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Review

Clinical Evidence on the Effects of GLP-1 Receptor Agonists and Dual GIP/GLP-1 Receptor Agonists on Appetite, Food Preferences, and Eating Behavior: A Narrative Review

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Abstract

Background: Incretin-based therapies have transformed obesity management, offering new opportunities to improve weight-related and cardiometabolic outcomes. However, their influence on eating behaviors and their core dimensions has not yet been comprehensively analyzed. Therefore, this narrative review aims to discuss the available clinical evidence regarding GLP-1 receptor agonists (GLP-1 RAs) and dual GLP-1/GIP receptor agonists (GIP/GLP-1 RAs) on eating behaviors in adults with obesity, including appetite, food preferences, food cravings, dietary intake, and diet quality. Methods: A narrative review of the literature was conducted to identify and analyze clinical studies assessing changes in eating behavior and diet-related outcomes during incretin-based pharmacological treatment for obesity. Studies evaluating GLP-1 RAs (e.g., liraglutide, semaglutide) and dual incretin agonists (tirzepatide) were included. Evidence from randomized clinical trials and observational studies examining appetite, energy intake, food preferences, and dietary behavior-related outcomes published before January 2026 was analyzed. Results: A total of 17 clinical studies met the eligibility criteria and were included in the narrative review. Available evidence suggests that incretin-based therapies reduced appetite and ad libitum energy intake, decreased cravings for energy-dense foods, and led to changes in food preferences. Some studies also report modifications in taste perception and greater control over eating behavior. Evidence regarding effects on dietary quality and macro- and micronutrient composition remains limited. Conclusions**:** GLP-1 RAs and GIP/GLP-1 RAs may affect multiple domains of eating behavior. Understanding these changes is important for optimizing dietary management during pharmacotherapy and for ensuring nutritionally balanced weight-loss strategies. Further research is needed to clarify the long-term effects of GLP-1 RAs and GIP/GLP-1 RAs on dietary habits and nutritional quality.

Keywords: obesity; anti-obesity medication; liraglutide; semaglutide; tirzepatide; appetite;dietary behavior; diet quality; food craving;emotional eating;

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1. Introduction

According to the World Health Organization, obesity is defined as "abnormal or excessive adipose tissue accumulation that presents a risk to health. It is a chronic,

relapsing, and progressive disease associated with increased risk of numerous health complications, including type 2 diabetes (T2D), cardiovascular diseases, several types of cancer (including breast, colorectal, pancreatic, and endometrial cancer), and musculoskeletal disorders. Consequently, obesity requires long-term, comprehensive management strategies that combine lifestyle modification with evidence-based medical treatment when appropriate [1,2].

Lifestyle intervention, including dietary modification and regular physical activity, remains the cornerstone of obesity treatment. However, lifestyle interventions alone are frequently insufficient to achieve and maintain clinically meaningful weight reduction. Therefore, current clinical guidelines recommend anti-obesity pharmacotherapy for adults with a body mass index (BMI) ≥ 30 kg/m^2 or≥ 27 kg/m^2 in the presence of at least one weight-related comorbidity, such as T2D, hypertension, or dyslipidemia, particularly when lifestyle interventions are ineffective [3].

In recent years, incretin-based therapies have transformed obesity management, offering new opportunities to improve weight-related and cardiometabolic outcomes.

Incretin-based anti-obesity agents include the glucagon-like peptide-1 receptor agonists (GLP-1 RAs), such as liraglutide and semaglutide, and the recently Food and Drug Administration (FDA)-approved oral non-peptide GLP-1 RA - orforglipron, as well asthedual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 RA – tirzepatide [4,5].

Liraglutide and semaglutide are GLP-1 RAs and structural analogues ofendogenous human GLP-1. This intestinal hormone, released postprandially, stimulates glucose-dependent insulin secretion from pancreatic β cells, thereby reducing postprandial hyperglycemia and contributing to glucose homeostasis*[6]*. In addition, GLP-1 suppresses glucagon secretion from pancreatic α cells, thereby reducinghepatic glucose production. Within the central nervous system, particularly the hypothalamus, GLP-1 regulates appetite by promoting satiety and reducing hunger. Furthermore, GLP-1 delaysgastric emptying, further prolonging the sensation of fullness and consequently reducing energy intake and promoting weight loss [7]. Emerging evidence also suggests that prolonged treatment with GLP-1 RAs may influence food preferences, although the underlying mechanisms and clinical significance require further investigation *[5]*.

Liraglutide (FDA-approved for obesity treatment in 2014) is a long-acting GLP-1 RA administered subcutaneously (s.c.) at a dose of 3.0 mg once daily (QD) [8]. Similarly, semaglutide (FDA-approved for obesity treatment in 2021) is a long-acting GLP-1 RA administered subcutaneously at a dose of 2.4 mg once weekly (QW) [9]. Both liraglutide and semaglutide have demonstrated significant effectiveness in the treatment of obesity. In the SCALE trial, liraglutide administration resulted in approximately 5–10% weight loss after 56 weeks, accompanied by improvements in glycemic control and cardiometabolic parameters [8].The efficacy of semaglutide has been demonstrated in the STEP clinical trial program. In the STEP 1 [10] trial, participants with overweight or obesity who receivedsemaglutide 2.4 mg QW for 65 weeks achieved a mean weight loss of 14.9%, while the placebo group lost an average of 2.4% [9]. In the STEP 5 trial, semaglutide led to an average weight loss of 15.2% after 104 weeks of treatment, compared with 2.6% in the placebo group [11]. These findings establishsemaglutideas one of the most effective pharmacological treatments currently available for obesity [9].

Tirzepatide is the first dualGIP/GLP-1 RA approved for obesity treatment in 2023 [12]. GIP and GLP-1 are endogenous incretin hormones that play complementary roles in regulating glucose metabolism and energy balance. By simultaneously activating both receptors, tirzepatide improves glycemic control while exerting potent effects on appetite regulation and body weight reduction. The clinical efficacy of tirzepatide has been demonstrated in the SURMOUNT [13,14] program in individuals with overweight or

obesity and in the SURPASS trial [15] in patients with obesity and T2D. Across these studies, treatment with tirzepatide at doses of 5 mg, 10 mg, or 15 mg QW resulted in a mean body weight reduction of approximately 15–20%, with some participants achieving weight loss approaching 25% [13–15].

Although incretin-based therapies have substantially improved obesity treatment, their use requires careful clinical monitoring because of potential adverse effects [16]. Despite their high efficacy in reducing body weight and improving glycemic control, GLP-1 RAs and dual GIP/GLP-1 RAs are commonly associated with gastrointestinal adverse events, including nausea, vomiting, diarrhea, and constipation, particularly during treatment initiation and dose escalation [13,15,17]. Clinical studies have reported that these symptoms occur in approximately 20–40% of treated patients, while 10–20% may require dose adjustments or treatment discontinuation due to poor tolerability [18]. In patients with pre-existing gastrointestinal disorders, the risk of more severe side effects, such as intestinal obstruction, gallbladder disease, or acute pancreatitis, may require careful clinical consideration.

Beyond their established metabolic effects, incretin-based pharmacotherapy appears to influence multiple dimensions of eating behavior by modulating appetite regulation, hunger and satiety perception, food cravings, emotional eating, and potentially shifting food preferences *[18]*. These behavioral adaptations may subsequently affect dietary quality and nutritional adequacy. Although numerous clinical studies have investigated selected aspects of these outcomes, the available evidence remains fragmented and has not yet been comprehensively analyzed. Moreover, most anti-obesity incretin-based therapies have been approved for clinical use only within the past decade, and their effects on food preferences, eating behavior, and dietary patterns are still being actively investigated, underscoring the need for the present review.

Therefore, this narrative review aims to comprehensively discuss the available clinical evidence regarding GLP-1 RAs and dual GLP-1/GIP RAs on eating behaviors in adults with obesity, including appetite, food preferences, food cravings, dietary intake, and diet quality. By integrating findings across these interrelated domains, this review seeks to provide a more comprehensive understanding of how incretin-based therapies influence eating behavior and identify priorities for future research and nutritional management.

2. Materials and Methods

2.1. Literature Search

This narrative review was based on a structured literature search conducted in the PubMed (MEDLINE), Embase, and Web of Science databases to identify relevant studies published between 2005 and February 2026. The search strategy included combinations of the following keywords and their synonyms: liraglutide, semaglutide, tirzepatide, GLP-1 receptor agonist, GIP/GLP-1 RAs, nutrition, eating behavior, feeding behavior, dietary intake, diet quality, appetite, food preference, food craving, and obesity. Boolean operators (AND/OR) were applied as appropriate. An example of search strings is presented in Table 1.

Table 1. Search strings example of searching in the Embase database.

Database	Search Terms
Embase	(‘excess body weight'/exp OR 'body weight, excess’ OR ‘excess body weight’ OR ‘overweight’ OR ‘obesity’/exp OR ‘obesity’) AND (‘liraglutide’/exp OR ‘glucagon like peptide 1 [7–36] [26 (6 n hexadecanoyl gamma glutamyllysine) 34 arginine]’ OR ‘liraglutide’ OR ‘liraglutide recombinant’ OR ‘saxenda’ OR ‘victoza’ OR

	'semaglutide'/exp OR 'glucagon like peptide 1 [7–36] [8 (2 amino 2 methylpropanoic acid) 26 [6 n [18 [n (17 carboxyheptadecanoyl) gamma glutamyl] 10 oxo 3, 6, 12, 15 tetraoxa 9, 18 diazaoctadecanoyl] lysine] 34 arginine]' OR 'ozempic' OR 'rybelsus' OR 'semaglutide' OR 'wegovy' OR 'tirzepatide'/exp OR 'mounjaro' OR 'mounjarokwikpen' OR 'tirzepatide' OR 'tyrosyl 2 methylalanyl alpha glutamylglycylthreonylphenylalanylthreonylseryl alpha aspartyltyrosylserylisoleucyl 2 methylalanylleucyl alpha aspartyllysylisoleucylalanylglutaminyl n6 (22, 42 dicarboxy 1, 10, 19, 24 tetraoxo 3, 6, 12, 15 tetraoxa 9, 18, 23 triazadotetracontan 1 yl) lysylalanylphenylalanylvalylglutaminyltryptophylleucylisoleucylalanylglycylglycylprolylserylserylglycylalanylprolylprolylprolylserinamide' OR 'zepbound' OR 'zepboundkwikpen' OR 'antiobesity agent'/exp OR 'anti obesity agents' OR 'anti-obesity agents' OR 'antiobesity agent' OR 'glucagon like peptide 1'/exp OR 'glp 1' OR 'glp-1' OR 'glucagon like peptide 1' OR 'glucagon like peptide I' OR 'glucagon-like peptide 1' OR 'glucagon-like peptide-1' OR 'glucagon like peptide 1 receptor agonist'/exp OR 'glp 1 agonist' OR 'glp 1 receptor agonist' OR 'glucagon like peptide 1 agonist' OR 'glucagon like peptide 1 receptor agonist' OR 'glucagon like peptide 1 receptor stimulating agent' OR 'glucagon-like peptide-1 receptor agonists' OR 'incretin mimetics' OR 'long acting glp 1 agonist' OR 'long acting glp 1 receptor agonist' OR 'long acting glucagon like peptide 1 agonist' OR 'long acting glucagon like peptide 1 receptor agonist' OR 'incretin mimetic agent'/exp) AND ('feeding behavior'/exp OR 'alimentary behavior' OR 'alimentary behaviour' OR 'behavior, alimentary' OR 'behavior, eating' OR 'behaviour, alimentary' OR 'behaviour, eating' OR 'eating behavior' OR 'eating behaviour' OR 'feeding behavior' OR 'feeding behaviour' OR 'feeding habit' OR 'feeding pattern' OR 'feeding time' OR 'food habit' OR 'food habits' OR 'meal time' OR 'nutrition habit' OR 'nutrition pattern' OR 'nutritional habit' OR 'appetite'/exp OR 'apetite' OR 'appetite' OR 'nutrition'/exp OR 'diet, food, and nutrition' OR 'nutrition' OR 'taste'/exp OR 'perception, taste' OR 'taste' OR 'taste perception' OR 'taste sense' OR 'food craving'/exp OR 'cravings for certain food' OR 'food craving' OR 'food cravings' OR 'food intake'/exp OR 'appetite regulation' OR 'feed intake' OR 'food consumption' OR 'food ingestion' OR 'food intake' OR 'food intake regulation' OR 'food uptake' OR 'meal ingestion' OR 'regulation, appetite' OR 'diet quality'/exp OR 'diet quality' OR 'dietary quality' OR 'emotional eating'/exp OR 'emotional eating')

2.2. Eligibility Criteria

Only human studies published in English and available in full text were considered eligible. Randomized controlled trials (RCTs), non-randomized clinical intervention studies, and observational studiesevaluating the effects of GLP-1 RAs or dual GIP/GLP-1 RAs on eating-related behaviors and dietary outcomes in adults (≥ 18 years) with overweight or obesity were included (BMI ≥25 kg/m^2). Given the limited number of studies investigating behavioral and dietary outcomes, studies were considered eligible regardless of intervention duration.

For the purpose of this review, eating-related behaviors were considered to encompass behavioral, perceptual, and dietary domains associated with food consumption. Therefore, studies regarding one or more of the following outcomes were considered eligible for inclusion:

- Appetite, hunger, satiety, fullness, prospective food consumption;
- Control of eating, food cravings, emotional eating;
- Food preferences, taste perception;
- Energy intake, diet quality, macronutrient intake, micronutrient intake, ultra-processed food consumption;
- Meal timing.

Because orforglipron received FDA approval after completion of the initial database search (April 1, 2026) [19], an additional targeted manual search was performed to identify any newly published studies relevant to the scope of this review. Only one eligible clinical study conducted in Japanese participants with T2D, reporting effects on appetite-related outcomes and meal intake was identified [20].

2.3. Study Selection and Data Synthesis

Titles and abstracts retrieved through the database search were screened for relevance, followed by full-text evaluation of potentially eligible studies. The selected studies were critically evaluated and narratively synthesized.

Data extracted from each included study included study design, study population, sample size, type and duration of incretin-based therapy, methods used to assess eating-related behaviors, and the principal outcomes.

The characteristics of the included studies are summarized in Table 2, whereas Table 3 synthesizes the reported effects of GLP-1 RAs and dual GIP/GLP-1 RAs on the analyzed behavioral and dietary outcomes.

Table 2. Characteristics of the studies included in the narrative review.

Reference	Study type	Population	Intervention	Outcomes Measured	Methods
Farr OM et al., 2016 [21]	Randomized, controlled trial (RCT), double-blind	Adults with type 2 diabetes (T2D), *n* = 21 (48.9 years; 31.5 kg/m^2; men (M): 50%))	Liraglutide 1.8 mg once a day (QD) (subcutaneous (s.c.)); for 17 days	Appetite, brain response to food cues,	Appetite ratings (visual analog scales (VAS)), functional magnetic resonance imaging (fMRI)
Tronieri JS et al., 2019 [22]	RCT	Adults with obesity, *n* = 113 (46.7 years; 38.8 kg/m^2; M: 23.9%)	Liraglutide 3.0 mg QD (s.c.) + intensive behavioral therapy (IBT) vs. IBT-alonevs. IBT + liraglutide3.0 mg QD (s.c.) + meal replacement diet for 52 weeks	Appetite (hunger, fullness), food preoccupation, meal liking, food craving	Appetite ratings (VAS)
Brindisi MC et al., 2019 [23]	Real-life clinical study	Adults with T2D and overweight/obesity, *n* =18 (56.5 years; 39.2 kg/m^2; M: 33.3%)	Liraglutide 1.2 mg QD (s.c.) for 12 weeks	Taste perception, Taste preference, olfactory food liking, and wanting	Taste preferences, food liking/wanting (VAS), PrefQuest, Three-Factor Eating Questionnaire (TFEQ)
Kadouh H et al., 2019 [24]	RCT	Adults with obesity, *n* = 40 (39.5 years; 35.9 kg/m^2; M: 10%)	Liraglutide 3.0 mg QD (s.c.) for 16 weeks	Appetite ratings (hunger, prospective food consumption, fullness/satiation, maximal tolerated volume (MTV)), taste preference,	Appetite (VAS, standardized nutrient drink test), and taste preference scores (VAS),
Coppin G et al., 2023 [25]	RCT, double-blinding	Women with obesity, *n* =73 (38.9 years, 35.4 kg/m^2; M: 0%)	Liraglutide 3.0 mg daily (s.c.) for 16 weeks	Food liking, neural response to food cues	Liking ratings; fMRI assessment of brain response to food stimuli

Silver HJ et al., 2023 [26]	RCT	Adults with obesity and prediabetes, *n* = 88 (50.3 years; 39.0 kg/m^2; M: 32%)	Liraglutide 1.8 mg QD (s.c.) vs. calorie restriction vs. sitagliptin (100 mg/d) for 14 weeks	Appetite (hunger), food preferences, dietary intake,	Appetite ratings and food preferences (VAS), three 24-h diet recalls,
Richardson KM et al., 2025 [27]	RCT (secondary analysis)	Adults with obesity and prediabetes, *n* = 70 (49.4 years; 39.5 kg/m^2; M: 31%)	Liraglutide 1.8 mg QD (s.c.) vs. caloric restriction vs. dipeptidyl peptidase-4 inhibitor (100 mg/day) for 14 weeks	Dietary intake, diet quality, dietary patterns	24-h dietary recall, Healthy Eating Index (HEI)-2020
Blundell J et al., 2017 [28]	RCT, double-blind, [cross-over]	Adults with obesity, *n* = 30 (42 years; 33.8 kg/m^2, M: 66.6%)	Semaglutide 1.0 mg QW (s.c.) for 12 weeks	Appetite (hunger, fullness, satiety, prospective food consumption), control of eating and food craving (over the past week), food preference, palatability of ad libitum meals, ad libitum energy intake during lunch meal, subsequent evening meal, and evening snack box, total day-time ad libitum energy intake, duration of ad libitum lunch,	Appetite rating (VAS); overall appetite suppression score (OAS), Control of Eating Questionnaire (CoEQ), palatability (taste, visual appearance, pleasantness) ratings (VAS), food preference and reward assessment (The Leeds Food Preference Task (LFPT)), ad libitum meal test;
Friedrichsen M et al., 2021 [29]	RCT [paralell]	Adults with obesity, *n* = 72 (42.8 years; 34.4 kg/ m^2, M: 61.1%)	Semaglutide 2.4 mg QW (s.c.)for 20 weeks	Appetite (satiety, fullness, prospective food consumption), food cravings, control of eating, food preference energy intake,	Appetite rating (VAS), CoEQ, standardized breakfast meal test, ad libitum lunch meal test;
Nicolau J et al., 2022 [30]	Observational prospective clinical study	Adults with obesity, *n* = 69 (43.7 years; 34.3 kg/ m^2, M: 17.3%)	Semaglutide 1.0 mg QW (s.c.) for 12 weeks	Emotional eating, food cravings, binge episodes	The Emotional Eater Questionnaire (EEQ), binge episodes, and food craving (sweet and savory) structured interview
Wharton S et al., 2023 *[10]*	RCT, double-blind	Adults with overweight/obesity, *n* = 174 (47 years; 38.7 kg/m^2; M: 22.4%)	Semaglutide 2.4 mg QW (s.c.) + lifestyle modification for 104 weeks	Control of eating	CoEQ
Gabe MBM et al., 2024 [31]	RCT, double-blind,	Adults with obesity, *n* = 61 (44 years; 34.9 kg/m^2; M: 65,6%)	Semaglutide 50 mg QD (oral) for 20 weeks	Appetite (hunger, fullness, satiety, prospective food consumption), control of eating, energy intake, and amount of food consumed,	Appetite ratings (VAS) after a standardized breakfast meal, OAS, CoEQ, appeal of the standardized breakfast, and ad libitum lunch (VAS), energy intake measurements during ad libitum lunch,

Jensterle M et al., 2025 [32]	RCT, single-blinded,	Women with obesity and PCOS, *n* = 30 (33.7 years; 36.4 kg/ m², M: 0%)	Semaglutide 1.0 mg QW (s.c.) for 16 weeks	Taste perception, brain response, gene expression analysis brain responses to visual cues of sweet and savory foods and to sweet solution dripping (on the tongue)	Taste recognition strip test; tongue biopsy, fMRI;
Heise T et al., 2023 [33]	RCT (secondary analysis)	Adults with T2D and obesity, *n* = 117 (61.7 years; 31.4 kg/m²; M: 73.5%)	Tirzepatide 15 mg QW(s.c.) vs. semaglutide 1.0 mg QD (s.c.) for 28 weeks	Appetite (hunger, satiety, fullness, and prospective food consumption), energy intake,	Fasting appetite ratings (VAS), ad libitum food intake during a 45-min buffet-style lunch,
Martin C et al., 2025 [34]	RCT (phase 1)	Adults with obesity, *n* = 114 (44.9 years; 36.2 kg/m²; M: 15%)	Tirzepatide 10 mg QW (s.c.) vs. liraglutide 3.0 mg QD (s.c.) vs. placebo for 6 weeks	Appetite, food craving, energy intake,	Appetite rating (VAS), Food Craving Inventory (FCI); Food Craving Questionnaire State (FCQ-S); Power of Food Scale (PFS); fRMI, ad libitum meal tests,
Kennedy A et al., 2025 [35]	RCT (secondary analysis)	Adults with obesity and at least one pre-specified metabolic impairment, *n* =55 (37.1 kg/m²; M: 16%)	Tirzepatide 15 mg QW (s.c.) for 18 weeks	Food craving, food preference	Food Preference Questionnaire (FPQ)FCI,
Ohwaki et al., 2026 [20]	RCT (phase 1)	Adults with T2D, *n*= 83 (56.1 years; 27.2 kg/m²; M: 96.7%)	Orforglipron 2 or 3 mg QD for 15 days (Phase A) and 12 mg, 24 mg, 45 mg QD for 84 days (Phase B)	Appetite (hunger, fullness, satiety, prospective food consumption), energy intake	Appetite ratings (VAS), 24-h diet recall;

Abbreviations: CoEQ—Control of Eating Questionnaire; FCI—food craving inventory; FCQ-S—Food Craving Questionnaire State; FPQ—food preference questionnaire; fRMI—functional magnetic resonance imaging; HEI—healthy eating index; IBT—intensive behavioral therapy; LFPT—The Leeds Food Preference Task; M—men; MTV—maximal tolerated volume; OAS—overall appetite suppression score; PFS—Power of Food Scale; PCOS—polycystic ovarian syndrome; QD—once a day; QW—one a week;s.c.subcutaneously; RCT—randomized-controlled trial; TFEQ—Three-factor Eating Questionnaire; T2D—type 2 diabetes; VAS, visual analog scale.

Table 3. Summary of the effects of GLP-1 RAs and dual GIP/GLP-1 RAs on eating-related behaviors and dietary outcomes in adults with obesity.

Reference	Intervention	Appetite	Control of Eating	Food Craving	Food Preference	Emotional Eating	Taste Perception	Energy Intake	Diet Quality	Macronutrient Intake
Farr OM et al., 2016 [21]	Liraglutide (0.6–1.8 mg QD (s.c.)	↑ fasting fullness (p< 0.006)	a trend towards less pleasant to eat food in the fasting state	↓inferior parietal cortex activation in response to highly desirable	NA	NA	NA	NA	NA	NA

	for 17 days		food cues (p< 0.001)							
Tronieri JS et al., 2019 [22]	Liraglutide 3.0 mg QD (s.c.) + intensive behavioral therapy (IBT) vs. IBT-alone vs. IBT + liraglutide + meal replacement diet for 52 weeks	in 6 and 24 weeks of intervention: greater ↓ in hunger (p =0.005; p =0.018) and greater ↑ in fullness (p =0.001, p = 0.01) in IBT + liraglutide vs. IBT-alone; ↔hunger and fullness between IBT-liraglutide and IBT—alone at weeks 40 or 52; in 6 and 24 weeks of intervention: greater ↓ food preoccupation in IBT + liraglutide vs. IBT-alone (p =0.002; p = 0.045) and in multicomponent vs. IBS-alone (p = 0.001; p= 0.022); IBT-liraglutide participants did not differ from multi-component participants in changes in appetite ratings at any assessment;	NA	reductions in cravings did not differ significantly among any of the groups at most time points	↓ meal liking in multicomponent groups (vs. IBT-alone through week 40; and vs. IBT-liraglutide through week 52); ↔ meal liking in IBT + liraglutide (vs. IBT alone); in IBT + liraglutide and IBT-alone groups meal liking increased slightly over the course of the 52-week trial;	NA	NA	NA	NA	NA

Brindisi MC et al., 2019 [23]	Liraglutide 1.2 mg QD (s.c.) for 3 months	↓ hunger (p =0.009)	NA	↓ wanting for sweet foods (p = 0.004); ↔ wanting for fatty foods	↓ preference for fatty foods (fatty-salty,p= 0.02; fatty-sweet,p= 0.04); ↔ preference for sweet foods; ↓preference for fat cake (borderline, p = 0.051)	NA	↑sweet taste sensitivity (p = 0.009); ↑ bitter taste sensitivity (p = 0.04); ↔ salty taste sensitivity;	NA	NA	NA
Kadouh H et al., 2020 [24]	Liraglutide 3 mg QD (s.c.) for 16 weeks	↓ overall appetite (p = 0.08), ↓ maximal tolerated volume (MTV) (p = 0.054), ↓ prospective food consumption (p = 0.03), ↑ perceived fullness (p = 0.02) vs. placebo	NA	NA	↓ preference to sweet (p = 0.02), salty (p = 0.005), savory (p = 0.006) or fatty food (p = 0.002);	NA	NA	NA	NA	NA
Coppin G et al., 2023 [25]	Liraglutide 3.0 mg QD(s.c.) for 16 weeks	NA	NA	NA	↔ food liking; ↔in reward-related neural responses	NA	NA	NA	NA	NA
Silver HJ et al., 2023 [26]	Liraglutide 1.8 mg QD (s.c.) vs. calorie restriction vs. sitagliptin (100 mg/d) for 14 weeks	↑ fullness (p = 0.003), ↔ hunger, prospective food consumption; ↔ satisfaction	NA	↔ desire for sweet and savory foods; ↔ desire for fatty/salty foods	NA	NA	NA	↓ energy intake (−293.8 kcal) (no significant vs. comparators)	NA	↔ fat; ↔ carbohydrates; ↔ protein; ↔ total and added sugars
Richardson KM et	Liraglutide	NA	NA	NA	NA	NA	NA	↓ energy intake (−284	↔in HEI-2020 score,	↔ in marco- and

al., 2025 [27]	1.8 mg/day for 14 weeks						kcal) (trend, $p = 0.062$) (no significant vs. comparators)	but negative changes in HEI-added sugar score (trend)	micronutrients intake
Blundell J et al., 2017 [28]	Semaglutide 1.0 mg QW (s.c.) for 12 weeks	↓ appetite (higher fasting overall appetite suppression scores in semaglutide; increasing difference towards the end of the 5 h postprandial period); ↓ hunger, ↓ meal duration	improved control of eating, including control of meal portion size	↓ food cravings; ↓ desire for savory foods, ↓ pleasantness of food	↓ preference for high-fat savory foods (↓ explicit liking of high-fat savory foods ($p = 0.0016$), ↓ implicit wanting of high-fat savory foods ($p = 0.0203$), and low-fat and sweet foods ($p = 0.0401$)), ↔explicit liking of high-fat sweet foods, low-fat foods, and implicit wanting of high-fat sweet foods and low-fat savory foods, no significant difference in palatability ratings between semaglutide and placebo for both ad libitumlunch and evening meal, except for taste of the ad libitum	NA	NA	↓ad libitum energy intake during lunch (−299.8 kcal; $p < 0.0001$) and during the subsequent evening meal (−175.6 kcal; $p = 0.0401$; 18%) and snacks (−245.5 kcal; $p = 0.0034$; 22%), 24% reduction in total energy intake across all ad libitum meals throughout the day (−725,1 kcal; $p < 0.0001$), lower intake from high-fat and non-sweet foods (−88.0 kcal; $p = 0.0184$; 35%) in the ad libitum evening snack box, ↔in energy intake from low-fat foods or high-fat and sweet foods	NA	↔ in macronutrient compositions of foods consumed in the ad libitum evening meal and evening snack (58 E% fat, 23 E% protein, 19 E% carbohydrate);

					lunch (p = 0.0398) and visual appearance of the ad libitum evening meal (p = 0.0432).					
Friedrichsen M et al., 2021 [29]	Semaglutide 2.4 mg QW (s.c.) for 20 weeks	↓ appetite (higher overall postprandial appetite suppression score in semaglutide vs. placebo (p = 0.001), ↓ hunger and prospective food consumption, ↑ fullness and satiety (versus placebo, all p<0.02).	better control of eating in semaglutide vs. placebo; ↑ fullness and contentment in semaglutide compared with placebo (p<0.05),	fewer/weaker food cravings in semaglutide vs. placebo (p<0.05); ↓ in desire and craving for savory foods, desire for sweet foods, and craving for dairy foods (versus placebo, all p<0.05).	NA	NA	NA	approx. 35% lower ad libitum energy intake during lunch in semaglutide vs. placebo (414.6 kcal vs. 639,15 kcal; p<0.0001); ↓ energy intake during lunch (−376.7 kcal in semaglutide vs. −152.1 kcal in placebo; p< 0.0001)	NA	NA
Nicolau J et al., 2022 [30]	Semaglutide 1.0 mg QW (s.c.) for 12 weeks	NA	NA	↓ sweet cravings (49.3% vs. 21.7%; p< 0.0001) and savory cravings (53.6% vs. 14.5%; p< 0.0001)	NA	↓ emotional eating (72.5% vs. 11.5%; p< 0.001), ↓ external eating (27.5% vs. 10.1%; p< 0.001), ↓ binge episodes (47.8% vs. 10.1%; p< 0.0001),	NA	NA	NA	NA
Wharton S et al., 2023 [10]	Semaglutide 2.4 mg QW (s.c.) + lifestyle modification	↓ hunger and ↓ fullness at week 20 (p < 0.001 for both)	↑craving control; ↑ control of eating; ↓ difficulty resisting cravings (weeks 20–	↓craving for savory foods at weeks 20, 52, and 104 (p<0.01), ↓ craving for sweet foods at	NA	NA	NA	NA	NA	NA

	for 104 weeks		104) (all $p<$ 0.05).	weeks 20 and 52 ($p<$ 0.05); ↓ desire to eat salty and spicy foods; ↓ cravings for dairy and starchy foods at 104 weeks (all $p<$ 0.05)						
Gabe MBM et al., 2024 [31]	Semaglutide 50 mg QW (oral) for 20 weeks	↓ appetite (higher OAS vs. placebo) ↓ hunger and prospective food consumption, ↑ fullness and satiety	improvement in control of eating	lower desire for sweet and savory foods, fewer cravings for sweet, dairy, starchy, and savory foods vs. placebo	NA	NA	NA	↓ ad libitum energy intake during lunch (−185.8 kcal, −33% vs. +55.2 kcal, +6.2% in placebo) ↓ food consumption	NA	NA
Jensterle M et al., 2022 [32]	Semaglutide 1.0 mg QW (s.c.) for 16 weeks	NA	NA	↓ reward-related response to visual food cues, particularly to calorie-dense, sweet, and savory foods); ↑ cortical response to sweet taste after a meal (all $p<$ 0.005)	NA	NA	↑ taste recognition, modulation of taste signaling–related gene expression	NA	NA	NA
Heise T et al., 2023 [33]	Tirzepatide 15 mg QW (s.c.) vs. semaglutide 1.0 mg QW (s.c.) for 28 weeks	↓ appetite in the tirzepatide and semaglutide group (p <0.001; no difference in appetite score reduction between the tirzepatide and semaglutide groups (p= 0.260); ↑ satiety and fullness and ↓	NA	NA	NA	NA	NA	↓ energy intake in tirzepatide (−348.4 kcal) and semaglutide (−284.1) group vs. placebo (−38.6 kcal; both $p<$ 0.001); Reduction in energy intake was numerically, but not significantly, greater with tirzepatide versus	NA	NA

		hunger and prospective food control in the tirzepatide and semaglutide groups					semaglutide (p = 0.187)	
Martin C et al., 2025 [34]	Tirzepatide 10 mg QW vs. placebo vs. liraglutide 3.0 mg QD for 6 weeks	↓ overall appetite), hunger, prospective food consumption (all, p<0.001), ↑ satiety (p< 0.01), and fullness (p<0.001) in tirzepatide, ↓ overall appetite, hunger, prospective food consumption (all, p< 0.01), ↔ in satiety and fullness in liraglutide	↓lack of eating control in tirzepatide and liraglutide (both, p< 0.01), ↓ disinhibition (tendency to overeat) in tirzepatide (0.0001) but not in the liraglutide group (p = 0.0711); ↔cognitive restraint in tirzepatide and liraglutide group (in liraglutide trend p =0.0723), ↓ BIS in tirzepatide (p = 0.0084), but not in liraglutide 0.3167)	↓ overall food craving (FCI), FCQ-S, ↓ physiological hunger (both, p = 0.001), in tirzepatide, ↓ overall food craving (p< 0.01), FCQ-S, and physiological hunger (p< 0.01) in liraglutide, ↓ desire to eat in tirzepatide (p< 0.001) and liraglutide (p< 0.01); ↓anticipation of positive and negative reinforcement in tirzepatide (both, p< 0.001) and liraglutide (p< 0.01 and p< 0.05, respectively); Greater improvement in overall craving, FCQ-S, and desire to eat in tirzepatide vs. liraglutide. Greater decrease in overall food craving, FCQ-S, in the tirzepatide vs. the liraglutide group. ↓ fMRI BOLD for high-fat/high sugar foods activation in the orbitofrontal cortex,	↓ craving for sweets, high-fats, high-fat foods, and and carbohydrates/starches (all, p< 0.001) in tirzepatide; ↓ craving for sweets, high-fats, CHO/starches (all, p< 0.05), and high-fat foods (p< 0.01) < 0.001) in liraglutide; ↔ craving for vegetables and fruits in tirzepatide and liraglutide; ↓ overall food responsiveness (PFS) and responsiveness for food available, food present, and food tasted in tirzepatide	↓ Eating to relieve negative emotions (↓ FCQ-S negative reinforcement)	NA	↓ energy intake at lunch in tirzepatide (−658 kcal) and liraglutide (−315 kcal) group vs. placebo (both p<0.001); Significant greater reduction in energy intake in tirzepatide vs. liraglutide (p< 0.001)	NA	↓ intake of fat, carbohydrates/sugar, protein (in ad libitummeals) in tirzepatide and liraglutide groups

			cingulate gyrus, hippocampus, and medial frontal gyrus in tirzepatide	(all, p = 0.001), ↓ overall food responsiveness (PFS) (p< 0.05) and responsiveness for food available (p< 0.001) and food tasted (p< 0.05) in liraglutide					
Kennedy A et al., 2025 [35]	Tirzepatide 15 mg QW (s.c.) for 6 weeks	NA	NA	↓overall food cravings, ↓ craving for sweets, carbohydrates (CHO)/starches, and fast-food fats (p < 0.05), ↔ preferences for high-fat or fruits and vegetables	↓ preference for high-fat and high-simple sugar and high-fat/high-simple sugar foods (p <0.001), ↔ preference for fat (p =0.0594)	NA	NA	NA	NA	NA
Ohwaki et al., 2026 [20]	Orforglipron 2 or 3 mg QD for 15 days (Phase A) and 12 mg, 24 mg, 45 mg QD for 84 days (Phase B)	↓ hunger and prospective food consumption ↑ fullness ↔ satiety	NA	NA	NA	NA	NA	↓ in energy intake (but no data about p value)	NA	NA

Meal timing, meal structure and ultra-processed food intake have not been assessed in analyzed studies; ↑ increase; ↓ decrease;↔ no significant change; Abbreviations: CHO—carbohydrates; FCI—food craving inventory; FCQ-S—Food Craving Questionnarie State; fRMI—functional magnetic resonance imaging; HEI—healthy eating index; IBT—intensive behavioral therapy; MTV—maximal tolerated volume; NA—not applicable; OAS—overall appetite suppression score; PFS—Power of Food Scale; QD—once a day; QW—one a week;s.c.—subcutaneously.

Because the objective of this study was to provide a comprehensive narrative synthesis of the available clinical evidence rather than to conduct a systematic review or meta-analysis, no formal assessment of methodological quality or risk of bias was performed.

3. Results

3.1. Characteristics of Included Studies

A total of 17 studies published between 2016 and 2026 met the eligibility criteria and were included in the narrative synthesis. Their characteristics are summarized in Table 2.

Most studies were RCTs, including double-blind, single-blind [32], crossover [28], phase 1 [20,34], and secondary-analysis designs [27,33,35]. Two studies were observational or real-life clinical studies [23,30].

Sample sizes ranged from 18 to 174 participants [10,23], and the study populations primarily comprised adults with overweight/obesity [10,22,24,25,29–31,34] or with overweight/obesity and obesity-related metabolic diseases, such as pediabetes [26,27], T2D [20,21,23,33], polycystic ovary syndrome [32], or other pre-specified metabolic impairment [35].

Liraglutide was evaluated in seven studies [21–27], with doses ranging from 1.2 to 3.0 mg/day and intervention periods of 12–52 weeks, whereas semaglutide was investigated in six studies [10,28–32] using subcutaneous doses of 1.0–2.4 mg/week or an oral dose of 50 mg/day, with intervention periods ranging from 12 to 104 weeks. Tirzepatide was assessed in three studies, using doses ranging from 10 to 15 mg/week [33–35], with intervention periods of 6–28 weeks. One phase 1 study evaluated oral orforglipron at different doses over 15- and 84-day treatment periods [20].

Several studies included active comparators or adjunctive interventions, such as intensive behavioral therapy (IBT) [22], caloric restriction [26,27], lifestyle modification[10], or other glucose-lowering medications [26,27].

The outcomes assessed varied across studies but predominantly focused on appetite and eating-related responses. Appetite-related measures included hunger, fullness, satiety, prospective food consumption, and overall appetite suppression, most commonly assessed using visual analogue scales and standardized meal or nutrient-drink tests. Other frequently investigated domains included food cravings, control of eating, food preferences, food liking and palatability, dietary intake, and energy intake. Several studies additionally assessed eating behavior using validated questionnaires, including the Control of Eating Questionnaire (CoEQ), Three-Factor Eating Questionnaire (TFEQ), Emotional Eater Questionnaire (EEQ), Food Craving Inventory (FCI), Food Preference Questionnaire (FPQ), and Leeds Food Preference Task (LFPT) [23,28–31]. Objective or neurobehavioral measures included ad libitum meal tests, dietary recalls, functional magnetic resonance imaging (fMRI) [25,32], and, in one study, taste recognition testing and gene expression analysis [32].

3.2. Appetite

One of the most consistently reported effects of incretin-based anti-obesity pharmacotherapy is appetite reduction. Many AOMs act through central and peripheral pathways involved in appetite regulation, leading to increased satiety and reduced hunger signals.

In a 16-week RCT, liraglutide 3 mg QD (s.c.), combined with dietary and behavioral counseling, was associated with lower prospective food consumption and greater perceived fullness compared to placebo. Trends toward reduced overall appetite ($p = 0.08$) and lower maximal tolerated volume during the standardized drink test ($p = 0.054$) were also observed, although these differences did not reach statistical significance. Similarly,

Tronieri et al. [22] demonstrated that intensive behavioral therapy (IBT) combined with liraglutide 3.0 mg QD was associated with a greater reduction in hunger and food preoccupation, as well as a greater increase in post-meal fullness, compared with IBT alone. These improvements persisted through week 24;however, between-group differences in appetite-related outcomes were no longer observed at week 52. Silver et al. [26] demonstrated a significant increase in fullness (p = 0.003) and a reduction in prospective food consumption (p= 0.02) in adults with obesity and prediabetes treated with liraglutide 1.8 mg QD (s.c.).

Evidence from RCTs demonstrated that semaglutide also improved multiple dimensions of appetite regulation. Across studies assessing appetite, most interventionsdemonstrated that semaglutide administration was associated with reduced appetite and hunger, accompanied by increased satiety and fullness [10,28,29,31]. Moreover, a decrease in prospective food consumption [29] was also observed, indicating a diminished anticipated desire to eat. These effects were evident after subcutaneous and oral administration of semaglutide at doses of 1.0 mg QW and 2.4 mg QW. Interestingly, in the study by Wharton et al. [10], treatment with semaglutide 2.4 mg QW led to a significant reduction in hunger and an increase in fullness at week 20; however, these changeswere no longer significant at weeks 52 and 104.

In a study by Martin et al. [34], 6 weeks of tirzepatide 10 mg QW (s.c.) led to highly significant reductions in overall appetite, hunger, and prospective food consumption, as well as increases in satiety and fullness. In the same study, positive effects of liraglutide 3 mg QD (s.c.) on overall appetite suppression, hunger, and prospective food consumption were also demonstrated; these effects were smaller than those observed in the tirzepatide group. Appetite suppression after 28 weeks of semaglutide 1.0 mg QW (s.c.) or tirzepatide 15 mg QW (s.c.) treatment, as well as improvement in hunger, prospective food consumption, satiety, and fullness, has also been observed in Heise et al. [33].

Objective assessments further supported these findings, demonstrating reduced ad libitumenergy intake and food consumption during the standardized meal test. Although the magnitude of these effects varied across studies, the direction of change was remarkably consistent, suggesting a sustained improvement in appetite regulation with GLP-1 RA and dual GIP/GLP-1 RA treatment.

3.3. Food Craving, Emotional Eating,and Control of Eating

Several studies have reported improvements in food craving and eating control during GLP-1 RAs and dual GIP/GLP-1 RAs.

Liraglutide has been associated with reductions in food-related preoccupation. Brindishi et al. [23] reported decreasedwanting for sweet foods and recalled liking for fatty foods in adults with T2D and obesity treated with liraglutide. Similarly, Tronieri et al. [22] found that IBT combined with liraglutide 3.0 mg QD resulted in a greater reduction in food preoccupation than IBT alone at weeks 6 and 24; however, this effect was no longer evident at week 52. Importantly, reductions in craving frequency did not consistently differ between treatment groups, suggesting that the effects of liraglutide on food-related preoccupation and craving may not be sustained over longer treatment periods. In contrast, Silver et al. [26] did not observe significant effects of liraglutide on the desire for sweet or savory foods. Neuroimaging studies provide additional evidence that liraglutide may influence responses to food-related cues. Farr et al. [21] demonstrated a significant reduction in activation of the inferior parietal cortex in response to highly desirable food cues following short-term liraglutide treatment.

Evidence for semaglutide similarly suggests effects on several dimensions of eating behavior, including improved control of eating [10,28,29,31] and reductions in craving for specific food categories [10,28–32].

Blundell et al. [28] reported improved control over eating, including better control of meal portion size,accompanied by reductions in food cravings, desire for savory foods, and the pleasantness of food inindividuals treated with semaglutide 1.0 mg QW (s.c.). Reductions in cravings for savory and sweet foods following semaglutide treatment have also been observed in other studies [32–34,37], while some studies have reported reductions in cravings for dairy, spicy, or starchy foods [10,29]. In addition, Jensterle et al. [32] reported that semaglutide 1.0 mg QW (s.c.) altered taste perception and neural responses to food cues in women with obesity and polycystic ovary syndrome (PCOS), including reduced reward-related brain activation in response to visual food cues.

Tirzepatide has also demonstrated effects on food craving. Kennedy et al. [35] reported significant reduction inoverall food craving, particularly for sweets, carbohydrates/starches and fast-food fats. Martin et al. [34] evaluated the multi-dimensional effect of 6-week tirzepatide or liraglutide administration on specific behavioral domains, and reported distinct therapeutic profiles for both incretin-based agents across craving, eating control, and emotional eating. In this study, tirzepatideat a dose of 10 mg QW (s.c.) demonstrated a markedly superior efficacy profile compared to liraglutide 3 mg QD (s.c.). Compared to placebo, both therapies significantly reduced overall cravings, but tirzepatide exerted a more profound suppressive effect on the physiological dimensions of craving, significantly mitigating the rewarding aspects of cravings, such as the general desire to eat and the anticipation of positive reinforcement. The observed reductions in psychological cravings were further supported by neurobiological data. At week 6, tirzepatide demonstrated a significant advantage over liraglutide by selectively decreasing brain BOLD fMRI activation in response to high-fat, high-sugar food stimuli within key reward-processing regions, including the medial frontal gyrus, cingulate gyrus, and orbitofrontal cortex. In contrast, liraglutide failed to induce any significant changes in brain activation to these hyper-palatable food cues compared to placebo [34].

Regarding the control of eating, both interventions exerted distinct temporal effects on dietary restriction and behavioral governance. In week 6, cognitive restraint levels remained statistically unchanged from baseline in tirzepatide and liraglutide groups, but a trend towards an increase was observed in liraglutide ($p = 0.0723$). Interestingly, a time-dependent divergence was observed earlier, after 3 weeks of administration, while a significant increase in cognitive restraint was achieved in liraglutide (dose 1.8 mg QD (s.c.)).

Emotional eating represents another potentially relevant behavioral domain, although the evidence is more limited than for food craving.

Nicolau et al. [30] reported significant reductions in emotional eating among individuals treated with semaglutide compared with baseline measures. In this prospective observational clinical study, administration of semaglutide 1.0 mg QW (s.c.) for 3 months resulted in reduced proportion of participant with abnormal eating patterns, including emotional eating (72.5% vs. 11.5%; $p< 0.001$), external eating (27.5% vs. 10.1%; $p< 0.001$); and binge episodes (47.8% vs. 10.1%; $p< 0.0001$) [30]. In the study byMartin et al. [34], 6 weeks of tirzepatide treatment also reduced disinhibition (decreased tendency to overeat in response to emotional or external cues). By contrast, the reduction in disinhibition observed in the liraglutide group did not reach statistical significance at week 6, remaining as a non-significant trend.

Overall, the available evidence suggests that semaglutide and tirzepatide may reduce emotional eating, whereas the effect of liraglutide remains inconclusive, and no study has specifically evaluated liraglutide in this context.

3.4. Food Preference and Taste Perception

Evidence regarding the effects of GLP-1 RAs and dual GIP/GLP-1 RAs on food preference patterns and taste perception remains limited.

In Kadouh et al. [24] study, 16 weeks of liraglutide 3.0 mg QD reduced preferences for sweet ($p = 0.02$), salty ($p = 0.005$), savory ($p = 0.006$), and fatty foods ($p = 0.002$), suggesting a reduction in preference for highly palatable foods. Similarly, a significant reduction in preference for fatty foods, including both fatty-salty and fatty-sweet foods, accompanied by increased sweet and bitter taste sensitivity after liraglutide treatment was observed by Brindishi et al. [23]. Contrary, Tronieri et al. [22] observed no significant differences between IBT-liraglutide and IBT-alone groups in meal liking, indicating that liraglutide has no sustained effect on the hedonic evaluation of meals over the 52-week intervention. Consistently, Coppin et al. [25] reported no differences in self-reported food liking or reward-related brain activation following liraglutide treatment compared to placebo.

Across studies on semaglutide, RCTs conducted by Blundell et al. [28] reported reduced desire for highly palatable foods, particularly sweet, savory, starchy, and dairy products during treatment. In women with obesity and PCOS [32], semaglutide improved overall recognition of basic tastes, accompanied by altered expression of taste signaling–related genes in tongue tissue. Semaglutide also reduced putamen activation in response to calorie-dense sweet and savory food cues and modulated cortical responses to sweet taste stimuli, suggesting changes in sensory processing of food.

Tirzepatide therapy has also been associated with shifts in food preference. The study by Martin et al. [34]demonstrated a shift in food preferences toward nutrient-dense choices under both treatments, driven by a selective reduction in hedonic desires for tirzepatide and liraglutide treatments. In this study, tirzepatide 10 mg QW (s.c.) and liraglutide 3 mg QD (s.c.) led to significant reductions in cravings for sweets, high-fat foods, and carbohydrates/starches, with no significant effects on cravings for vegetables and fruits. This shift in food preferences was accompanied by a marked downregulation of environmental food responsiveness, as measured by the Power of Food Scale (PFS). Similarly, Kennedy et al. [35] described decreased preference for high-fat and high-simple sugar and high-fat/high-simple sugar foods after tirzepatide treatment.

These findings suggest that pharmacotherapy may influence sensory and hedonic responses to food, potentially contributing to reduced intake of calorie-dense foods.

3.5. Dietary Intake, Energy Intake,and Diet Quality

Among the analyzed studies, only a few evaluated the effects of GLP-1 RAs and dual GIP/GLP-1 RAs on changes in the energy and nutritional value of the diet.

Silver et al. [26] reported that participants receiving liraglutide reduced their energy intake (about 300 kcal); however, the reduction was comparable to that observed in the calorie restriction and sitagliptin groups. Additionally, no significant changes in macronutrient distribution or total and added sugar intake were observed with liraglutide treatment. Similarly, secondary analysis of an RCT by Richardson et al. [27] revealed that participants receiving liraglutide reduced their energy intake (about 300 kcal); however, the magnitude of the reduction was comparable to that observed in the calorie restriction and dipeptidyl peptidase-4 inhibitor groups. In this study, liraglutide did not significantly alter dietary macronutrient distribution, total and added sugar intake, or overall diet quality, although participants across groups failed to meet recommended intakes for several vitamins and minerals. Only a small proportion of individuals in the liraglutide group achieved recommendations for vitamin D (9%), fiber (18%), potassium (8%), vitamin E (21%), vitamin C (35%), calcium (41%), and magnesium (41%), indicating potential risk of inadequate micronutrient intake.

Other incretin-based therapies have shown comparable effects. In the study by Blundell et al. [28], semaglutide significantly reduced ad libitum energy intake compared with placebo, with lower energy consumption observed at lunch, the evening meal, and during snacks, resulting in a 24% reduction in total daily energy intake. In addition, intake of high-fat, non-sweet foods during the evening snack was reduced, whereas no significant differences were observed for low-fat foods or high-fat sweet foods. Similarly, Friedrichsen et al. [29] in an RCT reported that treatment with semaglutide (2.4 mg weekly) resulted in a significantly lower ad libitumenergy intake at lunch compared with placebo, with participants receiving semaglutide consuming approximately 35% fewer calories during ad libitum meals.

Heise et al. [33] observed significant reductions in energy intake in patients treated with tirzepatide (−348.4 kcal) or semaglutide (−284.1 kcal), but reductions in energy intake were numerically, but not significantly, greater with tirzepatide compared to semaglutide ($p = 0.187$). Similarly, Martin et al. [34] demonstrated that both tirzepatide and liraglutide significantly reduced energy intake during standardized meal tests, but the reduction in energy intake was significantly greater with tirzepatidethan with liraglutide (−658 kcal vs. −315 kcal; $p< 0.001$). In this study, a reduction in energy intake was associated with a significant reduction in fat, protein, and carbohydrates in both groups.

Overall, available evidence suggests that incretin-based therapies consistently reduce energy intake, with more pronounced and statistically significant effects observed versus placebo, whereas in trials using active comparators such as caloric restriction, reductions in energy intake appear broadly comparable between treatment groups. However, evidence regarding their effects on dietary nutritional value and overall diet quality remains limited, highlighting the need for further research in this area.

4. Discussion

The available clinical evidence indicates that GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists affect eating behavior, involving not only reductions in hunger and a greater feeling of satiety but also influencing interconnected aspects of appetite regulation, including food cravings, emotional eating,energy intake,taste perception,and, potentially, food preferences. These effects seem clinically relevant since reductions in energy intake constitute an important component of pharmacologically induced weight loss, while changes in the hedonic value of food may facilitate sustained reductions in the consumption of energy-dense foods [10].

The results of this narrative review indicate that most AOMs (liraglutide, semaglutide, tirzepatide) increase satiety and reduce hunger signals through central and peripheral pathways involved in appetite regulation. Beyond appetite, GLP-1RAs appear to modify the motivational and hedonic aspects of eating. Semaglutide treatment has been associated with a lower relative preference for high-fat foods, as well as reductions in food cravings and food preoccupation [28]. Similar effects have been described with liraglutide. In individuals receiving liraglutide together with intensive behavioral therapy, hunger and food preoccupation decreased, and post-meal fullness increased more than with behavioral therapy alone during the earlier stages of treatment [22]. However, meal liking did not consistently change, suggesting that the medications may reduce motivation to eat without making food universally less pleasant. Nevertheless, these behavioral adaptations may subsequently affect dietary quality and nutritional adequacy. While incretin-based therapies may directly reduce appetite, food cravings, and food preoccupation, behavioral interventions may facilitate the translation of these physiological effects into more sustainable changes in eating habits. Consequently, some of the observed improvements in eating behavior may reflect the complementary effects of pharmacotherapy and behavioral support. This interaction may be particularly relevant for long-term weight

management, as behavioral strategies can promote self-monitoring, reinforce healthy eating patterns, and support adherence to dietary recommendations over time.

Changes in food preference and taste perception represent another potentially important mechanism of AOM action. It is suggested that GLP-1RAs may reduce the desire for highly palatable foods, particularly those rich in fat, sugar, or both.A study by Jamshed et al. [36] examined the effects of caloric restriction (CR) on appetite and cravings after 24 h of caloric deprivation. Findings revealed that individuals reported heightened cravings for high-calorie foods, particularly chocolate and sweet snacks. This suggests that even short-term caloric restriction can intensify feelings of hunger and amplify the desire for "forbidden" foods. Additionally, a long-term study on CR found that participants following a CR diet exhibited a skewed perception of food intake and often underestimated total caloric intake. In the study conducted by Dorling JL et al. [37], the impact of energy restriction - whether through intermittent or continuous dieting - on the constructs of 'liking' (pleasure derived from eating) and 'wanting' (motivation for food) was assessed. The primary outcome demonstrated a reduction in 'liking,' but a relatively stable level of desire for food, indicating that overall food cravings remained consistent despite changes in pleasure. Thus, it seems that CR may potentially disturb food preferences. On the contrary, the use of AOMs may influence sensory and hedonic responses to food, potentially contributing to reduced intake of calorie-dense foods. A study by Martin et al. [34] demonstrated that both tirzepatide and liraglutide medications selectively downregulate the desire for highly palatable, energy-dense foods without diminishing the appeal of healthier options. It should be noted that,while evidence supporting the effects of incretin-based therapies on appetite and energy intake is relatively consistent across studies, findings regarding food preferences and taste perception remain more heterogeneous. Differences in study design, intervention duration, and assessment methods may partly explain these inconsistencies.

Emotional eating refers to the tendency to consume food in response to emotional states rather than physiological hunger. This behavior is frequently observed in individuals with obesity and may contribute to overeating and difficulties in weight management. The available evidence suggests that semaglutide and tirzepatide may also reduce emotional eating, whereas the effect of liraglutide remains inconclusive, and no study has specifically evaluated liraglutide in this context.The mechanisms underlying these effects remain incompletely understood. However, growing experimental evidence suggests that incretin-based therapies may influence neural pathways involved not only in appetite regulation but also in food reward and motivational eating. Animal studies have demonstrated GLP-1 receptor signaling within brain regions associated with reward processing, including the nucleus accumbens, where activation of GLP-1 pathways reduces food intake and the rewarding value of palatable foods. These findings suggest that reductions in emotional eating may reflect not only decreased physiological hunger but also attenuated reward-driven and cue-induced eating behaviors. Given the important role of emotional eating in overeating and weight regain, modulation of food reward pathways may represent an additional mechanism contributing to the therapeutic effects of incretin-based therapies [38–40].

Despite growing evidence demonstrating the effects of anti-obesity medications on appetite and food cravings, relatively few studies have examined their impact on overall diet quality and nutrient intake. Understanding these aspects is important because changes in appetite and food preferences may influence dietary patterns and nutritional adequacy. A better understanding of these behavioral responses may also have implications beyond dietary management alone. Individuals whose obesity is characterized by increased appetite, heightened food reward, frequent cravings for energy-dense foods, or maladaptive eating behaviors may potentially represent subgroups particularly

responsive to incretin-based therapies. Although further validation is required, such characteristics may eventually support a more individualized approach to obesity treatment and dietary counseling.

During pharmacotherapy for obesity, treatment should be combined with lifestyle interventions, particularly dietary therapy. Therefore, appropriate dietary management is crucial, especially in the context of the individual pharmacotherapy course and the patient's clinical status. The diet should be tailored to current clinical needs, considering both reduced tolerance to certain foods and increased requirements for specific nutrients. Proper dietary adjustments can help alleviate side effects associated with pharmacotherapy, maintain a healthy weight, and improve overall well-being, all of which are essential for effective obesity treatment [41]. For patients using AOMs who experience reduced appetite, it is advisable to focus on small, frequent meals that provide essential nutrients without overwhelming the digestive system. It is recommended to consume regular, appropriately portioned meals throughout the day. Although incretin-based therapies often reduce appetite, excessive restriction of food intake or prolonged intervals between meals may result in compensatory hunger and subsequent overeating in some individuals. Regular meal patterns may therefore support adequate nutrient intake and improve adherence to dietary recommendations [42].

It is also crucial to maintain adequate protein intake to prevent muscle loss, as weight reduction with these treatments may lead to a decrease in lean body mass [43]. For individuals undergoing pharmacological treatment for obesity, it is recommended that their diet includes adequate protein intake, with a target of approximately 1.2–1.6 g of protein per kilogram of body weight per day (15–18%) to support satiety, preserve lean body mass, and improve metabolic outcomes. Nutritional guidelines also recommend that the diet of individuals with obesity include fats rich in polyunsaturated fatty acids, accounting for 35–45% of total daily caloric intake, with saturated fats comprising less than 10%. Additionally, carbohydrates should constitute 35–45% of total daily caloric intake [43,44]. Despite reduced appetite, the diet should remain balanced and include nutrient-dense foods [45].

Available studies indicate a significant impact of pharmacotherapy on eating behaviors and diet quality, but there are still notable gaps in knowledge. Most research focuses on short-term treatment effects, with limited long-term data available, and primarily emphasizes positive nutritional changes during pharmacological treatment, often overlooking potential negative effects such as deterioration in diet quality due to reduced appetite or altered taste perception. Additionally, there is a lack of consistent tools to assess diet quality in patients undergoing pharmacotherapy, and changes in taste preferences and food perception remain poorly studied, requiring further analysis in the context of individual differences. Despite these limitations, available evidence suggests that proper dietary support can significantly improve pharmacological treatment outcomes and enhance patients' quality of life [26].

Clinical Implications and Future Perspectives

The observed effects of incretin-based therapies may have implications extending beyond weight reduction alone. Current evidence suggests that GLP-RAs and dual GIP/GLP-1 RAs influence multiple dimensions of eating behavior, including appetite regulation, food reward, food cravings, emotional eating, taste perception, and food choice. These behavioral and sensory responses may become increasingly relevant as obesity management moves toward more individualized treatment strategies.

From a clinical perspective, a better understanding of these responses may facilitate the identification of patient subgroups who are particularly likely to benefit from incretin-based pharmacotherapy. Individuals whose obesity is predominantly driven by

hyperphagia, heightened food reward, hedonic eating, emotional eating, or persistent cravings for energy-dense foods may represent clinically relevant phenotypes with differing responsiveness to treatment. Consequently, behavioral phenotyping may, in the future, complement traditional clinical and metabolic assessment when selecting therapeutic approaches [46]. These observations align also with the concept of Precision Obesity Medicine, in which treatment is tailored not only to biological and metabolic characteristics but also to the behavioral determinants of energy intake. Future research should determine whether appetite regulation, food cravings, food reward responsiveness, taste perception, and other eating-behavior characteristics can serve as reliable predictors of treatment response and support the development of more personalized obesity treatment algorithms.

Furthermore, assessment of appetite-related and sensory characteristics could potentially contribute to predicting treatment response and optimizing therapeutic strategies. Such information may support individualized treatment selection, tailoring of dose-escalation schedules, and adjustment of treatment intensity according to patients' behavioral profiles. However, these potential applications remain hypothetical and require validation in prospective studies before they can be incorporated into routine clinical decision-making [46,47].

The findings of this review also highlight the importance of integrating pharmacotherapy with individualized nutritional counseling. Although incretin-based therapies may facilitate reductions in appetite and improve control over eating behavior, long-term treatment success remains dependent on maintaining adequate diet quality and nutritional status. Consequently, dietary assessment and nutritional support should be considered integral components of obesity pharmacotherapy rather than adjunctive interventions [48].

Finally, it should be noted that GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists are prescription medications that require medical supervision. Although most injectable formulations are intended for self-administration after appropriate patient education, misuse or unsupervised dose adjustments may result in excessive appetite suppression and substantially reduced food intake. Consequently, some individuals may be at risk of inadequate nutrient consumption, loss of lean body mass, and the development of micronutrient deficiencies. Furthermore, prolonged reliance on markedly reduced food intake may contribute to maladaptive eating behaviors, such as meal skipping, irregular eating patterns, and poor dietary quality, which could adversely affect long-term nutritional health. Therefore, appropriate nutritional counseling and clinical monitoring remain essential during treatment.

5. Strengths and Limitations

The strengths of this review include the comprehensive evaluation of currently availableevidence regarding the effects of GLP-1 RAs and dual GIP/GLP-1 RAs on appetite, food preferences, taste perception, and eating behavior. Although the metabolic effects of incretin-based therapies have been extensively investigated, their impact on eating and food-related outcomes has been addressed less frequently, and dedicated reviews focusing on these aspects remain limited. Furthermore, the review integrates findings across multiple behavioral domains that are often examined separately, providing a broader perspective on the mechanisms through which these therapies may influence dietary intake and weight management

This narrative review has also several limitations. Firstly, while both human and animal studies were included to illustrate physiological and behavioral mechanisms, the inclusion was based on relevance rather than a systematic quality assessment, which may introduce selection bias. Secondly, the current literature provides limited data on dietary

quality during treatment with GLP-1 RAs and GIP/GLP-1 RAs. Few studies assess not only the quantity but also the quality of food intake, making it difficult to evaluate how incretin-based pharmacotherapy influences overall dietary patterns. Additionally, there is a lack of follow-up research examining whether changes in eating behavior persist after discontinuation of pharmacotherapy, or how sustainable these changes are in real-life settings. Another limitation is insufficient evidence on the composition of diet during or after treatment — for instance, whether patients shift towards more nutrient-dense eating or continue with highly processed, energy-dense food consumption. An additional limitation should be considered when interpreting the findings of this review. Changes in food preferences, taste perception, and dietary behaviors observed during treatment with GLP-1 RAs and GIP/GLP-1 RAs may not be attributable exclusively to the pharmacological action of these agents. Individuals seeking weight reduction are often simultaneously engaged in broader lifestyle modifications, including dietary changes, increased physical activity, self-monitoring practices, participation in behavioral or nutritional counseling, and other health-promoting behaviors. Moreover, heightened motivation to lose weight and improved health following treatment initiation may independently influence eating behavior. Consequently, the relative contribution of medication-specific effects and concurrent behavioral factors cannot be fully disentangled based on the currently available evidence.

6. Conclusions

Current evidence consistently supports the beneficial effects of incretin-based therapies on appetite regulation and energy intake. Evidence regarding their influence on food preferences, taste perception, and overall diet quality is promising but remains less consistent and warrants further investigation.

Pharmacotherapy of obesity plays a key role in managing patients with obesity by regulating food intake and influencing taste preferences, in that way modifying appetite**.** Available studies suggest that GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists may facilitate healthier food choices, improve control over eating behavior, and support long-term weight management.Overall, incretin-based therapies appear to influence not only body weight but also several behavioral determinants of food intake. A better understanding of these effects may enhance the integration of pharmacological and nutritional approaches to obesity management and contribute to more effective long-term treatment strategies.

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Abbreviations

The following abbreviations are used in this manuscript:

BMI	body mass index
CHO	carbohydrates
CoEQ	Control of Eating Questionnaire
CR	caloric restriction
FCI	food craving inventory
FCQ-S	Food Craving Questionnarie State
FDA	Food and Drug Administration
FPQ	food preference questionnaire
fRMI	functional magnetic resonance imaging
GIP	glucose-dependent insulinotropic polypeptide
GLP-1	glucagon-like peptide 1
GLP-1 RAs	glucagon-like peptide-1 receptor agonists
HEI	healthy eating index
IBT	intensive behavioral therapy
LFPT	The Leeds Food Preference Task
MTV	maximal tolerated volume
NA	not applicable
PCOS	polycysic ovarian syndrome
PFS	Power of Food Scale
RCTs	Randomized controlled trials
s.c.	subcutaneously
TFEQ	Three-factor Eating Questionnaire
T2D	type 2 diabetes
OAS	overall appetite suppression score;
QD	once daily
QW	once weekly
VAS	visual analog scale

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