

Food Noise in Obesity: Current Evidence, Measurement, and Clinical Implications

Yeimer Ortiz-Martínez ¹

1. Endocrinology, Pontificia Universidad Javeriana, Bogota, COL

Corresponding author: Yeimer Ortiz-Martínez, yeimerortiz@gmail.com

Review began 09/21/2026

Review ended 10/13/2026

Published 10/18/2026

© Copyright 2026

Ortiz-Martínez. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118095

Abstract

Food noise has emerged as a patient-derived term describing frequent, persistent, or intrusive food-related thoughts that may be unwanted, distressing, or disruptive. Although the expression has rapidly entered public discourse, particularly alongside reports of appetite changes during glucagon-like peptide-1 receptor agonist (GLP-1RA) treatment, its scientific boundaries remain unsettled. This narrative review summarizes current definitions, epidemiologic observations, measurement instruments, proposed mechanisms, differential diagnosis, and responses to obesity interventions. Two dedicated instruments have been published: the five-item Food Noise Questionnaire (FNQ) and the Ro Allison Indiana Dhurandhar Food Noise Inventory (RAID-FN). Both demonstrate promising initial psychometric properties, but neither has established diagnostic thresholds, minimal clinically important differences, predictive validity, or sufficient evidence of responsiveness to treatment. Consumer surveys and selected cross-sectional studies suggest that burdensome food-related cognition is common in some populations; however, representative prevalence estimates are unavailable. Randomized trials show that glucagon-like peptide-1-based therapies reduce appetite, cravings, food-cue reactivity, and energy intake, whereas direct evidence of reduced food noise is currently limited primarily to retrospective and cross-sectional observations. Food noise should therefore be regarded as an emerging clinical dimension rather than a diagnosis, biomarker, or validated treatment target. Prospective studies using validated instruments are required to determine whether it provides information beyond established constructs and whether its reduction independently predicts weight-management, mental-health, or quality-of-life outcomes.

Categories: Internal Medicine, Endocrinology/Diabetes/Metabolism

Keywords: appetite, craving, food noise, food-related cognition, glp-1 receptor agonist, obesity, semaglutide, tirzepatide

Introduction And Background

Thinking about food is universal and necessary. People anticipate meals, make food-related decisions, respond to hunger, and derive pleasure and social meaning from eating. These thoughts become clinically relevant when they are frequent, intrusive, unwanted, emotionally distressing, or persistent enough to interfere with concentration, daily activities, or health-related behaviors. The term food noise has recently been adopted to describe this experience [1-4].

Eating behavior reflects interactions among homeostatic, hedonic, gut-brain, cognitive, and environmental processes [5,6]. Satiety ends an eating episode, whereas satiety inhibits further eating after a meal. Prospective food consumption is the amount a person anticipates they could or would eat, and food craving is an intense desire or urge for a particular food [5,6]. These are related but distinct experiences: hunger, satiety, and prospective food consumption may influence food-related cognition, whereas craving and food-cue responsiveness appear to overlap with, but are not equivalent to, food noise [6].

Food noise gained visibility through patient narratives and social media, particularly through reports that glucagon-like peptide-1 receptor agonists (GLP-1RAs) made persistent food-related thoughts “quieter” [7,8]. For some individuals, these thoughts may impose a substantial cognitive burden, compete for attention and daily activities, and make dietary interventions harder to sustain [2-4]. Internal states and external cues, including food availability and exposure to food cues, may contribute to these experiences, although the relative roles of these factors remain uncertain [1,5,6]. Scientific interest has since expanded to include proposed definitions, two questionnaires, observational studies, commentaries, and early mechanistic investigations. However, terminology has advanced faster than the evidence: it remains unclear whether food noise is a distinct construct, a clinically useful umbrella term, or a label for overlapping experiences such as craving, food preoccupation, cue responsiveness, and repetitive thinking [4,6,9-11].

This distinction matters because a construct intended for clinical use must be clearly defined, reliably measured, distinguishable from related conditions, and linked to patient-relevant outcomes. Food noise is not a recognized diagnosis. The Food Noise Questionnaire (FNQ) and the Ro Allison Indiana Dhurandhar Food Noise Inventory (RAID-FN) measure food-noise symptoms [2,9], but standardized diagnostic cutoffs and severity classifications remain unestablished. This narrative review critically evaluates the evidence on

How to cite this article

Ortiz-Martínez Y (October 18, 2026) Food Noise in Obesity: Current Evidence, Measurement, and Clinical Implications. Cureus 18(10): e118095. DOI 10.7759/cureus.118095

food-noise definitions, measurement, epidemiology, proposed mechanisms, differential diagnosis, and responses to obesity treatment, distinguishing direct measurements of food noise from indirect evidence on appetite, craving, energy intake, or neural responses to food.

Review

Search strategy

A narrative literature search was conducted on September 18, 2026, using PubMed, including MEDLINE-indexed records, and Scopus. The exact phrase “food noise” was searched in each database. The initial search retrieved 36 records in PubMed and 41 documents in Scopus. An updated search was performed on October 7, 2026, using the same approach to identify newly available literature. Titles, abstracts, and available full texts were reviewed for relevance to definition, measurement, epidemiology, mechanisms, obesity treatment, metabolic and bariatric surgery, stigma, or clinical assessment. Publications were considered for narrative inclusion when they provided original data, instrument-development or validation findings, or substantive conceptual or clinical information relevant to these domains. Duplicate publications and records in which food noise was mentioned only incidentally without relevant data or conceptual information were not prioritized. Additional references were identified from bibliographies of relevant publications and from foundational human studies of appetite, food craving, GLP-1-based treatment, and ingestive behavior. Recent conference evidence was considered when directly relevant to this rapidly evolving field and is identified as such. Because this was a narrative review, study selection was purposive rather than systematic, and we did not perform a formal risk-of-bias assessment, a Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram, or meta-analysis. Database counts represent retrieved records rather than unique included studies and include overlapping publications, commentaries, conference materials, and studies in which food noise was not the primary outcome. Using the exact phrase may also have excluded older studies that examined comparable experiences under different terminology.

Definition and Evolution of Food Noise

The first scholarly definition, proposed by Hayashi et al. in 2023, defined food noise as “heightened and/or persistent manifestations of food cue reactivity,” which may lead to intrusive food-related thoughts and maladaptive eating behaviors [1]. This definition was embedded in the Cue-Influencer-Reactivity-Outcome (CIRO) model, which proposes that internal and external food cues interact with individual and contextual influences to generate food-cue reactivity and downstream cognitive or behavioral outcomes [1].

Subsequent definitions shifted attention toward the cognitive experience itself. Diktas et al. described food noise as persistent, intrusive food-related thoughts that disrupt daily life and make healthy behaviors difficult [2]. Dhurandhar et al. defined it as persistent thoughts about food perceived as unwanted or dysphoric and potentially causing social, mental, or physical harm [3]. More recently, Umashanker and Mitchell placed food noise within a broader taxonomy of food-related thinking and proposed that clinical relevance depends on frequency, intrusiveness, emotional valence, functional impairment, and associated behavior [10].

These definitions share an emphasis on food-related cognition but differ in whether cue reactivity, negative emotional valence, behavioral consequences, or functional impairment are required. Accordingly, the most defensible current description is phenomenologic: food noise refers to frequent or persistent food-related thoughts that may be experienced as intrusive, unwanted, bothersome, or disruptive and may interfere with daily functioning or health-related behaviors (Figure 1). This description reflects the conceptual evolution from food-cue reactivity toward the cognitive and functional experience of food noise, without presuming a specific biological mechanism or implying that all food-related thinking is pathological. Table 1 summarizes the principal definitions and conceptual differences identified in the literature.

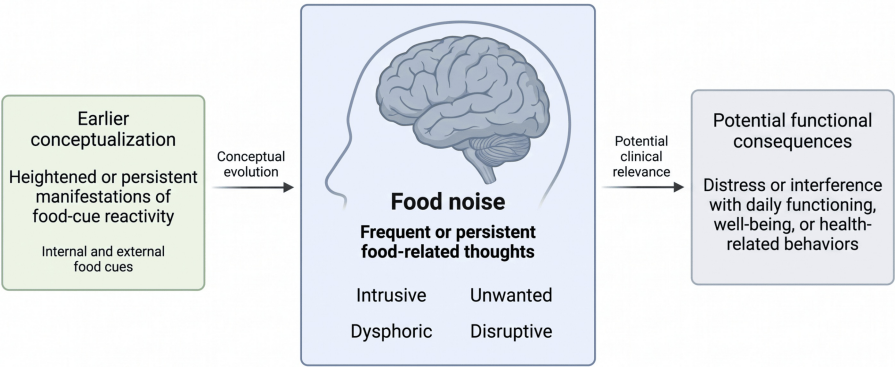


FIGURE 1: Conceptual evolution and potential clinical relevance of food noise

Food noise was initially conceptualized as heightened or persistent manifestations of food-cue reactivity arising in relation to internal and external food cues. Subsequent definitions have emphasized the cognitive experience of frequent or persistent food-related thoughts that may be intrusive, unwanted, dysphoric, or disruptive. Potential clinical relevance arises when these experiences cause distress or interfere with daily functioning, well-being, or health-related behaviors. Food noise remains an emerging construct and is not currently a clinical diagnosis.

Author’s own elaboration based on [1-4,9].

Image credit: Image was created using BioRender (BioRender.com, Toronto, ON, Canada)

Source	Definition or conceptualization	Primary emphasis	Principal uncertainty
Hayashi et al., 2023 [1]	Heightened or persistent manifestations of food-cue reactivity that may produce intrusive thoughts and maladaptive eating	Internal and external cues	Whether cue exposure is necessary
Diktas et al., 2025 [2]	Persistent, intrusive food-related thoughts that disrupt daily life and make healthy behaviors difficult	Intrusiveness and interference	Degree of overlap with craving and preoccupation
Dhurandhar et al., 2025 [3]	Persistent thoughts about food perceived as unwanted or dysphoric and potentially causing social, mental, or physical harm	Dysphoria and harm	Whether distress is required in all cases
Umashanker and Mitchell, 2026 [10]	Food-related thinking that becomes clinically relevant through frequency, intrusiveness, valence, impairment, or maladaptive behavior	Classification of food-related thoughts	Clinical thresholds remain undefined
Brewis et al., 2026 [11]	Emerging and culturally situated label overlapping established appetite-related phenomena	Conceptual and contextual interpretation	Incremental validity and risk of overmedicalization

TABLE 1: Published definitions and conceptualizations of food noise

Author’s own elaboration based on [1-4,9-11]

Epidemiology

The prevalence of food noise remains unknown because validated diagnostic or severity thresholds have not been established. A 2024 consumer survey conducted by WeightWatchers and the STOP Obesity Alliance reported that 57% of respondents living with overweight or obesity had experienced food noise [12]. This finding demonstrated that the term resonated with patients, but it should not be interpreted as a population prevalence estimate because the survey was commercially affiliated and did not use either of the subsequently validated instruments.

A nationally representative survey of 1,000 U.S. adults found that 50.89% reported perceived food noise at least occasionally and 17.98% reported it frequently (often or always) [13]. Because food noise was assessed

using a single lay-language item rather than the FNQ or RAID-FN, these estimates reflect perceived or self-identified food noise rather than clinically significant food noise defined by a validated threshold.

A 2026 cross-sectional study evaluated 593 healthcare students and professionals in Al-Ahsa, Saudi Arabia [14]. Using a bilingual 14-item scale adapted from the Food Thought Suppression Inventory, the authors reported a mean score of 41.9 ± 12.7 on a 14-70 scale. Based on study-defined thresholds, 75.9% had at least moderate food noise and 29.0% had high food noise. The scale was unidimensional and internally consistent, but it was not the FNQ or RAID-FN, and its severity thresholds were exploratory. Food-noise scores were associated with a positive SCOFF score, a five-item screening questionnaire designed to raise suspicion of a possible eating disorder rather than establish a diagnosis, and with higher perceived stress [15]. Convenience sampling, the young health-professionals population, and the use of an adapted instrument preclude extrapolation to students, health professionals, or the general population.

Social media has strongly influenced public understanding. An analysis of 99 highly viewed TikTok videos tagged with food noise found that most content consisted of personal testimonies, food noise was usually described negatively, and approximately half of the videos discussed medications, predominantly GLP-1-based therapies [7]. Only 22.2% of content creators were identified as health professionals. These videos offer insight into lived experience and the term's rapid diffusion, but engagement metrics cannot establish prevalence, causation, or therapeutic efficacy.

Current data therefore indicate that identification with the experience described as food noise is common in selected populations and has also been documented in a nationally representative U.S. sample. However, the prevalence of clinically meaningful food noise measured with validated instruments remains unknown. Obesity should not be treated as a proxy for food noise, and clinicians should not presume food noise is absent in people without obesity.

Differential Diagnosis

Food noise is not a formal diagnosis [3,4,10,11]. When a patient describes persistent thoughts about food, clinicians should determine the phenomenology, context, associated behaviors, and functional consequences rather than assuming that obesity itself explains the experience.

Physiologic hunger is generally linked to energy needs and tends to resolve after adequate intake. Craving involves a desire for a particular food and may be brief or cue-dependent. Food preoccupation describes sustained cognitive attention to eating, food, calories, or body-related concerns and can occur during dietary restriction or eating disorders. Binge-eating disorder requires recurrent objectively large eating episodes accompanied by loss of control and specified associated features; food noise alone does not satisfy these criteria [1,3]. Food addiction models emphasize addiction-like patterns of consumption and impairment, whereas food noise primarily describes cognition.

Persistent food thoughts may also arise in anorexia nervosa or other restrictive states, bulimia nervosa, obsessive-compulsive disorder, depression, anxiety, food insecurity, metabolic conditions, or medication-related appetite changes. Intentional caloric restriction itself can intensify food preoccupation. Evaluation should therefore include eating-disorder symptoms, dietary restriction, loss-of-control eating, mood and anxiety symptoms, medication use, sleep, stress, food access, and the patient's sociocultural environment. Food noise may coexist with any of these conditions but should not replace established diagnostic assessment [3,10,11].

Measurement of Food Noise

Two dedicated instruments have been published: the FNQ and the RAID-FN [2,9]. They operationalize partially different definitions and should not be considered interchangeable.

The FNQ is a brief five-item self-report measure addressing time spent thinking about food, persistence across the day, discomfort, adverse effects on life, and distraction. Items are rated from 0 to 4, producing a total score from 0 to 20. Initial validation supported a unidimensional structure, strong internal consistency, and test-retest reliability [2]. An Italian validation involving 1,087 adults also supported unidimensionality, high categorical omega reliability (0.917), and configural, metric, and scalar invariance across gender [16]. The correlation with food preoccupation was very strong ($r = 0.831$); however, this corresponded to substantial shared variance and underscored uncertainty about incremental validity.

The RAID-FN was developed through a multistep process involving expert input, cognitive interviewing, item reduction, and psychometric testing [9]. It includes a seven-item short form and a 23-item research version covering preoccupation, persistence, and dysphoria. The short form demonstrated high internal consistency and two-week test-retest reliability, although the retest sample was small. Strong correlations with craving, cue responsiveness, and eating-disorder-related quality of life support convergent validity while again raising questions about construct overlap.

Neither instrument currently has a validated diagnostic cutoff, severity classification, minimal clinically important difference, or demonstrated ability to predict treatment response, weight change, or long-term clinical outcomes. A lower score should therefore be interpreted as fewer endorsed symptoms, not proof of biological normalization or treatment success. Table 2 summarizes their structure, available psychometric evidence, and remaining limitations.

Instrument	Structure and scoring	Initial evidence	Important limitations
Food Noise Questionnaire [2]	Five items; 0-4 per item; total 0-20; unidimensional	Strong internal consistency and test-retest reliability; correlations with craving-related measures	No diagnostic cutoff, severity bands, MCID, predictive validity, or established treatment responsiveness
RAID-FN short form [9]	Seven items assessing preoccupation, persistence, and dysphoria	Cronbach's alpha = 0.90; test-retest ICC = 0.89; correlations with craving, cue responsiveness, and quality of life	Retest sample n = 27; no cutoff, sensitivity, specificity, MCID, or outcome prediction
RAID-FN research version [9]	Twenty-three items; multidimensional assessment	Broader coverage of persistence and emotional burden	Greater respondent burden; clinical interpretation and independent validation remain limited

TABLE 2: Food-noise instruments and reported psychometric properties

FNQ: Food Noise Questionnaire; RAID-FN: Ro Allison Indiana Dhurandhar Food Noise Inventory; ICC: intraclass correlation coefficient; MCID: minimal clinically important differences

Author's own elaboration based on [2,9]

Clinical Assessment

A brief clinical conversation may be structured around domains assessed by the FNQ [2] and the RAID-FN [9]. The questions presented in Table 3 were formulated solely to facilitate clinical history-taking. They are not verbatim questionnaire items, do not constitute a screening instrument, have not been independently validated or assigned a score, and should not replace the FNQ or RAID-FN when formal measurement is intended.

Domain	Suggested question	Clinical purpose
Frequency and persistence	"How often do thoughts about food come to mind, and how difficult are they to shift away from?"	Characterize frequency and persistence
Intrusiveness and emotional valence	"Do these thoughts appear when you are not hungry, and do they feel neutral, pleasant, unwanted, or distressing?"	Distinguish intrusive cognition from routine food thinking
Functional interference	"Do food-related thoughts interfere with work, study, sleep, relationships, or other daily activities?"	Identify functional impact
Behavioral relationship	"Are these thoughts associated with eating, restriction, loss-of-control eating, or avoidance?"	Clarify behavioral consequences and differential diagnoses
Context and treatment	"Do they change with hunger, stress, sleep deprivation, dieting, food insecurity, food cues, or treatment?"	Identify physiologic, environmental, and treatment-related influences
Safety	"Have you been severely restricting food, binge eating, compensating after eating, or experiencing substantial distress about food, weight, or body image?"	Identify symptoms requiring established eating-disorder assessment

TABLE 3: Suggested questions for clinical assessment of food noise

FNQ: Food Noise Questionnaire; RAID-FN: Ro Allison Indiana Dhurandhar Food Noise Inventory

Author's own elaboration based on the domains assessed by the FNQ and RAID-FN [2,9]. These questions have not been validated as a screening or diagnostic instrument, should not be scored, and should not replace the original questionnaires or established assessments for eating disorders

No biological pathway is specific to food noise. Appetite and eating behavior emerge from the integration of homeostatic and gut-brain signals with hedonic/reward-related processes, learning, attention, memory, affective states, and environmental food cues [5,6]. Food noise primarily describes a cognitive experience of persistent or intrusive food-related thoughts and overlaps conceptually and psychometrically with food preoccupation, craving, cue responsiveness, and repetitive thinking [2,6,9-11]. A recent theoretical review proposed five potentially interacting explanatory frameworks: homeostatic dysregulation, heightened food-cue reactivity, addiction-like processes, thought suppression, and impaired memory inhibition [6]. These remain hypotheses rather than established food-noise-specific mechanisms. Homeostatic signals such as ghrelin, leptin, insulin, cholecystokinin, peptide YY, and endogenous GLP-1 regulate appetite, but no hormonal or neural biomarker has been shown to map specifically onto FNQ or RAID-FN scores (Figure 2).

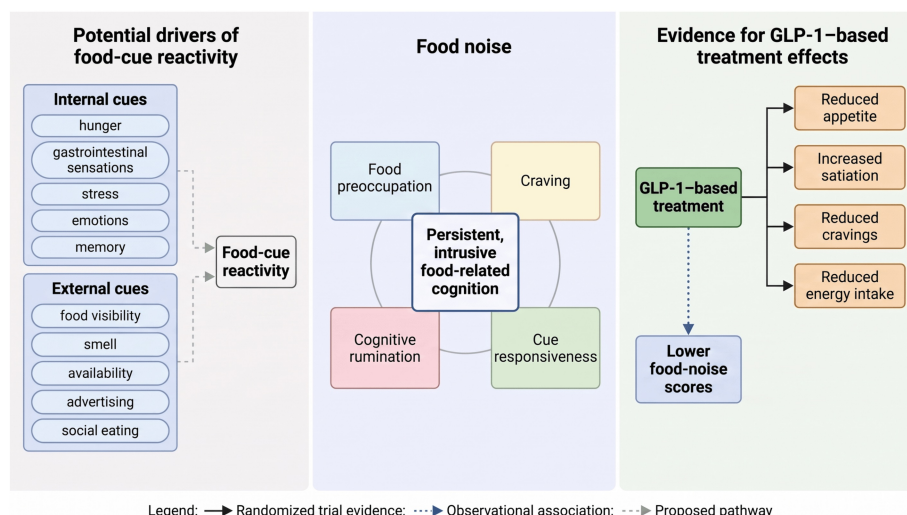


FIGURE 2: Potential drivers, overlapping domains, and treatment evidence relevant to food noise

Internal and external cues are proposed contributors to food-cue reactivity. Food noise is represented as persistent and intrusive food-related cognition that overlaps conceptually with food preoccupation, craving, cue responsiveness, and cognitive rumination; these relationships should not be interpreted as established causal pathways. Randomized trials demonstrate that GLP-1-based therapies reduce appetite, cravings, and energy intake and increase satiation, whereas evidence for lower food-noise scores remains predominantly observational. Solid arrows indicate randomized trial evidence, blue dotted arrows indicate observational associations, and gray dashed arrows indicate proposed pathways.

Source: Author's own elaboration based on [1,2,8,9].

Image credit: Image was created using BioRender (BioRender.com, Toronto, ON, Canada)

Central GLP-1 signaling has well-established effects on appetite and satiation through distributed hypothalamic, hindbrain, and reward-related pathways [17,18]. Human studies of GLP-1-based treatment have reported changes in food-cue responses in regions including the orbitofrontal and cingulate cortices, hippocampus, and nucleus accumbens [19,20]. These findings support central regulation of appetite and ingestive behavior but do not identify a food-noise-specific circuit. A randomized six-week tirzepatide trial showed reductions in energy intake, appetite, cravings, overeating tendencies, and environmental food-cue reactivity, with region- and food-category-specific functional imaging findings; however, it did not administer a validated food-noise instrument [19]. A report of intracranial nucleus accumbens activity in one individual receiving tirzepatide was hypothesis-generating but cannot establish a biomarker [20]. Likewise, a cross-sectional study of 79 adults found lower FNQ scores after approximately six months of GLP-1RA use, but chemosensory differences were largely limited to sweet-detection thresholds, providing little support for broad taste or olfactory alteration as the primary mechanism [21].

Responses to obesity interventions

Incretin-Based Pharmacotherapy

Randomized trials consistently demonstrate the effects of incretin-based medications on appetite and ingestive behavior. In a 20-week trial of 72 adults with obesity, semaglutide 2.4 mg reduced energy intake during an ad libitum meal by approximately 35% relative to placebo and improved hunger, fullness, satiety, control of eating, and food-craving outcomes [22]. A 12-week crossover trial of 30 adults similarly found that semaglutide 1.0 mg reduced energy intake, hunger, and food cravings [23]. These trials provide strong evidence that semaglutide modifies appetite-related outcomes, but they predated validated food-noise instruments and cannot establish an effect on food noise as presently defined.

The six-week randomized tirzepatide study also demonstrated reductions in energy intake and in several dimensions of appetite, craving, overeating, and cue reactivity [19]. Again, these findings are relevant to food noise but constitute indirect evidence because neither FNQ nor RAID-FN was used.

Direct evidence concerning measured food noise is predominantly observational. The INFORM survey retrospectively assessed 550 current users of injectable semaglutide for weight management; 547 completed all FNQ items [8]. Participants answered the same five FNQ questions for recalled pretreatment and current experiences. Median scores decreased from 13 to 6, and item endorsement fell markedly. For example, 63% recalled spending excessive time thinking about food before treatment compared with 15% during treatment, while constant thoughts throughout the day decreased from 62% to 16%. Participants also reported high treatment satisfaction and perceived improvements in health behavior and well-being.

In the cross-sectional chemosensory study, approximately six-month GLP-1RA users had lower FNQ scores than eligible nonusers, together with lower craving, cue responsiveness, perceived food reward, and energy intake [21]. Because participants were not followed longitudinally, medication exposure was confounded with baseline differences, weight loss, treatment expectations, and other factors.

Randomized evidence also extends these observations to combined amylin/GLP-1 receptor agonism. In a randomized, placebo-controlled clinical pharmacology study in adults with overweight or obesity, once-weekly CagriSema, a fixed-dose combination of cagrilintide and semaglutide, was evaluated for its effects on appetite, eating behavior, energy intake, and neural responses to food cues using functional MRI. After 52 weeks, CagriSema was associated with improvements in appetite, eating control, food-related thoughts, craving control, and food-cue reactivity, together with changes in functional brain responses to high-calorie food cues [24]. Because validated food-noise instruments such as the FNQ or RAID-FN were not administered, these findings should be considered indirect evidence relevant to food noise rather than direct evidence of changes in the construct itself.

Taken together, current evidence supports a clear hierarchy: randomized trials demonstrate reductions in appetite, cravings, food-cue reactivity, and energy intake, whereas reductions in directly measured food noise are supported primarily by retrospective and cross-sectional observations. Figure 3 summarizes these findings across the appetite-related and food-noise domains and distinguishes outcomes evaluated in randomized trials from those reported in observational studies. Using food-noise scores to guide medication selection, dose adjustment, maintenance therapy, microdosing, or treatment discontinuation remains investigational.

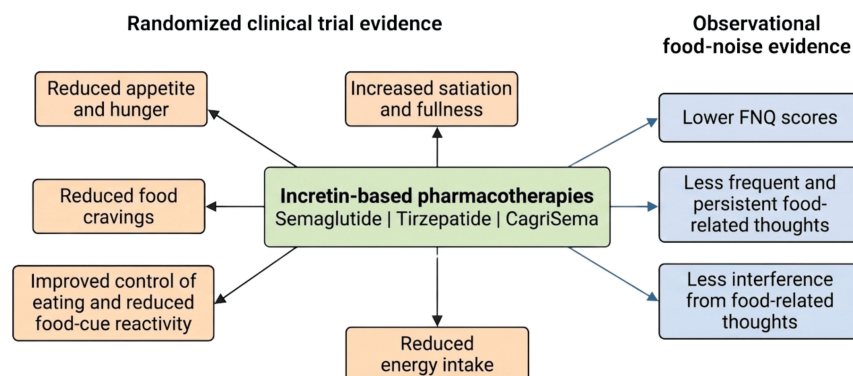


FIGURE 3: Evidence for incretin-based therapies across appetite and food-noise domains

FNQ: Food Noise Questionnaire

Randomized studies of semaglutide, tirzepatide, and CagriSema demonstrate improvements in appetite-related outcomes, cravings, food-cue reactivity, eating control, and/or energy intake. Observational studies also report lower FNQ scores and reduced frequency, persistence, and functional interference of food-related thoughts.

Source: Author's own elaboration based on [8,18,21-24]

Image credit: Image was created using BioRender (BioRender.com, Toronto, ON, Canada)

Metabolic and Bariatric Surgery

Metabolic and bariatric surgery alters eating behavior through interactions among gastrointestinal, neuroendocrine, behavioral, and central mechanisms. Roux-en-Y gastric bypass (RYGB) is associated with enhanced postprandial GLP-1 and peptide YY responses, which may contribute to greater satiety and reduced appetite [25]. Objective studies further indicate that RYGB and sleeve gastrectomy substantially reduce total energy intake, primarily through smaller meal sizes and slower eating rather than consistent changes in meal initiation or macronutrient selection [26].

Surgery also alters reward-related responses to food. A systematic review of 23 functional MRI studies found that food-cue reactivity in reward-related brain regions after RYGB often decreased or remained unchanged, though findings varied by procedure, nutritional state, food-cue paradigm, postoperative timing, and study design [27]. Longitudinal evidence also indicates that food cravings generally decrease after sleeve gastrectomy and RYGB, particularly in the early postoperative period, though trajectories vary over time [28].

Direct measurement of food noise after metabolic and bariatric surgery has now been reported. Bond and colleagues evaluated 60 adults during postoperative weight stability, at a mean of 13.3 ± 4.5 months after surgery, using the FNQ [29]. Higher weight self-stigma was associated with higher FNQ scores. Because the study was cross-sectional and lacked prospective preoperative FNQ measurement, it does not establish that surgery reduces food noise. However, it demonstrates the feasibility of measuring food noise after surgery and supports prospective evaluation of postoperative food-noise trajectories.

Behavioral and Dietary Interventions

Cognitive-behavioral and mindfulness-based interventions can improve binge-eating symptoms, emotional eating, and some craving-related outcomes in selected populations [30-32]. Nutritional counseling, stimulus-control strategies, sleep optimization, and modification of the food environment may also address circumstances that intensify hunger or exposure to food cues. However, these interventions have not been evaluated specifically as treatments for food noise using the FNQ or RAID-FN.

Evidence from related eating outcomes should therefore not be extrapolated as proof of efficacy for food noise. Behavioral and nutritional interventions remain appropriate components of comprehensive obesity care, but no specific psychological, dietary, or environmental intervention is currently an established food-noise treatment [3,4,9]. Table 4 summarizes the available evidence on pharmacological, surgical, and behavioral interventions.

Intervention or study	Design	Main findings	Relevance to food noise
Semaglutide 2.4 mg [22]	Randomized, placebo-controlled trial; 20 weeks; n = 72	Approximately 35% lower ad libitum energy intake; reduced hunger and cravings; increased fullness and improved control of eating	Strong indirect evidence; no validated food-noise measure
Semaglutide 1.0 mg [23]	Randomized crossover trial; 12 weeks; n = 30	Reduced energy intake, hunger, and food cravings	Indirect evidence only
Tirzepatide [19]	Randomized phase 1 trial; six weeks	Reduced energy intake, appetite, cravings, overeating tendency, and food-cue reactivity	Indirect evidence; food noise was not directly measured
CagriSema [24]	Randomized clinical pharmacology study	Improved appetite, eating behavior, food-related thoughts, craving control, and food-cue reactivity; altered functional brain responses to food cues	Indirect randomized evidence; validated food-noise instruments were not administered
INFORM semaglutide survey [8]	Retrospective cross-sectional survey; n = 550	Lower recalled pretreatment versus current FNQ scores; reported improvements in well-being and health behaviors	Direct measurement, but no prospective baseline or untreated comparator
Mixed GLP-1RA chemosensory study [21]	Cross-sectional study; n = 79	Lower FNQ scores, cravings, cue responsiveness, perceived food reward, and energy intake among approximately six-month users	Direct association; susceptible to confounding and selection bias
Metabolic and bariatric surgery [25-29]	Mechanistic, longitudinal, neuroimaging, and cross-sectional postoperative studies	Reduced energy intake and meal size, slower eating, altered gut-brain signaling and food-cue responses, and generally reduced cravings; postoperative FNQ measurement has been reported	Relevant mechanistic evidence and emerging direct measurement; no prospective pre-to-post evidence using validated food-noise scores
Behavioral interventions [30-32]	Randomized trials and systematic reviews addressing related eating outcomes	Cognitive-behavioral and mindfulness-based interventions may improve binge eating, emotional eating, or craving	No intervention has been established specifically for food noise

TABLE 4: Evidence of obesity interventions and food-related outcomes

FNQ: Food Noise Questionnaire; GLP-1RA: glucagon-like peptide-1 receptor agonist;

Author’s own elaboration based on [8,19,21-32]

Implications for Obesity Treatment

Asking about food-related cognitive burden may add a patient-centered dimension to obesity care. Two individuals with similar body mass index or weight trajectories may experience markedly different levels of cognitive interference, distress, or difficulty disengaging from food-related thoughts. Clinicians may document the frequency, persistence, intrusiveness, emotional valence, behavioral consequences, and functional impact of these experiences and monitor these domains alongside weight, metabolic outcomes, nutritional status, adverse effects, quality of life, and mental health [2-4,9].

Food-noise scores should not currently determine whether a patient receives obesity pharmacotherapy, which medication is selected, or which dose is prescribed. Proposed food-noise-guided therapy, including using symptom changes to guide microdosing, maintenance dosing, dose escalation, or treatment discontinuation, remains investigational [3,4,9]. No randomized trial has validated an FNQ- or RAID-FN-guided dosing algorithm or demonstrated that the lowest dose associated with food-noise suppression maintains weight loss or cardiometabolic benefits. Dose modification may be appropriate for established clinical reasons, including tolerability, treatment response, contraindications, or individualized risk-benefit assessment, but should not be presented as an evidence-based food-noise protocol. More broadly, interpretation of obesity pharmacotherapy should preserve the dose regimens actually studied, because flexible-dose or maximum-tolerated-dose regimens should not be interpreted as evidence for a single fixed-dose exposure [33].

The proposal that incretin-based therapy might provide a temporary neurobiological scaffold for developing nutritional, culinary, or cognitive-behavioral skills is conceptually appealing [34]. Pharmacological

reductions in hunger, cravings, and food-cue responsiveness may create an opportunity to implement sustainable behavioral changes. Nevertheless, no trial has established that time-limited incretin therapy combined with an integrated behavioral intervention is equivalent to or superior to continued pharmacotherapy.

In the STEP 1 extension, participants regained about two-thirds of their prior weight loss within one year after semaglutide withdrawal [35]. Similarly, SURMOUNT-4 showed substantial weight regain in participants switched from tirzepatide to placebo, whereas continued treatment maintained and further reduced weight [36]. These findings do not preclude individualized dose reduction or discontinuation, but they indicate that a temporary-scaffold model requires prospective evaluation rather than clinical assumption.

Weight regain after treatment withdrawal may also be accompanied by a return of appetite-related pressures. However, STEP 1 extension and SURMOUNT-4 did not measure food noise with validated instruments; whether food noise recurs or intensifies after medication withdrawal therefore remains unknown and should be tested prospectively [35,36].

Longitudinal food-noise monitoring may eventually help characterize treatment response, symptom recurrence, or the need for additional clinical support. Before such monitoring can guide treatment, research must establish that changes in FNQ or RAID-FN scores are reproducible, clinically interpretable, and independently associated with patient-important outcomes, irrespective of hunger, cravings, energy intake, and body weight [2-4,9].

In low- and middle-income countries (LMICs), socioeconomic circumstances and health-system constraints may shape access to obesity care, including medication affordability and availability, facility-level access, and geographic barriers. These factors may also affect access to dietary support and the environments in which food-related thoughts occur. Because direct food-noise research in LMICs is limited, findings from high-income settings should not be assumed to generalize across populations or health systems [37,38].

Food Noise and Stigma

The language of food noise may reduce blame by validating an experience that some patients describe as difficult to control. Recognition of interacting biological, psychological, and environmental influences may counter explanations of obesity based solely on willpower. However, labels can also distinguish, stereotype, and separate individuals, thereby contributing to stigma, status loss, or discrimination [39]. Categorizing individuals as having “high” or “low” food noise could reinforce perceptions of deficient self-control or imply that pharmacological suppression is required for acceptable eating behavior. These possibilities remain theoretical because researchers have not yet measured food-noise-specific stigma empirically [3].

Food noise should not be conflated with obesity. Intrusive or persistent food-related thoughts may occur across the body-size spectrum, whereas not every person with obesity experiences them [3,4]. Similar experiences may occur in eating disorders, restrictive dieting, food insecurity, or other clinical and social circumstances. Their interpretation therefore requires attention to context, emotional valence, associated behaviors, nutritional status, and functional impairment, not body size alone. Evidence from weight-stigma and food-addiction research demonstrates that causal labels can have complex effects: biological explanations may reduce personal blame while also affecting perceived control, self-efficacy, or expectations regarding treatment [40-41].

Clinicians and researchers should use neutral, person-centered language and ask patients how they understand their experiences. Lower food-related cognition should not be presumed to be universally beneficial. In individuals with restrictive eating disorders, inadequate intake, or high nutritional risk, appetite suppression or reduced attention to food may be harmful. Clinical assessment should therefore include screening for severe dietary restriction, binge eating, compensatory behaviors, body-image distress, and other eating-disorder symptoms. Future research should evaluate whether the food-noise label produces validation or, conversely, self-stigma, shame, concealment, or pressure to pursue pharmacotherapy. Studies should include participants across body sizes, cultural groups, gender identities, and socioeconomic circumstances and incorporate established strategies for reducing weight stigma in healthcare [42,43].

Limitations of the evidence and future directions

The food-noise literature remains small, recent, and heterogeneous. Definitions have evolved rapidly, validated instruments overlap with established measures of food preoccupation, craving, cue responsiveness, and repetitive thinking, and few studies include prospective baseline assessments [1-4,9-11]. Much of the early treatment narrative originated from patient testimonials, social-media discourse, retrospective recall, and cross-sectional comparisons. Although randomized trials demonstrate that GLP-1-based therapies modify appetite, cravings, food-cue responsiveness, control of eating, and energy intake, validated food-noise instruments have rarely been included as randomized outcomes [8,19,21-23]. Available direct studies are vulnerable to selection bias, retrospective recall, expectancy effects, residual confounding,

and limited demographic representation.

Several research priorities follow. First, definitions should be refined through patient involvement and cross-cultural research, without assuming that all frequent food-related cognition is pathological [3,4]. Second, the FNQ and RAID-FN require independent replication, normative data, longitudinal evaluation, cross-cultural validation, minimal clinically important differences, and evidence of incremental and predictive validity beyond related constructs [2,9]. Third, prospective randomized trials should measure food noise concurrently with hunger, satiation, satiety, prospective food consumption, craving, cue responsiveness, eating-disorder symptoms, energy intake, quality of life, weight, and metabolic outcomes. Mediation analyses could then assess whether changes in food noise contribute independently to clinical outcomes.

Ecological momentary assessment may help characterize within-day variability and determine whether food-related thoughts arise spontaneously or in response to hunger, stress, dietary restriction, sleep disturbance, or environmental food cues. This methodology has previously been used to study food cravings in daily life and could be adapted for validated food-noise instruments [44]. Experimental studies should examine attention, memory, repetitive thinking, reward valuation, and food-cue processing without assuming a single food-noise neural circuit.

Prospective studies of behavioral treatment, metabolic and bariatric surgery, medication withdrawal, and weight recurrence are also needed [25–36]. Across these settings, research should determine whether changes in validated food-noise scores offer predictive or explanatory value beyond hunger, craving, food preoccupation, cue responsiveness, eating behavior, and body weight. Establishing this incremental clinical value will be essential before incorporating food-noise measurement into treatment algorithms.

Future studies should clarify how homeostatic dysregulation interacts with reward, attention, memory, learning, and environmental cues, and should investigate the brain circuits associated with food-related cognitive burden without presuming a food-noise-specific pathway. They should also establish standardized assessment procedures, clinically interpretable thresholds, and diagnostic criteria, or determine whether food noise is better treated as a dimensional patient-reported experience rather than a diagnosis.

Conclusions

Food noise is an emerging patient-reported cognitive construct describing frequent, persistent, or intrusive food-related thoughts that may feel unwanted, distressing, or disruptive to daily functioning. The development of the FNQ and RAID-FN is an important step toward standardized measurement, but diagnostic thresholds, severity categories, and minimal clinically important differences have not been established. Importantly, food noise substantially overlaps with food preoccupation, craving, cue responsiveness, and broader appetite-related cognition, and current evidence does not yet show that it is empirically distinguishable from these established domains.

Incretin-based therapies consistently modify appetite, cravings, food-cue reactivity, eating control, and energy intake in randomized studies, while observational evidence suggests lower directly measured food noise. Future prospective studies should determine whether changes in food-noise scores provide information beyond established appetitive constructs and independently predict weight-management, mental-health, or quality-of-life outcomes. Until then, food noise may provide a useful patient-centered description of the cognitive experience surrounding food, while its independent clinical significance continues to be defined.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Yeimer Ortiz-Martínez

Acquisition, analysis, or interpretation of data: Yeimer Ortiz-Martínez

Drafting of the manuscript: Yeimer Ortiz-Martínez

Critical review of the manuscript for important intellectual content: Yeimer Ortiz-Martínez

Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** YOM declare(s) personal fees from

Abbott, Amgen, Bayer, AstraZeneca, Boehringer Ingelheim, Novartis, Novo Nordisk, and Sanofi. YOM has received honoraria for speaking engagements from Abbott, Amgen, Bayer, AstraZeneca, Boehringer Ingelheim, Novartis, Novo Nordisk, and Sanofi. These relationships had no role in the conception or preparation of this manuscript. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

- Hayashi D, Edwards C, Emond JA, Gilbert-Diamond D, Butt M, Rigby A, Masterson TD: What is food noise? A conceptual model of food cue reactivity. *Nutrients*. 2023, 15:4809. [10.3390/nu15224809](https://doi.org/10.3390/nu15224809)
- Diktas HE, Cardel MI, Foster GD, et al.: Development and validation of the Food Noise Questionnaire . *Obesity* (Silver Spring). 2025, 33:289-97. [10.1002/oby.24216](https://doi.org/10.1002/oby.24216)
- Dhurandhar EJ, Maki KC, Dhurandhar NV, et al.: Food noise: definition, measurement, and future research directions. *Nutr Diabetes*. 2025, 15:30. [10.1038/s41387-025-00382-x](https://doi.org/10.1038/s41387-025-00382-x)
- Umashanker D, Mitchell JE: Food noise: current knowledge and future research directions . *Obesity* (Silver Spring). 2026, 34:762-4. [10.1002/oby.70149](https://doi.org/10.1002/oby.70149)
- Blundell J, de Graaf C, Hulshof T, et al.: Appetite control: methodological aspects of the evaluation of foods . *Obes Rev*. 2010, 11:251-70. [10.1111/j.1467-789X.2010.00714.x](https://doi.org/10.1111/j.1467-789X.2010.00714.x)
- Wylie F, Mahmut MK, Francis HM, Davidson T, Stevenson RJ: Food noise: a theoretical review . *Appetite*. 2026, 228:108791. [10.1016/j.appet.2026.108791](https://doi.org/10.1016/j.appet.2026.108791)
- Hayashi D, Kort J, Robles-Martinez IM, Orabueze D, Bakhl K, Masterson TD: "And just like that, quiet": a content analysis of TikTok videos on food noise. *Nutr Diabetes*. 2026, 16:16. [10.1038/s41387-026-00423-z](https://doi.org/10.1038/s41387-026-00423-z)
- Arnaut T, Duncan S, Faurby M, Hahn-Pedersen JH, Kvist K, Steenackers N, Buse JB: Retrospective assessment of food noise changes after initiation of injectable semaglutide for weight management in the USA: the INFORM survey. *Adv Ther*. 2026, 43:3649-59. [10.1007/s12325-026-03636-x](https://doi.org/10.1007/s12325-026-03636-x)
- Dhurandhar EJ, Maki KC, Dhurandhar NV, et al.: Development and rigorous multistep validation of a psychometric tool to measure food noise. *Appetite*. 2026, 217:108339. [10.1016/j.appet.2025.108339](https://doi.org/10.1016/j.appet.2025.108339)
- Umashanker D, Mitchell J: Food noise is a type of thinking . *Diabetes Obes Metab*. 2026, 28:9735-8. [10.1111/dom.71203](https://doi.org/10.1111/dom.71203)
- Brewis A, Hayashi D, Gualano B, et al.: Food noise: conceptual, methodological, and ethical considerations. *Appetite*. 2026, 226:108700. [10.1016/j.appet.2026.108700](https://doi.org/10.1016/j.appet.2026.108700)
- Understanding food noise. (2024). Accessed: October 7, 2026: <https://stop.publichealth.gwu.edu/LFD-jan24>.
- Hayashi D, Masterson TD: Prevalence and correlates of perceived food noise among U.S. adults in a nationally representative sample. *Obes Sci Pract*. 2026, 12:e70201. [10.1002/osp4.70201](https://doi.org/10.1002/osp4.70201)
- Alarfaj S, Alqahtani M, Almaqhawi A: Food noise among healthcare students and healthcare professionals in Al-Ahsa, Saudi Arabia: a cross-sectional study. *Nutrients*. 2026, 18:2824. [10.3390/nu18172824](https://doi.org/10.3390/nu18172824)
- Morgan JF, Reid F, Lacey JH: The SCOFF questionnaire: assessment of a new screening tool for eating disorders. *BMJ*. 1999, 319:1467-8. [10.1136/bmj.319.7223.1467](https://doi.org/10.1136/bmj.319.7223.1467)
- Mocini E, Di Vincenzo O, D'angelo C, Baldari C, Migliaccio S, Zagaria A: Psychometric properties of the Italian Food Noise Questionnaire (FNQ) for the assessment of intrusive food-related thoughts. *Behav Sci (Basel)*. 2026, 16:609. [10.3390/bs16040609](https://doi.org/10.3390/bs16040609)
- Drucker DJ: Mechanisms of action and therapeutic application of glucagon-like peptide-1 . *Cell Metab*. 2018, 27:740-56. [10.1016/j.cmet.2018.03.001](https://doi.org/10.1016/j.cmet.2018.03.001)
- van Bloemendaal L, Iljerman RG, Ten Kulve JS, et al.: GLP-1 receptor activation modulates appetite- and reward-related brain areas in humans. *Diabetes*. 2014, 63:4186-96. [10.2337/db14-0849](https://doi.org/10.2337/db14-0849)
- Martin CK, Carmichael OT, Carnell S, et al.: Tirzepatide on ingestive behavior in adults with overweight or obesity: a randomized 6-week phase 1 trial. *Nat Med*. 2025, 31:3141-50. [10.1038/s41591-025-03774-9](https://doi.org/10.1038/s41591-025-03774-9)
- Choi W, Nho YH, Qiu L, et al.: Brain activity associated with breakthrough food preoccupation in an individual on tirzepatide. *Nat Med*. 2025, 31:4038-43. [10.1038/s41591-025-04035-5](https://doi.org/10.1038/s41591-025-04035-5)
- Rutigliani G, Mattes RD: The effects of glucagon-like peptide-1 receptor agonists on chemosensory function and ingestive behavior. *Physiol Behav*. 2026, 316:115486. [10.1016/j.physbeh.2026.115486](https://doi.org/10.1016/j.physbeh.2026.115486)
- Friedrichsen M, Breitschaft A, Tadayon S, Wizert A, Skovgaard D: The effect of semaglutide 2.4 mg once weekly on energy intake, appetite, control of eating, and gastric emptying in adults with obesity. *Diabetes Obes Metab*. 2021, 23:754-62. [10.1111/dom.14280](https://doi.org/10.1111/dom.14280)
- Blundell J, Finlayson G, Axelsen M, Flint A, Gibbons C, Kvist T, Hjerpsted JB: Effects of once-weekly semaglutide on appetite, energy intake, control of eating, food preference and body weight in subjects with obesity. *Diabetes Obes Metab*. 2017, 19:1242-51. [10.1111/dom.12932](https://doi.org/10.1111/dom.12932)
- Effects of CagriSema on appetite, eating behaviour and food cue reactivity using functional MRI in adults with overweight or obesity. (2026). Accessed: October 7, 2026: <https://sciencehub.novonordisk.com/congresses/easd2026/goldstone.html>.
- le Roux CW, Welbourn R, Werling M, et al.: Gut hormones as mediators of appetite and weight loss after Roux-en-Y gastric bypass. *Ann Surg*. 2007, 246:780-5. [10.1097/SLA.0b013e3180caa3e3](https://doi.org/10.1097/SLA.0b013e3180caa3e3)
- Contreras Hernández F, Villalbeitia I, Levine I, le Roux CW, Miras AD, Price RK: Effects of bariatric surgery and obesity medicines on human eating behavior. *Annu Rev Nutr*. 2026, 46:339-56. [10.1146/annurev-nutr-070225-105258](https://doi.org/10.1146/annurev-nutr-070225-105258)
- Alabdulkader S, Al-Alsheikh AS, Miras AD, Goldstone AP: Obesity surgery and neural correlates of human eating behaviour: a systematic review of functional MRI studies. *Neuroimage Clin*. 2024, 41:103563. [10.1016/j.nicl.2024.103563](https://doi.org/10.1016/j.nicl.2024.103563)
- Koball AM, Ames GE, Fitzsimmons AJ, Kallies KJ, Bennie BA: Food cravings after bariatric surgery: comparing laparoscopic sleeve gastrectomy and Roux-en-Y gastric bypass. *Eat Weight Disord*. 2024, 29:7. [10.1007/s40519-023-01636-2](https://doi.org/10.1007/s40519-023-01636-2)
- Bond DS, Wu Y, Papasavas PK, et al.: Weight self-stigma and food noise during post-metabolic bariatric surgery weight stability. *Obesity* (Silver Spring). 2026, 1-6. [10.1002/oby.70318](https://doi.org/10.1002/oby.70318)
- Wilson GT, Wilfley DE, Agras WS, Bryson SW: Psychological treatments of binge eating disorder . *Arch Gen*

- Psychiatry. 2010, 67:94-101. [10.1001/archgenpsychiatry.2009.170](https://doi.org/10.1001/archgenpsychiatry.2009.170)
31. Katterman SN, Kleinman BM, Hood MM, Nackers LM, Corsica JA: Mindfulness meditation as an intervention for binge eating, emotional eating, and weight loss: a systematic review. *Eat Behav.* 2014, 15:197-204. [10.1016/j.eatbeh.2014.01.005](https://doi.org/10.1016/j.eatbeh.2014.01.005)
 32. O'Reilly GA, Cook L, Spruijt-Metz D, Black DS: Mindfulness-based interventions for obesity-related eating behaviours: a literature review. *Obes Rev.* 2014, 15:453-61. [10.1111/obr.12156](https://doi.org/10.1111/obr.12156)
 33. Ortiz-Martínez Y: The challenge of dose-specific comparisons in obesity pharmacotherapy. *Obesity (Silver Spring)*. 2026, 1-1. [10.1002/oby.70296](https://doi.org/10.1002/oby.70296)
 34. Rizwan AA, Galyean S: Food noise as a missing piece in obesity treatment: toward an integrated intervention framework. *Int J Obes (Lond)*. 2026, [10.1038/s41366-026-02185-1](https://doi.org/10.1038/s41366-026-02185-1)
 35. Wilding JP, Batterham RL, Davies M, et al.: Weight regain and cardiometabolic effects after withdrawal of semaglutide: the STEP 1 trial extension. *Diabetes Obes Metab.* 2022, 24:1553-64. [10.1111/dom.14725](https://doi.org/10.1111/dom.14725)
 36. Aronne LJ, Sattar N, Horn DB, et al.: Continued treatment with tirzepatide for maintenance of weight reduction in adults with obesity: the SURMOUNT-4 randomized clinical trial. *JAMA.* 2024, 331:38-48. [10.1001/jama.2023.24945](https://doi.org/10.1001/jama.2023.24945)
 37. Link BG, Phelan JC: Conceptualizing stigma. *Annu Rev Sociol.* 2001, 27:363-85. [10.1146/annurev.soc.27.1.363](https://doi.org/10.1146/annurev.soc.27.1.363)
 38. Fatoye F, Mbada C, Niyi-Odumosu F, Fatoye C, Useh U, Hakimi Z, Gebrye T: The clinical and economic burden of obesity in low- and middle-income countries: a systematic review. *Int J Obes (Lond)*. 2025, 49:2453-61. [10.1038/s41366-025-01913-3](https://doi.org/10.1038/s41366-025-01913-3)
 39. Wong B, Wight L, Gauvreau CL, et al.: Exploring factors affecting access to non-communicable disease medicines in low- and middle-income countries: a systematic review. *BMJ Public Health.* 2026, 4:e002497. [10.1136/bmjph-2024-002497](https://doi.org/10.1136/bmjph-2024-002497)
 40. Hill B, Bergmeier H, Incollingo Rodriguez AC, et al.: Weight stigma and obesity-related policies: a systematic review of the state of the literature. *Obes Rev.* 2021, 22:e13333. [10.1111/obr.13333](https://doi.org/10.1111/obr.13333)
 41. Cassin SE, Buchman DZ, Leung SE, Kantarovich K, Hawa A, Carter A, Sockalingam S: Ethical, stigma, and policy implications of food addiction: a scoping review. *Nutrients.* 2019, 11:710. [10.3390/nu11040710](https://doi.org/10.3390/nu11040710)
 42. Latner JD, Puhl RM, Murakami JM, O'Brien KS: Food addiction as a causal model of obesity. Effects on stigma, blame, and perceived psychopathology. *Appetite.* 2014, 77:77-84. [10.1016/j.appet.2014.03.004](https://doi.org/10.1016/j.appet.2014.03.004)
 43. Talumaa B, Brown A, Batterham RL, Kalea AZ: Effective strategies in ending weight stigma in healthcare. *Obes Rev.* 2022, 23:e13494. [10.1111/obr.13494](https://doi.org/10.1111/obr.13494)
 44. Roefs A, Boh B, Spanakis G, Nederkoorn C, Lemmens LH, Jansen A: Food craving in daily life: comparison of overweight and normal-weight participants with ecological momentary assessment. *J Hum Nutr Diet.* 2019, 32:765-74. [10.1111/jhn.12693](https://doi.org/10.1111/jhn.12693)