematic review focusing on NAFLD, metabolic syndrome, and insulin resistance, reinforcing the notion that UPF consumption correlates with these metabolic risk factors.

Beyond liver health, epidemiological data also point to a connection between UPFs and cardiometabolic health outcomes. Juul et al. [67] reviewed evidence from diverse populations, finding consistent associations between UPF intake and increased risks of metabolic syndrome, hypertension, type 2 diabetes, obesity, and cardiovascular disease. These findings are echoed by Lane et al. [68], who performed an umbrella review of meta-analyses, concluding that higher UPF exposure correlates with adverse health outcomes, including cardiometabolic disorders, with evidence graded as moderate to high quality. The mechanistic pathways proposed include the high energy density, added sugars, and unhealthy fats prevalent in UPFs, which contribute to weight gain, insulin resistance, and systemic inflammation. Inflammatory processes are also implicated in the relationship between UPFs and chronic diseases. Vissers et al. [69] reviewed evidence suggesting that UPFs, characterized by high fat, sugar, and additive content, may play a role in the rising incidence of IBD, a chronic inflammatory condition of the gastrointestinal tract. Gubatan et al. [70] further discussed dietary interventions in IBD, noting that diets rich in unprocessed foods tend to improve inflammatory biomarkers, whereas UPFs may exacerbate inflammation, although current evidence is limited by study heterogeneity. Yao et al. [71] identified emerging evidence from prospective cohort studies indicating that UPFs may be involved in the development of Crohn's disease, a subtype of IBD, but not ulcerative colitis, suggesting disease-specific effects.

The potential carcinogenic implications of UPF consumption are also under investigation. Kliemann et al. [72] highlighted the global shift towards UPFs as a factor contributing to obesity and cancer risk, emphasizing the need for further research into the dual harms posed by UPFs on health and the environment. Anastasiou et al. [73] explored the link between UPFs and obesity-related cancers, proposing biological mechanisms such as endocrine disruption, inflammation, and epigenetic alterations as mediators of increased cancer risk. Fajkić et al. [74] introduced the Trojan horse model, suggesting that UPFs deliver endocrine-disrupting chemicals that may promote carcinogenesis through chronic low-grade inflammation and microbiome alterations. The role of specific food additives in UPFs, such as inorganic phosphate additives, has been examined as a mechanistic link to cardiorenal diseases. Calvo et al. [75] discussed how phosphate additives, commonly used in UPFs, may contribute to cardiovascular and bone diseases, especially among individuals with chronic kidney disease, further illustrating the complex pathways through which UPFs may influence chronic disease risk. Despite the accumulating evidence,



some researchers question whether the concept of 'UPFs' adds value beyond traditional nutrient profiling. Astrup and Monteiro [76] argued that many health effects attributed to UPFs can be explained by conventional dietary factors such as energy density, fiber content, and glycemic load, suggesting that the classification may not provide additional insights for dietary guidelines. Conversely, Temple [3] emphasized the need to understand the broader relationship between UPFs, obesity, and other chronic diseases, advocating for policies that address the food environment and consumer behavior.

In summary, epidemiological studies consistently demonstrate associations between UPF consumption and a spectrum of chronic diseases, including NAFLD, metabolic syndrome, cardiovascular diseases, IBD, and certain cancers. The evidence underscores the importance of considering UPFs as a significant dietary factor influencing chronic disease risk, although debates remain regarding the mechanistic explanations and the added value of the UPF classification system. Future research directions include elucidating biological mechanisms, refining exposure assessments, and developing targeted public health interventions to mitigate the health impacts of UPFs.

Clinical Correlates: UPF Addiction and Eating Disorders

The clinical landscape surrounding UPF addiction and eating disorders has garnered increasing scholarly attention, emphasizing the complex interplay of neurobiological, psychological, and behavioral factors. A growing body of research underscores the prevalence, clinical correlates, and potential treatment approaches for these intertwined conditions, highlighting the necessity for nuanced understanding and tailored interventions. Research by Oliveira et al. [77] emphasizes that food addiction, including UPF addiction, remains an evolving field with ongoing efforts to elucidate its subjective experience and clinical correlates. Their systematic review underscores the importance of understanding the mechanisms underlying food addiction and its psychological dimensions, which are crucial for developing effective diagnostic and treatment strategies. Similarly, Praxedes et al. [78] conducted a systematic review with meta-analysis to determine the prevalence of food addiction using the Yale Food Addiction Scale, revealing a notable prevalence across various populations and emphasizing the importance of standardized assessment tools in clinical settings.

The conceptualization of UPF addiction as a clinical entity akin to substance-use disorders is further supported by Vasiliu [17], who highlights the high prevalence of food addiction in the general population—up to 20%—and advocates for its recognition as a distinct diagnostic category. This perspective aligns with the neurobiological models discussed by Araç [58], which propose that certain eating behaviors towards UPFs mirror the neurobiological pathways involved in substance addictions, such as reward system dysregulation. These models suggest that UPF addiction may involve alterations in reward processing, similar to those observed in substance use disorders, thereby reinforcing the addiction framework. The relationship between UPF addiction and eating disorders is complex and multifaceted. Sankaranarayanan et al. [79] systematically reviewed disordered eating among individuals with schizophrenia spectrum disorders, revealing that disordered eating behaviors are prevalent in this population, which may include or overlap with UPF addiction symptoms. Bodell and Racine [80] further elaborate on reward processing alterations in binge-type eating disorders, proposing a mechanistic staging model that accounts for changes in reward sensitivity over the course of illness.

This model suggests that reward system alterations—such as increased 'wanting' without corresponding 'liking'—may underpin compulsive eating behaviors characteristic of binge episodes and UPF addiction.

The clinical correlates of UPF addiction extend to weight-related factors. Wiss [81] observed a J-shaped relationship between UPF addiction and body mass index, with heightened prevalence among underweight individuals, indicating that UPF addiction may not be solely associated with obesity but also with other weight classes. This nuanced relationship underscores the importance of considering individual differences and comorbidities when assessing UPF addiction in clinical populations. Eating disorders often co-occur with UPF addiction, complicating diagnosis and treatment. The review by Dennis et al. [82] highlights that UPF addiction is especially common among individuals seeking treatment for eating disorders, substance use disorders, and psychiatric comorbidities. They advocate comprehensive assessment protocols, such as the Food Addiction Symptom Inventory developed by LaFata et al. [83], which provides clinicians with a structured approach to diagnosing UPF addiction. Such tools are vital for identifying individuals at risk and tailoring interventions accordingly.

The neurobiological and psychological overlap between UPF addiction and eating disorders is further elaborated by Ifland and Brewerton [21], who discuss phenomenological similarities and the potential for shared treatment protocols. They suggest that traditional substance-use disorder recovery strategies, such as harm reduction or abstinence, could be adapted for UPF addiction and binge-type eating disorders, although current practices often lack consensus. This is echoed by Thompson et al. [84], who present case vignettes illustrating the potential benefits of abstinence-based treatments, emphasizing individualized care that considers genetic, neurobiological, and psychosocial factors. Psychobehavioral factors also play a significant role in UPF addiction and eating disorders. Barros et al. [85] examined how psychological variables, such as body image perception and anxiety, influence eating behaviors, including the tendency to consume UPFs rapidly. These factors may modulate the severity and manifestation of UPF addiction symptoms, suggesting that psychological interventions targeting these domains could be beneficial. The impact of early life experiences on UPF addiction and eating disorders is highlighted by Wiss et al. [86], who found that adverse childhood experiences are significant risk factors for UPF addiction and related psychopathologies. Their structural equation modeling indicates that adverse childhood experiences may influence the development of UPF addiction through neurobiological and psychological pathways, emphasizing the importance of trauma-informed approaches in treatment.

From a policy and clinical perspective, Wiss and LaFata [24] discuss the broader implications of UPFs on mental health, including their role in depression and other psychiatric conditions. They advocate for nuanced treatment considerations, especially in cases where UPF addiction coexists with eating disorders, and highlight the emerging consensus on the need for specialized assessment and intervention protocols.

In summary, the literature converges on the recognition of UPF addiction as a clinically significant condition with substantial overlap with various eating disorders. The evidence underscores the importance of standardized diagnostic tools, neurobiological models of reward dysregulation, and individualized treatment approaches—ranging from abstinence-based strategies to trauma-informed care. As research continues to evolve, integrating these insights into clinical



practice holds promise for improving outcomes for individuals affected by UPF addiction and comorbid eating disorders.

Clinical Studies

The addictive properties of UPFs have been increasingly studied, with clinical evidence suggesting that these foods can trigger behaviors similar to those seen in substance-use disorders. UPFs, characterized by their high content of refined carbohydrates, fats, and additives, are designed to be hyper-palatable, which can lead to compulsive eating behaviors.

A study by Loch et al. [87], conducted in July 2022 using the University of Michigan National Poll on Healthy Aging, investigated the prevalence of UPF addiction among older adults in the US and its associations with various health domains. The overall prevalence of UPF addiction was found to be 12.4% among older adults aged 50 to 80 years in the US. Prevalence was notably higher among women (16.9%) compared to men (7.5%). The highest rate of UPF addiction was observed in women aged 50 to 64, reaching 21%. Men who reported being overweight were 19.14 times more likely to meet the criteria for UPF addiction (95% confidence interval (CI) 5.26 to 69.66). Women who reported being overweight were 11.44 times more likely to meet the criteria for UPF addiction (95% CI 4.56 to 28.71). Women reporting worse physical health were 1.93 times more likely to meet the criteria for UPF addiction (95% CI 1.26 to 2.98). Men reporting worse physical health were 2.99 times more likely to meet the criteria for UPF addiction (95% CI 1.70 to 5.26). Women reporting worse mental health were 2.78 times more likely to meet the criteria for UPF addiction (95% CI 1.79 to 4.32). Men reporting worse mental health were 4.02 times more likely to meet the criteria for UPF addiction (95% CI 2.19 to 7.38). Women reporting feelings of social isolation were 3.40 times more likely to meet the criteria for UPF addiction (95% CI 2.16 to 5.34). Men reporting feelings of social isolation were 3.35 times more likely to meet the criteria for UPF addiction (95% CI 1.83 to 6.14). In summary, the study indicates that UPF addiction is prevalent among older adults in the US, with a disproportionately higher rate among women. Furthermore, UPF addiction is significantly associated with poorer self-reported physical health, mental health, and feelings of social isolation across both genders, and particularly with being overweight.

A study (NCT03407053) by Hall et al. [88] investigated the impact of UPFs on energy intake and weight in adults admitted to the NIH Clinical Center. Participants consumed significantly more calories when on an ultra-processed diet, with an average increase of 508 ± 106 kcal/d compared to the unprocessed diet. The increased energy intake was primarily driven by higher consumption of carbohydrates (280 ± 54 kcal/d). Fat intake also increased significantly (230 ± 53 kcal/d) during the ultra-processed diet. Protein consumption, however, did not show a significant change (-2 ± 12 kcal/d). Weight changes were strongly correlated with energy intake, showing a correlation coefficient of $r = 0.8$. During the ultra-processed diet, participants gained an average of 0.8 ± 0.3 kg. Conversely, during the unprocessed diet, participants lost an average of 1.1 ± 0.3 kg. The study involved 20 weight-stable adults with a mean age of 31.2 ± 1.6 years and a body mass index of 27 ± 1.5 kg/m². It was an inpatient randomized controlled trial where subjects received either ultra-processed or unprocessed diets for 2 weeks, followed by the alternate diet for another 2 weeks. Meals were carefully matched for presented calories, energy density, macronutrients, sugar, sodium, and fiber, and participants were allowed to eat as much or as little as desired.

A study by Figueiredo et al. [89] found a significant positive association between increased UPF intake and a higher risk of non-restrictive eating disorder types, specifically bulimic, binge eating, and 'other' eating disorders. For every 10-percentage point increase in UPF intake, the odds ratios were 1.08 for bulimic eating disorder, 1.21 for binge eating disorder, and 1.11 for 'other' eating disorders. The highest risk was observed for binge eating disorder, with a 21% higher risk compared to individuals without eating disorder. No significant association was detected between UPF intake and restrictive eating disorders. The final sample included 43,993 participants from the French NutriNet-Santé web-cohort. The mean age was 51.0 years, and 76.1% were women. Out of the total participants, 5,967 (13.6%) were categorized as likely eating disorder cases. This included 444 with restrictive eating disorder, 1,575 with bulimic eating disorder, 3,124 with binge eating disorder, and 824 with 'other' eating disorder. The average UPF intake constituted $16.0\% \pm 8.0\%$ of the total food consumed by the participants. The main results regarding the association between UPF intake and eating disorder types generally remained consistent across sensitivity analyses. When excluding participants using medication for anxiety/depression, the associations for bulimic and 'other' eating disorders became statistically non-significant, while binge eating disorder remained significantly associated. Excluding participants with fewer than six 24-hour dietary records did not substantially change the main results. This epidemiological study, the first of its kind using NOVA-derived UPF intake in a large adult French population, establishes significant positive associations between UPF consumption and several types of eating disorders, specifically bulimic, binge eating, and 'other' eating disorders. While restrictive eating disorder showed no such link, the findings highlight the potential detrimental impact of UPF on mental health outcomes related to eating behaviors.

A study by Lopes et al. [90] involved 3414 participants, with a significant majority (88.1%) being female. The average age was 56.7 ± 11.8 years, and participants had an average of 7.2 ± 4.1 years of education. On average, individuals had participated in the Health Academy Program for 19.7 ± 15.3 months. More than half (61%) of the participants had recently attempted to lose weight. At baseline, the mean contribution of calories from UPF consumption in the highest quartile (fourth quartile) was 47.7% (95% CI 47.2% to 48.3%). In contrast, the first quartile (lowest consumption) showed a 9.7% contribution (95% CI 9.4% to 10%). There was no significant difference in UPF caloric contribution between the control and intervention groups ($p = 0.406$). When stratifying UPF consumption by quartiles, individuals in the control group with higher UPF consumption (last quartile) were older (55.3 ± 12.1 years vs 52.9 ± 12.4 years; $p = 0.021$), had higher education levels (8.1 ± 4.1 years vs 7.8 ± 3.9 years; $p < 0.001$), and higher income (BRL 1017.5 ± 874.0 vs BRL 830.0 ± 668.9 ; $p = 0.001$). They also had a longer participation time in Health Academy Program (20.7 ± 16.2 months vs 15.5 ± 13.5 months; $p < 0.001$) compared to their counterparts in the intervention group. Additionally, the control group's last quartile had a lower prevalence of weight loss attempts (62.3% vs 64.2%; $p = 0.047$). Among individuals in the fourth quartile, which represented the highest UPF consumption, there was a positive weight variation of 0.178 kg (95% CI: -0.081 to 0.436). The difference between the control group and intervention group in this quartile was notable, with the control group experiencing -0.096 kg (95% CI -0.459 ; 0.267) and the intervention group experiencing 0.531 kg (95% CI 0.169; 0.894; $p = 0.018$). After adjusting for various factors, a comparison with the lowest UPF consumption group (first quartile) revealed that individuals in the second, third, and fourth quartiles experienced positive weight variations. Specifically,



these variations were 0.363 kg (95% CI 0.038; 0.689; $p = 0.029$) for the second quartile, 0.467 kg (95% CI 0.159; 0.776; $p = 0.003$) for the third quartile, and 0.389 kg (95% CI 0.061; 0.717; $p = 0.020$) for the fourth quartile. No significant differences were observed between the control and intervention groups in these adjusted analyses. In summary, the study found a clear association between higher UPF consumption and positive weight change (weight gain) over a 12-month period, particularly in the second, third, and fourth quartiles of consumption compared to the lowest quartile (Figure 1). This relationship persisted even after adjusting for various covariates, highlighting the impact of UPFs on body weight in the studied population.

A study by Hamano et al. [91] demonstrated that consuming UPFs leads to significant weight gain and increased energy intake, which is also associated with a decreased chewing frequency. These results suggest that reducing UPF consumption could be a valuable strategy in medical nutritional therapy for obesity prevention. All 9 eligible participants who were randomly assigned to either UPF or non-UPF diets completed the study. Participants experienced a significant weight gain of 1.1 kg more during the UPF consumption period compared to the non-UPF period. This finding had a 95% CI of 0.2 to 2.0 and a p -value of 0.021. Daily energy intake was substantially higher during the UPF period, with participants consuming an average of 813.5 kcal more per day. The 95% CI for this difference was 342.4 to 1284.7, and the P -value was 0.0041. The study observed a significant reduction in chewing frequency, specifically the number of chews per calorie, during the UPF consumption period ($p = 0.016$) (Figure 2).

A study by Dicken et al. [92] concluded that while both minimally processed food (MPF) and UPF diets adhering to national dietary guidelines resulted in weight loss, the MPF diet led to significantly greater weight loss and more favorable changes in body composition and craving control (Figure 3). Both MPF and UPF diets, when following UK dietary guidelines, resulted in weight loss over 8 weeks. Specifically, the MPF diet led to a mean percentage weight change (%WC) of -2.06% (95% CI $-2.99, -1.13$), while the UPF diet resulted in -1.05% (95% CI $-1.98, -0.13$). The MPF diet demonstrated significantly greater weight loss compared to the UPF diet, with a within-participant difference in %WC of -1.01% (95% CI $-1.87, -0.14$; $p = 0.024$). Both diets led to significantly lower weight and BMI at 8 weeks from baseline. However, reductions in weight (-0.96 kg) and

body mass index (-0.34 kg/m²) were significantly greater on the MPF diet compared to the UPF diet. Waist circumference did not show significant differences between diets. Fat mass, body fat percentage, visceral fat rating, and total body water mass were significantly lower at 8 weeks on the MPF diet, but not on the UPF diet. These reductions were significantly greater on the MPF diet compared to the UPF diet. Other body composition measures, such as muscle mass and bone mass, did not differ significantly between diets. Systolic and diastolic blood pressure were significantly lower on the MPF diet but not the UPF diet. Heart rate was significantly lower on the UPF diet but not the MPF diet. Changes in blood pressure and heart rate did not differ significantly between diets. Total cholesterol, HDL-C, and non-HDL-C were significantly lower on both diets at 8 weeks. Triglycerides and glycated hemoglobin were significantly lower on the MPF diet only, while fasting glucose and LDL-C were significantly lower on the UPF diet only. Changes in triglycerides were significantly lower on the MPF than UPF diet, and changes in LDL-C were significantly lower on the UPF than MPF diet. The Power of Food Scale scores (food present, tasted, and total) and control of eating questionnaire (CoEQ) measures (craving for sweet, savory, and difficulty resist craved nominated food) were significantly lower at 8 weeks on the MPF diet, but not the UPF diet. CoEQ craving control was significantly higher on both diets. Improvements in CoEQ craving for savory, difficulty to resist craving nominated food, and craving control were significantly greater on the MPF diet than the UPF diet. Self-reported energy intake was significantly lower during both MPF and UPF diets compared to baseline, but significantly more so on the MPF diet (-327.3 kcal/day). MPF adherence was 84.5% and UPF adherence was 91.2%. Adherence was higher in the first periods of the crossover. While there were no significant differences in overall diet ratings, flavors and tastes, and delivery and preparation were rated significantly lower on the MPF versus UPF diet. Mild gastrointestinal adverse events were common on both diets but did not differ significantly by diet. Notably, constipation, dyspepsia/gastroesophageal reflux, fatigue, sleep-related adverse events, and infections were more frequently reported on the UPF diet.

Assessment Tools for UPF Addiction and Withdrawal

The assessment of UPFs addiction and withdrawal is a growing area of research, reflecting the increasing recognition of the addictive potential of these foods. Various tools have been developed to measure

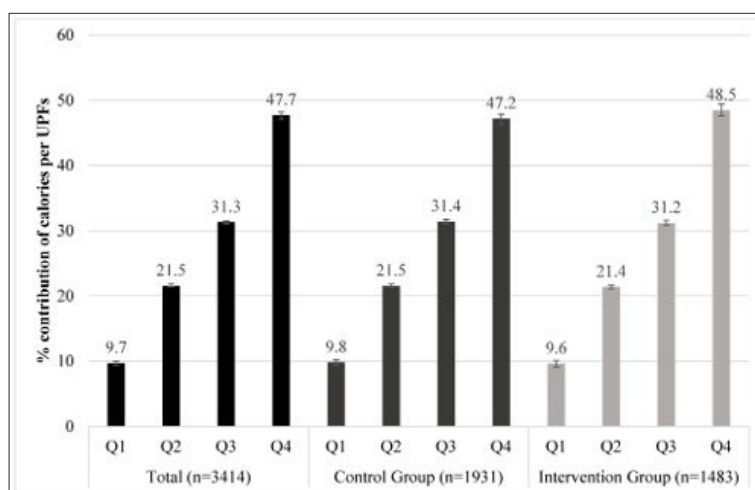


Figure 1: Baseline caloric contribution from UPFs in a fruit and vegetable community-based randomized controlled trial, Belo Horizonte-MG, Brazil (2013 - 2017) [90].

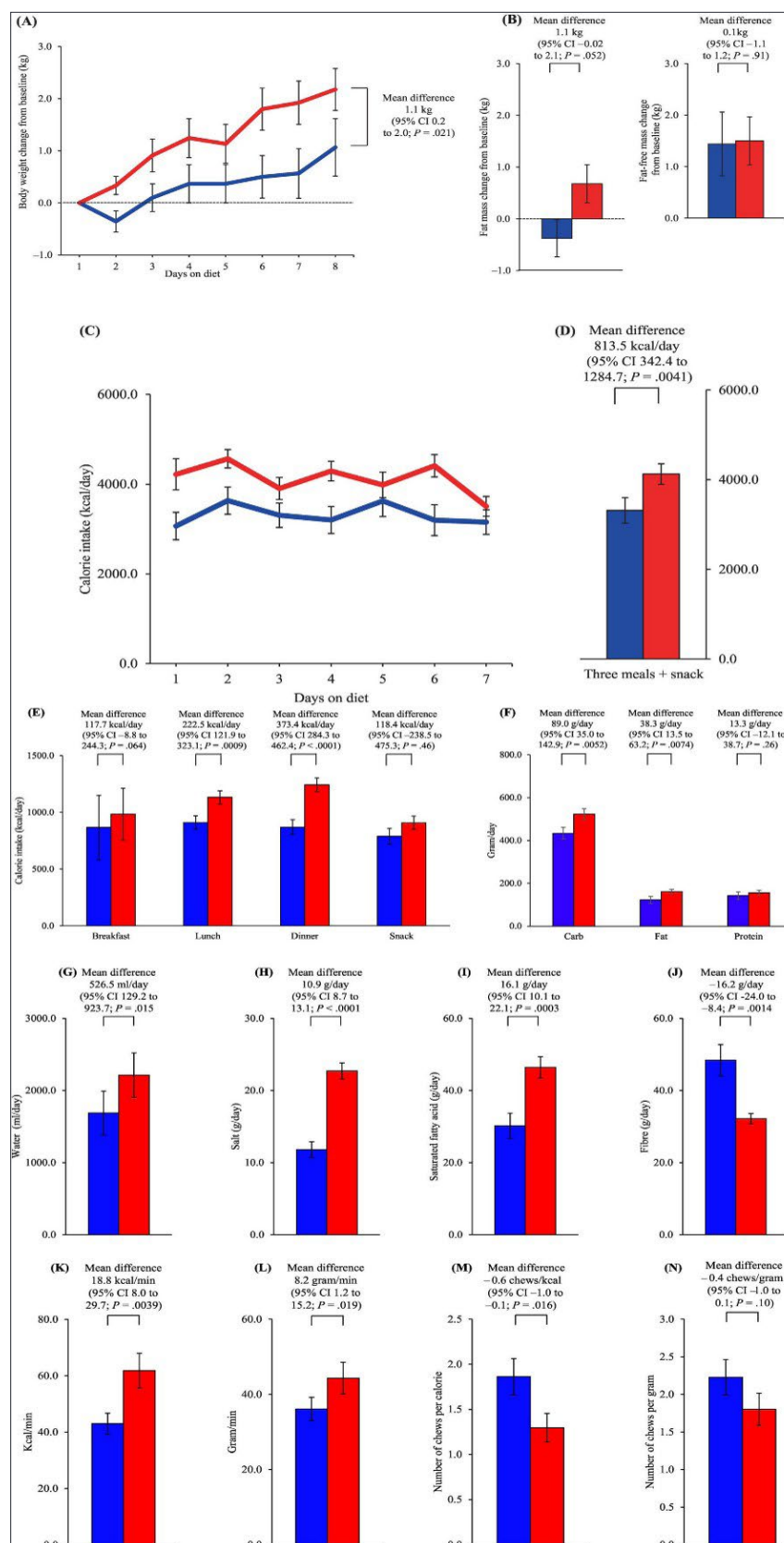


Figure 2: Paired analyses were conducted to assess changes in body weight (A), body composition (B), and dietary intake patterns. These included daily and average calorie intake (C, D), meal-specific calorie consumption (E), and intake of macronutrients (F), water (G), salt (H), saturated fatty acids (I), and fiber (J). Eating microstructure was analyzed via calories (K) and grams (L) ingested per minute, and chews per calorie (M) and per gram (N) [91].

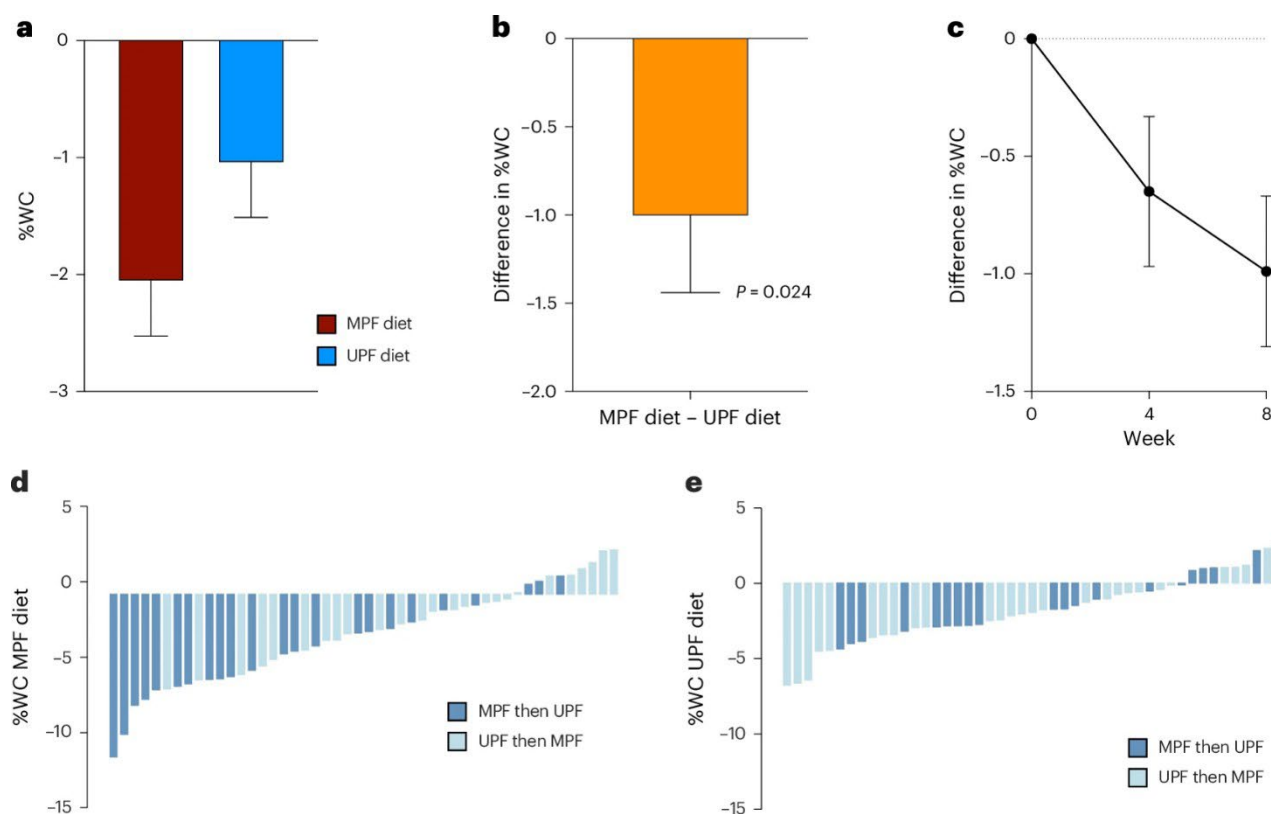


Figure 3: Estimated marginal means (and standard errors) for %WC were derived from mixed-effects models, adjusted for randomization arm and nightshift status, with a diet-by-arm interaction term and a participant-level random effect (Intention-to-treat N = 50). Panel (a) shows %WC on MPF and UPF diets. Panel (b) displays the mean difference in %WC between diets, with a 2-sided t-test (df = 46.1) indicating a significant reduction on the MPF diet (Cohen's d = -0.48, 95% CI -0.91, -0.06; p = 0.024, uncorrected). Panel (c) presents the estimated difference from the repeated-measures model. Panels (d) and (e) show unadjusted individual %WC values on the MPF and UPF diets, respectively [92].

Table 2: Overview of assessment instruments for UPF addiction and withdrawal symptoms.

Tool name (abbreviation)	Primary purpose and constructs measured	Key features and validation	Clinical and research utility
Yale Food Addiction Scale 2.0 (YFAS 2.0)	Diagnoses UPF/food addiction based on DSM-5 criteria for substance use disorders (e.g., loss of control, continued use despite consequences, cravings).	- Widely validated, high internal reliability. - Used in diverse populations (e.g., US older adults, adolescents). - Quantifies symptom count and provides diagnostic threshold.	Gold-standard for epidemiological and clinical studies; helps establish prevalence and correlates of UPF addiction.
mProWS	Measures physical and psychological withdrawal symptoms upon reducing UPF intake (e.g., irritability, cravings, fatigue).	7-item short form derived from 29-item ProWS. - Validated for reliability and convergent validity with YFAS 2.0. - Designed for low participant burden (e.g., ecological momentary assessment).	Useful in intervention studies to monitor withdrawal; supports addiction model by quantifying abstinence effects.
Food Addiction Symptom Inventory (FASI)	Structured clinical assessment of UPF addiction symptoms and severity.	- Developed clinical settings to aid diagnosis. - Complements YFAS 2.0 with a focus on practical, in-clinic application.	Help clinicians systematically evaluate UPF addiction, especially in patients with eating or substance use disorders.
Power of Food Scale (PFS)	Measures appetite for, and psychological responsiveness to, HPFs in the environment.	- Assesses hedonic hunger (food present, food tasted). - Used in studies linking UPF intake to craving and reward sensitivity.	Captures susceptibility to food cues, which is elevated in UPF addiction and binge-type eating disorders.
CoEQ	Assesses cravings, control overeating, and mood related to food intake over previous 7 days.	- Includes subscales for craving types (sweet, savory) and difficulty resisting food. - Sensitive to dietary interventions (e.g., MPF vs UPF diets).	Tracks changes in craving and control during dietary trials; useful for evaluating treatment efficacy.
NOVA food classification system	Categorizes foods by degree of processing (Group 4 = ultra-processed).	- Not a psychometric tool, but a framework for quantifying UPF exposure in diets via dietary records. - Enables calculation of % calories from UPFs.	Essential for operationalizing 'UPF intake' in research; links consumption levels to health outcomes.

addiction-like symptoms and withdrawal effects associated with UPFs (Table 2). These tools are crucial for understanding the impact of UPFs on health and for developing interventions to address food addiction.

- mProWS: The mProWS is a concise 7-item scale derived from the original 29-item ProWS. It measures physical and psychological

withdrawal symptoms when reducing UPF consumption. It has been validated for reliability and convergent validity with addictive-like eating behaviors, such as those measured by the Yale Food Addiction Scale 2.0 (YFAS 2.0). The mProWS is particularly useful in studies with high participant burden, such as ecological momentary assessments, due to its brevity [93].



- **Yale Food Addiction Scale 2.0 (YFAS 2.0):** The YFAS 2.0 is a widely used tool for diagnosing UPF addiction, applying DSM-5 criteria for substance use disorders to food consumption [94]. It has demonstrated high internal reliability and construct validity, making it a robust measure for assessing food addiction across various populations [95]. The scale has been used to identify the prevalence of UPF addiction in different demographic groups, such as older adults in the USA, where it showed a higher prevalence among women [87].
- **Assessment in specific populations:** Research has shown that UPF addiction is prevalent among adolescents and is linked to obesity and other health issues. Assessment tools like the YFAS 2.0 are used to study these patterns and guide preventive measures [96]. During the COVID-19 pandemic, UPF addiction was found to mediate the relationship between psychological stress and emotional eating, highlighting the importance of addressing food addiction in public health strategies [97].
- **Withdrawal symptoms and research directions:** Withdrawal symptoms from UPFs are a critical component of addiction assessment. Evidence suggests that both animals and humans experience withdrawal-like symptoms, although more experimental research is needed to fully understand these effects in humans. Future research should focus on longitudinal studies to better understand the addictive potential of UPFs and their impact on health, particularly in children [12].

In summary the tools mentioned provide valuable insights into UPF addiction and withdrawal, there is ongoing debate about the classification of food addiction as a substance-based or behavioral addiction. This distinction has implications for clinical and policy approaches to addressing UPF consumption. Additionally, the NOVA classification system, which categorizes foods based on processing levels, may need refinement to better align with findings on food addiction.

Environmental and Societal Influences on UPF Consumption

The consumption of UPFs has become a significant concern globally, driven by a complex interplay of environmental, societal, and individual factors. The literature underscores that these influences are deeply embedded within broader socio-economic and environmental contexts, which shape dietary patterns and health outcomes across populations. Environmental factors play a pivotal role in the proliferation of UPF consumption. Matos et al. [98] highlight the rapid growth of the UPF market in Latin America, emphasizing how environmental and social determinants contribute to increased intake. The expansion of UPF industries, coupled with environmental impacts such as increased greenhouse gas emissions, water, and land use, further exacerbate sustainability concerns. García et al. [99] provide longitudinal evidence linking higher UPF consumption to increased greenhouse gas emissions, water, energy, and land use, illustrating the environmental footprint of dietary shifts towards UPFs. Similarly, Kesse-Guyot et al. [100] explore environmental impacts along the entire value chain of UPF consumption, reinforcing the notion that environmental sustainability is compromised by the widespread adoption of these foods.

Societal influences are equally significant. Socioeconomic disparities, educational inequalities, and cultural factors shape UPF consumption patterns. Crepaldi et al. [101] examine how educational inequality intersects with sex and race/skin color in Brazil, revealing

that marginalized groups tend to consume more UPFs, often due to limited access to MPFs. This pattern is echoed in Cunha et al. [102], who utilize a socioecological model to analyze Brazilian adolescents' dietary behaviors, finding that individual and environmental factors collectively influence high UPF intake. Woods et al. [103] further emphasize that in Canada, nearly half of daily caloric intake comes from UPFs, with socio-economic status and food environment playing crucial roles. These societal factors are compounded by marketing strategies and the availability of UPFs, which often target vulnerable populations, thereby perpetuating health disparities. Cultural and behavioral factors also influence UPF consumption. Quinn et al. [104] discuss how dietary guidelines often fail to adequately communicate the harms associated with processed foods, which may influence public perceptions and behaviors. The rationales embedded within dietary advice can either upstreamly address broader determinants or downstreamly focus on individual choices, affecting societal attitudes towards UPFs. Moreover, the consumption of plant-based meat alternatives, as studied by Ohlau et al. [105], indicates a shift in dietary patterns that may be driven by sustainability concerns but also raises questions about the degree of processing involved and its implications for health and environmental sustainability.

Health-related societal impacts are evident in the association between UPF consumption and non-communicable diseases. Matos et al. [98] link high UPF intake to obesity, type 2 diabetes, hypertension, cardiovascular diseases, and cancer in Latin America. These health outcomes are further supported by Wang et al. [106], who demonstrate that maternal consumption of UPFs increases the risk of offspring developing overweight or obesity, highlighting intergenerational health effects. Woods et al. [103] also note the increased risk of chronic diseases associated with UPF consumption in Canada, emphasizing the societal burden of dietary patterns dominated by processed foods. The environmental and societal influences are interconnected, as the production and consumption of UPFs contribute significantly to environmental degradation while simultaneously perpetuating health inequalities. For instance, Garzillo et al. [107] show that higher UPF intake correlates with increased diet water footprints, which are primarily driven by energy intake associated with processed foods. Similarly, de Cruz et al. [108] evaluate the environmental impacts of beef and UPF consumption in Brazil, illustrating how dietary choices influence ecological sustainability.

Addressing these multifaceted influences requires comprehensive policy and societal interventions. Quinn et al. [104] advocate for improved communication within dietary guidelines to better inform the public about the harms of UPFs, considering upstream determinants such as food environment and marketing. The socioecological model applied by Cunha et al. [102] underscores the importance of multi-level strategies that target individual behaviors, community environments, and broader policy frameworks to reduce UPF consumption.

In summary, the literature demonstrates that environmental and societal factors are deeply intertwined in shaping UPF consumption patterns. Environmental impacts, such as increased resource use and greenhouse gas emissions, are compounded by societal inequalities that limit access to healthier, MPFs. Cultural norms, marketing, and policy gaps further influence dietary behaviors, contributing to the global rise in non-communicable diseases linked to UPF intake. Effective mitigation strategies must therefore adopt a holistic approach, addressing both environmental sustainability and social equity to promote healthier and more sustainable dietary patterns.



Conclusion

The evidence in this review strongly suggests that UPFs, particularly those engineered to be hyper-palatable, exhibit addictive properties capable of driving compulsive consumption patterns. Convergent findings from neurobiological, behavioral, epidemiological, and clinical studies indicate that UPFs activate reward pathways, reinforce intake, and can lead to maladaptive eating behaviors that mirror substance use disorders. This addictive potential is underpinned by specific food formulations, impaired inhibitory control, and measurable withdrawal symptoms, supporting the conceptualization of UPF addiction as a distinct and clinically relevant condition.

Recognizing the addictive nature of UPFs carries significant implications for public health policy, clinical practice, and future research. There is a pressing need for regulatory measures—such as clearer labeling, marketing restrictions, and reformulation standards—akin to those applied to other addictive substances. Clinically, integrating validated assessment tools and adapting treatment strategies from addiction medicine, including abstinence-based or harm-reduction approaches, may improve outcomes for individuals with UPF addiction and comorbid eating disorders. Future investigations should prioritize longitudinal and mechanistic research to elucidate causal pathways, while interdisciplinary efforts must address the environmental and socioeconomic drivers that perpetuate UPF consumption. Ultimately, a comprehensive strategy that bridges science, policy, and clinical innovation is essential to mitigate the profound impact of UPFs on individual health and societal well-being.

Acknowledgments

None.

Conflict of Interest

None.

References

- Mesas AE, Giroto E, Rodrigues R, Martínez-Vizcaino V, Jiménez-López E, et al. (2024) Ultra-processed food consumption is associated with alcoholic beverage drinking, tobacco smoking, and illicit drug use in adolescents: a nationwide population-based study. *Int J Ment Health Addict* 22: 3109-3132. <https://doi.org/10.1007/s11469-023-01038-6>
- Çelik ÖM, Güler Ü, Ekici EM (2025) Factors affecting ultra-processed food consumption: hedonic hunger, food addiction, and mood. *Food Sci Nutr* 13: e70248. <https://doi.org/10.1002/fsn3.70248>
- Temple NJ (2024) Making sense of the relationship between ultra-processed foods, obesity, and other chronic diseases. *Nutrients* 16: 4039. <https://doi.org/10.3390/nu16234039>
- Wiss DA, LaFata EM (2025) Structural equation modeling of adverse childhood experiences, ultra-processed food intake, and symptoms of post-traumatic stress disorder, ultra-processed food addiction, and eating disorder among adults seeking nutrition counseling in Los Angeles, CA. *Appetite* 208: 107938. <https://doi.org/10.1016/j.appet.2025.107938>
- McCausland HC, Gearhardt AN, Peralta JM, LaFata EM (2025) A critical evaluation of the terms used to describe foods implicated in addictive-like eating. *Curr Addict Rep* 12: 1-14. <https://doi.org/10.1007/s40429-025-00689-w>
- Monteiro CA, Louzada ML, Steele-Martinez E, Cannon G, Andrade GC, et al. (2025) Ultra-processed foods and human health: the main thesis and the evidence. *Lancet* 406: 2667-2684. [https://doi.org/10.1016/s0140-6736\(25\)01565-x](https://doi.org/10.1016/s0140-6736(25)01565-x)
- Ahmed A, Imran A, Wei CR, Nadeem R, Shankar A, et al. (2024) Contribution of ultra-processed foods to increased obesity and non-communicable diseases. *Cogent Food Agric* 10: 1-13. <https://doi.org/10.1080/23311932.2024.2438405>
- Machado-Rodrigues AM, Padez C, Rodrigues D, Dos Santos EA, Baptista LC, et al. (2024) Ultra-processed food consumption and its association with risk of obesity, sedentary behaviors, and well-being in adolescents. *Nutrients* 16: 3827. <https://doi.org/10.3390/nu16223827>
- Yates J, Kadiyala S, Deeney M, Carriedo A, Gillespie S, et al. (2024) A toxic relationship: ultra-processed foods & plastics. *Glob Health* 20: 1-7. <https://doi.org/10.1186/s12992-024-01078-0>
- Unwin J, Giaeever H, Avena N, Kennedy C, Painschab M, et al. (2025) Toward consensus: using the Delphi method to form an international expert consensus statement on ultra-processed food addiction. *Front Psychiatry* 16: 1-6. <https://doi.org/10.3389/fpsy.2025.1542905>
- Wilcox CE (2021) Neurobiology and cognitive neuroscience of hedonic eating. In *Food addiction, obesity, and disorders of overeating: an evidence-based assessment and clinical guide*. Springer International Publishing, pp 109-125.
- Gearhardt AN, Schulte EM (2021) Is food addictive? A review of the science. *Annu Rev Nutr* 41: 387-410. <https://doi.org/10.1146/annurev-nutr-110420-111710>
- Gearhardt AN, DiFeliceantonio AG (2023) Highly processed foods can be considered addictive substances based on established scientific criteria. *Addiction* 118: 589-598. <https://doi.org/10.1111/add.16065>
- Contreras-Rodriguez O, Solanas M, Escorihuela RM (2022) Dissecting ultra-processed foods and drinks: do they have a potential to impact the brain? *Rev Endocr Metab Disord* 23: 697-717. <https://doi.org/10.1007/s11154-022-09711-2>
- Kelly AL, Baugh ME, Oster ME, DiFeliceantonio AG (2022) The impact of caloric availability on eating behavior and ultra-processed food reward. *Appetite* 178: 106274. <https://doi.org/10.1016/j.appet.2022.106274>
- Ho D, Verdejo-Garcia A (2021) Interactive influences of food, contexts and neurocognitive systems on addictive eating. *Prog Neuropsychopharmacol Biol Psychiatry* 110: 110295. <https://doi.org/10.1016/j.pnpbp.2021.110295>
- Vasiliu O (2022) Current status of evidence for a new diagnosis: food addiction—a literature review. *Front Psychiatry* 12: 1-10. <https://doi.org/10.3389/fpsy.2021.824936>
- Mehr JB, Mitchison D, Bowrey HE, James MH (2021) Sleep dysregulation in binge eating disorder and “food addiction”: the orexin (hypocretin) system as a potential neurobiological link. *Neuropsychopharmacology* 46: 2051-2061. <https://doi.org/10.1038/s41386-021-01052-z>
- Srouf B, Kordahi MC, Bonazzi E, Deschasaux-Tanguy M, Touvier M, et al. (2022) Ultra-processed foods and human health: from epidemiological evidence to mechanistic insights. *Lancet Gastroenterol Hepatol* 7: 1128-1140. [https://doi.org/10.1016/s2468-1253\(22\)00169-8](https://doi.org/10.1016/s2468-1253(22)00169-8)
- Forde CG (2023) Beyond ultra-processed: considering the future role of food processing in human health. *Proc Nutr Soc* 82: 406-418. <https://doi.org/10.1017/s0029665123003014>
- Ifland J, Brewerton TD (2025) Binge-type eating disorders and ultra-processed food addiction: phenomenology, pathophysiology and treatment implications. *Front Psychiatry* 16: 1-10. <https://doi.org/10.3389/fpsy.2025.1584891>
- Hu S, Gearhardt AN, LaFata EM (2024) Development of the modified highly processed food withdrawal scale (mProWS). *Appetite* 198: 107370. <https://doi.org/10.1016/j.appet.2024.107370>
- Hebebrand J, Albayrak Ö, Adan R, Antel J, Dieguez C, et al. (2014) “Eating addiction”, rather than “food addiction”, better captures addictive-like eating behavior. *Neurosci Biobehav Rev* 47: 295-306. <https://doi.org/10.1016/j.neubiorev.2014.08.016>
- Wiss DA, LaFata EM (2024) Ultra-processed foods and mental health: where do eating disorders fit into the puzzle? *Nutrients* 16: 1955. <https://doi.org/10.3390/nu16121955>
- Vásquez F, Tiscornia C, Aicardi V, Vásquez S (2026) Dietary transitions and the rising global burden of chronic kidney disease: insights from nutritional epidemiology. *Nutrients* 18: 911. <https://doi.org/10.3390/nu18060911>
- Dong L, Rashkova I (2024) Healthy Food Consumption: Challenges and the Path Forward. In Tang CS (ed) *Responsible and Sustainable Operations: The New Frontier*. Springer Nature, Switzerland, pp 119-143.
- Monda A, de Stefano MI, Villano I, Allocca S, Casillo M, et al. (2024) Ultra-processed food intake and increased risk of obesity: a narrative review. *Foods* 13: 2627. <https://doi.org/10.3390/foods13162627>
- White JS (2014) Sucrose, HFCS, and Fructose: History, Manufacture, Composition, Applications, and Production. In Rippe J (ed) *Fructose, High Fructose Corn Syrup, Sucrose and Health*. Springer, New York, pp 13-33.
- Gardner C, Wylie-Rosett J, Gidding SS, Steffen LM, Johnson RK, et al. (2012) Nonnutritive sweeteners: current use and health perspectives: a scientific statement



- from the American Heart Association and the American Diabetes Association. *Circulation* 126: 509-519. <https://doi.org/10.2337/dc12-9002>
30. Hull SC, Mszar R, Ostfeld RJ, Ferrucci LM, Mucci LA, et al. (2025) Diet and prevention of cardiovascular disease and cancer: JACC: cardiooncology state-of-the-art review. *Cardio Oncol* 7: 649-667. <https://doi.org/10.1016/j.jacc.2025.07.008>
 31. Ramasinghe C, Bordiga M, Xu B (2025) A comprehensive review of the triangular relationship among diet, gut microbiota, and aging. *Int J Mol Sci* 26: 8785. <https://doi.org/10.3390/ijms26188785>
 32. Davidson TL, Jones S, Roy M, Stevenson RJ (2019) The cognitive control of eating and body weight: it's more than what you "think". *Front Psychol* 10: 1-22. <https://doi.org/10.3389/fpsyg.2019.00062>
 33. Francis HM, Stevenson RJ (2015) The Effect of Western Diet on Cognition in Humans. In *Diet and Exercise in Cognitive Function and Neurological Diseases*.
 34. Fazzino TL, Dorling JL, Apolzan JW, Martin CK (2021) Meal composition during an ad libitum buffet meal and longitudinal predictions of weight and percent body fat change: the role of hyper-palatable, energy dense, and ultra-processed foods. *Appetite* 167: 105592. <https://doi.org/10.1016/j.appet.2021.105592>
 35. Demeke S, Rohde K, Chollet-Hinton L, Sutton C, Kong KL, et al. (2023) Change in hyper-palatable food availability in the US food system over 30 years: 1988–2018. *Public Health Nutr* 26: 182-189. <https://doi.org/10.1017/s1368980022001227>
 36. Ghosh I, Gutka H, Krause ME, Clemens R, Kashi RS (2023) A systematic review of commercial high concentration antibody drug products approved in the US: formulation composition, dosage form design and primary packaging considerations. *mAbs* 15: 1-20. <https://doi.org/10.1080/19420862.2023.2205540>
 37. Ahrné L, Chen H, Henry CJ, Kim HS, Schneeman B, et al. (2025) Defining the role of processing in food classification systems—the IUFOST formulation & processing approach. *NPJ Sci Food* 9: 1-17. <https://doi.org/10.1038/s41538-025-00395-x>
 38. Jun D, Knowles K, Fazzino TL (2025) Examination of hyper-palatable foods and their nutrient characteristics using globally crowdsourced data. *PLoS One* 20: 1-16. <https://doi.org/10.1371/journal.pone.0325479>
 39. Sutton CA, Stratton M, L'Insalata AM, Fazzino TL (2024) Ultraprocessed, hyper-palatable, and high energy density foods: prevalence and distinction across 30 years in the United States. *Obesity* 32: 166-175. <https://doi.org/10.1002/oby.23897>
 40. Could a Definition of 'Hyper-palatable' Food Help the Food Industry Create Healthy Products? [<https://insights.figlobal.com/diet-health/could-a-definition-of-hyper-palatable-food-help-the-food-industry-create-healthy-products->] [Accessed September 15, 2026]
 41. Ben-Porat T, Avshalom T, Mahajna H, Kaloti D, Subhi H, et al. (2026) Binge eating and addictive-like eating behaviors seven years after sleeve gastrectomy: implications for long-term weight loss outcomes. *Obes Surg* 36: 3675-3684. <https://doi.org/10.1007/s11695-026-08751-w>
 42. The Bliss Effect in Ultra-Processed Food Formulation. *FoodTimes*. [<https://www.foodtimes.eu/consumers-and-health/bliss-effect-ultra-processed-food/>] [Accessed September 15, 2026]
 43. Ilieva G, Yankova T, Ruseva M, Dzhabarova Y, Klisarova-Belcheva S, et al. (2025) Consumer perceptions and attitudes towards ultra-processed foods. *Appl Sci* 15: 3739. <https://doi.org/10.3390/app15073739>
 44. Nguyen L, Walters J, Spies B, Coppus A, Massie J, et al. (2025) Food for thought: a systematic review and meta-analysis on the effects of ultra-processed foods on cognition in children and adolescents. *Food Front* 6: 1838-1866. <https://doi.org/10.1002/fft2.70064>
 45. Ekici EM, Eroğlu FE, Çelik ÖM (2025) Association between ultra-processed food consumption, carbon footprint awareness and sustainable and healthy eating behaviors in adult: a cross-sectional study. *BMC Public Health* 25: 1-13. <https://doi.org/10.1186/s12889-025-22918-7>
 46. Heuchan GN, Buck C, Conway R, Dicken S, Brown AC, et al. (2025) Development, content and planned evaluation of a behavioural support intervention to reduce ultraprocessed food intake and increase physical activity in UK healthcare workers: UPDATE trial stage 2 study protocol. *BMJ Open* 15: 1-12. <https://doi.org/10.1136/bmjopen-2025-107435>
 47. Via E, Contreras-Rodríguez O (2023) Binge-eating precursors in children and adolescents: neurodevelopment, and the potential contribution of ultra-processed foods. *Nutrients* 15: 2994. <https://doi.org/10.3390/nu15132994>
 48. Dicken S, Makaronidis J, van Tullemen C, Jassil FC, Hall K, et al. (2024) UPDATE trial: investigating the effects of ultra-processed versus minimally processed diets following UK dietary guidance on health outcomes: a protocol for an 8-week community-based cross-over randomised controlled trial in people with overweight or obesity, followed by a 6-month behavioural intervention. *BMJ Open* 14: 1-14. <https://doi.org/10.1136/bmjopen-2023-079027>
 49. Sun Y, Correia PE, Teixeira PP, Spiazzi BF, Brietzke E, et al. (2025) Ultra-processed food intake as an effect modifier in the association between depression and diabetes in Brazil: a cross-sectional study. *Nutrients* 17: 2454. <https://doi.org/10.3390/nu17152454>
 50. Wang ZY, Lu CC, Liu WD, Cui L, Deng XX, et al. (2024) Trends and hotspots of global ultra-processed food research (2010–2023): a scientometric study. *Food Health* 6: 1-15. <https://doi.org/10.53388/fh2024001>
 51. Cortes ML, Louzado JA, Oliveira MG, Bezerra VM, Mistro S, et al. (2021) Unhealthy food and psychological stress: the association between ultra-processed food consumption and perceived stress in working-class young adults. *Int J Environ Res Public Health* 18: 3863. <https://doi.org/10.3390/ijerph18083863>
 52. Teo PS, Lim AJ, Goh AT, Janani R, Choy JYM, et al. (2022) Texture-based differences in eating rate influence energy intake for minimally processed and ultra-processed meals. *Am J Clin Nutr* 116: 244-254. <https://doi.org/10.1093/ajcn/nqac068>
 53. Coletro HN, Mendonça RD, Meireles AL, Machado-Coelho GLL, de Menezes MC (2022) Ultra-processed and fresh food consumption and symptoms of anxiety and depression during the COVID-19 pandemic: COVID inconfindentes. *Clin Nutr ESPEN* 47: 206-214. <https://doi.org/10.1016/j.clnesp.2021.12.013>
 54. Lane MM, Lotfaliany M, Hodge AM, O'Neil A, Travica N, et al. (2023) High ultra-processed food consumption is associated with elevated psychological distress as an indicator of depression in adults from the Melbourne Collaborative Cohort Study. *J Affect Disord* 335: 57-66. <https://doi.org/10.1016/j.jad.2023.04.124>
 55. Lane KE, Davies IG, Darabi Z, Ghayour-Mobarhan M, Khayatzadeh SS, et al. (2022) The association between ultra-processed foods, quality of life and insomnia among adolescent girls in Northeastern Iran. *Int J Environ Res Public Health* 19: 6338. <https://doi.org/10.3390/ijerph19106338>
 56. Silva JB, Elias BC, Warkentin S, Mais LA, Konstantyner T (2022) Factors associated with the consumption of ultra-processed food by Brazilian adolescents: National Survey of School Health, 2015. *Rev Paul Pediatr* 40: 1-10. <https://doi.org/10.1590/1984-0462/2022/40/2020362>
 57. Santos GCJ, Fernandes MSS, Carniel PG, Garcêz AS, Leandro CG, et al. (2024) Dietary intake in children and adolescents with food addiction: a systematic review. *Addict Behav Rep* 19: 100531. <https://doi.org/10.1016/j.abrep.2024.100531>
 58. Araç E (2024) The intersection of obesity and behavioral addictions: a comprehensive narrative review of neurobiological mechanisms and clinical implications. *DAHUDER Med J* 5: 148-154. <https://doi.org/10.56016/dahudermj.1793707>
 59. Yuan S, Zhu T, Gu J, Hua L, Sun J, et al. (2025) Associations of ultra-processed food intake and its circulating metabolomic signature with mental disorders in middle-aged and older adults. *Nutrients* 17: 1582. <https://doi.org/10.3390/nu17091582>
 60. Neves ACM, Menezes-Júnior LAAD, Mendonça RDD, Meireles AL, Carraro JCC (2024) Anxiety symptoms influence food consumption differently depending on nutritional status during the COVID-19 pandemic: a longitudinal study with university students. *J Am Nutr Assoc* 43: 704-712. <https://doi.org/10.1080/27697061.2024.2378085>
 61. Zheng W, Xiong J, Huang B, Kong Q (2025) Associations between ultra-processed food consumption and duration of exercise with psychological symptoms in Chinese adolescents: a nationwide cross-sectional survey. *Front Nutr* 12: 1-16. <https://doi.org/10.3389/fnut.2025.1591909>
 62. Jesus JIFS, Monfort-Pañego M, Santos GVA, Monteiro YC, Nogueira SM, et al. (2025) Food, quality of life and mental health: a cross-sectional study with federal education workers. *Nutrients* 17: 2519. <https://doi.org/10.3390/nu17152519>
 63. Prescott SL, Logan AC, LaFata EM, Naik A, Nelson DH, et al. (2024) Crime and nourishment: a narrative review examining ultra-processed foods, brain, and behavior. *Dietetics* 3: 318-345. <https://doi.org/10.3390/dietetics3030025>
 64. Zhang S, Gan S, Zhang Q, Liu L, Meng G, et al. (2022) Ultra-processed food consumption and the risk of non-alcoholic fatty liver disease in the Tianjin Chronic Low-grade Systemic Inflammation and Health Cohort Study. *Int J Epidemiol* 51: 237-249. <https://doi.org/10.1093/ije/dyab174>
 65. Henney AE, Gillespie CS, Alam U, Hydes TJ, Cuthbertson DJ (2023) Ultra-processed food intake is associated with non-alcoholic fatty liver disease in adults: a systematic review and meta-analysis. *Nutrients* 15: 2266. <https://doi.org/10.3390/nu15102266>
 66. Grinshpan LS, Eilat-Adar S, Ivancovsky-Wajeman D, Kariv R, Gillon-Keren M, et al. (2024) Ultra-processed food consumption and non-alcoholic fatty liver disease,



- metabolic syndrome and insulin resistance: a systematic review. *JHEP Rep* 6: 100964. <https://doi.org/10.1016/j.jhepr.2023.100964>
67. Juul F, Deierlein AL, Vaidean G, Quatromoni PA, Parekh N (2022) Ultra-processed foods and cardiometabolic health outcomes: from evidence to practice. *Curr Atheroscler Rep* 24: 849-860. <https://doi.org/10.1007/s11883-022-01061-3>
 68. Lane MM, Gamage E, Du S, Ashtree DN, McGuinness AJ, et al. (2024) Ultra-processed food exposure and adverse health outcomes: umbrella review of epidemiological meta-analyses. *BMJ* 384. <https://doi.org/10.1136/bmj-2023-077310>
 69. Vissers E, Wellens J, Sabino J (2022) Ultra-processed foods as a possible culprit for the rising prevalence of inflammatory bowel diseases. *Front Med* 9: 1-11. <https://doi.org/10.3389/fmed.2022.1058373>
 70. Gubatan J, Kulkarni CV, Talamantes SM, Temby M, Fardeen T, et al. (2023) Dietary exposures and interventions in inflammatory bowel disease: current evidence and emerging concepts. *Nutrients* 15: 579. <https://doi.org/10.3390/nu15030579>
 71. Yao CK, Fitzpatrick J, Machado P, Staudacher HM (2024) Is there an “optimal” diet for prevention of inflammatory bowel disease? *JGH Open* 8: 1-11. <https://doi.org/10.1002/jgh3.70016>
 72. Kliemann N, Al Nahas A, Vamos EP, Touvier M, Kesse-Guyot E, et al. (2022) Ultra-processed foods and cancer risk: from global food systems to individual exposures and mechanisms. *Br J Cancer* 127: 14-20. <https://doi.org/10.1038/s41416-022-01749-y>
 73. Anastasiou IA, Kounatidis D, Vallianou NG, Skourtis A, Dimitriou K, et al. (2025) Beneath the surface: the emerging role of ultra-processed foods in obesity-related cancer. *Curr Oncol Rep* 27: 390-414. <https://doi.org/10.1007/s11912-025-01654-6>
 74. Fajkić A, Lepara O, Jahić R, Hadžović-Džuvo A, Belančić A, et al. (2025) Ultra-processed diets and endocrine disruption, explanation of missing link in rising cancer incidence among young adults. *Cancers* 17: 2196. <https://doi.org/10.3390/cancers17132196>
 75. Calvo MS, Dunford EK, Uribarri J (2023) Industrial use of phosphate food additives: a mechanism linking ultra-processed food intake to cardiorenal disease risk? *Nutrients* 15: 3510. <https://doi.org/10.3390/nu15163510>
 76. Astrup A, Monteiro CA (2022) Does the concept of “ultra-processed foods” help inform dietary guidelines, beyond conventional classification systems? *No. Am J Clin Nutr* 116: 1482-1488. <https://doi.org/10.1093/ajcn/nqac230>
 77. Oliveira J, Colombarolli MS, Cordas TA (2021) Prevalence and correlates of food addiction: systematic review of studies with the YFAS 2.0. *Obes Res Clin Pract* 15: 191-204. <https://doi.org/10.1016/j.orcp.2021.03.014>
 78. Praxedes DR, Silva-Júnior AE, Macena ML, Oliveira AD, Cardoso KS, et al. (2022) Prevalence of food addiction determined by the Yale Food Addiction Scale and associated factors: a systematic review with meta-analysis. *Eur Eat Disord Rev* 30: 85-95. <https://doi.org/10.1002/erv.2878>
 79. Sankaranarayanan A, Johnson K, Mammen SJ, Wilding HE, Vasani D, et al. (2021) Disordered eating among people with schizophrenia spectrum disorders: a systematic review. *Nutrients* 13: 3820. <https://doi.org/10.3390/nu13113820>
 80. Bodell LP, Racine SE (2023) A mechanistic staging model of reward processing alterations in individuals with binge-type eating disorders. *Int J Eat Disord* 56: 516-522. <https://doi.org/10.1002/eat.23875>
 81. Wiss D (2022) Clinical considerations of ultra-processed food addiction across weight classes: an eating disorder treatment and care perspective. *Curr Addict Rep* 9: 255-267. <https://doi.org/10.1007/s40429-022-00411-0>
 82. Dennis K, Barrera S, Bishop N, Nguyen C, Brewerton TD (2024) Food addiction screening, diagnosis and treatment: a protocol for residential treatment of eating disorders, substance use disorders and trauma-related psychiatric comorbidity. *Nutrients* 16: 2019. <https://doi.org/10.3390/nu16132019>
 83. LaFata EM, Worwag K, Derrigo K, Hessler C, Allison KC, et al. (2024) Development of the food addiction symptom inventory: the first clinical interview to assess ultra-processed food addiction. *Psychol Assess* 36: 654-664. <https://doi.org/10.1037/pas0001340>
 84. Thompson SP, Jacobs J, Wiss DA (2025) Abstinence-based treatment of comorbid eating disorders and ultra-processed food addiction. *Front Psychiatry* 16: 1-9. <https://doi.org/10.3389/fpsy.2025.1586490>
 85. Barros LDM, Peixoto VA, Carvalho GCO, Pereira MR, Carnaúba RTL, et al. (2025) Association between psychobehavioral factors and the increased eating rate of ultra-processed versus non-ultra-processed meals in individuals with obesity: a secondary analysis of a randomized trial. *Nutrients* 17: 2236. <https://doi.org/10.3390/nu17132236>
 86. Wiss DA, Tran CD, LaFata EM (2025) The association between cumulative adverse childhood experiences and ultra-processed food addiction is moderated by substance use disorder history among adults seeking outpatient nutrition counseling. *Front Psychiatry* 16: 1-11. <https://doi.org/10.3389/fpsy.2025.1543923>
 87. Loch LK, Kirch M, Singer DC, Solway E, Roberts JS, et al. (2025) Ultra-processed food addiction in a nationally representative sample of older adults in the USA. *Addiction* 121: 510-521. <https://doi.org/10.1111/add.70186>
 88. Hall KD, Ayuketah A, Brychta R, Cai H, Cassimatis T, et al. (2019) Ultra-processed diets cause excess calorie intake and weight gain: an inpatient randomized controlled trial of ad libitum food intake. *Cell Metab* 30: 67-77. <https://doi.org/10.1016/j.cmet.2020.08.014>
 89. Figueiredo N, Kose J, Srour B, Julia C, Kesse-Guyot E, et al. (2022) Ultra-processed food intake and eating disorders: cross-sectional associations among French adults. *J Behav Addict* 11: 588-599. <https://doi.org/10.1556/2006.2022.00009>
 90. Lopes MS, Freitas PPD, Lopes ACS (2025) The results of ultra-processed food consumption on weight change: a randomized controlled community trial in a health promotion program. *Nutrients* 17: 638. <https://doi.org/10.3390/nu17040638>
 91. Hamano S, Sawada M, Aihara M, Sakurai Y, Sekine R, et al. (2024) Ultra-processed foods cause weight gain and increased energy intake associated with reduced chewing frequency: a randomized, open-label, crossover study. *Diabetes Obes Metab* 26: 5431-5443. <https://doi.org/10.1111/dom.15922>
 92. Dicken SJ, Jassil FC, Brown A, Kalis M, Stanley C, et al. (2025) Ultraprocessed or minimally processed diets following healthy dietary guidelines on weight and cardiometabolic health: a randomized, crossover trial. *Nat Med* 31: 3297-3308. <https://doi.org/10.1038/s41591-025-03842-0>
 93. Varnado A, Smith A, Mason TB, Smith KE (2024) The ecological validity of the Yale Food Addiction Scale 2.0 and momentary food addiction symptoms. *Psychol Addict Behav* 38: 628-636. <https://doi.org/10.1037/adb0001014>
 94. Thompson SP, Thaw AK (2024) The Badly Behaving Brain: How Ultra-Processed Food Addiction Thwarts Sustained Weight Loss. In *Weight Loss - A Multidisciplinary Perspective*. Intechopen.
 95. Meule A, Gearhardt AN (2019) Ten years of the Yale Food Addiction Scale: a review of version 2.0. *Curr Addict Rep* 6: 218-228. <https://doi.org/10.1007/s40429-019-00261-3>
 96. Wang Z (2024) The effects of ultra-processed foods on food addiction in adolescents and the mechanism of prevention. *Highlights Sci Eng Technol* 109: 86-90. <https://doi.org/10.54097/vedd7v82>
 97. Stariolo JB, Lemos TC, Khandpur N, Pereira MG, Oliveira LD, et al. (2024) Addiction to ultra-processed foods as a mediator between psychological stress and emotional eating during the COVID-19 pandemic. *Psicol Reflex Crit* 37: 1-13. <https://doi.org/10.1186/s41155-024-00322-1>
 98. Matos RA, Adams M, Sabaté J (2021) The consumption of ultra-processed foods and non-communicable diseases in Latin America. *Front Nutr* 8: 1-10. <https://doi.org/10.3389/fnut.2021.622714>
 99. García S, Pastor R, Monserrat-Mesquida M, Álvarez-Álvarez L, Rubin-García M, et al. (2023) Ultra-processed foods consumption as a promoting factor of greenhouse gas emissions, water, energy, and land use: a longitudinal assessment. *Sci Total Environ* 891: 164417. <https://doi.org/10.1016/j.scitotenv.2023.164417>
 100. Kesse-Guyot E, Allès B, Brunin J, Fouillet H, Dussiot A, et al. (2023) Environmental impacts along the value chain from the consumption of ultra-processed foods. *Nat Sustain* 6: 192-202. <https://doi.org/10.1038/s41893-022-01013-4>
 101. Crepaldi BVC, Okada LM, Claro RM, Louzada MLDC, Rezende LF, et al. (2022) Educational inequality in consumption of in natura or minimally processed foods and ultra-processed foods: the intersection between sex and race/skin color in Brazil. *Front Nutr* 9: 1-13. <https://doi.org/10.3389/fnut.2022.1055532>
 102. Cunha RO, do Carmo AS, Silva UM, Canella DS (2025) Association between the food environment of Brazilian public and private schools and the consumption of ultra-processed foods: analysis of the National Student Health Survey (PeNSE) 2019. *Food Policy* 134: 102871. <https://doi.org/10.1016/j.foodpol.2025.102871>
 103. Woods N, Gilliland J, Seabrook JA (2023) Applicability of the Socioecological Model for understanding and reducing consumption of ultra-processed foods in Canada. *Can J Diet Pract Res* 84: 38-42. <https://doi.org/10.3148/cjdp-2022-027>
 104. Quinn M, Jordan H, Lacy-Nichols J (2021) Upstream and downstream explanations of the harms of ultra-processed foods in national dietary guidelines. *Public Health Nutr* 24: 5426-5435. <https://doi.org/10.1017/s1368980021003505>
 105. Ohlrau M, Spiller A, Risius A (2022) Plant-based diets are not enough? Understanding



- the consumption of plant-based meat alternatives along ultra-processed foods in different dietary patterns in Germany. *Front Nutr* 9: 1-17. <https://doi.org/10.3389/fnut.2022.852936>
106. Wang Y, Wang K, Du M, Khandpur N, Rossato SL, et al. (2022) Maternal consumption of ultra-processed foods and subsequent risk of offspring overweight or obesity: results from three prospective cohort studies. *BMJ* 379: e071767. <https://doi.org/10.1136/bmj-2022-071767>
107. Garzillo JMF, Poli VFS, Leite FHM, Steele EM, Machado PP, et al. (2022) Ultra-processed food intake and diet carbon and water footprints: a national study in Brazil. *Rev Saude Publica* 56: 1-9.
108. da Cruz GL, Louzada MLC, Silva JTD, Garzillo JMF, Rauber F, et al. (2024) The environmental impact of beef and ultra-processed food consumption in Brazil. *Public Health Nutr* 27: 1-10. <https://doi.org/10.1017/s1368980023002975>