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Review

Nutritional and lifestyle strategies in obesity management during incretin-based therapy: a narrative review with practical recommendations

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ABSTRACT

The growing use of glucagon-like peptide 1 receptor agonists and dual glucagon-like peptide 1/gastric inhibitory polypeptide receptor agonists in obesity management has highlighted the need for structured nutritional care alongside pharmacotherapy. This narrative review describes the main nutritional pillars in the care of patients receiving incretin-based therapies and proposes a practical framework for nutritional follow-up. PubMed, Embase, and the Cochrane Library were searched from inception to May 2026. We independently selected studies on dietary intake, body composition, lean mass, sarcopenia, micronutrient inadequacies, gastrointestinal symptoms, and nutritional strategies and graded the evidence using the Oxford Centre for Evidence-Based Medicine 2011 levels. Specialized nutritional therapy, ideally delivered by a registered dietitian/nutritionist within a multidisciplinary team, is central to preserving muscle mass, preventing nutritional inadequacies, improving gastrointestinal tolerability, and sustaining weight loss. Adequate protein intake (1.2–1.6 g/kg/d), body composition monitoring, resistance exercise, hydration, and micronutrient surveillance are particularly relevant. We propose the preservation of muscle mass, routine screening for deficiencies, adequate hydration, tailored nutritional therapy, and observation of gastrointestinal symptoms framework; the Incretin Plate Model; and the Metabolic Hourglass Model. These expert-derived tools have not yet been validated. Individualized, systematized nutritional care may maximize the benefits of incretin-based therapies while reducing nutritional and functional risks. Prospective studies are needed to validate the proposed models and to establish standardized monitoring and supplementation protocols.

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Introduction

The global prevalence of obesity and type 2 diabetes mellitus has risen substantially, increasing the burden of cardiovascular disease, metabolic complications, and impaired quality of life. Incretin-based therapies—namely, glucagon-like peptide 1 (GLP-1) receptor agonists (GLP-1RAs) and dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonists—have transformed obesity management, with landmark trials such as the Semaglutide Treatment Effect in People with Obesity (STEP) program and the SURMOUNT program of tirzepatide demonstrating substantial weight loss alongside glycemic, cardiovascular, and functional benefits [1–3].

Despite this efficacy, the effects of these therapies on dietary intake, nutritional status, and body composition remain incompletely understood. Appetite suppression, delayed gastric emptying, and gastrointestinal symptoms may reduce energy intake (by approximately 35% in a controlled feeding study of semaglutide 2.4 mg [4]), compromise macronutrient and micronutrient adequacy, and contribute to lean mass loss; these concerns are particularly relevant in older adults and individuals with sarcopenic obesity [5].

GLP-1 receptor agonists are now central agents in obesity pharmacotherapy, supported by robust trial and body composition evidence [6–8]. Real-world data show that weight loss is generally more modest than in trials, although comparable outcomes are achievable with high adherence; discontinuation within the first year remains common, largely owing to gastrointestinal intolerance, cost, and maintenance challenges [9]. Gastrointestinal adverse events remain the most frequent safety concern, with no

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consistent evidence linking these agents to pancreatitis, pancreatic cancer, thyroid disease, depression, or self-injurious behavior [9,10].

Nutritional care is therefore a fundamental component of optimizing outcomes in patients with obesity treated with incretin-based agents, particularly regarding lean mass preservation, nutritional adequacy, gastrointestinal symptom management, and long-term metabolic sustainability. Obesity care is team-based, and current guidance recommends that dietary counseling during GLP-1–based therapy be provided by registered dietitians/nutritionists working within the multidisciplinary team [1,11]. This review aimed to synthesize current evidence and propose structured, evidence-informed nutritional and lifestyle recommendations for patients receiving GLP-1 receptor agonists or dual GLP-1/GIP receptor agonists [12].

Methods

Design

This narrative review adopted a translational approach focused on the nutritional care of patients with obesity undergoing treatment with GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists. Because the objective was to support clinical practice rather than to estimate pooled effects, a narrative design with a structured, reproducible search was chosen. The manuscript was prepared following the Scale for the Assessment of Narrative Review Articles (Supplementary Table S1) [13].

Search strategy

A bibliographic search was conducted in PubMed, Embase, and the Cochrane Library for publications available from the inception of each database through May 31, 2026, using the descriptors "GLP-1 receptor agonists," "GIP/GLP-1 dual agonists," "nutrition," "dietary intake," "lean body mass," "sarcopenia," and "obesity" individually and in combination with the Boolean operators AND and OR. No language restrictions were applied, although English language studies were prioritized. No start date limit was applied; however, because the first GLP-1 receptor agonist for chronic weight management (liraglutide 3 mg) was approved in December 2014 following its pivotal trial [14], the eligible literature on nutritional care during incretin-based obesity therapy was published from 2015 onward, with earlier publications addressing GLP-1 receptor agonists mainly in type 2 diabetes. Publications not retrievable with these descriptors but required for the review—namely, dietary reference standards and clinical guidelines, methodological references, studies on dietary behaviors, and the Bariatric Plate Model on which the Incretin Plate Model is based [15]—were added purposively by us.

Eligibility and study selection

Eligible designs included randomized controlled trials, observational studies, meta-analyses, systematic reviews, and relevant narrative reviews addressing dietary intake, body composition, lean mass loss, sarcopenia risk, macronutrient and micronutrient inadequacies, hydration status, gastrointestinal symptoms, and nutritional strategies associated with incretin-based therapies. Clinical practice guidelines and consensus statements from scientific societies were also eligible but only as sources of practice recommendations, not as evidence of efficacy. Letters to the editor, conference abstracts, editorials, animal and in vitro studies, and investigations not addressing nutritional or lifestyle outcomes were excluded as evidence for recommendations. Short commentaries were cited only to provide clinical context and were never used as the sole support for quantitative recommendations; one preclinical study was retained exclusively to illustrate a mechanistic hypothesis on muscle reversibility [16].

We independently screened titles and abstracts and then assessed the full texts of potentially eligible publications; disagreements were resolved by discussion until consensus was reached. Because the aim was translational rather than exhaustive, when several publications addressed the same question, the most recent, comprehensive, or highest-level evidence was retained (e.g., systematic reviews/meta-analyses and randomized trials over observational studies and observational studies over expert consensus). This prioritization explains the focused number of references relative to the breadth of the search.

Appraisal of the evidence

The level of evidence of each primary study, meta-analysis, and guideline was graded independently by us using the Oxford Centre for Evidence-Based Medicine 2011 levels of evidence [17]. The characteristics and evidence levels of the key studies are summarized in Table 1, practical protein intake recommendations are presented in Table 2, and the evidence supporting each component of the proposed model is mapped in Table 3. Consistent with the narrative design, a formal risk-of-bias assessment of individual studies (e.g., the Cochrane Risk of Bias 2 [RoB 2] tool) was not performed; this is acknowledged as a limitation. In addition to synthesizing the available evidence, this review proposes a structured nutritional care model aimed at preserving muscle mass, ensuring nutritional adequacy, managing gastrointestinal symptoms, and supporting the metabolic sustainability of weight loss during incretin-based obesity treatment.

Evidence synthesis

Clinical indications, nutritional assessment, and the role of the dietitian

GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists are indicated for individuals with a body mass index (BMI) $\geq$27 kg/m^2 with at least one weight-related comorbidity or BMI $\geq$30 kg/m^2 regardless of comorbidities [12]. Nutritional assessment should encompass clinical history, comorbidities, weight trajectory, dietary patterns, gastrointestinal symptoms, eating disorders, food preferences, and nutritional inadequacy risk alongside physical assessment (weight, height, circumference, muscle strength, body composition) and relevant biochemical parameters [12,24]. Follow-up should ideally begin before or concurrently with pharmacotherapy, particularly during dose escalation, with periodic monitoring of body composition, and macronutrient and micronutrient status, as well as screening for sarcopenia. Dietary tracking applications, food diaries, and telehealth platforms may support adherence and longitudinal monitoring [24].

Obesity treatment relies on a team-based model, and the registered dietitian/nutritionist is the professional best placed to implement the nutritional care described in this review. The joint advisory of four scientific societies recommends pairing GLP-1 therapy with dietary counseling by a registered dietitian/nutritionist to support medication adherence, prevent or manage adverse gastrointestinal effects, and promote nutrient adequacy within multidisciplinary care that may also include physicians, exercise professionals, and behavioral health specialists [1]. Dietitian-delivered weight management interventions improve body weight,

Table 1
Characteristics and level of evidence of the key studies included in the evidence synthesis

Study	Design	Population	Intervention; duration	Main findings relevant to nutritional care	Evidence level
STEP 1 [2]	Phase 3 RCT	Adults with overweight/obesity without diabetes ($N = 1961$); DXA substudy $n = 140$	Semaglutide 2.4 mg/wk + lifestyle vs placebo; 68 wk	Weight −14.9% vs −2.4%; DXA fat mass −19.3%, lean mass −9.7%	2
SURMOUNT-1 [3]	Phase 3 RCT	Adults with obesity/overweight without diabetes ($N = 2539$)	Tirzepatide 5, 10, 15 mg vs placebo; 72 wk	Weight −15.0%, −19.5%, −20.9% vs −3.1%	2
SURMOUNT-1 DXA [6]	DXA substudy of RCT	$N = 160$ (124 tirzepatide, 36 placebo)	Tirzepatide vs placebo; 72 wk	Fat mass −33.9%, lean mass −10.9%; ~75% of weight lost was fat and ~25% was lean mass	2
STEP 3 [18]	Phase 3 RCT	Adults with overweight/obesity ($N = 611$)	Semaglutide 2.4 mg vs placebo + 30 intensive behavioral therapy visits with a registered dietitian, 8-wk meal replacement diet and then 1200–1800 kcal/d; 68 wk	Weight −16.0% vs −5.7%	2
STEP UP [8]	Phase 3b RCT	Adults with obesity ($N = 1407$; 5:1:1)	Semaglutide 7.2 mg vs 2.4 mg vs placebo; 72 wk	Weight −18.7% vs −15.6% vs −3.9% (treatment policy estimand)	2
Friedrichsen et al. [4]	RCT	Adults with obesity ($N = 72$)	Semaglutide 2.4 mg vs placebo; 20 wk	Ad libitum energy intake 35% lower; weight −9.9% vs −0.4%; less hunger and fewer food cravings	2
S-LITE [19]	RCT	Adults with obesity after an 8-wk low-calorie diet (−13.1 kg) (N = 195)	Exercise, liraglutide 3 mg, combination, or placebo; 52 wk	Exercise plus liraglutide vs placebo, from randomization to 52 wk: additional weight loss of 9.5 kg and a 3.9-percentage-point greater reduction in body-fat percentage	2
SURMOUNT-OSA [20]	Secondary analysis of 2 RCTs	Adults with OSA and obesity ($N = 469$)	Tirzepatide vs placebo; 52 wk	Improvement in sleep-disordered breathing associated with weight reduction	3
SEMALEAN [7]	Prospective real-world cohort (uncontrolled)	Severe obesity, mean BMI 46.3 kg/m^2 ($N = 106$)	Semaglutide 2.4 mg; 12 mo	Weight −13%; fat mass −18%; lean mass stabilized after initial decline; handgrip +4.5 kg; sarcopenic obesity 49%→33%	4
Johnson et al. [21]	Cross-sectional	GLP-1RA users for ≥1 mo ($N = 69$)	Not applicable (cross-sectional dietary intake assessment)	Intakes below recommendations for fiber (14.5 g/d), calcium, vitamin D, iron, magnesium, potassium; fat 39.9% of energy; protein below needs in g/kg	4
Sinha and Ghosal [22]	Systematic review and meta-analysis	9 studies: 3 RCTs (n = 430) and 6 observational studies	GLP-1RAs; not applicable (meta-analysis)	RCTs: no significant reduction in alcohol consumption; observational studies: fewer alcohol-related events (HR, 0.64)	1 (RCTs); 3 (observational studies)
Morgan-Bathke et al. [23]	Systematic review and meta-analysis	Adults with overweight/obesity	Weight management interventions delivered by dietitians vs usual care not applicable (meta-analysis)	Greater percent weight loss (MD −4.0%); lower BMI, waist circumference, and blood pressure; better quality of life	1
Jeromson et al. [16]	Preclinical (mice)	Mice with diet-induced obesity	Semaglutide vs calorie-matched restriction; 4 wk (plus 6-wk withdrawal follow-up)	Similar reductions in muscle size and strength; recovery after discontinuation	Not graded (mechanistic context only)

BMI, body mass index; DXA, dual-energy x-ray absorptiometry; GLP-1RA, glucagon-like peptide 1 receptor agonist; HR, hazard ratio; MD, mean difference; OSA, obstructive sleep apnea; RCT, randomized controlled trial STEP, Semaglutide Treatment Effect in People with Obesity.
Levels of evidence adapted from to the Oxford Centre for Evidence-Based Medicine [17]: 1) systematic review of randomized trials, 2) randomized trial, 3) non-randomized cohort or secondary analysis, 4) cross-sectional study or uncontrolled cohort, and 5) mechanism-based reasoning or expert consensus; guidelines and consensus statements used for recommendations are graded in Table 3

Table 2
Practical protein intake recommendations during incretin-induced weight loss

Clinical objective	Protein target	Notes
Minimum recommended intake (general population)	0.8 g/kg/d	Recommended dietary allowance for healthy adults
Critical minimum threshold	0.4–0.5 g/kg/d	Values below this range may predispose to muscle loss
Active weight loss	1.2–1.6 g/kg/d	Recommended range to preserve lean mass during energy restriction; in obesity, preferably calculated on adjusted or reference body weight
Patients with obesity and available body composition data	~1.5 g/kg lean mass per day	May be more precise than calculations based on total body weight
Alternative practical target	80–120 g/d	Equals 16–24% of energy on the 2000-kcal/d reference diet of the source advisory; scaled to the 1200- to 1800-kcal/d range used in this review (≈80 g at 1200 kcal; ≈120 g at 1800 kcal), corresponds to ≈25–30% of energy *
Prolonged high intake	≥2.0 g/kg/d	Should be avoided or used only with individualized indication and monitoring

*Illustrative macronutrient distribution within the energy range adopted in this review: 1200 kcal/d = protein 80 g (27% of energy), carbohydrate 135 g (45%), fat 37 g (28%); 1800 kcal/d = protein 120 g (27%), carbohydrate 210 g (47%), fat 53 g (27%); both examples respect the recommended carbohydrate (45–65% and ≥130 g/d) and fat (20–35%) ranges [31].Adapted from Mozaffarian et al. [1].

waist circumference, blood pressure, and quality of life [23], and in the STEP 3 trial semaglutide was combined with 30 intensive behavioral therapy visits with a registered dietitian, resulting in a mean weight change of −16.0% versus −5.7% with placebo at 68 wk [18].

Pharmacodynamic effects and nutritional implications

GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists act through central appetite modulation, delayed gastric emptying, and glucose-dependent insulin secretion, promoting spontaneous reductions in energy intake and clinically significant weight loss [1]. Class representatives include semaglutide, liraglutide, dulaglutide, and tirzepatide (dual GLP-1/GIP agonism); trials report reductions in caloric intake of 16% to 39% and weight loss of 5% to 18%. In a randomized trial, semaglutide 2.4 mg reduced ad libitum energy intake by 35% versus placebo after 20 wk, accompanied by less hunger, fewer food cravings, and better control of eating [4].

Tirzepatide enables weight reduction ≥20% of initial body weight with a tolerability profile comparable to selective GLP-1 receptor agonists, with additional benefits in obstructive sleep apnea, metabolic dysfunction–associated steatohepatitis, and heart failure with preserved ejection fraction as well as reduced progression to type 2 diabetes [25]. In the STEPUP trial, semaglutide 7.2 mg produced greater weight loss than 2.4 mg (20.7% versus 17.5%, trial product estimand, 18.7% versus 15.6% treatment-policy estimand, with more patients achieving losses ≥20% and ≥25% [8]. In SURMOUNT-OSA, tirzepatide improved obstructive sleep apnea alongside weight loss [20]; the SURPASS (type 2 diabetes) and SURMOUNT (obesity) programs demonstrated dose-dependent reductions in glycated hemoglobin and body weight, with unprecedented efficacy among pharmacologic obesity therapies [26]. Despite these benefits, marked reductions in energy intake raise the risk of subclinical nutritional inadequacy. In a cross-sectional study of GLP-1 receptor agonist users, insufficient intake of fiber, calcium, iron, magnesium, and potassium as well as vitamins A, C, E, and D was observed even when relative protein intake appeared adequate as a percentage of energy [21], a concern also discussed in a narrative review of dietary intake in this population [27].

Lean mass loss and sarcopenia risk

GLP-1 receptor agonist–induced weight loss may include significant lean mass reduction, raising sarcopenia concerns, particularly in older adults, patients with low protein intake, and those with preexisting sarcopenic obesity. Reviews estimate that 15% to 40% of total weight loss corresponds to fat-free mass (FFM) reduction [28], approaching magnitudes seen with severe calorie restriction [29]. In the dual-energy x-ray absorptiometry substudies of

Table 3
Evidence map of the PRATO framework with supporting evidence and level of evidence for each pillar

PRATO pillar	Recommendation	Main supporting evidence	Highest evidence level	Nature of the evidence
P: Preservation of muscle mass	Protein 1.2–1.6 g/kg/d (adjusted body weight); resistance training; body composition and handgrip monitoring	DXA substudies quantifying lean mass loss [2,6]; RCT of exercise combined with a GLP-1RA [19]; protein targets from consensus [1,12]	2 (lean mass loss, exercise); 5 (protein target)	RCT data plus expert consensus
R: Routine screening for deficiencies	Laboratory assessment at baseline and at 3, 6, and 12 mo	Cross-sectional intake data [21]; narrative reviews [27,33]	4	Extrapolated from bariatric surgery practice
A: Adequate hydration	At least 2–3 L/d in small volumes between meals	Expert recommendations [38,39]	5	Expert consensus
T: Tailored nutritional therapy	Individualized plan within 1200–1800 kcal/d; Incretin Plate Model; delivered by a registered dietitian/nutritionist	Obesity guideline [11]; meta-analysis of dietitian-delivered interventions [23]; RCT with dietitian-led behavioral therapy [18]	1 (dietitian-delivered care); 5 (plate model)	Systematic evidence plus untested model
O: Observation of gastrointestinal symptoms	Smaller, more frequent meals; lower-fat meals; fluids between meals; gradual soluble fiber	Multidisciplinary consensus [38]; reviews [9,10]; RCT on appetite and gastric emptying [4]	5 (dietary measures)	Expert consensus

DXA, dual-energy x-ray absorptiometry; GLP-1RA, glucagon-like peptide 1 receptor agonist; RCT, randomized controlled trial
Levels of evidence adapted from to the Oxford Centre for Evidence-Based Medicine [17]: 1) systematic review of randomized trials, 2) randomized trial, 3) non-randomized cohort or secondary analysis, 4) cross-sectional study or uncontrolled cohort, and 5) mechanism-based reasoning or expert consensus; the PRATO framework itself is an expert-derived model that has not been validated

the pivotal trials, semaglutide reduced total fat mass by 19.3% and lean mass by 9.7% [2], and tirzepatide reduced fat mass by 33.9% and lean mass by 10.9%, so that approximately 75% of the weight lost was fat and 25% was lean mass [6]. However, part of this FFM reduction may reflect decreased total body water and physiological adaptation rather than functional muscle impairment, and body composition can improve despite absolute lean mass loss when accompanied by substantial visceral fat loss.

In a preclinical study in mice, semaglutide produced muscle size and strength reductions similar to calorie restriction, although it was more efficient at reducing fat, and lean mass loss reversed after treatment discontinuation, suggesting an adaptive, reversible process [16]; this hypothesis has not been tested in humans. In the SEMALEAN study, a prospective real-world cohort of 106 patients with severe obesity (68.9% women; mean BMI 46.3 kg/m^2) treated with semaglutide 2.4 mg for 12 mo, patients lost 13% of body weight (59% achieving ≥10% loss), fat mass fell 18%, lean mass stabilized after an initial modest decline, handgrip strength increased (+4.5 kg), sarcopenic obesity prevalence fell from 49% to 33%, and resting energy expenditure adjusted for lean mass improved [7]. In a randomized trial, combining supervised exercise with liraglutide after diet-induced weight loss reduced body fat percentage more than either intervention alone (−3.9 percentage points versus placebo) [19]. These findings suggest the clinical impact of lean mass loss depends on muscle quality and functional capacity, not just absolute quantity, reinforcing the importance of body composition monitoring, protein adequacy, and resistance training, particularly in patients at higher risk of frailty.

Dietary planning during incretin-based therapy

Dietary planning should prioritize nutritional quality, lean mass preservation, and prevention of deficiencies; cardiometabolically favorable patterns (Mediterranean, DASH, or plant-predominant, minimally processed diets) are recommended, provided that protein and micronutrient adequacy are addressed [12,24]. As practical references, energy intake may range from 1200 to 1500 kcal/d for women and 1500 to 1800 kcal/d for men [11], individualized by age, body composition, activity, comorbidities, and response; restriction below these thresholds increases the risk of inadequate intake of iron, calcium, magnesium, and zinc as well as vitamins A, D, E, K, B1, B12, and C [1], and added sugar should remain below 10% of total energy [30].

Adequate protein density is the central nutritional priority during incretin-induced weight loss, using reduced dietary volume as an opportunity to improve diet quality rather than as a driver of deficiency (Table 2) [28,29]. Because protein targets in the source advisory were expressed for a 2000-kcal/d reference diet (80–120 g/d, 16–24% of energy) [1], they were recalculated for the 1200- to 1800-kcal/d range adopted in this review. Scaling the absolute target to energy intake (approximately 80 g/d at 1200 kcal and 120 g/d at 1800 kcal) corresponds to approximately 25% to 30% of energy, whereas carbohydrate and fat remain within their recommended ranges (Table 2). In individuals with obesity, gram per kilogram targets calculated on actual body weight may exceed what is achievable within these energy levels; adjusted or reference body weight or lean mass–based targets (approximately 1.5 g/kg lean mass per day) are therefore preferable [1]. Protein distribution across meals rather than total intake alone is clinically relevant: an early protein-rich meal can help patients with satiety or nausea meet daily targets [29]. When intake is insufficient, whey protein supplementation may be considered individually, and creatine monohydrate may support muscle function alongside resistance training, taking into account renal function and patient context.

The dimension of dietary behavior also matters: reduced appetite and food preoccupation create a window for nutrition education (distinguishing physiological from emotional hunger, reducing ultra-processed food intake, and reintroducing whole foods) [24], moving beyond restriction toward sustainable dietary patterns [12]. Nutritional care must therefore integrate protein adequacy, dietary quality, micronutrient monitoring, and dietary behavior strategies to maximize fat loss while preserving lean mass and function [1,5].

Macronutrient and micronutrient adequacy during incretin-based therapy

Although GLP-1 receptor agonists may reduce FFM, this does not necessarily reflect proportional skeletal muscle or functional loss, as FFM includes body water, organs, and connective tissue [32]. Reduced dietary intake may increase subclinical nutritional inadequacy, particularly with prolonged calorie restriction, low dietary diversity, or persistent gastrointestinal symptoms [1,27,33]; vitamin B12, iron, magnesium, and vitamin D warrant closer monitoring given their roles in muscle function, energy metabolism, hematopoiesis, and quality of life. Omega-3 fatty acids may potentiate metabolic benefits and support muscle preservation through anti-inflammatory and metabolic mechanisms [32,34], although clinical data in GLP-1 receptor agonist users are lacking.

Resistance training with adequate protein remains the primary non-pharmacologic strategy for muscle preservation [1,12,24], although evidence specific to incretin-based therapy remains limited [19]. Branched-chain amino acids, particularly leucine, creatine, vitamin D, and omega-3 fatty acids have been proposed as adjuncts for patients at higher sarcopenia risk or unable to meet protein targets, although universal recommendations are not yet supported and individualization is needed [34]. Novel pharmacologic approaches are also under investigation, including bimagrumab, a monoclonal antibody that inhibits the activin type II receptor to modulate myostatin/activin signaling, with early-phase data suggesting a benefit for muscle preservation alongside fat loss [34]. Overall, the nutritional approach should extend beyond energy restriction to encompass dietary quality, protein adequacy, micronutrient monitoring, and targeted strategies for muscle preservation throughout treatment.

Dietary fiber and carbohydrate quality

Adequate fiber intake should be encouraged for its metabolic, cardiometabolic, satiety, and gastrointestinal benefits, prioritizing vegetables, legumes, and whole fruit over juices for their higher fiber content and lower glycemic load [24]. Recommended intake is 14 g per 1000 kcal, corresponding to approximately 25 g/d for women and 30 to 38 g/d for men [31], with intake of approximately 35 g/d beneficial in prediabetes and diabetes [35]; in GLP-1 receptor agonist users, fiber intake was found to be well below these targets (mean 14.5 g/d) [21]. Emerging evidence suggests that fiber may modulate the therapeutic response to GLP-1 receptor agonists via gut microbiota interactions and colonic fermentation metabolites, potentially improving weight loss maintenance and reducing regain risk after dose reduction or discontinuation [36]. Carbohydrates should represent 45% to 65% of energy intake and not fall below the recommended dietary allowance of 130 g/d [31], prioritizing whole, minimally processed sources, with

reduced refined sugar and ultra-processed food intake, particularly in patients with insulin resistance or diabetes [24].

Dietary fat quality and lipid intake

Fat intake should represent approximately 20% to 35% of total energy (approximately 27–70 g/d within the 1200- to 1800-kcal/d range) [31], emphasizing monounsaturated and polyunsaturated sources (olive oil, canola oil, nuts, seeds, avocados, and fatty fish) for their cardiometabolic and anti-inflammatory benefits, whereas saturated fat should remain below 10% of energy [37]. In GLP-1 receptor agonist users, fat was found to provide a mean of 39.9% of energy—more than the recommended range [21].

Gastrointestinal tolerability must also be considered, as high-fat meals may worsen nausea, fullness, reflux, and discomfort owing to treatment-induced delayed gastric emptying [38]. Distributing fat intake across meals, favoring lower-fat cooking methods, and prioritizing higher-quality fat sources may improve both adherence and gastrointestinal tolerability.

Hydration

Adequate hydration is an important component of nutritional support given the high prevalence of nausea, vomiting, early satiety, and constipation associated with delayed gastric emptying; insufficient intake may worsen symptoms and increase dehydration risk, particularly with markedly reduced dietary intake [24,38]. Fluid should be distributed throughout the day, preferably between meals and in small volumes, with a practical target of at least 2 to 3 L/d [39], individualized by body weight, activity, comorbidities, medications, and gastrointestinal losses. Water, sugar-free flavored water, and unsweetened herbal teas should be preferred; combined with adequate fiber intake, hydration is particularly relevant for reducing constipation risk [36,38].

Micronutrient supplementation and muscle preservation

Sustained reductions in energy intake may increase macronutrient and micronutrient inadequacy, particularly with prolonged hypocaloric diets, low dietary diversity, or persistent gastrointestinal symptoms, warranting periodic biochemical and nutritional monitoring in higher-risk patients [1,24]. B-complex vitamins (thiamine, pyridoxine, folate, B12) and fat-soluble vitamins A, D, E, and K along with iron, calcium, magnesium, zinc, and potassium are the nutrients most frequently affected, especially with insufficient intake, persistent vomiting, or highly restrictive diets [21,27,33]. A complete multivitamin with calcium and vitamin D has been suggested to reduce deficiency risk when intake is insufficient [39].

Sibal et al. [33] reported that GLP-1 receptor agonist therapy consistently reduces caloric intake, often with inadequate protein consumption and increased risk of lean mass loss and sarcopenia. Deficiencies, particularly of vitamin D, thiamine, and other B-complex vitamins, may emerge within the first 12 mo in the absence of standardized monitoring protocols, a gap the authors suggest could be addressed using nutritional surveillance strategies analogous to postbariatric surgery follow-up.

Beyond protein intake and resistance training, adjunct nutrients such as, branched-chain amino acids (particularly leucine), creatine, vitamin D, and omega-3 fatty acids may benefit selected individuals (older adults, sarcopenic patients, those with low habitual protein intake), although evidence remains limited [32,34]. Supplementation should therefore be individualized according to dietary intake, body composition, gastrointestinal symptoms, activity level, comorbidities, and nutritional risk and integrated with clinical laboratory monitoring to optimize weight loss quality and reduce lean mass–related complications.

Dietary behavior and alcohol consumption

GLP-1 receptor agonists have been proposed to reduce alcohol cravings and consumption, potentially through central effects on reward circuitry and motivation and attenuation of hedonic hunger [40]. However, the evidence is inconsistent: in a recent meta-analysis, pooled randomized trials (three trials, 430 participants) showed no significant reduction in total alcohol consumption (standardized mean difference, −0.24; 95% confidence interval, −0.70 to 0.23), whereas the observational studies included in the same meta-analysis associated GLP-1 receptor agonist use with fewer alcohol-related events (hazard ratio, 0.64; 95% confidence interval, 0.59–0.69) [22]. Reduced alcohol intake would offer additional nutritional benefit given its high calorie density and adverse effects on hydration, nutrient absorption, dietary quality, body composition, and sleep, potentially improving dietary adherence and self-regulation. Current data remain insufficient for specific recommendations, and further research is needed; meanwhile, approaches targeting dietary behaviors, such as mindful eating and improved recognition of hunger and satiety cues, may support more sustainable dietary change [41].

Management of gastrointestinal symptoms and dietary behaviors

Delayed gastric emptying requires dietary adaptation, particularly during dose escalation; nausea, vomiting, postprandial fullness, constipation, and dyspepsia are the most common adverse effects and may compromise intake, diet quality, and adherence [9,10]. Smaller, more frequent meals as well as reduced high-fat intake, better tolerated food preparations, adequate fluid intake between meals, and gradual introduction of soluble fiber may improve tolerability, constipation, and microbiota modulation [9,10,24,38].

Strategies targeting dietary behaviors, such as slow, deliberate chewing as well as mindful eating, recognition of hunger and satiety cues, and prioritization of nutrient-dense foods, may improve tolerability, self-regulation, and long-term sustainability [12,24]. Lifestyle factors, including stress management, sleep, physical exercise, and social support, further contribute to adherence, quality of life, and sustained outcomes.

Discussion

The efficacy of GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists in reducing body weight and improving glycemic control is well established [2,3], yet important gaps remain in the systematization of nutritional care, particularly regarding energy and protein adequacy, muscle mass preservation, prevention of deficiencies, and management of gastrointestinal symptoms related to delayed gastric emptying [24,33]. The spontaneous reduction in energy intake induced by these therapies affects not only dietary volume but also diet quality, dietary behavior, and macronutrient and micronutrient intake [21,24,27], underscoring the need for structured interventions that extend beyond pharmacologic efficacy alone [1,12].

An important finding of this review is the asymmetry of the evidence base (Tables 1 and 3). The effects of incretin-based therapies on weight, energy intake, and body composition derive from randomized trials [2–4,6,8], whereas most specific nutritional recommendations (protein targets, hydration, micronutrient surveillance schedules, and gastrointestinal management) are based on cross-

sectional data, extrapolation from other weight loss settings, and expert consensus [1,12,38,39]. Clinicians should therefore regard these recommendations as pragmatic, evidence-informed guidance rather than as interventions of proven efficacy in GLP-1 receptor agonist users.

The impact on body composition remains one of the most debated aspects of incretin-based therapy. Although FFM reductions of up to 40% of total weight loss have been reported, data from the SEMALEAN cohort suggest that functional improvements in muscle strength and reduced sarcopenic obesity prevalence can occur even alongside modest lean mass loss [7,28], reinforcing the value of functional assessments (handgrip strength, physical performance, and lean mass–adjusted resting energy expenditure) alongside absolute lean mass measurements.

Protein adequacy (1.2–1.6 g/kg/d during active weight loss, individualized by body composition, activity, and sarcopenia risk) remains the cornerstone nutritional priority [1,29]; distributing protein across meals is particularly relevant given that early satiety and nausea limit intake per meal, and supplementation (protein, creatine) may be considered individually when dietary targets are not met [29]. Micronutrient deficiency risk is an underrecognized concern: Sibal et al. [33] found that inadequacies, particularly of vitamin D, thiamine, and other B-complex vitamins, may emerge within the first 12 mo of treatment in the absence of standardized monitoring, supporting the case for postbariatric surgery–style surveillance protocols in this population [27,33].

Dietary fiber and carbohydrate quality also warrant attention beyond their established glycemic and cardiovascular benefits: fiber may modulate endogenous GLP-1 secretion via gut microbiota interactions, potentially amplifying therapeutic effects and supporting long-term weight loss sustainability, positioning fiber optimization as a complementary rather than merely an adjunct strategy [36]. Investigational pharmacologic approaches targeting muscle preservation, such as bimagrumab and other activin/myostatin pathway inhibitors, are promising but require validation in larger, longer-term trials [34].

Attention to dietary behaviors is an equally important and often overlooked dimension of care: the appetite suppression and reduced food preoccupation induced by these agents create a valuable window for dietary education (e.g., mindful eating, recognition of hunger and satiety cues, and reduced ultra-processed food intake) that clinicians should actively leverage to establish sustainable long-term patterns rather than reinforce restriction alone [24,41]. This work is best performed by registered dietitians/nutritionists integrated into the obesity care team, whose interventions have demonstrated benefit [18,23]; the preservation of muscle mass, routine screening for deficiencies, adequate hydration, tailored nutritional therapy, and observation of gastrointestinal symptoms (PRATO) framework is intended as a structure for this dietitian-led care. The potential influence of GLP-1 receptor agonists on alcohol consumption through central modulation of reward circuitry and hedonic hunger is a novel hypothesis with possible additional nutritional benefits (dietary quality, sleep, adherence), although randomized evidence is not yet confirmatory, and evidence remains insufficient for specific recommendations and warrants prospective study [22,40].

Limitations

As a narrative review, this work lacks a formal systematic methodology (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) and quantitative meta-analysis, limiting the strength of its conclusions. Although the search was structured and selection was performed independently by us, selection and citation bias cannot be excluded, and the prioritization of English language publications and the most recent evidence may have omitted relevant studies. A formal risk-of-bias assessment of individual studies was not performed; evidence levels were graded with the Oxford Centre for Evidence-Based Medicine scale, and a substantial proportion of the recommendations rest on expert consensus. Included studies are heterogeneous in design, populations, follow-up duration, and outcomes, precluding direct comparison, and the predominance of short- to medium-term trials restricts extrapolation to long-term treatment. The proposed PRATO framework, Incretin Plate Model, and Metabolic Hourglass Model are expert-derived and have not been validated or prospectively tested. No standardized nutritional monitoring protocols currently exist for patients receiving incretin-based therapies—a significant clinical gap that future prospective studies and randomized trials should address.

Proposed nutritional care model

Building on the evidence synthesized earlier, this review proposes a structured nutritional care framework for patients with obesity undergoing incretin-based pharmacotherapy [1,12,24]. The model, designated PRATO, comprises five integrated clinical pillars that systematize nutritional assessment, monitoring, and intervention across the phases of GLP-1 receptor agonist and dual GLP-1/GIP receptor agonist therapy, addressing its metabolic, functional, and behavioral dimensions. The framework is intended to be implemented by a registered dietitian/nutritionist working within the multidisciplinary obesity care team [1,11].

Importantly, the PRATO framework, the Incretin Plate Model, and the Metabolic Hourglass Model are expert-derived, evidence-informed tools designed to organize clinical practice; they have not been validated or tested prospectively. Table 3 maps each component to its supporting evidence and level of evidence, making explicit which elements derive from randomized data and which derive from expert consensus. Before these models can be considered evidence-based, their content validity should be assessed (e.g., through a Delphi panel of dietitians and obesity specialists) followed by feasibility and usability studies and pragmatic trials evaluating clinical outcomes such as lean mass, muscle function, nutrient status, gastrointestinal tolerability, and adherence.

The PRATO framework

The PRATO model comprises five pillars—preservation of muscle mass, routine screening for nutritional deficiencies, adequate hydration as a therapeutic component, tailored nutritional therapy, and observation and management of gastrointestinal symptoms—each grounded in the evidence reviewed earlier and addressing a distinct, interrelated domain of care (Fig. 1). Preservation of muscle mass emphasizes adequate protein intake (1.2–1.6 g/kg/d during active weight loss, preferably calculated on adjusted body weight), resistance training, and longitudinal body composition monitoring as the primary non-pharmacologic strategies against lean mass loss, particularly in older adults and patients with sarcopenic obesity or low habitual protein intake [28,32,34].

Routine screening for nutritional deficiencies advocates periodic laboratory assessment of micronutrients most susceptible to depletion (vitamin D, thiamine, vitamin B12, folate, iron, calcium, magnesium, zinc, and potassium) following a surveillance protocol analogous to postbariatric monitoring, with assessments at baseline and at 3, 6, and 12 mo [27,33]; this schedule is extrapolated from bariatric practice and has not been tested in GLP-1 receptor agonist users. Adequate hydration treats fluid intake as an active therapeutic component: at least 2 to 3 L/d [39], distributed

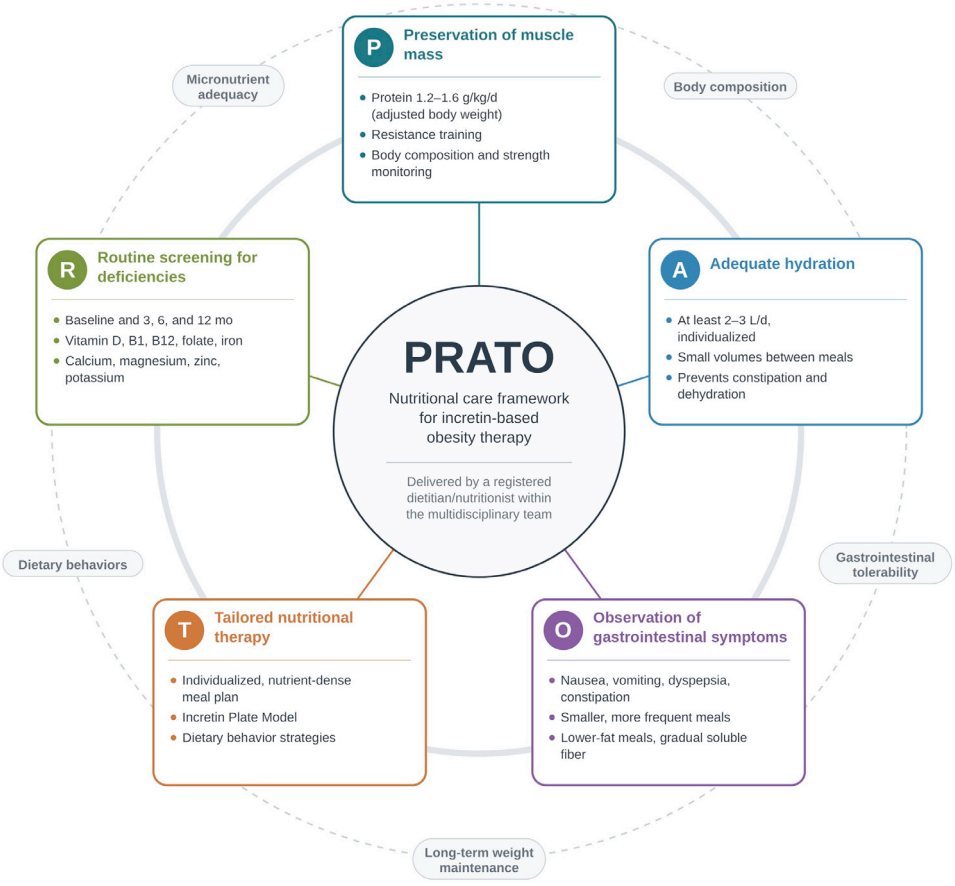

Fig. 1. The PRATO framework, a structured nutritional care model for patients undergoing incretin-based obesity therapy, intended to be delivered by a registered dietitian/nutritionist within the multidisciplinary team. The five integrated pillars—preservation of muscle mass (P), routine screening for nutritional deficiencies (R), adequate hydration (A), tailored nutritional therapy (T), and observation of gastrointestinal symptoms (O)—collectively address body composition, micronutrient adequacy, gastrointestinal tolerability, dietary behaviors, and long-term weight maintenance. Protein targets refer to adjusted body weight. Expert-derived model; not yet validated (Table 3). Figure generated with the assistance of artificial intelligence (Claude; Anthropic, San Francisco, CA, USA).

between meals in small volumes, to mitigate gastrointestinal symptoms, reduce constipation risk, and support tolerability [38].

Tailored nutritional therapy encompasses individualized dietary planning based on cardiometabolically favorable patterns, protein density optimization, micronutrient adequacy, and dietary behavior strategies using 1200 to 1500 kcal/d (women) and 1500 to 1800 kcal/d (men) as practical references, individualized by age, body composition, activity, and clinical response [1,11]. The observation and management of gastrointestinal symptoms pillar addresses dietary adaptations for dose escalation tolerability: smaller, more frequent meals as well as reduced high-fat intake, gradual introduction of soluble fiber, and mindful eating [9,10,38].

Nutritional monitoring protocol

To operationalize the PRATO framework in practice, a quarterly structured follow-up protocol is proposed for the first year of treatment. During active weight loss, assessments should include anthropometrics, body composition analysis, laboratory evaluation, dietary intake review, gastrointestinal symptom monitoring, and adherence evaluation (Fig. 2). During the maintenance phase, monitoring shifts toward sustained metabolic surveillance, with continued emphasis on dietary quality, adherence, dietary behaviors, muscle preservation, and prevention of weight regain (Fig. 3).

The Incretin Plate Model

To translate the PRATO framework into practical meal planning, an adapted plate model inspired by the Bariatric Plate Model [15] prioritizes high nutritional density within reduced dietary volumes, accounting for delayed gastric emptying, early satiety, and reduced energy intake. The proportions refer to the visual distribution of the plate and are intended for dietary counseling; they have not been tested as a standalone intervention. For breakfast and snacks, protein-rich, lower-fat dairy products and eggs comprise approximately 50% of the meal for their protein, calcium, and tolerability; minimally processed whole fruit with a lower glycemic index comprises 30%; and quality carbohydrates from whole grains comprise the remaining 20% (Fig. 4).

For lunch and dinner, lean, iron-rich protein sources (lean meats, poultry, fish, eggs, legumes, tofu) comprise approximately 50% of the plate, favoring lean mass preservation and satiety; fiber-rich, colorful vegetables comprise 30%; and quality carbohydrates from minimally processed sources (whole grains, tubers, legumes) comprise the remaining 20%, complemented by small portions of healthy fats (olive oil, avocados, nuts, seeds). Adequate hydration and satiety-based portion control are emphasized as cross-cutting principles (Fig. 5). Together, the PRATO framework and the Incretin Plate Model translate current evidence into practical, scalable clinical tools,



Fig. 2. Structured nutritional monitoring cycle during the active weight loss phase in patients receiving GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists. The four-quadrant cycle encompasses 1) comprehensive nutritional assessment, 2) dietary optimization based on individual food preferences and the Incretin Plate Model within 1200 to 1800 kcal/d, 3) management of GI symptoms, and 4) maximization of body fat loss through lean mass preservation, resistance training, and adequate protein intake (1.2–1.6 g/kg/d, corresponding to approximately 25–30% of total energy intake). GI, gastrointestinal; GIP, glucose-dependent insulinotropic polypeptide; GLP-1, glucagon-like peptide 1. (Based on data from [1].) Figure generated with the assistance of artificial intelligence (Claude; Anthropic, San Francisco, CA, USA).

emphasizing dietary quality, protein adequacy, functional preservation, and the long-term metabolic sustainability of weight loss.

Conceptual proposal: The Metabolic Hourglass Model

As a conceptual and educational representation of nutritional care during GLP-1 receptor agonist and dual GLP-1/GIP receptor agonist therapy, this review proposes the Metabolic Hourglass Model (Fig. 6). The hourglass metaphor illustrates a single clinical message: because incretin-based therapy narrows the volume of food a patient can eat, every meal must carry more nutrients. Each level of the hourglass is paired with the clinical actions and monitoring parameters it implies so that the figure can be used directly during consultations.

The upper chamber (intake) represents nutrient-dense eating within reduced volumes: protein first followed by fiber-rich, minimally processed carbohydrates; mostly unsaturated fats; and micronutrients at particular risk of inadequacy (vitamin B12, iron, magnesium, zinc, selenium, calcium, and vitamin D). Food groups are drawn as layers illustrated with representative foods. Protein forms the base layer so that it is the first to pass the constriction—a visual cue for the "protein first" principle; blue circles represent at-risk micronutrients distributed across all food groups. The

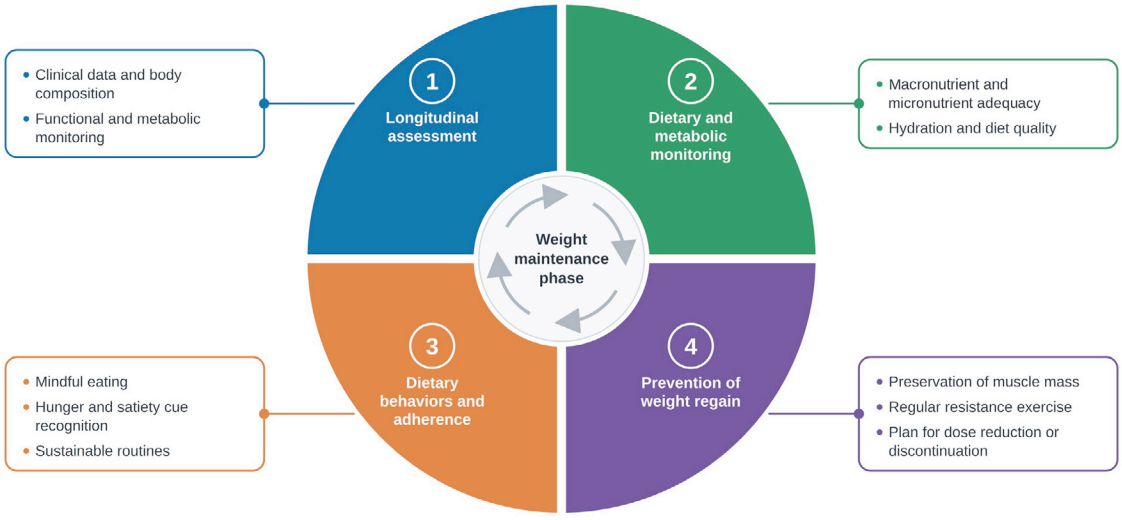

Fig. 3. Integrated nutritional monitoring cycle during the weight maintenance phase in patients receiving incretin-based therapy. The model integrates four key domains: 1) longitudinal nutritional assessment; 2) dietary and metabolic monitoring, including macronutrient and micronutrient adequacy, hydration, and diet quality; 3) dietary behaviors and adherence strategies; and 4) prevention of weight regain through lean mass preservation, regular resistance exercise, and planning for dose reduction or discontinuation. (Adapted from Mozaffarian et al. [1].) Figure generated with the assistance of artificial intelligence (Claude; Anthropic, San Francisco, CA, USA).

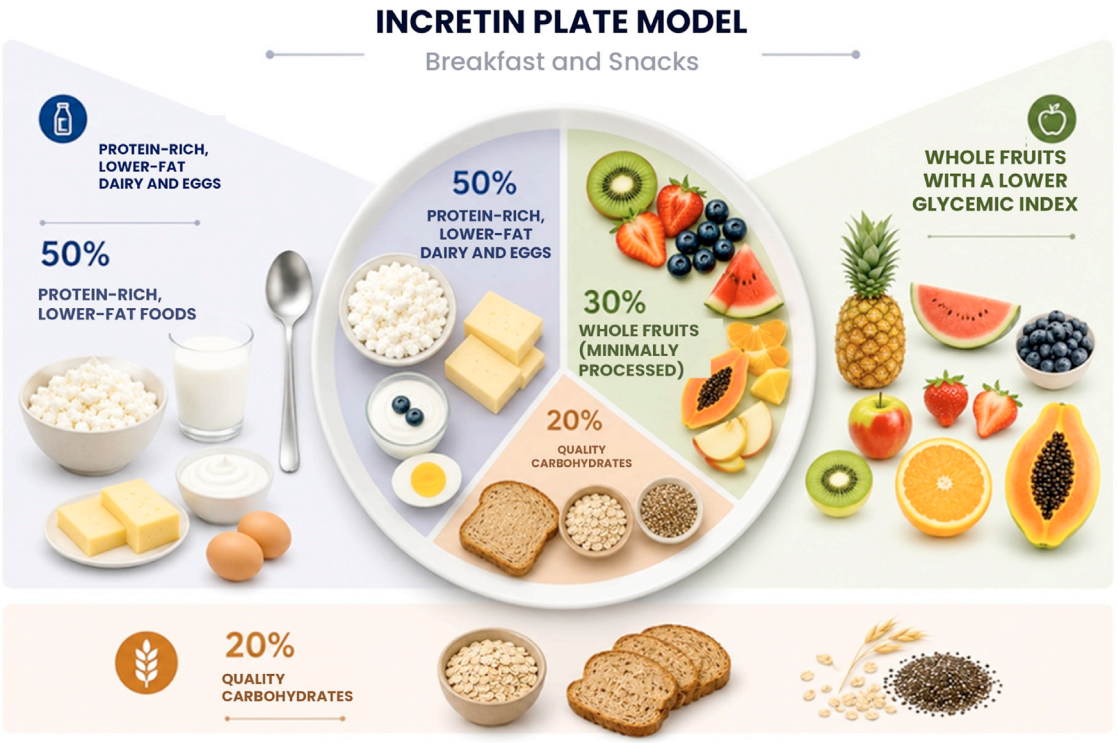

Fig. 4. Incretin Plate Model for breakfast and snacks in patients receiving GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists. The model prioritizes protein-rich, lower-fat dairy products and eggs (50%), minimally processed whole fruits with a lower glycemic index (30%), and quality carbohydrates from whole grains (20%), emphasizing protein density, micronutrient adequacy, and gastrointestinal tolerability. GIP, gastric inhibitory polypeptide; GLP-1, glucagon-like peptide 1. (Based on the concept of the Bariatric Plate Model (Cambi and Baretta, 2018) [15].) Figure generated with the assistance of artificial intelligence (Claude; Anthropic, San Francisco, CA, USA).

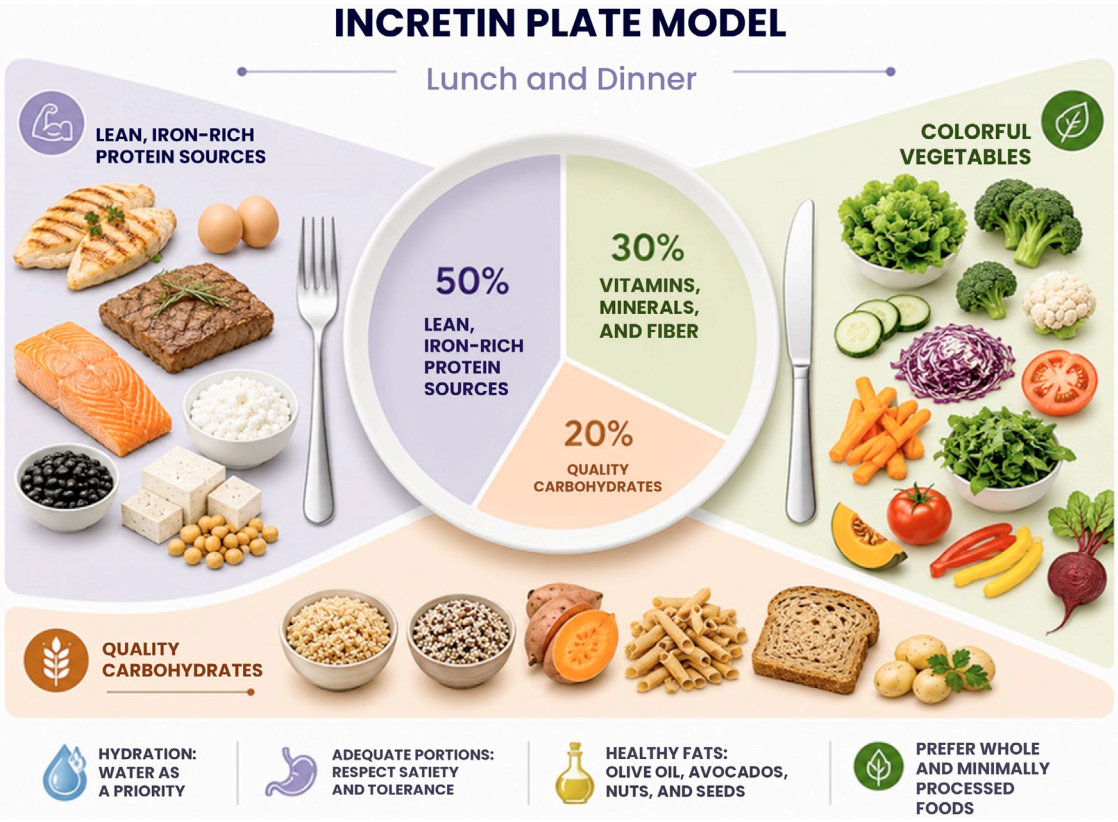

Fig. 5. Incretin Plate Model for lunch and dinner in patients undergoing incretin-based obesity therapy. The model distributes meal volume as follows: lean, iron-rich protein sources (50%); fiber-rich, colorful vegetables providing vitamins, minerals, and antioxidant compounds (30%); and quality carbohydrates from minimally processed sources (20%), with cross-cutting principles of hydration, satiety-based portions, small amounts of healthy fats, and preference for minimally processed foods. (Based on the concept of the Bariatric Plate Model (Cambi and Baretta, 2018) [15].) Figure generated with the assistance of artificial intelligence (Claude; Anthropic, San Francisco, CA, USA).

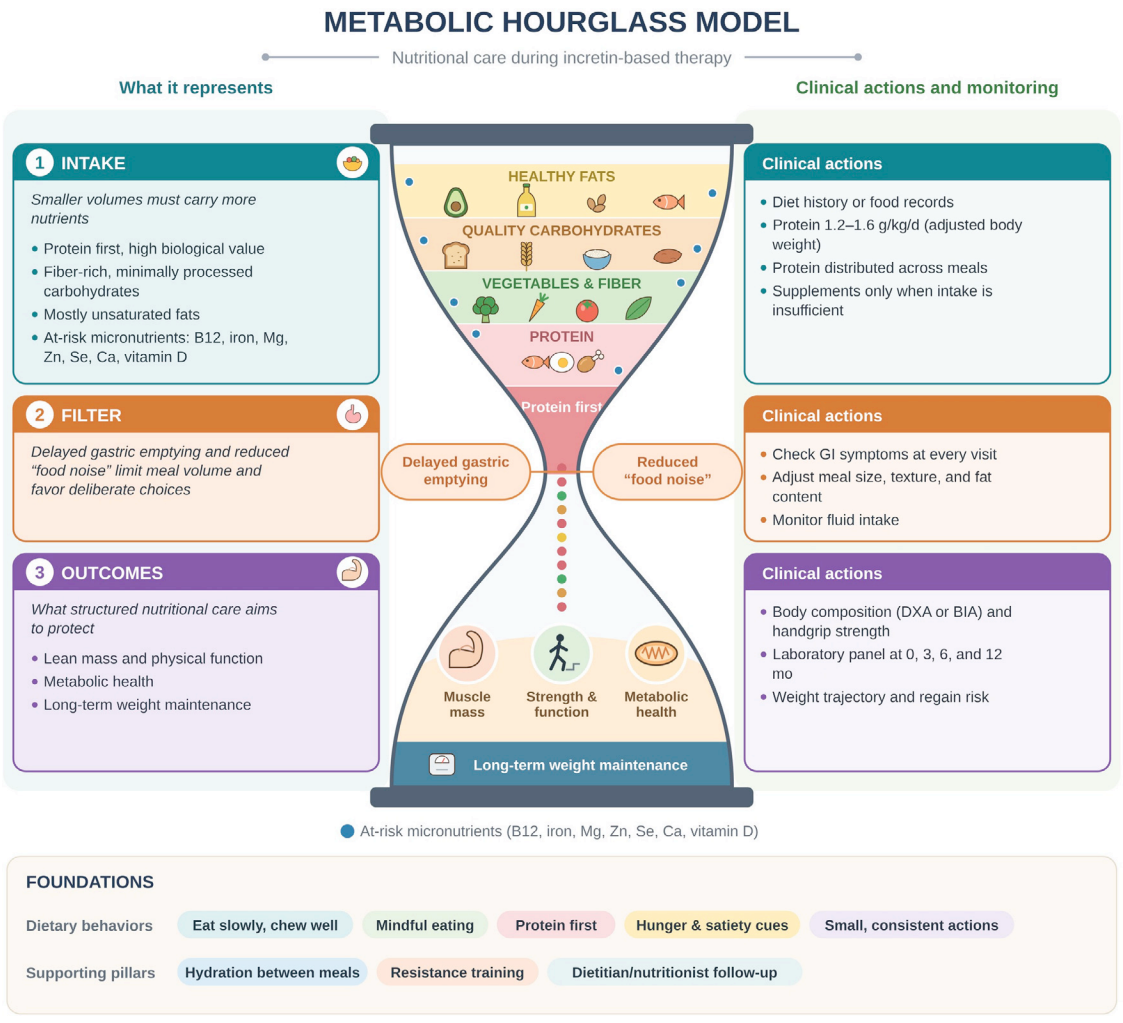

Fig. 6. The Metabolic Hourglass, a conceptual counseling model for patients undergoing incretin-based obesity therapy, with what each level represents shown on the left and the corresponding clinical actions and monitoring parameters shown on the right. The upper chamber (intake) depicts nutrient-dense eating within reduced food volumes as food group layers illustrated with representative foods (healthy fats, quality carbohydrates, vegetables and fiber, and protein), with protein at the base to signal the "protein first" principle; blue circles represent at-risk micronutrients (vitamin B12, iron, Mg, Zn, Se, Ca, and vitamin D). The central constriction (filter) represents delayed gastric emptying and reduced "food noise." The lower chamber (outcomes) depicts muscle mass, strength and physical function, and metabolic health, resting on long-term weight maintenance. The base lists the foundations, comprising dietary behaviors and supporting pillars (hydration between meals, resistance training, and dietitian/nutritionist follow-up). Expert-derived model; not yet validated. BIA, bioelectrical impedance analysis; Ca, calcium; DXA, dual-energy x-ray absorptiometry; GI, gastrointestinal; Mg, magnesium; Se, selenium; Zn, zinc. Figure generated with the assistance of artificial intelligence (Claude; Anthropic, San Francisco, CA, USA).

corresponding clinical actions are dietary assessment, a protein target of 1.2 to 1.6 g/kg/d distributed across meals, and supplementation only when intake is insufficient.

The central narrowing (filter) represents delayed gastric emptying and reduced "food noise" (diminished intrusive preoccupation with food), which limit meal volume and favor more deliberate food choices. Clinically, this level corresponds to checking gastrointestinal symptoms at every visit and adjusting meal size, texture, fat content, and fluid intake.

The lower chamber (outcomes) represents what structured nutritional care aims to protect: lean mass and physical function, metabolic health, and long-term weight maintenance. These outcomes are monitored through body composition (dual-energy x-ray absorptiometry or bioelectrical impedance analysis); handgrip strength; laboratory assessment at 0, 3, 6, and 12 mo; and weight trajectory.

The base of the model lists its foundations: dietary behaviors (eating slowly; mindful eating; protein first; recognizing hunger and satiety cues; and small, consistent actions) and supporting pillars (hydration and electrolytes between meals, regular resistance training, and follow-up by a registered dietitian/nutritionist). The Metabolic Hourglass is intended as a patient education and counseling aid that complements rather than replaces the PRATO framework; like the other models, its usefulness and comprehensibility should be evaluated with patients and clinicians.

Conclusions

Nutritional care in patients receiving GLP-1 receptor agonists and dual GLP-1/GIP receptor agonists is a fundamental component of contemporary obesity management. Beyond weight reduction, these therapies produce clinically relevant changes in dietary intake, dietary behavior, body composition, and gastrointestinal tolerability, requiring a structured, continuous, and individualized nutritional approach throughout treatment, ideally delivered by a registered dietitian/nutritionist as part of a multidisciplinary team.

Nutritional follow-up should prioritize lean mass and metabolic function preservation, prevention of macronutrient and micronutrient inadequacies, gastrointestinal symptom management, and sustained adherence. Adequate protein intake, body composition monitoring, hydration optimization, individualized dietary planning, and dietary behavior interventions appear central to optimizing clinical and metabolic outcomes. Integrating pharmacotherapy with structured nutritional education, continuous monitoring, and healthy lifestyle habits may support long-term weight loss sustainability and reduce nutritional complications; conceptual tools such as the PRATO framework and the Metabolic Hourglass Model may facilitate patient understanding, engagement, and adherence, although they require validation before routine adoption.

Significant gaps remain in the standardization of nutritional monitoring protocols for this population. Future prospective studies and randomized controlled trials are needed to validate the proposed models and to define structured protocols for nutritional surveillance, body composition assessment, supplementation strategies, and targeted interventions for lean mass preservation during accelerated weight loss, supporting a more precise, safe, and sustainable approach to obesity treatment.

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Declaration of generative artificial intelligence and artificial intelligence–assisted technologies in the writing process

During the preparation of this manuscript, the authors used Claude (Anthropic, San Francisco, CA, USA) to assist in the development of illustrative figures (conceptual diagrams and schematic illustrations in Figs. 1–6) and to improve the English language writing. Following the use of this tool, the authors carefully reviewed and edited all content and assume full responsibility for the content of this manuscript.

Data availability statement

No new data sets were generated or analyzed during the preparation of this narrative review. The evidence supporting this review is derived from the published studies cited in the reference list.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Maria Paula Carlin Cambi: Writing – review & editing, Writing – original draft, Project administration, Methodology, Investigation, Conceptualization, Visualization. **Daniela Vicinansa Monaco-Ferreira:** Writing – review & editing, Writing – original draft, Validation, Supervision, Conceptualization, Investigation, Visualization.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.nut.2026.113452.

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