Nutritional, functional, and psychological considerations for incretin-based therapies in adults—an EASO, EFAD, and ECPO Consensus Statement

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Incretin-based therapies, including GLP-1 receptor agonists and dual GLP-1–GIP receptor agonists, have transformed obesity management, producing substantial weight loss and cardiometabolic benefits, with potential improvements in obesity-related complications, physical function, quality of life, and psychological wellbeing for many individuals. However, reduced appetite, rapid weight loss, gastrointestinal adverse effects, and changes in eating behaviour might create nutritional, functional, or psychological risks in some individuals. This EASO–EFAD–ECPO Consensus Statement outlines pragmatic nutritional, functional, and psychological considerations during incretin-based therapy treatment. We synthesise evidence on medical nutrition therapy, including protein targets during weight loss, dietary quality, and mitigation of gastrointestinal adverse events. We discuss pragmatic approaches that could help support preservation of fat-free mass and physical function during weight loss, including adequate protein intake and progressive resistance exercise. We emphasise the psychological and identity-related challenges during incretin-based therapy, including shifts in food reward, coping, and social connection, and recommend psychological screening, with integrated support where needed. We propose pragmatic monitoring during weight loss, including diet quality, micronutrient risk, and functional measures, with body-composition assessment where indicated. We suggest approaches to address disparities in access and adherence to ensure equity. We discuss the importance of shared decision making with the patient in decisions to start, up-titrate, delay, pause, down-titrate, or discontinue incretin-based therapies, considering the physical and psychological risks of treating versus not treating. Finally, we identify future research priorities, including longitudinal research on micronutrient status, macronutrient requirements, changes in dietary patterns and food choices, musculoskeletal outcomes, unique dosing schedules, and post-cessation maintenance to optimise safety, efficacy, and the patient experience.

Lancet Diabetes Endocrinol 2026

Published Online
July 8, 2026
https://doi.org/10.1016/S2213-8587(26)00122-1

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Introduction

Obesity is a chronic, relapsing disease and a key driver of type 2 diabetes, cardiovascular disease, and many other conditions.[1] Incretin-based therapies, particularly GLP-1 receptor agonists and dual GLP-1 and GIP receptor agonists, represent a paradigm shift in obesity management, with their weight-lowering and glucose-lowering effects now approaching metabolic–bariatric surgery.[2–4]

Recently, four North American societies published an advisory statement regarding nutritional priorities to support GLP-1 receptor agonists for obesity management,[5] and the Academy of Nutrition and Dietetics underscored the crucial role of registered dietitians (hereafter termed dietitians) in supporting medication adherence, managing side-effects, and ensuring nutritional adequacy during incretin-based therapy.[6] In 2025, WHO published its first guideline on GLP-1 therapies for obesity, issuing two conditional recommendations that (1) GLP-1 therapies can be used as a long-term treatment option for adults living with obesity; and (2) when these agents are prescribed, they should be integrated with intensive behavioural therapy as part of a comprehensive multimodal clinical algorithm.[7] This Consensus Statement from the European Association for the Study of Obesity (EASO), European Federation of the Associations of Dietitians (EFAD), and European Coalition for People Living with Obesity (ECPO) suggests pragmatic, evidence-informed nutritional, functional, and psychological considerations for individuals receiving incretin-based therapies, recognising that, where direct evidence is limited, some considerations are informed by expert consensus. Additionally, it emphasises the patient perspective, particularly expectation management and long-term behavioural support.

Methods

This Consensus Statement was developed (April–Dec, 2025) via an iterative, multidisciplinary process, including a European Congress of Obesity 2025 workshop (May 12, 2025) and regular online meetings to appraise evidence and refine statements. Statements are based on available evidence and expert consensus. This Consensus Statement does not apply to individuals who are planning pregnancy, currently pregnant, or breastfeeding; or to those intending to lose weight specifically before conception (further details are provided in the appendix pp 3–4).

Throughout this Consensus Statement, we use the term considerations to denote evidence-informed issues that clinicians and services might consider during incretin-based therapy, rather than mandatory recommendations for all patients. Where direct

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incretin-based therapy-specific evidence is scarce, statements are based on expert consensus, biological plausibility, extrapolation from related evidence in obesity, malnutrition, exercise, metabolic–bariatric surgery, mental health, and lived experience. These considerations are intended to support pragmatic, individualised, and proportionate care, not to imply universal specialist referral, routine body-composition testing, routine formal psychological intervention, or routine extensive biochemical testing for all people receiving incretin-based therapies. In most patients, basic monitoring and standard behavioural support might be sufficient, with escalation to dietetic, psychological, physiotherapy, body-composition, or biochemical assessment where baseline vulnerability, rapid or excessive weight loss, poor intake, persistent gastrointestinal intolerance, functional decline, psychosocial complexity, or patient preference indicates additional needs. We did not undertake a health economic evaluation; therefore, local implementation should consider service capacity, affordability, and health-care equity.

Overview of incretin-based therapies

Incretin-based therapies include pharmacological agents targeting incretin pathways, particularly GLP-1 receptors. They enhance glucose-dependent insulin secretion, suppress glucagon and thus endogenous hepatic glucose output, slow gastric emptying, and act centrally in the hypothalamus to increase satiety and reduce energy intake.[8,9] Mechanistic studies report mean energy intake reductions of approximately 300 kcal per day with incretin-based therapies (~348 kcal per day with tirzepatide and ~284 kcal per day with semaglutide), with substantial interindividual variability.[10–13]

These mechanisms contribute to significant glycaemic-lowering and weight-lowering efficacy. Mathematical modelling shows that incretin-based therapies attenuate the appetite-feedback control loop, achieving weight loss more effectively than energy restriction alone by shifting the bodyweight equilibrium and delaying the onset of weight-loss plateaus.[14] Amylin, co-secreted with insulin, also regulates energy intake and glucose homoeostasis; although not approved for obesity, amylin administration for type 2 diabetes is associated with weight loss and warrants further study.[15]

Liraglutide, semaglutide, and tirzepatide are the incretin-based therapies currently licensed in Europe, with independent indications for both obesity and type 2 diabetes. These medications are recommended in combination with dietary intervention and physical activity for obesity and type 2 diabetes and are substantially effective for weight loss.[16] Clinically meaningful weight regain (≥5%), is common after cessation.[17] Additionally, trials infrequently report on fat versus lean mass loss. In clinical trials, semaglutide (2·4 mg) and tirzepatide (15 mg) achieved maximum average weight loss of 14·8% and 20·8%, respectively, versus placebo.[2,3,18,19] Newer generations of multiagonists and co-formulations might deliver greater weight loss.

Beyond weight reduction, incretin-based therapies confer broad cardiometabolic benefits, including improved cardiovascular and kidney outcomes, and have demonstrated efficacy across key obesity-related complications such as heart failure with preserved ejection fraction, obstructive sleep apnoea, and metabolic dysfunction-associated steatohepatitis.[20–26]

Incretin-based therapies consistently reduce appetite and improve certain eating behaviours.[27,28] Preclinical studies suggest tirzepatide reduces preference for high-fat palatable foods, and early clinical trial data show significant reductions in ad-libitum energy intake and appetite or craving measures versus placebo.[13,29] Behaviourally, tirzepatide reduces eating disinhibition and reactivity to the food environment without increasing cognitive restraint.[30] Patients report becoming more attuned to physiological hunger and satiety cues and less driven by emotional, situational, or external sensory triggers after initiating incretin-based therapies.[31] Reductions in overeating, meal frequency, and food cravings during the weight-loss phase have been documented.[30] Some individuals report reduced food noise (ie, persistent unwanted or dysphoric food-related thoughts): a phenomenon for which the neurobehavioural basis remains poorly characterised.[32]

Similar to metabolic–bariatric surgery, attenuated food reward pathways might trigger shifts to maladaptive coping strategies if psychological distress is unaddressed or healthier coping mechanisms are not developed.[33,34] In qualitative focus-group data, some patients also report distress about losing their sense of self or former coping mechanisms despite weight-loss success.[35] Overall, these issues suggest that prescribing incretin-based therapies might benefit from a multidisciplinary approach that includes dietitian-delivered medical nutrition therapy, assessment of wellbeing and therapeutic needs, and, where indicated, structured psychological support to help patients adapt to altered appetite, reward responses, and the impact of a changing relationship with food.

Medical nutrition therapy: the role of registered dietitians

Dietitian-delivered medical nutrition therapy during incretin-based therapy

Medical nutrition therapy delivered by a registered dietitian is central to obesity care and complements incretin-based therapies.[36] It is an evidence-based, iterative process in which dietitians assess, diagnose, and manage obesity through personalised nutrition and behaviour change support, delivered using a person-centred, culturally sensitive approach that recognises weight bias, prioritises health beyond weight, and is grounded in shared decision making to agree and support treatment goals.[37–39]

Risk-stratified and stepped-care implementation

Although incretin-based therapies are effective, real-world outcomes are limited by tolerability, access, and attrition. There is currently no direct randomised controlled trial evidence showing that universal dietitian-delivered medical nutrition therapy improves outcomes in all patients. However, absence of trial evidence is not evidence of no risk in some subgroups at higher nutritional risk. In practice, core nutritional advice and management of common gastrointestinal adverse effects might be delivered by appropriately trained members of the multidisciplinary team, with escalation to dietitian-delivered medical nutrition therapy for patients with greater nutritional risk, complexity, or emerging concerns. In patients at low risk with good treatment tolerance, adequate intake, no evident nutritional risk factors, and an expected weight-loss trajectory, structured dietary advice and routine multidisciplinary team follow-up might be sufficient, with dietitian input reserved for emerging nutritional concerns or greater clinical complexity. Within this model, dietitians might also provide leadership, supervision, and protocol development to support safe delivery of nutritional care by the wider multidisciplinary team.

Treatment aims and evidence base

Within this risk-stratified framework, dietitian-delivered medical nutrition therapy might support tolerability, adherence, nutritional adequacy, and longer-term durability by proactively addressing nutrition-related risks during appetite suppression.[12,40,41] In the context of incretin-based therapies, medical nutrition therapy aims to improve gastrointestinal tolerance and adherence;[42] maintain macronutrient and micronutrient adequacy;[5,6,12,43] support preservation of fat-free mass while optimising functional capacity;[44] reinforce healthy dietary patterns and reduce disordered-eating risk;[36,37] and support sustained behaviour change and improvements to quality of life.[39]

Dietitian-led medical nutrition therapy improves BMI, waist circumference, blood pressure, and quality of life in obesity management compared with no intervention or usual care,[45] and adjunctive strategies (eg, dietary self-monitoring alongside liraglutide [3·0 mg]) have been associated with greater weight loss than no self-monitoring.[46] During incretin-based therapies, reduced appetite might compromise dietary quality and nutrient adequacy; structured review of intake and targeted monitoring might enable earlier identification of deficiencies and support long-term safety and adherence.[5,44] Although data are limited, dietitian involvement might improve detection of nutritional risk and potential deficiencies, yet dietitians are included in less than half of incretin-based therapy trials, underscoring the need for prospective studies evaluating medical nutrition therapy effects on safety, tolerability, and attrition.[44,47] Practical macronutrient and micronutrient targets are summarised in table 1, with key dietary approaches discussed in panel 1 and a full description on the role of dietitians and medical nutrition therapy implementation provided in the appendix (pp 5–11).

Medical nutrition therapy management of incretin-based therapy-related side-effects

The most common side-effects of incretin-based therapies are gastrointestinal, and include nausea, reflux, vomiting, constipation, and diarrhoea.[42] These are often dose-dependent, worsen following dose escalation, improve with maintenance,[60] and largely result from delayed gastric emptying and enhanced satiety signalling.[5] Although side-effects are usually mild to moderate, they lead to treatment discontinuation in approximately 4–7% of participants receiving semaglutide or tirzepatide in pivotal obesity trials.[2,3,12] The appendix (pp 53–55) provides comparison data of side-effect profiles related to incretin-based therapies.

Dose escalation

A gradual dose-escalation schedule should be used to minimise gastrointestinal side-effects. If significant side-effects occur, delaying titration, dose reduction, or temporarily pausing treatment should be considered,[42,70] noting that extending titration intervals can aid tolerability. However, even with these strategies some patients might only tolerate lower doses.

In a real-world observational study integrating intensive behavioural therapy with personalised GLP-1 receptor agonist dosing, a treat-to-target approach (withholding dose escalation when weekly weight loss was >0·5% and satiety was adequate) achieved 16·7% weight loss at 64 weeks on a mean semaglutide dose of 1·08 mg per week, indicating that individualised dosing can deliver substantial weight loss.[71]

Fluid management

Incretin-based therapies slow intestinal motility and increase constipation risk.[72] Moreover, experimental data show that the GLP-1 receptor agonist dulaglutide reduces fluid intake and urine output.[73] Vomiting or diarrhoea increase dehydration risk, compounding incretin-based therapy-related appetite and thirst suppression.

Appropriate fluid balance is essential, typically aiming for 2·0–2·5 L per day,[65,66] with consideration towards adjusting upwards in warm climates or with vigorous physical activity, and treatment should be individualised where fluid restriction is indicated (eg, heart failure and chronic kidney disease).[67,74,75] Patients should be counselled to prioritise water to maintain hydration, using other calorie-free fluids as adjuncts and noting that high caffeine intake and alcohol consumption can induce acute diuresis, which worsens dehydration.[76] Although there is little direct trial evidence specifically linking incretin-based therapy treatment to

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See **Online** for appendix

	Recommendation	Foods and nutrients to emphasise	Foods and components to limit or avoid
Energy intake[53–55]	Energy intake should be individualised, noting that intake often decreases spontaneously during incretin-based therapy treatment; where estimation of energy requirements is clinically useful, indirect calorimetry is the preferred method, but its routine use might be limited by cost, time, and availability; in practice, predictive approaches (eg, Mifflin–St Jeor equation using actual bodyweight) provide a pragmatic alternative; NIDDK Body Weight Planner might also be considered, as it incorporates energy intake, physical activity, resting energy expenditure, body-composition change over time, and metabolic adaptation; if intake falls below 800 kcal per day for a sustained period or nutritional requirements remain unmet despite appropriate adjunctive nutritional support, the multidisciplinary team should consider incretin-based therapy down-titration, temporary treatment pause, or, in severe circumstances, discontinuation using shared decision making with the patient due to risk of malnutrition and disordered eating	NA	NA
Protein[44,53,56–59]	Individualise protein recommendation via medical nutrition therapy assessment to clinical context, current incretin-based therapies, needs, and comorbidities; general recommendation during weight loss phase: 1·0–1·5 g/kg adjusted bodyweight*†; recommendation during weight maintenance phase: potential target could be 0·8–1·0 g/kg adjusted bodyweight§; minimum 60 g per day; EFSA proposes ~75 g per day of high-quality protein can maintain nitrogen balance during energy restriction¶; high-quality protein intake should be distributed across meals (25–30 g protein per main meal) to optimise muscle protein synthesis; include protein-rich snacks between meals to meet protein requirements	High-quality sources: prioritise a variety of lean proteins; seafood: tuna, white fish (eg, haddock, pollock, and cod), prawns, and other seafood; lean red meat in small quantities; poultry: skinless chicken and turkey; eggs and dairy: eggs, natural yoghurt, low-fat milk, quark, and cottage cheese; plant-based: legumes (eg, beans, lentils, and chickpeas), soy, soy products (eg, tofu and tempeh), Quorn, and moderate amounts of unsalted nuts and seeds (~30 g per day); protein supplementation (eg, whey, casein, or soy protein powder, ready-to-drink shakes, and protein bars) should be considered if dietary intake is insufficient, as assessed by the registered dietitian	Processed and high-fat meats: limit high-fat meats; limit processed meats (eg, sausages, bacon, deli meats, and hot dogs)
Carbohydrates	Emphasise complex carbohydrates (eg, whole grains, vegetables, and legumes); limit refined carbohydrates (eg, sugars and refined grains)	Whole, unprocessed sources; focus on complex carbohydrates rich in fibre; vegetables: abundant intake of a wide variety, including leafy greens (eg, spinach and kale), colourful vegetables (eg, peppers and tomatoes), okra, cruciferous vegetables (eg, broccoli and bok choy), and root vegetables or tubers (eg, carrots, beetroot, parsnips, yams, cassava, taro, potatoes, and plantain); fruits: include whole fruits including berries, apples, and citrus; whole grains: oats, quinoa, brown rice, barley, buckwheat, maize, millet, stone-ground cornmeal, whole corn flour, teff, sorghum, and whole-wheat bread, pasta, chapati, and bulgur; legumes: beans, lentils, and chickpeas are excellent sources of both carbohydrates and protein	Refined and processed carbohydrates; added sugars: limit foods and drinks with high added-sugar content (eg, pastries, candies, and desserts); sugar-sweetened beverages: avoid fizzy drinks and sodas, sweetened juices, and sugary coffees and teas (eg, syrups or added sugars); refined grains: limit sugary cereals, white bread, white rice, and products made exclusively with white flour (eg, chapati)
Dietary fibre[12,60–64]	≥25 g per day‡; aim for a balanced intake of soluble (found in oats, legumes, fruits, and psyllium) and insoluble (found in whole grains, wheat bran, and vegetables) fibre; it can be challenging to meet fibre requirements when intake ≤1500 kcal; prioritise food-based fibre sources first; if constipation persists despite food-based strategies and adequate fluid intake, consider an evidence-based fibre supplement (eg, psyllium), titrated gradually according to tolerance; if increasing fibre intake, ensure regular fluid intake; fibre has several health effects and can also enhance the sensation of satiety	Fibre-rich vegetables (eg, broccoli, kale, Brussels sprouts, cauliflower, and cabbage), root vegetables and tubers (eg, carrot, beetroot, parsnip, yam, and cassava), legumes (eg, beans, lentils, chickpeas, and peas), whole fruits (eg, apples, pears, citrus fruits, figs, prunes, and kiwi), berries (eg, raspberries and strawberries), whole grains (eg, oats, quinoa, brown rice, barley, maize, millet, teff, sorghum; whole-wheat bread, pasta, chapati, and bulgur; and buckwheat, stone-ground cornmeal, whole corn flour), and wheat and oat bran; if constipation persists despite food-based strategies, consider an evidence-based fibre supplement (eg, psyllium; aiming for ~10 g per day supplementation, with gradual titration, starting with 3–5 g per day with adequate fluids); increase gradually to reduce gastrointestinal discomfort; adequate intake of fluid is necessary to prevent dehydration-related dizziness, weakness, and constipation	Limit white bread, pasta, and cereals made from refined flour, and white rice
Fats	Focus on quality, with emphasis on monounsaturated fats from olive oil and rapeseed oil, and polyunsaturated fat especially from oily fish (omega-3); limit saturated fat, and fried and highly processed fats	Unsaturated fats; plant-based: extra virgin olive oil (as a primary fat source), rapeseed oil, avocados, nuts, and seeds; seafood: oily fish is especially rich in omega-3s (eg, salmon, herring, and sardines)	Total fat intake, especially saturated fats; limit fried foods, fatty meats, butter, ghee, lard, full-fat dairy, crisps, chips, baked goods, pastries, etc

(Table 1 continues on next page)

	Recommendation	Foods and nutrients to emphasise	Foods and components to limit or avoid
(Continued from previous page)			
Fluids[65–67]	2·0–2·5 L per day from non-caloric sources limiting caffeinated drinks (2·5 L per day for males; 2·0 L per day for females)	Water: primary source of hydration; other: herbal teas, green tea, black tea, and coffee in moderation	Sugary drinks: as noted above, these should be avoided; limit fruit juice and smoothies; carbonated drinks (eg, sparkling water, soda, and fizzy beverages) can increase bloating, burping, and nausea due to GLP-1 receptor agonist-associated delayed gastric emptying; although this isn't harmful, it can cause discomfort
Micronutrients[12,53,68,69]	Encourage nutrient-dense foods; consider complete multivitamin supplementation if intake is <1200 kcal per day; there are higher baseline deficiency rates in people with obesity (ie, the double burden of obesity); complete multivitamin–mineral supplementation can reduce deficiency prevalence in this group, especially in those with a low energy intake (<1500 kcal per day) and with poor dietary quality	Variety and balanced, nutrient-rich meals are key; it is not necessary to include all nutrients in every meal; it is the overall daily or weekly intake that matters; different countries provide tools to support healthy eating; for example, the Eatwell guide from the UK gives advice on how to achieve a healthy, balanced diet and an adequate intake of vitamins and minerals; the Eatwell guide encourages at least five portions of a variety of fruit and vegetables every day; it also emphasises that one-third of food we eat should be higher fibre or wholegrain, and potatoes should be eaten with the skins on; it also recommends pulses, lean fish, meat and mince, less red and processed meat (eg, ham, sausages, and bacon), low-fat and low-sugar dairy foods, and the reduction of sugary, salty, and high-fat foods; the plate model is common in Nordic countries and is designed to assist creating balanced meals emphasising nutritional quality; half the plate: colourful plant foods, ensuring a wide array of vitamins, minerals, and beneficial phytochemicals (eg, polyphenols and antioxidants); one-quarter of the plate: lean protein or plant-based alternative; one-quarter of the plate: whole grains or starchy carbohydrate	Nutrient-poor foods; energy-dense-foods high in fat, salt, and/or sugar; often high in energy but low in essential nutrients; sugary drinks, candy and sweets, crisps, cheese puffs, Bombay mix, ice cream, desserts, pastries and cakes, processed meats (eg, sausages), and fried foods (eg, fries); important to note that when the energy intake is reduced, the need for vitamins, minerals, and other protective nutrients remains the same or even might increase

Targets are starting points to support nutritional adequacy during appetite suppression and should be individualised according to clinical context (eg, age, comorbidities, previous metabolic–bariatric surgery, and dietary pattern). This table can be used as a practical guide to build culturally acceptable, affordable meals that prioritise minimally processed foods, adequate protein, whole-grain carbohydrates and fibre, and unsaturated fats. The table is illustrative (not exhaustive) and should be tailored to preferences, allergies and intolerances, ethical and religious diets, and clinical needs. The limit and avoid column refers to routine intake; small amounts might fit within an overall high-quality pattern. For seafood choices, follow local sustainability and safety advice. NIDDK=National Institute of Diabetes and Digestive and Kidney Diseases. EFSA=European Food Safety Authority. *Chronic kidney disease might require protein 0·8–1·0 g/kg adjusted bodyweight. †Avoid prolonged very low (<0·5 g/kg adjusted bodyweight) or very high (>2 g/kg adjusted bodyweight) protein intakes. ‡Fibre should be titrated to promote comfortable daily stool; calculate individually as part of medical nutrition therapy; fibre intake recommendations differ by country. §Adjusted bodyweight = reference bodyweight + (0·33 × [actual bodyweight – reference bodyweight]). Reference bodyweight is defined as bodyweight at a BMI of 25 kg/m². ¶EFSA total diet replacement guidance provides contextual evidence for protein adequacy during substantial energy restriction. However, incretin-based therapy-specific protein targets should be individualised and should not override disease-specific protein guidance, particularly in chronic kidney disease or other conditions requiring tailored protein intake.

Table 1: **Nutritional and food-based guidance for patients on incretin-based therapies[5,12,14,48–52]**

Panel 1: **Dietary approaches for patients on incretin-based therapies**

Core principles and key foods

- Focus on foods that are rich in fibre such as fruits, vegetables, whole grains, legumes, moderate amounts of unsalted nuts and seeds (approximately 30 g per day); emphasis on taking healthy foods (eg, fruit and dairy) for in-between-meal snacks
- Use liquid unsaturated fats for cooking
- Prefer local, seasonal foods whenever possible
- Prioritise high-quality protein sources such as fish, seafood, poultry, eggs, and plant-based protein sources; choose lean meat in moderation and limit processed meats
- Daily consumption of lean dairy
- Limit snacks and sweet snacks while prioritising minimally processed whole foods
- Prioritise water as the main beverage source alongside other non-caloric drinks and limit drinks containing calories
- Pay attention to portion sizes and meal regularity
- Practice mindful eating by paying attention to the food you are eating and noticing hunger and fullness cues
- Enjoy meals as part of the health-related behaviours including physical activity, restorative sleep, and social connections

Below are examples of dietary approaches with evidence of broader health benefit, which can be adapted to individual preferences, cultural context, comorbidities, and nutritional risk.

Mediterranean diet

- Focus: high intake of fruits, vegetables, whole grains, legumes, nuts, and seeds; extra virgin olive oil is the principal source of fat
- Protein: fish and seafood are consumed regularly; poultry, eggs, and low-fat dairy in moderation
- Limit: red and processed meats and sweets, and high-fat dairy should be consumed infrequently

DASH diet

- Focus: designed to lower blood pressure; emphasises fruits, vegetables, and low-fat dairy products
- Include: whole grains, poultry, fish, and nuts
- Limit: saturated fat, cholesterol, red meat, sweets, and sugary beverages; has a specific focus on reducing sodium intake

Healthy Nordic diet

- Focus: based on traditional foods from Nordic countries; emphasises sustainability and local sourcing
- Key foods: green leafy vegetables, root vegetables (eg, potatoes), fruits (especially berries), whole grains (eg, rye, oats, and barley), fish, legumes, and low-fat dairy. Rapeseed oil (eg, canola) is a primary fat source

Traditional Asian dietary patterns

- General pattern: high in vegetables, soy products, rice, and noodles; protein is often from fish and legumes rather than red meat; emphasises soups and fermented foods
- Key principles: often includes multiple small portion dishes, promotes satiety, and incorporates herbs and spices (eg, garlic and ginger)

DASH=Dietary Approaches to Stop Hypertension.

dehydration-mediated acute kidney injury, severe gastrointestinal adverse effects or markedly reduced intake might plausibly increase acute kidney injury risk through volume depletion.[76–80] There is currently insufficient evidence to suggest that increasing fluid intake alone is beneficial for chronic constipation.[64]

Adequate fibre intake

Incretin-based therapies suppress appetite, potentially lowering dietary fibre intake, contributing to constipation. Aim for ≥25 g per day, individualised via medical nutrition therapy (fibre density of 12·6 g/1000 kcal or 3·0 g/MJ), recognising that meeting this target is challenging when energy intake is <1500 kcal per day (6·3 MJ per day).[61,62] For constipation, first-line advice should focus on gradual food-based fibre optimisation, adequate fluids, regular movement, and specific evidence-based food options where appropriate (eg, kiwifruit or prunes). If constipation persists despite these measures, consider an evidence-based fibre supplement such as psyllium, titrated gradually with adequate fluids and monitored for tolerability; supplements should complement rather than replace food-based strategies (table 1).[12,60,63,64] Furthermore, maintaining high dietary fibre intake remains crucial for broader health improvements.[81]

Hair loss

Rapid weight loss from any cause can trigger telogen effluvium, compounded by nutritional deficiencies (ie, iron, zinc, folate, or low protein intake).[82] However, direct evidence linking incretin-based therapies to alopecia is scarce.[83] Therefore, hair loss should be considered as a nutritional sign, prompting review of weight-loss rate and nutritional adequacy (including protein and iron intake and zinc where clinically indicated), with thyroid function and androgen evaluation considered only if clinically indicated or symptoms persist.

Vomiting

Persistent vomiting can lead to dehydration, electrolyte imbalances, and nutrient loss. Thiamine has limited stores (1–3 weeks), and protracted vomiting (>1 week) post-metabolic–bariatric surgery can precipitate deficiency and Wernicke encephalopathy.[84,85] Protracted vomiting or markedly reduced intake during incretin-based therapy treatment could precipitate thiamine deficiency and has been associated with Wernicke encephalopathy in case reports.[86]

In patients receiving incretin-based therapies who develop persistent nausea or vomiting, management should include prompt clinical review, hydration and nutritional assessment, and consideration of incretin-based therapy dose reduction or temporary treatment interruption. Where vomiting is prolonged, severe, or prevents maintenance of hydration or nutritional intake, down-titration, temporary cessation, or discontinuation should be considered. Empirical thiamine supplementation should be considered, with biochemical testing for deficiency where available. Symptomatic management might include short courses of as-needed antiemetics (ie, 5-HT3 antagonists such as ondansetron or H1 antagonists such as cyclizine).[42,70]

Patient education

Health-care professionals (including dietitians) should educate patients on managing expected gastrointestinal side-effects through dietary modifications, hydration, and slow-dose titration. Patients should be counselled about rare serious adverse events (eg, cholecystitis, acute pancreatitis, and bowel obstruction), but they should also be informed that placebo-controlled randomised controlled trial meta-analyses do not show a clear increased risk of acute pancreatitis or bowel obstruction with GLP-1 receptor agonists.[87] Short courses of medications for reflux and nausea might help, but are rarely required once incretin-based therapy doses stabilise (table 2).[6,12,42]

Summary

Gastrointestinal side-effects are common but are transient with incretin-based therapies, and can be mitigated with slow titration, low-fat meals, fluids, and fibre. Patients should be monitored in cases of prolonged vomiting, and should be educated about symptoms of concern (tables 1–3).

Clinical and nutritional considerations during incretin-based therapies: nutrient deficiency prevention and malnutrition risk management

Nutritional deficiencies and malnutrition risk

People with obesity frequently have co-existing nutritional inadequacies, reflecting a double burden in which excess adiposity coexists with undernutrition, including deficiencies or insufficiencies in key micronutrients, driven by poor diet quality, altered nutrient metabolism, and polypharmacy.[88]

Restrictive or self-directed dietary interventions and food insecurity can worsen deficits, especially when nutrient-dense food intake is limited.[5,89,90] Populations at higher risk of nutritional deficiency include older adults, post-metabolic–bariatric surgery patients, and individuals with chronic diseases or pre-existing nutritional deficiencies.[91] Although direct evidence linking incretin-based therapies to protein energy malnutrition is scarce, appetite suppression, gastrointestinal side-effects, and reduced energy intake provide plausible mechanisms by which nutritional inadequacy can develop, especially in food insecurity and other populations at high risk.[12]

	Medical nutrition therapy strategy
General advice	Eat regular meals (ie, 3–6 smaller meals spaced throughout the day) and avoid skipping meals; eat fat in moderation, and intake might need to be reduced if gastrointestinal symptoms are present; stay hydrated; avoid drinking fluids while eating; carbonated drinks might increase bloating, burping, and nausea due to GLP-1 receptor agonist's effect of slowing stomach emptying (although this is not harmful, it can cause discomfort); try to keep away from cooking smells if feeling nauseous; use a smaller plate as a way to manage portion sizes; take a short walk in the fresh air or open the window for a while before a meal; consider foods containing ginger; choose bland, dry, and cold or room-temperature foods to minimise odours; avoid strong smells, greasy and very fatty or sweet foods; tailor fibre consumption; keep food and symptom diary to track foods that might cause or worsen symptoms
Nausea and vomiting	In addition to general advice: if prescribed anti-nausea medication, take as directed and time meals for when they are most effective; in mornings, eat a plain item of food on waking
Persistent vomiting	Consider the risk of dehydration, electrolyte imbalances and nutrient loss that might result in weakness and arrhythmias; thiamine is crucial for energy metabolism and has limited stores (1–3 weeks); after metabolic–bariatric surgery, protracted vomiting (>1 week) can precipitate thiamine deficiency, which can lead to Wernicke encephalopathy; persistent vomiting might plausibly increase acute kidney injury risk through volume depletion, although direct trial evidence is limited; persistent vomiting should prompt urgent clinical review; consider incretin-based therapy dose reduction, delay of escalation, temporary pause, or discontinuation if hydration or nutritional intake cannot be maintained or symptoms are prolonged or severe; oral rehydration solution might be required to prevent dehydration; consider and assess for any current or new eating-disorder behaviour particularly if there is a suggestion that the vomiting may be self-induced
Constipation	Gradually increase food-based fibre intake towards ~25 g per day, ensure hydration (2·0–2·5 L per day), encourage regular movement, and consider specific evidence-based food options such as kiwifruit or prunes where appropriate; encourage use of a hot beverage, hot cereal, or other high-fibre foods to stimulate bowel movement; if constipation persists despite these measures, consider an evidence-based fibre supplement (eg, psyllium), titrated gradually with adequate fluids; discuss medications that affect bowel function with your clinician
Diarrhoea	Based on medical nutrition therapy assessment might require a low-fat and/or low-lactose meal plan, avoiding gas-producing foods; high-fat foods might worsen diarrhoea and gastrointestinal discomfort during incretin-based therapies; adjust fibre intake; small, regular meals; limit sugar alcohols (eg, sorbitol and xylitol), caffeine, and alcohol; keep a food and symptom diary to identify problem foods; include soluble fibre from fruits, vegetables, oats, psyllium, and bulking agents; ensure adequate hydration
Early satiety	Eat small, frequent meals and avoid skipping meals; nutrient-rich, balanced meals (eg, eat protein first); soft or easily digestible meals (eg, oatmeal, mashed potatoes and vegetables, omelette, yoghurt with berries and muesli); consume liquids after or between meals instead of before and with meals
Dyspepsia	Small, frequent meals; choose low-fat foods; limit spicy foods, fatty foods, caffeinated beverages, carbonated drinks, and alcohol; eat slowly and chew well
Fatigue	Eat regular meals, choose complex carbohydrates, adequate protein intake, stay hydrated, focus on micronutrients (eg, iron and B vitamins), and limit sugar; if this continues speak with a health-care professional about appropriate blood tests and further investigations
Headache	Stay hydrated and eat regular meals
Dizziness	Stay hydrated, eat small, frequent meals, and avoid standing up too quickly
Eructation (belching)	Eat slowly and chew well, avoid carbonated drinks, avoid chewing gum, and limit gas-producing foods

Targets for fibre (≥25 g per day) and fluids (2·0–2·5 L per day) refer to total daily intake; increase gradually to tolerance. Interventions should be individualised following medical nutrition therapy assessment, taking account of comorbidities and concurrent medicines. Patients should be advised to seek clinical review for persistent, severe, or progressive symptoms, inability to maintain hydration or nutrition, or suspected complications. Evidence for specific fibre supplements is largely derived from chronic constipation populations rather than incretin-based therapy-specific trials, so selection should follow local guidance and individual tolerability. Recommendations are based on the cited sources[6,12,42,64,70] and expert consensus. General note: these strategies are guidance only; personalisation within a structured medical nutrition therapy plan is essential for effective and safe side-effect management.

Table 2: **Dietary and behavioural measures recommended as first-line adjuncts to mitigate treatment-emergent symptoms during incretin-based therapies**

For more on the **GRASP calculator** see https://grasp.chu-clermontferrand.fr/home

Although there is currently no direct randomised controlled trial evidence demonstrating that nutritional deficiency risk is attributable to incretin-based therapy, emerging observational and modelling data, biological plausibility, and expert consensus support a pragmatic, proportionate approach to nutritional screening and prevention. Nutritional risk management should be targeted, particularly to patients with clinical features suggesting nutritional vulnerability, with dietitian-delivered medical nutrition therapy, biochemical monitoring, and supplementation used where clinically indicated rather than applied universally (appendix pp 56–61).[92]

Post-metabolic–bariatric surgery nutritional frameworks might provide a pragmatic template to inform incretin-based therapy-related care, while recognising that surgical anatomical changes limit direct translation. Detailed post-metabolic–bariatric surgery screening, lifelong supplementation, and monitoring considerations are summarised in the appendix (pp 12–14, 56–61).[93–98]

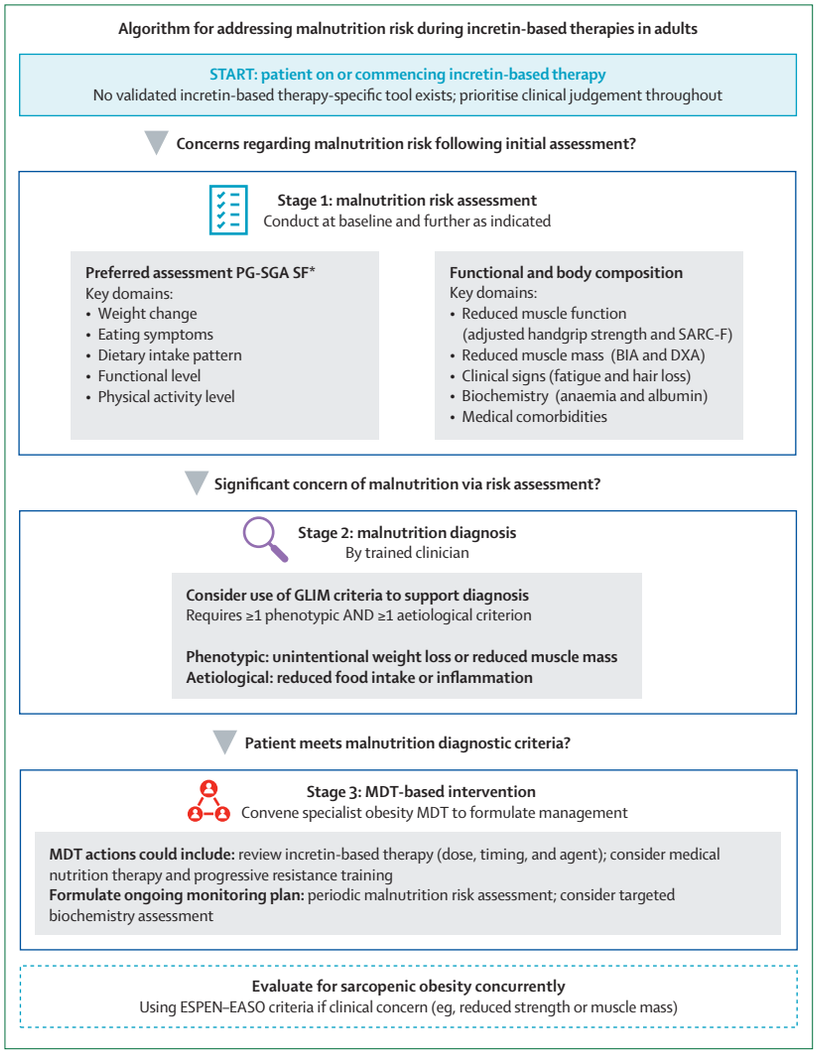

Figure 1: **Proposed algorithm for addressing malnutrition risk during incretin-based therapies in adults**
Principle: No validated incretin-based therapy-specific malnutrition-risk tool currently exists. Given the limited evidence available, this proposed algorithm reflects our expert consensus and may evolve as new data emerge. Measures of body composition and muscle function are not uniformly available across settings. The functional, anthropometric, and body-composition elements listed below are intended as a toolkit; clinicians should select locally feasible measures, prioritising function (eg, adjusted handgrip strength or sit-to-stand) and using body-composition assessment (eg, BIA or DXA) where available, with escalation to specialist assessment when concerns persist or risk is high. Stage 1 involves baseline and interval screening using a suitable nutrition-risk tool, supported where feasible by functional assessment, anthropometry, targeted biochemistry, and body-composition measures. Stage 2 confirms and stratifies risk using GLIM criteria, with emphasis on reduced intake, disease burden, muscle function, and muscle mass rather than BMI alone. Stage 3 involves multidisciplinary assessment and intervention, including review of incretin-based therapy dose or indication, dietitian-led medical nutrition therapy, correction of deficiencies, protein optimisation, resistance exercise, and ongoing monitoring. Sarcopenic obesity should be evaluated in parallel where clinically indicated, using muscle strength and muscle mass assessment in line with ESPEN–EASO guidance. Full details on suggested tools, thresholds, and implementation considerations are provided in the appendix (pp 29–31). BIA=bioelectrical impedance analysis. DXA=dual-energy x-ray absorptiometry. ESPEN=European Society for Clinical Nutrition and Metabolism. EASO=European Association for the Study of Obesity. GLIM=Global Leadership Initiative on Malnutrition. MDT=multidisciplinary team. PG-SGA SF=Patient-Generated Subjective Global Assessment–Short Form. SARC-F=Strength–Assistance–Rise–Climb–Falls. *If using malnutrition screening tool.

Screening and monitoring for malnutrition risk during incretin-based therapies

A proactive, risk-stratified approach to assessing malnutrition risk before and during incretin-based therapy might be appropriate, particularly in patients with features suggesting nutritional vulnerability. These might include, but are not limited to, older age or frailty, post-metabolic–bariatric surgery, pre-existing malnutrition or sarcopenia, restrictive dietary patterns, food insecurity, sustained very low energy intake, rapid or excessive weight loss, protracted gastrointestinal intolerance, eating disorder symptoms, or comorbidities that increase nutritional risk. However, many conventional screening tools might underperform in people with obesity because they rely on BMI and unintentional weight loss.[12,68,91,99,100] The Patient-Generated Subjective Global Assessment Short Form might be preferable to traditional malnutrition screens in people with obesity due to its greater ability to detect malnutrition risk.[101,102]

For malnutrition diagnosis, the Global Leadership Initiative on Malnutrition uses a two-step process: initial risk screening, followed by phenotypic and aetiological criteria.[103] However, its applicability in obesity is limited by reliance on low BMI thresholds and percentage weight loss, minimal attention to dietary quality and obesity-specific risks (eg, including nutritional complications related to obesity management such as metabolic–bariatric surgery), and challenges in assessing muscle mass in obesity.[68,103,104]

Late clinical signs (eg, fatigue, glossitis or neuropathy, alopecia, and impaired wound healing) and biochemical signatures (eg, anaemia, elevated C-reactive protein, and low albumin) also signal malnutrition risk,[12,104,105] while unintentional weight loss might indicate underlying disease.[12,70,104,105] No validated incretin-based therapy-specific tool exists, underscoring the need for new models that integrate functional and body composition assessment.[103,104,106,107] Given the very limited evidence available,

the proposed algorithm for addressing malnutrition risk during incretin-based therapies (figure 1) reflects our expert consensus and might evolve as new data emerge.

Targeted medical nutrition therapy to mitigate nutritional deficiency risk

With few incretin-based therapy-specific nutrition standards, medical nutrition therapy should draw on established obesity and post-metabolic–bariatric surgery principles.[12] Before and during incretin-based therapy, dietitian-led medical nutrition therapy focused on a balanced, nutrient-dense dietary pattern should be provided (table 1) and current dietary intake should be reviewed to identify and reduce nutritional deficiency risk.[108] Micronutrient adequacy is crucial during energy restriction. Pre-existing micronutrient inadequacy is common in obesity, although most of the evidence is observational.[109,110] Emerging data suggest that dietary quality and food choice might improve in some individuals during GLP-1 receptor agonist treatment, but evidence remains limited and heterogeneous. However, appetite suppression and altered taste are common and might compound the risk of inadequate intake in some patients. These should be addressed with medical nutrition therapy and multivitamin–mineral supplementation as required.[44] A pragmatic approach to nutritional screening, monitoring, and supplementation is therefore appropriate.

Laboratory monitoring

In the absence of trial evidence to support universal nutritional surveillance, we recommend a pragmatic approach. This should not be interpreted as routine-extensive biochemical screening for all incretin-based therapy users. Rather, testing should remain proportionate and targeted to patients with clinical features suggesting nutritional vulnerability. Baseline or periodic biochemistry (eg, full blood count, ferritin and iron studies, and vitamin B12) should be considered where clinically indicated (eg, symptoms or signs of deficiency, protracted gastrointestinal intolerance, sustained very low intake, older age, restrictive diets or food insecurity, comorbidity, or other vulnerabilities), with additional tests (including but not limited to serum folate, 25-hydroxyvitamin D, calcium, parathyroid hormone, magnesium, and albumin) targeted to clinical context and local resources. Patients using incretin-based therapies post-metabolic–bariatric surgery should follow standard post-metabolic–bariatric surgery nutritional monitoring protocols.[96]

Multivitamin–mineral supplementation

Multivitamin–mineral supplementation is not routinely required for all patients receiving incretin-based therapies, but supplementation should be considered selectively according to nutritional risk. If micronutrient intake is inadequate or deficiency risk is high, a daily complete multivitamin–mineral supplement should be considered. Use energy intake thresholds to guide actions: energy of <1500 kcal per day (6·3 MJ) signals heightened nutritional deficiency risk; <1200 kcal per day (5 MJ) warrants consideration of a daily complete multivitamin–mineral and additional protein supplementation.[12,53,68,69] For patients consuming >1200 kcal per day but with poor dietary quality, a daily complete multivitamin–mineral should be considered.

Incretin-based therapy dose reduction or temporary treatment pauses should be considered in severe cases of poor micronutrient intake or high deficiency risk.[44,111] Multivitamin–mineral preparations should be tailored according to clinical context (eg, chronic kidney disease, age, and diet quality) following medical nutrition therapy assessment. Total micronutrient exposure should be reviewed to avoid exceeding upper recommended limits, especially when combining products, and to ensure post-metabolic–bariatric surgery patients receive supplements as per guidelines.[96,112]

Protein requirements during incretin-based therapy

Incretin-based therapies lead to reduced intake across all macronutrients, including protein,[30] yet adequate protein remains essential for preserving muscle mass.[44] Evidence defining optimal protein targets specifically during incretin-based therapy treatment is scarce, and recommendations are therefore pragmatic and individualised rather than prescriptive. European Food Safety Authority guidance on total diet replacements provides useful contextual evidence for protein adequacy during substantial energy restriction. However, direct translation to incretin-based therapy treatment requires caution because incretin-based therapies differ from formula-based total diet replacement.[53]

Based on expert consensus during this project, consider aiming for 1·0–1·5 g/kg adjusted bodyweight per day during weight loss with incretin-based therapies (minimum ≥60 g per day), as intakes <1·0 g/kg adjusted bodyweight per day are linked to increased risk of muscle mass loss.[48–57] This higher range is intended as a pragmatic, individualised target during active weight loss in selected patients without contraindications and should not supersede disease-specific protein recommendations; in patients with chronic kidney disease or other conditions requiring protein restriction, lower targets and specialist dietetic assessment are required. For weight maintenance, individualise protein targets via medical nutrition therapy to the clinical context, current incretin-based therapy, and comorbidities; a typical range is approximately 0·8–1·0 g/kg adjusted bodyweight per day (adjusted bodyweight = reference bodyweight + [0·33 × (actual bodyweight − reference bodyweight)]; reference bodyweight is defined as a BMI of 25 kg/m²).[48,58] High biological-quality protein intake should be prioritised and distributed across meals, beginning meals with a small portion of protein to optimise satiety and muscle protein

synthesis.[12,53,59] Higher protein diets aid weight-loss maintenance and better preserve lean mass than lower protein diets, which is of particular relevance in older adults at risk of sarcopenia.[113,114] Evidence regarding intakes beyond recommended intakes for lean-mass preservation remain limited, particularly with incretin-based therapies. Key practical recommendations are summarised in table 3, with additional implementation details (including approaches for patients with nausea or constrained dietary patterns) provided in the appendix (pp 12–14).

Disease-specific nutritional considerations

Selected comorbidities might require tailored nutritional oversight during incretin-based therapies (appendix pp 12–14).

	Considerations
Role of medical nutrition therapy delivered by dietitians	
1a	Dietitian-delivered medical nutrition therapy is an important component of care for patients receiving incretin-based therapies, particularly for those with greater nutritional risk or clinical complexity, and might include assessment, diagnosis, intervention, and follow-up with personalised meal planning and behavioural support
1b	Where dietetic resources are limited, use a stepped-care approach to prioritise dietitian input for higher risk or more complex presentations, with routine follow-up and protocolised support delivered by other trained multidisciplinary team members
2	For patients receiving incretin-based therapies, medical nutrition therapy should prioritise dietary quality aligned with established healthy patterns, support gastrointestinal side-effect tolerance and medication adherence, maintain macronutrient and micronutrient adequacy, preserve lean mass and function, and promote healthy eating patterns and long-term behavioural change for weight maintenance and overall health improvements
3	Baseline and follow-up nutrition assessment should be considered where clinically indicated; dietitian input should be prioritised for older adults, patients with other chronic conditions, previous metabolic–bariatric surgery, restrictive dietary patterns (eg, vegans), food insecurity, food allergies, suspected malnutrition risk, or persistent treatment-related nutritional concerns
4	If energy intake remains below 800 kcal per day for a sustained period (eg, ≥1 week), or nutritional requirements remain unmet despite appropriate nutritional support, the multidisciplinary team should, via shared decision making with the patient, consider incretin-based therapy down-titration, a temporary pause, or discontinuation
Incretin-based therapy side-effects	
1	Adopt a gradual, flexible dose-escalation schedule to minimise gastrointestinal adverse effects; if substantial intolerance occurs, delay titration, revert to the previously tolerated dose, temporarily pause treatment, or discontinue if clinically necessary
2	Consider providing structured dietitian-led patient education on dietary strategies to prevent and manage incretin-based therapy-related gastrointestinal symptoms
3	Aim for approximately 2·0–2·5 L per day fluid intake, limit high-caffeine drinks and alcohol intake, and individualise by climate or vigorous physical activity and clinical context
4	Titrate dietary fibre to ≥25 g per day, increasing gradually with adequate fluids, to prevent and manage constipation during incretin-based therapy; individualise to national guidelines and patient preference
5	Consider monitoring for deficiencies: certain side-effects can signal or might cause nutritional problems; for instance, hair loss during rapid weight loss might indicate protein, iron, or zinc deficiency, and prolonged vomiting can deplete thiamine and is associated with Wernicke encephalopathy in case reports; such signs should prompt consideration of micronutrient biochemistry and supplementation
Clinical and nutritional recommendations	
1	When malnutrition is suspected or identified, consider assessment by appropriately trained clinicians (eg, dietitian); for high-risk or confirmed cases, consider convening the specialist multidisciplinary team to review incretin-based therapy indication, dosing, titration, initiate nutritional and functional interventions, and arrange structured follow-up, with repeat multidisciplinary team reviews for complex presentations
2a	Baseline assessment and correction: before initiating incretin-based therapies, consider assessing and correcting common micronutrient deficiencies (eg, vitamin D, iron, and vitamin B12) where clinically indicated and in line with local guidance
2b	Risk stratified monitoring: in the absence of trial evidence supporting universal surveillance, adopt a pragmatic, risk-stratified approach; this should not be interpreted as routine extended biochemical screening for all incretin-based therapy users, and testing should remain proportionate to clinical risk; consider baseline and/or periodic biochemistry (eg, full blood count, ferritin and iron studies, and vitamin B12) only where clinically indicated (eg, symptoms or signs of deficiency, protracted gastrointestinal intolerance, sustained very low intake, older age, restrictive diets or food insecurity, comorbidity, or other vulnerability) and, guided by clinical context and local resources, add further tests as needed (including but not limited to folate, 25-hydroxyvitamin D, calcium, parathyroid hormone, magnesium, and albumin)
2c	Response: throughout treatment, monitor nutritional adequacy where clinically indicated, recognising that reduced appetite and intake might unmask or exacerbate deficiencies, and consider multivitamin–mineral and/or targeted supplementation according to clinical context and local resources
3a	Set protein at ~1·0–1·5 g/kg adjusted bodyweight per day (minimum 60 g per day) during active weight loss on incretin-based therapies in selected patients without contraindications; this is an individualised pragmatic target and must not override disease-specific protein recommendations; direct evidence for specific protein thresholds in incretin-based therapy-treated populations remains limited; for weight maintenance, individualise protein intake to ~0·8–1·0 g/kg adjusted bodyweight per day via dietitian-led medical nutrition therapy; In chronic kidney disease or other conditions requiring protein restriction, use lower disease-specific targets with specialist (including renal dietitian) input; avoid protein intakes <0·4–0·5 g/kg per day and prolonged intakes >2 g/kg per day
3b	Emphasise high biological-quality protein, distributed across meals; consider dietitian-guided protein supplementation where intake is insufficient, particularly in the context of persistent nausea or early satiety; in plant-based diets, provide additional support to achieve adequate protein quantity and quality
4	Dietitian-led medical nutrition therapy should develop an individualised, nutrient-dense eating plan to meet protein and micronutrient needs despite reduced appetite
5	Routine multivitamin–mineral supplementation is not required for all patients receiving incretin-based therapies; for energy intake <1200 kcal per day, consider recommending a daily complete multivitamin–mineral supplement; for patients with energy intake ≥1200 kcal per day but poor dietary quality, consider a daily complete multivitamin–mineral supplement
6	Consider the need for a separate vitamin D supplement (dosing based on the country's national guidelines) and the prevalence of Vitamin D insufficiency and deficiency in the country of residence
7	Protein supplements (if assessed to be necessary) should be seen as a complement to whole foods, rather than a substitute, since they frequently lack other valuable nutrients such as fibre, vitamins, and minerals

(Table 3 continues on next page)

	Considerations
(Continued from previous page)	
8	Ensure that patients who have undergone metabolic–bariatric surgery and use incretin-based therapies receive the appropriate supplementation and monitoring as recommended after metabolic–bariatric surgery
Nutritional psychology	
1	Dietitians should routinely screen for disordered-eating behaviours and eating disorders and provide anticipatory guidance at initiation, dose changes, plateaus, maintenance and after cessation; where disordered-eating behaviours and eating disorders are present, consider discussing potential symptom change and agree relapse-prevention strategies; consider involving eating disorder-informed psychology when symptoms are pronounced
2	Consider exploring identity and adjustment after weight loss; if appropriately trained health-care professionals are available offer brief behavioural and psychological interventions and co-develop alternative coping strategies and connections beyond food
3	Consider monitoring for reward substitution, providing brief interventions, liaising with addiction services, and coordinating additional psychological support where indicated
4	Consider, if trained, using psychologically informed techniques delivered with respectful, weight-inclusive communication to reduce internalised stigma, counter incretin-based therapy-related and weight-related stigma, support body acceptance, develop realistic and acceptable maintenance goals, and sustain healthy eating patterns
Mental health considerations during treatment with incretin-based therapies	
1	For patients with greater psychosocial complexity, positive screens, or emerging concerns, consider using a complexity-readiness framework to guide proportionate psychological input; reassess at key clinical timepoints such as initiation, titration, plateaus, or discontinuation; consider applying reasonable adjustments for neurodivergence and learning disability, and establish shared care with other services and organisations as appropriate
2	Consider the use of brief validated screening tools to support assessment of psychological factors, particularly for individuals with greater clinical complexity or more complex mental health needs; positive screens and complex presentations (eg, recent suicidality, pronounced disordered-eating behaviours and eating disorders, complex severe mental illness, considerable adversity with unmanaged trauma symptoms, and hazardous substance use) might warrant consideration of 1:1 psychology and/or psychiatry review
3	Access to incretin-based therapies should not be routinely delayed on the basis of severe mental illness alone; instead, support and monitoring can be adapted to enable safe initiation, recognising the high burden of obesity-related morbidity and the risk of therapeutic inequity
4	Consider assessing baseline suicide risk and monitoring mood where indicated, particularly at initiation, dose escalation, and cessation; update safety plans and confirm engagement with appropriate services alongside monitoring for reward substitution
5a	Simultaneous titration of psychotropics is uncommon and not contraindicated; because delayed gastric emptying and vomiting might theoretically affect oral psychotropic absorption (eg, antipsychotics, lithium, and mood stabilisers), consider planning with psychiatry
5b	If mental state deterioration or gastrointestinal intolerance raises concern for reduced oral psychotropic absorption (eg, lithium, antipsychotics, and mood stabilisers), coordinate with psychiatry and consider pragmatic safeguards (eg, plasma levels where appropriate, dose adjustment, slower incretin-based therapy titration, or non-oral formulations), without delaying access to incretin-based therapies
6	Where patients are under psychiatric care or taking psychotropics, consider an integrated care approach with two-way multidisciplinary team communication with clear escalation plans, documented within shared-care arrangements where relevant; consider early check-ins (1–2 weeks) after initiation and dose changes to review mood, adherence, and gastrointestinal effects that could affect drug absorption
7	Consider assessment of disordered-eating behaviours (including binge or loss-of-control eating, purging, and restrictive patterns) at baseline and follow-up, and liaison with eating-disorder services, with titration adjusted where clinically indicated
8	Consider food as a ritual, emotional regulator, and connector; anticipate disrupted food-related coping or rituals and reward substitution (eg, alcohol and substance use); provide brief anticipatory guidance and signpost or refer to addiction and psychology services when indicated
9	In individuals with current or recent substance or alcohol misuse, consider structured monitoring, coordination with addiction and mental health services, and brief interventions with clear escalation pathways
Body composition, lean mass, and sarcopenic obesity	
1	Routine monitoring during incretin-based therapies should include simple anthropometric and functional assessment, with more detailed body-composition assessment (including fat and lean-mass distribution) considered in patients at high risk or where clinical concerns arise
2	Where body-composition data are available, a pragmatic aim during weight reduction might be to favour proportionally greater fat loss than fat-free mass loss; for example, an approximate ≥3:1 fat:fat-free mass loss ratio might be used as an illustrative guide; however, direct evidence supporting a specific threshold in incretin-based therapy-treated populations remains limited, so this should be interpreted cautiously and not as a validated universal standard
3	Begin routine monitoring with a minimum set comprising bodyweight and waist circumference or waist-to-height ratio, a simple functional assessment (eg, handgrip strength or sit-to-stand test), and a brief patient-reported question on perceived weakness or difficulty with daily activities; repeat these measures during therapy, with frequency adjusted to baseline risk, weight-loss rate, and weight-loss magnitude; more detailed body-composition and/or functional assessment should be considered where decline from baseline, rapid or substantial weight loss, or persistent clinical concerns arise
4	Key interventions that might help support more favourable body-composition changes include progressive resistance exercise and adequate protein intake; consider tailoring protein intake and resistance-training prescriptions to the individual's ongoing assessment findings and multidisciplinary team clinical judgement; however, direct evidence in incretin-based therapy-treated populations remains limited, and specific protein and exercise thresholds should be regarded as pragmatic, consensus-based considerations that require more rigorous evaluation in prospective clinical trials
5	Consider adjusting management for groups at high risk; in older adults and individuals with pre-frailty or frailty, consider adopting a personalised, cautious approach: arrange closer monitoring of body composition and functional status, titrate incretin-based therapy more slowly, involve the multidisciplinary team (including registered physiotherapists) for support, and consider the need for personalised exercise programmes
6	Use pragmatic anthropometric and functional measures (eg, waist circumference or waist-to-height ratio alongside a simple functional measure such as handgrip strength or sit-to-stand test) as the default minimum monitoring set in routine care, with DXA or bioelectrical impedance analysis used as step-up tools where available and clinically indicated
7	Consider prioritising functional status (eg, activities of daily living, mobility, and strength) alongside, or where appropriate over, anthropometry when judging treatment benefit or potential harm, particularly in older adults and those with pre-frailty or frailty
8	Consider escalation to multidisciplinary team or specialist review when there is rapid or excessive weight loss, emerging functional decline, suspected sarcopenia, or inability to meet protein intake or exercise goals due to intolerance or comorbidity

(Table 3 continues on next page)

	Considerations
(Continued from previous page)	
Bone health	
1a	Because GLP-1 receptor agonists are associated with modest, weight loss-proportional reductions in bone mineral density, consider counselling patients on incretin-based therapies to maintain adequate calcium and vitamin D and to perform regular resistance and weight-bearing exercise, which might help mitigate bone loss
1b	Note that evidence that calcium and/or vitamin D supplementation alone reduces fracture risk is limited; therefore, supplementation should be individualised and aligned with local guidance
2a	For patients with osteoporosis risk factors, consider evaluating bone health when starting an incretin-based therapy; this could include FRAX or a DXA scan if available locally and warranted
2b	Interpret FRAX cautiously because fracture prediction tools are unvalidated during considerable weight loss and FRAX might underestimate risk in obesity
3a	If a patient has low bone mineral density and/or high fracture risk, consider commencing osteoporosis treatment in parallel with incretin-based therapy and consider referring those at high risk of falls to falls prevention services.
3b	Where gastrointestinal intolerance or absorption concerns are present, consider whether non-oral osteoporosis therapies (eg, intravenous bisphosphonates) are preferable, particularly around incretin-based therapy initiation and titration
Physical activity and exercise	
1	Beyond weight loss, regular physical activity confers broad health benefits, reduces cardiovascular risk independent of weight, and enhances physical function and mental wellbeing
2	During weight loss with incretin-based therapies, resistance training is potentially important to preserving lean body mass and strength
3	Encourage gradual progression from baseline activity and capacity; for many patients, the initial goal might simply be to start performing some bodily movement as well as reduce sedentary time; public health targets such as 150–300 mins/week of moderate-intensity aerobic activity, together with muscle-strengthening activities on ≥2 days/week, should be presented as long-term goals rather than immediate expectations for most patients; personalise the programme to baseline activity and fitness, comorbidities or limitations, previous experience and preferences, and socioenvironmental factors, adjusting as needed; clinically meaningful benefits occur even below guideline targets; aerobic options might include high-intensity interval training in selected patients where appropriate and supervised to prevent any injury
4	Escalate to multidisciplinary team, registered physiotherapist, or exercise specialist input when pain limits progression, recurrent injury occurs, there is rapid functional decline, suspected sarcopenia or frailty, or the patient cannot meet protein or resistance-training goals due to intolerance or comorbidity
5	Reduce sedentary time and encourage breaking up prolonged sitting, emphasising that clinically meaningful benefits accrue even below guideline targets
6	Use a structured progression approach (eg, FITT-VP principles) and reassess at key milestones (initiation, dose escalation, plateaus, and maintenance) to support adherence and minimise injury risk
Socioeconomic considerations	
1	Consider the effect of cost and the health-care infrastructure in driving inequalities in access to incretin-based therapies and to provide comprehensive nutritional, functional, and psychological support
2	Food insecurity both contributes to obesity and undermines treatment; therefore, consider routinely screening for it and tailor nutritional guidance and support accordingly

Summary of consensus-based considerations and practical recommendations for the safe and effective use of incretin-based therapies alongside registered dietitian-led medical nutrition therapy, physical activity, and psychological support where clinically indicated. Statements apply to adults unless stated and should be individualised through shared decision making, with heightened caution in chronic kidney disease, older adults or adults with frailty, post-metabolic–bariatric surgery, and other complex comorbidities. These considerations are not intended to represent a universal package of specialist assessment, biochemical testing, body-composition measurement, or psychological intervention for all patients receiving incretin-based therapies. Rather, they should be implemented using a proportionate, stepped-care approach: basic monitoring and standard behavioural support for most patients, with escalation to dietitian-led medical nutrition therapy, additional biochemical assessment, body-composition testing, physiotherapy or exercise specialist input, or psychological support where baseline vulnerability, emerging symptoms, rapid or excessive weight loss, persistent gastrointestinal intolerance, poor dietary intake, functional decline, psychosocial complexity, or patient preference indicates additional need. Local implementation should consider service capacity, affordability, and equity. Numbering indicates item order within sections and not strength or hierarchy of recommendations and considerations. Protein prescriptions are expressed as grams per kilogram of adjusted bodyweight; emphasise high-quality protein distributed across meals. Biochemical monitoring refers to at least annual basic tests with additional panels guided by dietitian-led assessment and clinical context. Fluid and fibre targets should be adapted for clinical indications (eg, heart failure and chronic kidney disease). Bone-health notes reflect weight-loss–proportional changes in bone mineral density; combine adequate calcium and vitamin D with resistance and weight-bearing exercise. Screening tools are examples and should be used pragmatically with locally validated alternatives. Recommendations and considerations relating to protein targets, resistance training, and body-composition preservation are largely consensus-based and pragmatic; direct evidence supporting specific thresholds in incretin-based therapy-treated populations remains limited and requires prospective clinical trial evaluation. DXA=dual-energy x-ray absorptiometry. FRAX=Fracture Risk Assessment Tool. FITT-VP=Frequency, Intensity, Time, Type, Volume, and Progression.

***Table 3*: Summary of considerations and recommendations**

Medication review requirements

Some concomitant medicines might need review and dose adjustment during incretin-based therapies (appendix p 15).

Summary

Evidence on dietary quality and nutrient intake during incretin-based therapy treatment remains scarce. Food choice might improve in some individuals, whereas reduced intake or nutritional inadequacy might occur in some patients, particularly when energy intake is very low, gastrointestinal intolerance is clinically significant, or pre-existing nutritional vulnerability is present. Individualised dietary advice should be provided for all, which should be delivered by appropriately trained multidisciplinary team members where suitable, with escalation to dietitian-delivered medical nutrition therapy, protein optimisation, and laboratory monitoring where clinically indicated (table 3).

Nutritional psychology: appetite, reward, and eating behaviour during incretin-based therapies

Disordered eating, food reward, and coping

People with obesity have higher rates of disordered eating behaviours and eating disorders than those with healthy weight.[115–117] Evidence on eating disorders with incretin-based therapies is scarce and heterogenous;

some studies suggest fewer binge or purging episodes, but long-term data are lacking.[118,119] Weight loss and appetite suppression with incretin-based therapies might resemble eating disorder-like behaviours.[120] If food previously served as a coping strategy, unaddressed psychological distress might shift to other maladaptive behaviours.[33,34,120] Therefore, brief routine inquiry regarding disordered eating behaviours might be considered as part of routine clinical follow-up, with escalation to more structured assessment or specialist referral where concerns are identified.[121]

Incretin-based therapies are associated with improved mental health (eg, reduced depressive symptoms and improved wellbeing or quality of life).[122,123] Broader psychological benefits include improved self-esteem, mood, hope, and interpersonal relationships, and reduced anxiety, depression, and compulsive behaviours; visible weight change might reinforce these effects.[124] Nonetheless, unanticipated emotional difficulties might occur: fear of losing access to treatment, unmet treatment-related expectations, loss of identity, loss of food-based coping strategies, and worries about perceived stigma, criticism, and uninvited attention.[35]

Weight loss with incretin-based therapies can interact with pervasive obesity-related and incretin-based therapy-related stigma and identity change, sometimes contributing to internalised shame, reduced wellbeing, and poorer engagement; where needed, proportionate psychological support might help patients navigate these adaptations and optimise outcomes.[125–129]

If trained, consider using behavioural and psychological techniques (eg, motivational interviewing, person-centred counselling, acceptance and commitment therapy, and cognitive behavioural therapy) to help patients navigate identity shifts. Interventions incorporating mindfulness, emotion regulation, and self-compassion, and education regarding obesity's multifactorial, progressive, and relapsing nature, can reduce internalised stigma, promote body acceptance, and encourage a sustainable relationship with food.[130,131]

Furthermore, dietitians and other health-care professionals should counter self-stigmatising narratives with respectful, empowering, weight-inclusive communication that recognises health beyond the scales.[132] Centring care on the patient's lived experience during incretin-based therapy-induced weight loss supports long-term psychological wellbeing and behaviour change. Without this support, isolation, diminished self-efficacy, and reduced adherence might undermine outcomes. Structured psychological support for mental health and body image might be helpful for selected patients to support behaviour change and long-term efficacy, particularly where screening or clinical assessment identifies psychological distress, body-image concerns, or disordered eating. However, direct trial evidence isolating this component during incretin-based therapy treatment remains scarce.[7,133,134]

Nutritional psychology considerations for incretin-based therapies

The multidisciplinary team should remain vigilant for disordered eating behaviours and eating disorders and incorporate brief inquiry at baseline and during follow-up, with targeted use of validated tools (eg, Dutch Eating Behaviour Questionnaire, Binge Eating Scale [BES], Seven-Item Binge Eating Disorder Screener [BEDS-7], and SCOFF Questionnaire) where clinically indicated (eg, previous eating disorder, loss-of-control eating, purging behaviours, marked dietary restraint, substantial psychological distress, or concerning weight-loss or behavioural patterns).[121,135] Screening tools should be complemented by clinical assessment and ongoing monitoring for specific indicators, including rapid or excessive weight loss, indicators of poor nutritional adequacy, and disproportionate focus on weight goals at the expense of nutrition, function, and overall health. Anticipatory guidance should be provided regarding potential changes in appetite, coping strategies, and eating patterns, which should be reassessed particularly at treatment initiation, dose escalation, and when clinical concerns emerge. Where clinically significant symptoms are identified, agree relapse-prevention strategies, involve eating disorder-informed psychology, and consider specialist referral. Support adjustment after weight loss and alternative coping strategies and social connection beyond food should be encouraged (appendix pp 62–63).[44]

Further background material, including epidemiology, qualitative lived-experience findings, stigma considerations, and patient-reported outcome frameworks, are provided in the appendix (pp 62–63).

Mental health considerations during treatment with incretin-based therapies

Incretin-based therapies can produce substantial weight loss, but their effects on appetite and reward pathways might interact with pre-existing psychological vulnerabilities and patterns of eating.[136] Individuals vary in their psychological profile, readiness for treatment, and the role that eating plays in their lives. For individuals with greater psychosocial complexity, positive screens, or emerging concerns, a biopsychosocial multidisciplinary approach using a complexity-readiness framework might help proportionate assessment and monitoring of psychological wellbeing during incretin-based therapy treatment. These considerations do not imply formal psychological intervention for all patients. For many individuals, expectation-setting, brief inquiry or screening, and routine review might be sufficient, with escalation to psychological or psychiatric support for those with severe mental illness, current or recent risk to self, eating disorder symptoms, substance or alcohol misuse, psychosocial instability, or emerging distress during treatment. Consideration of psychological and emotional factors might also be relevant to understanding treatment response and weight

trajectories after discontinuation. Recent evidence suggests clinically meaningful weight regain after stopping treatment,[137] although the role of behavioural and psychological processes remains uncertain and is probably multifactorial.

Proposed framework for psychological support during incretin-based therapy treatment in adults

Stage 1: psychological assessment

Clinical complexity	Treatment readiness
Higher complexity Current or recent risk to self, including suicidal behaviour or deliberate self-harm, persistent suicidal ideation, severe psychological concerns or eating disorder symptoms, or marked psychosocial instability	**Higher readiness** Nuanced treatment understanding of incretin-based therapies, awareness of emotional drivers of eating, less severe or no disordered eating, adaptive coping skills, and consistent past engagement
Medium complexity Stable psychiatric conditions, intermittent disordered eating, and developing awareness of how emotions affect eating and mood	**Medium readiness** Low but modifiable treatment understanding, unrealistic treatment expectations, emotional eating or episodic disordered eating, and inconsistent past engagement
Lower complexity Mild-to-well managed psychiatric conditions, intermittent mild disordered eating, stable social support, and awareness of how emotions affect eating and mood	**Lower readiness** Low fixed or inaccurate treatment understanding and unrealistic expectations, frequent disordered eating with maladaptive emotional regulation, and poor past engagement

Stage 2: therapeutic input matrix

	Lower readiness	Medium readiness	Higher readiness
Higher complexity	**Higher input needs** Consider psychological intervention before initiating incretin-based therapy, depending on context with very close monitoring	**Medium–higher input needs** Consider initiating incretin-based therapy with closer monitoring and additional psychological input where indicated	**Medium input needs** Consider initiating incretin-based therapy with more frequent psychologically informed support and close monitoring
Medium complexity	**Medium–higher input needs** Consider readiness-enhancing psychological support before or alongside incretin-based therapy and close monitoring	**Medium input needs** Consider initiating incretin-based therapy with structured psychological support and regular monitoring	**Lower–medium input needs** Consider initiating incretin-based therapy with routine monitoring and concurrent psychoeducation
Lower complexity	**Medium input needs** Consider initiating incretin-based therapy with close monitoring and psychoeducation to build readiness	**Lower–medium input needs** Consider initiating incretin-based therapy with routine monitoring, and brief psychoeducation if needed	**Lower input needs** Consider initiating incretin-based therapy with routine monitoring

Figure 2: **Proposed framework for psychological support during incretin-based therapy treatment in adults**
This clinical decision-support tool sets out a two-dimensional framework for proportionate psychological risk management during incretin-based therapy treatment. It is intended to guide psychological input where clinically indicated, rather than to imply that formal structured psychological support or repeated structured screening is required for all patients. Routine care should include psychological awareness and brief inquiry, while more structured screening, monitoring, and use of this framework should be reserved for patients with greater psychosocial complexity, positive screens, or emerging concerns. Stage 1 involves assessment of two domains: clinical complexity and treatment readiness. Clinical complexity includes factors such as coexisting mental health difficulties, disordered eating, risk issues, functional limitations, social context, and other stressors. Treatment readiness reflects understanding of treatment, expectations, emotion-regulation skills, previous adherence, and engagement with support. Stage 2 integrates these domains within a matrix to guide proportionate escalation, including monitoring, multidisciplinary team review, or referral to psychological or mental health services where appropriate. The framework should be revisited at initiation, dose escalation, weight-loss plateau, and discontinuation. Further implementation detail is provided in the appendix (pp 32–33, 69–75).

Alcohol use and addictive behaviours

Alcohol use should be assessed at baseline and monitored where clinically indicated (eg, current or recent misuse, previous substance-use disorder, or emerging concerns during treatment). Evidence for broader reward substitution behaviours during incretin-based therapies is limited; clinicians should remain alert to clinically meaningful behavioural change and respond with brief intervention and coordinate with addiction and mental health services when needed.[138–141]

Suicide risk

Trials and observational data do not indicate an increased risk of suicidality with incretin-based therapies. However, evidence in severe mental illness remains scarce due to trial exclusions and small subgroup studies. Accordingly, clinical monitoring should be risk-stratified, with particular attention for patients at high risk and during periods of rapid weight change (appendix pp 64–68).[122,136,142–146]

Psychological risk assessment framework

We propose a two-dimensional framework for psychological risk management during incretin-based therapies: clinical complexity (ie, psychiatric comorbidity, eating disorder or disordered eating behaviour severity, frequency and functional impact, social support, and medical or functional factors); and treatment readiness (ie, treatment understanding, flexible expectations, emotion-regulation skills, and current or past adherence). Both are dynamic and might be revisited at key clinical timepoints across the pathway, particularly during incretin-based therapy dose escalation, plateaus, and discontinuation. These spectra form a therapeutic input matrix (figure 2; appendix pp 69–73). This framework is intended to support triage and proportional escalation of psychological support, rather than routine formal psychological intervention for all patients receiving incretin-based therapies.

Tiered psychological pathways

Where feasible, brief validated screening tools might support assessment of psychological complexity (eg, Patient Health Questionnaire [PHQ]-9 and PHQ-8 for depression; Generalised Anxiety Disorder-7 for anxiety; BEDS-7, BES, or Three-Factor Eating Questionnaire-R18 for disordered eating behaviours; and Alcohol Use Disorders Identification Test for alcohol risk).[147] Positive screens or presentations indicating greater psychological complexity (eg, recent suicidality, pronounced disordered eating behaviours or eating disorders, complex or severe mental illness, substantial body image issues, substantial adversity with unmanaged trauma-related symptoms, and hazardous substance use) should prompt further structured clinical assessment conducted within the health-care professional's competencies, with referral to specialist psychological or psychiatric input when indicated or if more detailed assessments are needed.

Access to incretin-based therapies should not be delayed in severe mental illness, for which antipsychotic-associated weight gain often drives discontinuation.[148] Plans should reflect local capacity, be shared across primary and secondary care, and include clear shared-care agreements with multidisciplinary team documentation to support implementation.

Supplementary practical psychological considerations, including the supporting background evidence underpinning these recommendations (eg, reward substitution and psychiatric safety signals) are provided in the appendix (pp 18–20, 76–78),[139–141,149,150] with practical recommendations presented in table 3.

Summary

Incretin-based therapies are often associated with improved wellbeing and quality of life. A stepped-care approach can help identify individuals who might benefit from further psychological input, supporting proportionate care based on clinical complexity, treatment readiness, and findings from brief psychological assessment where indicated (table 3).

Body composition, lean mass, and sarcopenic obesity

Definition and risks associated with sarcopenic obesity

Sarcopenic obesity, characterised by excess adiposity with reduced muscle mass and function, worsens physical limitations and adverse health outcomes in older adults and those with pre-frailty.[151,152] Pharmacologically induced weight loss reduces fat-free mass, including skeletal muscle, potentially exceeding that seen with dietary intervention alone and similar to losses observed with metabolic–bariatric surgery.[153] Across incretin-based therapy trials, lean-mass changes were proportional to total weight loss and varied by agent and population.[2,3]

Meta-analysis reports that lean-mass loss accounts for approximately 24–30% of total weight loss.[154,155] Notably, incretin-based therapy trials consistently show net cardiometabolic benefit, and evidence from randomised controlled trials has not established that lean-mass loss associated with incretin-based therapy is clinically detrimental at a population level per se; however, muscle function and longer-term outcomes are not consistently assessed, and groups at higher risk are under-represented.

Accordingly, the clinical significance of lean-mass loss for strength, function, and bone health remains uncertain and might be most relevant in subgroups at higher risk (eg, older adults, adults with low baseline muscle mass and function, and adults with pre-frailty or frailty), particularly with rapid weight loss.[4,59,156,157]

Body-composition and functional assessment

Older adults and other patients at high risk (eg, those with low baseline muscle mass or function, pre-frailty or frailty, pre-existing sarcopenia or malnutrition, rapid weight loss, or persistent functional concerns) might warrant closer body-composition and functional monitoring.[4] Some aspect of basic functional monitoring should remain part of routine care during incretin-based therapy treatment. Pragmatic options include adjusted handgrip strength, the five-times sit-to-stand test, or both, together with a brief patient-reported question regarding new weakness or greater difficulty with daily tasks. Absolute handgrip strength might decline with weight loss and should therefore be interpreted in context; where handgrip strength is used, scoring adjusted for age, sex, and body size might improve interpretability in adults with obesity.[158–160]

Clinically, weight loss accompanied by emerging functional decline should prompt review of dietary adequacy, resistance training, and treatment intensity within the multidisciplinary team. Routine monitoring should begin with a minimum set comprising regular bodyweight and waist circumference, a simple functional assessment (eg, adjusted handgrip strength, five-times sit-to-stand test, or both), and a brief patient-reported question on perceived weakness or increased difficulty with daily activities (ie, do you feel weaker or have more difficulty performing daily tasks since starting the medication?); decline from baseline on these pragmatic measures should prompt further assessment. Routine body-composition monitoring is not required for all patients; rather, bioelectrical impedance analysis or dual-energy x-ray absorptiometry should be regarded as step-up tools where available and clinically indicated, particularly in patients at high risk or where functional concerns arise. Where body-composition data are available, a pragmatic aim might be to favour proportionally greater fat loss than lean-mass loss, for example an approximate 3:1 ratio of fat loss to lean-mass loss (ie, for every 4 kg lost, ≥3 kg fat and ≤1 kg fat-free mass). However, direct evidence supporting a specific threshold in incretin-based therapy-treated populations remains limited, so this should be interpreted cautiously and not as a validated universal standard. Accordingly, dietitian-delivered medical nutrition therapy and progressive resistance training might help support preservation of lean mass and function (appendix pp 79–83).[161]

Beyond the minimum set, body-composition assessment (fat and lean-mass distribution), ideally paired with a simple functional measure (eg, adjusted handgrip strength), can help guide and optimise obesity management during incretin-based therapies in patients at high risk or where clinical concerns arise.[162–164] Where available, dual-energy x-ray absorptiometry is the reference standard, with bioelectrical impedance analysis as a pragmatic alternative in routine care; if handgrip is unavailable, consider the five-times sit-to-stand or Strength–Assistance–Rise–Climb–Falls tests (appendix pp 84–90).[153,165,166]

Clinical implementation of body-composition monitoring

Consistent with the EASO diagnostic framework, moving beyond BMI alone should be considered by incorporating central adiposity (eg, waist circumference or waist-to-height ratio) and a pragmatic measure of muscle function (eg, adjusted handgrip strength or five-times sit-to-stand test), with dual-energy x-ray absorptiometry or bioelectrical impedance analysis used where available.[167,168] Figure 3 provides a pragmatic, risk-stratified toolkit rather than mandatory assessments: all patients should undergo the minimum monitoring set, while more detailed body-composition and functional assessment should be reserved for patients at high risk, with rapid or substantial weight loss, or with persistent functional concerns. Monitoring should be risk-stratified and responsive to the rate and magnitude of weight loss, prioritising functional status where this best reflects day-to-day capability, and using findings to guide adjustment of protein targets and resistance-training prescriptions with multidisciplinary team input. Further operational detail on measures and monitoring frequency is provided in figure 3 and the appendix (pp 21–23, 84–90).

Figure 3: **Integrating body-composition and functional assessment during incretin-based therapies in adults**

This risk-stratified framework provides a pragmatic toolkit for monitoring body-composition and physical function during incretin-based therapy, rather than mandating universal testing. It reflects expert consensus and should be adapted to local resources. Patients at higher risk of clinically important muscle loss may include older adults and those with frailty, low baseline muscle mass or function, pre-existing malnutrition or sarcopenia, and rapid weight loss. Stage 1 establishes the baseline and minimum monitoring assessment, comprising bodyweight, waist circumference or waist-to-height ratio, a simple functional measure such as adjusted handgrip strength and/or the five-times sit-to-stand test, and a brief patient-reported question on weakness or difficulty with daily activities. Additional body-composition assessment, such as bioelectrical impedance analysis or dual-energy x-ray absorptiometry, can be considered where available and clinically indicated. Stage 2 stratifies the risk of clinically important muscle loss or functional decline using factors such as older age, frailty, low baseline muscle mass or function, pre-existing malnutrition or sarcopenia, rapid or substantial weight loss, and emerging functional concerns. Stage 3 combines incretin-based therapy with medical nutrition therapy, adequate protein, hydration, fibre, fruit and vegetables, and progressive resistance exercise. Stage 4 repeats monitoring at regular intervals. Stage 5 uses trends to guide continuation, escalation, or multidisciplinary team review. The overall goal of this framework is to optimise the therapeutic benefits of incretin-based therapies while proactively mitigating the risks of sarcopenic obesity and functional decline. Further details are provided in the appendix (pp 34–35). ADL=activities of daily living. BIA=bioelectrical impedance analysis; DXA=dual-energy x-ray absorptiometry. EASO=European Association for the Study of Obesity. GRASP=Grip and Sarcopenia Prediction. SARC-F=Strength–Assistance–Rise–Climb–Falls. SPPB=short physical performance battery. *Indicates minimum monitoring set.

Older adults, frailty, and weight regain

Evidence in older adults (especially those older than 80 years) is scarce. In those with frailty or sarcopenia, consider balancing benefits and risks through shared decision making; consider cautious dose escalation, reviewing polypharmacy and monitoring body composition. Geriatric or frailty specialists should be involved where appropriate.[169,170]

Evidence on body-composition changes during weight regain after intentional weight loss is mixed.[44,171] In adults with overweight or obesity, pooled longitudinal data do not support a universal statement that regain is disproportionately adipose. However, some studies in

older or postmenopausal women who regain weight suggest greater fat-mass regain than lean-mass recovery.[172,173] Direct evidence after incretin-based therapy cessation remains scarce. Progressive resistance training and 1·0–1·5 g/kg adjusted bodyweight per day high-quality protein intake during incretin-based therapy might help preserve lean mass and muscle mass and limit weight regain,[114] although these currently remain hypotheses and require rigorous testing in prospective clinical trials.[161] Additional background on sarcopenic obesity, assessment modalities, and implementation of body-composition and functional monitoring is provided in the appendix (pp 21–23).

Summary

Incretin-based therapy-induced weight loss includes fat and lean-mass loss; pragmatic anthropometric and functional monitoring should be used in routine care, body-composition assessment should be considered in patients at high risk or where concerns arise, and lean-mass preservation should be supported with resistance training and adequate protein intake. The impact of pre-frailty or frailty should also be considered (table 3).

Physical activity and exercise in obesity management

Physical activity is central to obesity management, with benefits far beyond weight loss.[174] Compared with the absence of training, exercise produces only modest weight loss, but it does confer substantial cardiometabolic, functional, and psychological improvements.[174,175] During weight loss, resistance training is essential for lean-mass preservation.[174] Combined aerobic and resistance training optimises both cardiorespiratory fitness and muscular strength (panel 2), addressing the dual risks of sarcopenic obesity.[174,175]

Overall, exercise delivers modest weight loss but major health gains; aerobic and resistance training should be combined to preserve lean mass and improve function and adherence, and treatments should be individualised and progress gradually (table 3). Additional detail on the rationale, definitions, cardiometabolic and quality-of-life outcomes, and clinical recommendations is provided in the appendix (pp 24–26).

Panel 2: Physical activity and exercise clinical considerations

- Encourage any movement (some is better than none) and limit sedentary time,[174,176] with emphasis on sustaining physical activity in the long term to support weight-loss maintenance
- For overall health, adapt general population guidelines to the individual patient; encourage gradual progression from baseline activity and capacity; for many patients, the initial goal might simply be to start performing some bodily movement as well as reduce sedentary time; public health targets such as 150–300 mins/week of moderate-intensity aerobic activity (or 75–150 mins/week of vigorous-intensity physical activity), combined with muscle-strengthening activities on two or more days per week, can be presented as long-term goals to be obtained through a progressive increase rather than immediate expectations for most patients;[176,177] in the STEP-3 clinical trial, physical activity began at 100 mins/week (spread across 4–5 days) and was progressed by 25 mins every 4 weeks to 200 mins/week, illustrating a pragmatic and graded approach rather than immediate attainment of higher activity targets[178]
- Emphasise and teach moderate-intensity to high-intensity muscle-strengthening training (eg, resistance training) to help reduce lean body mass loss during obesity management;[176,179] training should target the major muscle groups
- Personalise and progress gradually, recognising clinically meaningful benefits occur below guideline targets; tailor to baseline activity and fitness, comorbidities and limitations, previous experience, and preferences, and use a structured progression framework such as FITT-VP (Frequency, Intensity, Time, Type, Volume, and Progression) with regular reassessment[174,180,181]
- High-intensity interval training might be a time-efficient option when appropriate and supervised; consider power training in selected patients to improve muscle quality[174,182]

Bone health in the context of incretin-based therapies

Bone health, bodyweight change, and fracture risk

Obesity is associated with higher bone mineral density but, paradoxically, higher fracture risk, whether through reduced bone quality, metabolic effects, or biomechanics.[183,184] Weight loss is associated with bone loss and increased fracture incidence, whether from lifestyle changes or metabolic–bariatric surgery.[185–188]

In pharmacotherapy studies, observed bone mineral density reductions appear largely weight-mediated,[187–189] with a lack of fracture endpoint data.[190] Baseline fracture risk assessment should be considered at incretin-based therapy initiation (eg, Fracture Risk Assessment Tool, dual-energy x-ray absorptiometry, or both if clinically indicated); patients with high risks of falls should be referred to falls services; and low bone mineral density should be managed as per osteoporosis guidance.[191]

Assessment and mitigation strategies

Current guidance for incretin-based therapy-associated bone loss is largely extrapolated from the general population and post-metabolic–bariatric surgery data. Ensuring adequate calcium and vitamin D is reasonable, although neither calcium nor vitamin D supplementation (individually or combined) has shown fracture-risk reduction in community-dwelling adults in the general

population, and direct fracture trial data for incretin-based therapies are lacking.[192–196] Alongside this, resistance and weight-bearing exercise might help mitigate bone loss, and patients at higher fracture risk should be assessed and managed according to osteoporosis guidance.[197,198] For individuals requiring active bone management, consider parenteral osteoporosis therapies (eg, intravenous bisphosphonates) if gastrointestinal intolerance or absorption concerns are present, including during incretin-based therapy. Further details on bone health during incretin-based therapy weight loss, fracture risk, bone mineral density, and mitigation strategies are provided in the appendix (pp 27–28).

Summary

Overall, incretin-based therapy-induced weight loss reduces bone mineral density and may increase fracture risk; fracture risk should be assessed, ensuring that nutrition (including protein intake) and resistance training are appropriate for the individual and treat patients at high risk (table 3).

Socioeconomic considerations and health-care equity

Obesity disproportionately affects minority ethnic groups and lower socioeconomic populations, who also have reduced access to specialist services (eg, in the UK) and incretin-based therapies (eg, in North America).[199,200] Structural determinants of health, including out-of-pocket expenses, commissioning variation, service capacity, navigation barriers, and provider biases, drive both lower uptake and higher discontinuation rates.[90,201] Socioeconomic deprivation and reliance on public insurance are associated with poorer adherence, while private-pay models further exacerbate affordability barriers.[41,202]

Provision is also influenced by regional regulations and prescribing guidance. People with obesity without diagnosed complications are often disadvantaged compared with those with diagnosed obesity-related complications.[203] Policy should expand incretin-based therapy coverage and address stigma, while clinical services should improve access to dietitian-led medical nutrition therapy, which could reduce inequalities and improve adherence and outcomes (appendix pp 91–93).

Food insecurity, the lack of physical or economic access to safe, nutritious food meeting dietary needs, and food preferences for an active healthy life[204] substantially affect weight management. European prevalence of food insecurity is 5–20%, disproportionately affecting women, children, older adults, and single-parent households.[5,90] Paradoxically, obesity is more prevalent in people with food insecurity, driven not by lack of food but by lack of affordable healthy options,[205] and UK data link cost-of-living pressures to poorer diet quality.[206]

Various options are available to screen for food insecurity (eg, two-item nutritional security screener and Food Foundation's three-item tool);[207] screening must always be done sensitively (appendix pp 91–93).

A limitation of this Consensus Statement is that we did not undertake health economic modelling. The cost-effectiveness of different models of nutritional, functional, and psychological support during incretin-based therapy treatment remains uncertain and might vary by health-care system, drug access model, workforce capacity, and the baseline risk profile of the treated population. Universal intensive monitoring or specialist referral could create opportunity costs and inadvertently restrict access. Therefore, we favour a proportionate stepped-care model, in which basic monitoring is available to most patients and higher-intensity input is targeted to those with clinical vulnerability, emerging concerns, or patient-identified needs.

Future research directions

A systematic review of 417 incretin-based therapy randomised controlled trials showed that less than 20% of trials reported dietary intake or nutritional biomarkers and less than 5% reported bone, micronutrient, or physical-function outcomes, leaving major gaps across nutritional, functional, and psychological domains.[208] Research priorities include a core outcome set for incretin-based therapy trials,[208] robust nutritional safety surveillance (including malnutrition screening tools), evaluation of muscle function and performance,[4,114,209] mobility and strength outcomes, strategies to preserve

Search strategy and selection criteria

We evaluated the effect of incretin-based therapies, specifically GLP-1 receptor agonists and related agents, on nutritional status, body composition, physical function, psychological health, and socioeconomic equity. We searched PubMed for studies written in English from inception to Dec 15, 2025. Search terms for pharmacotherapy (including "GLP-1 receptor agonists", "dual/triple agonists", "semaglutide", "tirzepatide", "liraglutide", and "cagrilintide") were combined with terms across five key domains: nutrition (eg, "nutrition therapy", "diet quality", "micronutrients", "malnutrition", and "gastrointestinal tolerability"), body composition and function (eg, "sarcopenia", "skeletal muscle", "fat-free mass", "bone health", and "physical function and performance"), physical activity (eg, "exercise", "resistance training", and "aerobic training"); psychological and psychiatric health (eg, "food noise", "cravings", "food reward", "mental health", "suicidal ideation", and "disordered eating"), and equity (eg, "healthcare access", "health equity", "social determinants of health", "food insecurity", and "economic burden"). We restricted results to studies involving adults and identified additional references through author collaboration.

lean mass and bone,[161] optimal protein intake during incretin-based therapy treatment, psychological outcomes and targeted supports, dose-titration methods that reduce gastrointestinal discontinuation, haematological and pharmacokinetic safety (including psychotropic co-initiation and narrow therapeutic-index drugs), mixed-methods studies of lived experience, and post-cessation, post-metabolic–bariatric surgery, and special-population studies (appendix pp 94–96).

Conclusion

Incretin-based therapies represent a paradigm shift in obesity care. Implementation should be individualised, with escalation to dietitian-led medical nutrition therapy and integrated psychological and functional support, particularly where nutritional risk, psychosocial complexity, or functional vulnerability are present. Priorities include mitigating gastrointestinal side-effects, reducing micronutrient risk, and preserving lean mass via adequate protein, fibre, fluids, and nutrient-dense foods, alongside resistance training, targeted supplementation, and regular monitoring, with attention to identity, coping, and disordered eating. Further research is needed to evaluate long-term nutritional, functional, and psychological outcomes, develop obesity-specific malnutrition tools, and test strategies to protect muscle and bone.

Contributors

The methodology was designed by LJD with input from all other authors across various meetings. The literature review and evidence appraisal was performed by LJD with support from LT and all other authors in their respective areas of expertise. LJD led the initial and revised drafts with support from LT; all authors provided critical revisions throughout the manuscript development. LJD developed the figures and tables for the manuscript with support from all other authors. The project was coordinated by LJD. Project supervision was provided by BM, AC, MH, and VDY. All authors approved the final version of the manuscript and accept responsibility for the integrity of the work from inception to publication.

Declaration of interests

VDY has provided educational sessions or served on advisory boards for Eli Lilly, Novo Nordisk, Regeneron, and Rhythm Pharmaceuticals. BM is a shareholder in Reset Health and performs advisory and educational work for Novo Nordisk and advisory work for Lilly, Pfizer, and Johnson & Johnson. KC is on the patient advisory board for Novo Nordisk and Boehringer Ingelheim; has received consulting fees from Eli Lilly; declares lecture fees from Apollo Endo Surgery, Novo Nordisk, and I&J Ethicon; is chair of the European Coalition for People Living with Obesity (ECPO) and Weight Loss Surgery Information; and is director of Operations of Obesity UK. GOM reports receiving honoraria, paid to the Royal College of Surgeons in Ireland Obesity Research and Care Group, for educational events and research projects from the European Association for the Study of Obesity (EASO) and Novo Nordisk. SB is on an advisory board for Boehringer Ingelheim and Novo Nordisk; has received honoraria for educational events from the National Prevention and Cardiovascular Health, Apollo Endo Surgery, Radcliff Medical Education, Novo Nordisk Ireland, Safefood, and University College Dublin; and consultancy fees from ECPO. LJD has received funding, paid to King's College London, from Novo Nordisk for independent research investigating the social determinants of obesity care; has received honorarium for educational activities from Eli Lilly; and travel funding to present at international conferences from EASO. HM reports receiving support for attending meetings and travel from the Nutrition Society and the European Federation of the Associations of Dietitians (EFAD); holds unpaid leadership roles as a public health council member for the Nutrition Society and a committee member of the Obesity Group of the British Dietetic Association. JLB performs advisory and educational work for Novo Nordisk, with all fees paid to her institute. AB declares researcher-led grants from the National Institute for Health Research, Rosetrees Trust, Medical Research Council, INNOVATE UK, British Dietetic Association, British Association of Parenteral and Enteral Nutrition, Biotechnology and Biological Sciences Research Council, the Office of Health Improvement and Disparities, and Novo Nordisk; reports honoraria from Novo Nordisk, Eli Lilly, and Mac Nutrition outside the submitted work; and is on the medical advisory board and is a shareholder of Reset Health Clinics. DA has previously received honoraria for educational and training events from Novo Nordisk. ES has previously received honoraria for educational events from Novo Nordisk, Boston Scientific, and Ethicon (Johnson & Johnson). All other authors declare no competing interests.

Acknowledgments

The project was conceived and designed by the EASO Obesity Management Working Group (BM, AC, and LJD), EASO Nutrition Working Group (MB and MW), and EFAD's European Specialist Dietetic Network Obesity Committee (MH, EG, HM, LT, and DOM). The EASO Physical Activity, Fitness, and Function Working Group (JMO and GOM), the ECPO (KC, VM, and SB), and other contributors were involved as priorities emerged.

References

1 Dobbie LJ, Coelho C, Crane J, McGowan B. Clinical evaluation of patients living with obesity. *Intern Emerg Med* 2023; **18:** 1273–85.

2 Wilding JPH, Batterham RL, Calanna S, et al. Once-weekly semaglutide in adults with overweight or obesity. *N Engl J Med* 2021; **384:** 989–1002.

3 Jastreboff AM, Aronne LJ, Ahmad NN, et al. Tirzepatide once weekly for the treatment of obesity. *N Engl J Med* 2022; **387:** 205–16.

4 Linge J, Birkenfeld AL, Neeland IJ. Muscle mass and glucagon-like peptide-1 receptor agonists: adaptive or maladaptive response to weight loss? *Circulation* 2024; **150:** 1288–98.

5 Mozaffarian D, Agarwal M, Aggarwal M, et al. Nutritional priorities to support GLP-1 therapy for obesity: a joint advisory from the American College of Lifestyle Medicine, the American Society for Nutrition, the Obesity Medicine Association, and the Obesity Society. *Am J Clin Nutr* 2025; **122:** 344–67.

6 Gigliotti L, Warshaw H, Evert A, et al. Incretin-based therapies and lifestyle interventions: the evolving role of registered dietitian nutritionists in obesity care. *J Acad Nutr Diet* 2025; **125:** 408–21.

7 Celletti F, Farrar J, De Regil L. World Health Organization Guideline on the Use and Indications of Glucagon-Like Peptide-1 Therapies for the Treatment of Obesity in Adults. *JAMA* 2025; **335:** 434–38.

8 Genua I, Cusi K. Pharmacological approaches to nonalcoholic fatty liver disease: current and future therapies. *Diabetes Spectr* 2024; **37:** 48–58.

9 Yabut JM, Drucker DJ. Glucagon-like peptide-1 receptor-based therapeutics for metabolic liver disease. *Endocr Rev* 2023; **44:** 14–32.

10 Heise T, DeVries JH, Urva S, et al. Tirzepatide reduces appetite, energy intake, and fat mass in people with type 2 diabetes. *Diabetes Care* 2023; **46:** 998–1004.

11 Friedrichsen M, Breitschaft A, Tadayon S, Wizert A, Skovgaard D. The effect of semaglutide 2·4 mg once weekly on energy intake, appetite, control of eating, and gastric emptying in adults with obesity. *Diabetes Obes Metab* 2021; **23:** 754–62.

12 Almandoz JP, Wadden TA, Tewksbury C, et al. Nutritional considerations with antiobesity medications. *Obesity* 2024; **32:** 1613–31.

13 Martin CK, Carmichael OT, Carnell S, et al. Tirzepatide on ingestive behavior in adults with overweight or obesity: a randomized 6-week phase 1 trial. *Nat Med* 2025; **31:** 3141–50.

14 Hall KD. Physiology of the weight-loss plateau in response to diet restriction, GLP-1 receptor agonism, and bariatric surgery. *Obesity* 2024; **32:** 1163–68.

15 Walker CS, Aitken JF, Vazhoor Amarsingh G, Zhang S, Cooper GJS. Amylin: emergent therapeutic opportunities in overweight, obesity and diabetes mellitus. *Nat Rev Endocrinol* 2025; **21:** 482–94.

16 Wilding JPH. Beyond lifestyle interventions: exploring the potential of anti-obesity medications in the UK. *Clin Obes* 2018; **8:** 211–25.
17 Wilding JPH, Batterham RL, Davies M, et al. Weight regain and cardiometabolic effects after withdrawal of semaglutide: the STEP 1 trial extension. *Diabetes Obes Metab* 2022; **24:** 1553–64.
18 Rubino D, Abrahamsson N, Davies M, et al. Effect of continued weekly subcutaneous semaglutide *vs* placebo on weight loss maintenance in adults with overweight or obesity: the STEP 4 randomized clinical trial. *JAMA* 2021; **325:** 1414–25.
19 Wadden TA, Chao AM, Machineni S, et al. Tirzepatide after intensive lifestyle intervention in adults with overweight or obesity: the SURMOUNT-3 phase 3 trial. *Nat Med* 2023; **29:** 2909–18.
20 Lincoff AM, Brown-Frandsen K, Colhoun HM, et al. Semaglutide and cardiovascular outcomes in obesity without diabetes. *N Engl J Med* 2023; **389:** 2221–32.
21 Bonaca MP, Catarig AM, Houlind K, et al. Semaglutide and walking capacity in people with symptomatic peripheral artery disease and type 2 diabetes (STRIDE): a phase 3b, double-blind, randomised, placebo-controlled trial. *Lancet* 2025; **405:** 1580–93.
22 Perkovic V, Tuttle KR, Rossing P, et al. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. *N Engl J Med* 2024; **391:** 109–21.
23 Malhotra A, Grunstein RR, Fietze I, et al. Tirzepatide for the treatment of obstructive sleep apnea and obesity. *N Engl J Med* 2024; **391:** 1193–205.
24 Sanyal AJ, Newsome PN, Kliers I, et al. Phase 3 trial of semaglutide in metabolic dysfunction-associated steatohepatitis. *N Engl J Med* 2025; **392:** 2089–99.
25 Loomba R, Hartman ML, Lawitz EJ, et al. Tirzepatide for metabolic dysfunction-associated steatohepatitis with liver fibrosis. *N Engl J Med* 2024; **391:** 299–310.
26 Kosiborod MN, Abildstrøm SZ, Borlaug BA, et al. Semaglutide in patients with heart failure with preserved ejection fraction and obesity. *N Engl J Med* 2023; **389:** 1069–84.
27 Quast DR, Nauck MA, Schenker N, Menge BA, Kapitza C, Meier JJ. Macronutrient intake, appetite, food preferences and exocrine pancreas function after treatment with short- and long-acting glucagon-like peptide-1 receptor agonists in type 2 diabetes. *Diabetes Obes Metab* 2021; **23:** 2344–53.
28 Blundell J, Finlayson G, Axelsen M, et al. Effects of once-weekly semaglutide on appetite, energy intake, control of eating, food preference and body weight in subjects with obesity. *Diabetes Obes Metab* 2017; **19:** 1242–51.
29 Geisler CE, Antonellis MP, Trumbauer W, et al. Tirzepatide suppresses palatable food intake by selectively reducing preference for fat in rodents. *Diabetes Obes Metab* 2023; **25:** 56–67.
30 Bettadapura S, Dowling K, Jablon K, Al-Humadi AW, le Roux CW. Changes in food preferences and ingestive behaviors after glucagon-like peptide-1 analog treatment: techniques and opportunities. *Int J Obes* 2024; **49:** 418–26.
31 Cheney C, Hunter K, Klein M. Impact of GLP-1 receptor agonists on perceived eating behaviors in response to stimuli. *Diabetes Metab Syndr Obes* 2025; **18:** 1411–18.
32 Dhurandhar EJ, Maki KC, Dhurandhar NV, et al. Food noise: definition, measurement, and future research directions. *Nutr Diabetes* 2025; **15:** 30.
33 Conason A, Teixeira J, Hsu CH, Puma L, Knafo D, Geliebter A. Substance use following bariatric weight loss surgery. *JAMA Surg* 2013; **148:** 145–50.
34 Brown A, Mellor D, Makaronidis J, Shuttlewood E, Miras AD, Pournaras DJ. "From evidence to practice"—insights from the multidisciplinary team on the optimal integration of GLP-1 receptor agonists in obesity management services. *Nutr Bull* 2024; **49:** 257–63.
35 Farrell E, Nadglowski J, Hollmann E, le Roux CW, McGillicuddy D. "What would be left of me?" Patient perspectives on the risks of obesity treatment: an innovative health initiative stratification of obesity phenotypes to optimise future obesity therapy (IMI2 SOPHIA) qualitative study. *Obes Pillars* 2024; **12:** 100129.
36 Hassapidou M, Vlassopoulos A, Kalliostra M, et al. European Association for the Study of Obesity Position Statement on Medical Nutrition Therapy for the Management of Overweight and Obesity in Adults Developed in Collaboration with the European Federation of the Associations of Dietitians. *Obes Facts* 2023; **16:** 11–28.
37 Morgan-Bathke M, Raynor HA, Baxter SD, et al. Medical nutrition therapy interventions provided by dietitians for adult overweight and obesity management: an Academy of Nutrition and Dietetics evidence-based practice guideline. *J Acad Nutr Diet* 2023; **123:** 520–45.e10.
38 Swan WI, Vivanti A, Hakel-Smith NA, et al. Nutrition care process and model update: toward realizing people-centered care and outcomes management. *J Acad Nutr Diet* 2017; **117:** 2003–14.
39 Raynor HA, Morgan-Bathke M, Baxter SD, et al. Position of the Academy of Nutrition and Dietetics: medical nutrition therapy behavioral interventions provided by dietitians for adults with overweight or obesity, 2024. *J Acad Nutr Diet* 2024; **124:** 408–15.
40 Miller CK. Medical nutrition therapy: still relevant in the era of pharmacotherapy for obesity care. *J Acad Nutr Diet* 2024; **125:** 595–99.
41 Gasoyan H, Butsch WS, Schulte R, et al. Changes in weight and glycemic control following obesity treatment with semaglutide or tirzepatide by discontinuation status. *Obesity* 2025; **33:** 1657–67.
42 Wharton S, Davies M, Dicker D, et al. Managing the gastrointestinal side effects of GLP-1 receptor agonists in obesity: recommendations for clinical practice. *Postgrad Med* 2022; **134:** 14–19.
43 Scott Butsch W, Sulo S, Chang AT, et al. Nutritional deficiencies and muscle loss in adults with type 2 diabetes using GLP-1 receptor agonists: a retrospective observational study. *Obes Pillars* 2025; **15:** 100186.
44 Mechanick JI, Butsch WS, Christensen SM, et al. Strategies for minimizing muscle loss during use of incretin-mimetic drugs for treatment of obesity. *Obes Rev* 2024; **26:** e13841.
45 Morgan-Bathke M, Baxter SD, Halliday TM, et al. Weight management interventions provided by a dietitian for adults with overweight or obesity: an evidence analysis center systematic review and meta-analysis. *J Acad Nutr Diet* 2023; **123:** 1621–61.e25.
46 Tronieri JS, Fabricatore AN, Wadden TA, et al. Effects of dietary self-monitoring, physical activity, liraglutide 3·0 mg, and placebo on weight loss in the SCALE IBT trial. *Obes Facts* 2020; **13:** 572–83.
47 Moetaz I, Abumweis S, Alqadi S, AbuGhoush M. The role of the dietitian in weight management of adults with obesity without diabetes using glucagon like peptide-1 agonist receptors: a systematic review and meta-analysis of randomised controlled clinical trials. *Clin Obes* 2025; **15:** e70030.
48 Bischoff SC, Barazzoni R, Busetto L, et al. European Guideline on Obesity Care in Patients with Gastrointestinal and Liver Diseases—joint ESPEN/UEG guideline. *Clin Nutr* 2022; **41:** 2364–405.
49 Rondanelli M, Faliva MA, Gasparri C, et al. Current opinion on dietary advice in order to preserve fat-free mass during a low-calorie diet. *Nutrition* 2020; **72:** 110667.
50 Mehrtash F, Dushay J, Manson JE. Integrating diet and physical activity when prescribing GLP-1s—lifestyle factors remain crucial. *JAMA Intern Med* 2025; **185:** 1151–52.
51 Al-Najim W, Raposo A. BinMowyna MN, le Roux CW. Unintended consequences of obesity pharmacotherapy: a nutritional approach to ensuring better patient outcomes. *Nutrients* 2025; **17:** 1934.
52 Spreckley M, Ruggiero CF, Brown A. Nutrition strategies for next-generation incretin therapies: a systematic scoping review of the current evidence. *Obes Rev* 2026; **27:** e70079.
53 European Food Safety Authority Panel on Dietetic Products, Nutrition and Allergies. Scientific opinion on the essential composition of total diet replacements for weight control. *EFSA J* 2015; **13:** 3957.
54 Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. *J Am Diet Assoc* 2005; **105:** 775–89.
55 Hall KD, Sacks G, Chandramohan D, et al. Quantification of the effect of energy imbalance on bodyweight. *Lancet* 2011; **378:** 826–37.
56 Weijs PJM, Wolfe RR. Exploration of the protein requirement during weight loss in obese older adults. *Clin Nutr* 2016; **35:** 394–98.
57 Kokura Y, Ueshima J, Saino Y, Maeda K. Enhanced protein intake on maintaining muscle mass, strength, and physical function in adults with overweight/obesity: a systematic review and meta-analysis. *Clin Nutr ESPEN* 2024; **63:** 417–26.
58 Singer P, Blaser AR, Berger MM, et al. ESPEN guideline on clinical nutrition in the intensive care unit. *Clin Nutr* 2019; **38:** 48–79.

59 Stefanakis K, Kokkorakis M, Mantzoros CS. The impact of weight loss on fat-free mass, muscle, bone and hematopoiesis health: implications for emerging pharmacotherapies aiming at fat reduction and lean mass preservation. *Metabolism* 2024; **161:** 156057.

60 He L, Li Q, Yang Y, et al. Pharmacovigilance study of GLP-1 receptor agonists for metabolic and nutritional adverse events. *Front Pharmacol* 2024; **15:** 1416985.

61 Carlsen H, Pajari AM. Dietary fiber—a scoping review for Nordic Nutrition Recommendations 2023. *Food Nutr Res* 2023; **67:** 9979.

62 European Food Safety Authority Panel on Dietetic Products, Nutrition and Allergies. Scientific opinion on dietary reference values for carbohydrates and dietary fibre. *EFSA J* 2016; **8:** 1462.

63 van der Schoot A, Drysdale C, Whelan K, Dimidi E. The effect of fiber supplementation on chronic constipation in adults: an updated systematic review and meta-analysis of randomized controlled trials. *Am J Clin Nutr* 2022; **116:** 953–69.

64 Dimidi E, van der Schoot A, Barrett K, et al. British Dietetic Association Guidelines for the Dietary Management of Chronic Constipation in Adults. *J Hum Nutr Diet* 2025; **38:** e70133.

65 Markland AD, Palsson O, Goode PS, Burgio KL, Busby-Whitehead J, Whitehead WE. Association of low dietary intake of fiber and liquids with constipation: evidence from the National Health and Nutrition Examination Survey. *Am J Gastroenterol* 2013; **108:** 796–803.

66 Wang DC, Peng XF, Chen WX, Yu M. The association of moisture intake and constipation among US adults: evidence from NHANES 2005-2010. *BMC Public Health* 2025; **25:** 399.

67 European Food Safety Authority Panel on Dietetic Products, Nutrition and Allergies. Scientific opinion on dietary reference values for water. *EFSA J* 2016; **8:** 1459.

68 Cederholm T, Bosaeus I. Malnutrition in adults. *N Engl J Med* 2024; **391:** 155–65.

69 Allen LH. Micronutrients—assessment, requirements, deficiencies, and interventions. *N Engl J Med* 2025; **392:** 1006–16.

70 Dobbie LJ, Parretti HM, Fallows E, et al. Ten top tips for the management of GLP-1 receptor agonists in adults within primary care. *Obes Facts* 2025; **18:** 535–40.

71 Seier S, Stamp-Larsen K, Jensen SBK, Torekov SS, Gudbergsen H. Treat to target in weight management with semaglutide: real-world evidence from an eHealth clinic. *Diabetes Obes Metab* 2025; **27:** 6979–87.

72 Jalleh RJ, Marathe CS, Rayner CK, et al. Physiology and pharmacology of effects of GLP-1-based therapies on gastric, biliary and intestinal motility. *Endocrinology* 2024; **166:** 155.

73 Winzeler B, Sailer CO, Coynel D, et al. A randomized controlled trial of the GLP-1 receptor agonist dulaglutide in primary polydipsia. *J Clin Invest* 2021; **131:** e151800.

74 Lambert K, Neale E, Nichols L, et al. Interventions for improving adherence to dietary salt and fluid restrictions in people with chronic kidney disease (stage 4 and 5). *Cochrane Database Syst Rev* 2022; **10:** CD015181.

75 Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines. *J Am Coll Cardiol* 2022; **79:** e263–421.

76 Hobson RM, Maughan RJ. Hydration status and the diuretic action of a small dose of alcohol. *Alcohol Alcohol* 2010; **45:** 366–73.

77 Mehta RL, Burdmann EA, Cerdá J, et al. Recognition and management of acute kidney injury in the International Society of Nephrology 0by25 Global Snapshot: a multinational cross-sectional study. *Lancet* 2016; **387:** 2017–25.

78 Natale P, Green SC, Tunnicliffe DJ, Pellegrino G, Toyama T, Strippoli GFM. Glucagon-like peptide 1 (GLP-1) receptor agonists for people with chronic kidney disease and diabetes. *Cochrane Database Syst Rev* 2025; **2:** CD015849.

79 Dong S, Sun C. Can glucagon-like peptide-1 receptor agonists cause acute kidney injury? An analytical study based on post-marketing approval pharmacovigilance data. *Front Endocrinol* 2022; **13:** 1032199.

80 Fishkin A, Rozenberg A, Schechter M, et al. Kidney outcomes with glucagon-like peptide-1 receptor agonists versus other glucose-lowering agents in people with type 2 diabetes: a systematic review and meta-analysis of real-world data. *Diabetes Metab Res Rev* 2025; **41:** e70066.

81 Veronese N, Gianfredi V, Solmi M, et al. The impact of dietary fiber consumption on human health: an umbrella review of evidence from 17 155 277 individuals. *Clin Nutr* 2025; **51:** 325–33.

82 Zhang W, Fan M, Wang C, et al. Hair loss after metabolic and bariatric surgery: a systematic review and meta-analysis. *Obes Surg* 2021; **31:** 2649–59.

83 Desai DD, Sikora M, Nohria A, et al. GLP-1 agonists and hair loss: a call for further investigation. *Int J Dermatol* 2024; **63:** 1128–30.

84 Pacei F, Tesone A, Laudi N, et al. The relevance of thiamine evaluation in a practical setting. *Nutrients* 2020; **12:** 1–17.

85 Karimi Behnagh A, Eghbali M, Abdolmaleki F, Abbasi M, Mottaghi A. Pre- and post-surgical prevalence of thiamine deficiency in patients undergoing bariatric surgery: a systematic review and meta-analysis. *Obes Surg* 2024; **34:** 653–65.

86 Gras C, De Wit V, Oussedik N, et al. Semaglutide-induced Wernicke encephalopathy: a comprehensive analysis. *Eur J Clin Nutr* 2025; **79:** 1160–63.

87 Chiang CH, Jaroenlapnopparat A, Colak SC, et al. Glucagon-like peptide-1 receptor agonists and gastrointestinal adverse events: a systematic review and meta-analysis. *Gastroenterology* 2025; **169:** 1268–81.

88 Barazzoni R, Gortan Cappellari G. Double burden of malnutrition in persons with obesity. *Rev Endocr Metab Disord* 2020; **21:** 307–13.

89 Churuangsuk C, Griffiths D, Lean MEJ, Combet E. Impacts of carbohydrate-restricted diets on micronutrient intakes and status: a systematic review. *Obes Rev* 2019; **20:** 1132–47.

90 Carrillo-Álvarez E. Perspective: food and nutrition insecurity in Europe: challenges and opportunities for dietitians. *Adv Nutr* 2023; **14:** 995–1004.

91 ElSayed NA, McCoy RG, Aleppo G, et al. 13. Older adults: standards of care in diabetes—2025. *Diabetes Care* 2025; **48** (suppl 1)**:** S266–82.

92 Kanai R, Kinoshita S, Kanbe I, et al. Once-weekly semaglutide administered after laparoscopic sleeve gastrectomy: effects on body weight, glycemic control, and measured nutritional metrics in Japanese patients having both obesity and type 2 diabetes. *Obes Pillars* 2024; **9:** 100098.

93 Spreckley M, Ruggiero CF, Brown A. Bridging the nutrition guidance gap for GLP-1 receptor agonist therapy assisted weight loss: lessons from bariatric surgery. *Int J Obes* 2025; **2025:** 1–3.

94 Bal BS, Finelli FC, Shope TR, Koch TR. Nutritional deficiencies after bariatric surgery. *Nat Rev Endocrinol* 2012; **8:** 544–56.

95 Mechanick JI, Apovian C, Brethauer S, et al. Clinical practice guidelines for the perioperative nutrition, metabolic, and nonsurgical support of patients undergoing bariatric procedures—2019 update: cosponsored by American Association of Clinical Endocrinologists/American College of Endocrinology, the Obesity Society, American Society for Metabolic & Bariatric Surgery, Obesity Medicine Association, and American Society of Anesthesiologists. *Surg Obes Relat Dis* 2020; **16:** 175–247.

96 O'Kane M, Parretti HM, Pinkney J, et al. British Obesity and Metabolic Surgery Society Guidelines on Perioperative and Postoperative Biochemical Monitoring and Micronutrient Replacement for Patients Undergoing Bariatric Surgery—2020 update. *Obes Rev* 2020; **21:** e13087.

97 O'Brien PE, MacDonald L, Anderson M, Brennan L, Brown WA. Long-term outcomes after bariatric surgery: fifteen-year follow-up of adjustable gastric banding and a systematic review of the bariatric surgical literature. *Ann Surg* 2013; **257:** 87–94.

98 Cohen RV, Busetto L, Levinson R, et al. International Consensus Position Statement on the Role of Obesity Management Medications in the Context of Metabolic Bariatric Surgery: expert guideline by the International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO). *Br J Surg* 2024; **111:** znae283.

99 Skipper A, Coltman A, Tomesko J, et al. Position of the Academy of Nutrition and Dietetics: malnutrition (undernutrition) screening tools for all adults. *J Acad Nutr Diet* 2020; **120:** 709–13.

100 Petra G, Kritsotakis EI, Gouvas N, et al. Multicentre prospective study on the diagnostic and prognostic validity of malnutrition assessment tools in surgery. *Br J Surg* 2025; **112:** znaf013.

101 van Vliet IMY, Gomes-Neto AW, de Jong MFC, Bakker SJL, Jager-Wittenaar H, Navis GJ. Malnutrition screening on hospital admission: impact of overweight and obesity on comparative performance of MUST and PG-SGA SF. *Eur J Clin Nutr* 2021; **75:** 1398–406.

102 De Groot LM, Lee G, Ackerie A, van der Meij BS. Malnutrition screening and assessment in the cancer care ambulatory setting: mortality predictability and validity of the patient-generated subjective global assessment short form (PG-SGA SF) and the GLIM criteria. *Nutrients* 2020; **12:** 1–13.

103 Jensen GL, Cederholm T, Correia MITD, et al. GLIM consensus approach to diagnosis of malnutrition: a 5-year update. *JPEN J Parenter Enteral Nutr* 2025; **49:** 414–27.

104 Cederholm T, Jensen GL, Correia MITD, et al. GLIM criteria for the diagnosis of malnutrition—a consensus report from the global clinical nutrition community. *Clin Nutr* 2019; **38:** 1–9.

105 Cristancho C, Mogensen KM, Robinson MK. Malnutrition in patients with obesity: an overview perspective. *Nutr Clin Pract* 2024; **39:** 1300–16.

106 Compher C, Cederholm T, Correia MITD, et al. Guidance for assessment of the muscle mass phenotypic criterion for the Global Leadership Initiative on Malnutrition diagnosis of malnutrition. *JPEN J Parenter Enteral Nutr* 2022; **46:** 1232–42.

107 Uhlmann K, Schaller F, Lehmann U. Current practice of assessing and monitoring muscle strength, muscle mass and muscle function during nutritional care by dietitians in Switzerland—an online survey. *Nutrients* 2022; **14:** 1741.

108 Espinosa-Salas S, Gonzalez-Arias M. Nutrition: micronutrient intake, imbalances, and interventions. StatPearls, 2023.

109 Astrup A, Bügel S. Overfed but undernourished: recognizing nutritional inadequacies/deficiencies in patients with overweight or obesity. *Int J Obes* 2019; **43:** 219–32.

110 Jun S, Cowan AE, Bhadra A, et al. Older adults with obesity have higher risks of some micronutrient inadequacies and lower overall dietary quality compared to peers with a healthy weight, National Health and Nutrition Examination Surveys (NHANES), 2011-2014. *Public Health Nutr* 2020; **23:** 2268–79.

111 Baldwin C, Smith R, Gibbs M, Weekes CE, Emery PW. Quality of the evidence supporting the role of oral nutritional supplements in the management of malnutrition: an overview of systematic reviews and meta-analyses. *Adv Nutr* 2021; **12:** 503–22.

112 Busetto L, Dicker D, Azran C, et al. Practical Recommendations of the Obesity Management Task Force of the European Association for the Study of Obesity for the Post-Bariatric Surgery Medical Management. *Obes Facts* 2017; **10:** 597–632.

113 Drummen M, Tischmann L, Gatta-Cherifi B, et al. High compared with moderate protein intake reduces adaptive thermogenesis and induces a negative energy balance during long-term weight-loss maintenance in participants with prediabetes in the postobese state: a PREVIEW study. *J Nutr* 2020; **150:** 458–63.

114 Neeland IJ, Linge J, Birkenfeld AL. Changes in lean body mass with glucagon-like peptide-1-based therapies and mitigation strategies. *Diabetes Obes Metab* 2024; **26** (suppl 4)**:** 16–27.

115 Melville H, Lister NB, Libesman S, et al. The prevalence of eating disorders and disordered eating in adults seeking obesity treatment: a systematic review with meta-analyses. *Int J Eat Disord* 2025; **58:** 1644–61.

116 Nagata JM, Garber AK, Tabler JL, Murray SB, Bibbins-Domingo K. Prevalence and correlates of disordered eating behaviors among young adults with overweight or obesity. *J Gen Intern Med* 2018; **33:** 1337–43.

117 Duncan AE, Ziobrowski HN, Nicol G. The prevalence of past 12-month and lifetime DSM-IV eating disorders by BMI category in US men and women. *Eur Eat Disord Rev* 2017; **25:** 165–71.

118 Aoun L, Almardini S, Saliba F, et al. GLP-1 receptor agonists: a novel pharmacotherapy for binge eating (binge eating disorder and bulimia nervosa)? A systematic review. *J Clin Transl Endocrinol* 2024; **35:** 100333.

119 Radkhah H, Rahimipour Anaraki S, Parhizkar Roudsari P, et al. The impact of glucagon-like peptide-1 (GLP-1) agonists in the treatment of eating disorders: a systematic review and meta-analysis. *Eat Weight Disord* 2025; **30:** 10.

120 Bartel S, McElroy SL, Levangie D, Keshen A. Use of glucagon-like peptide-1 receptor agonists in eating disorder populations. *Int J Eat Disord* 2024; **57:** 286–93.

121 House ET, Lister NB, Seidler AL, et al. Identifying eating disorders in adolescents and adults with overweight or obesity: a systematic review of screening questionnaires. *Int J Eat Disord* 2022; **55:** 1171–93.

122 Chen X, Zhao P, Wang W, Guo L, Pan Q. The antidepressant effects of GLP-1 receptor agonists: a systematic review and meta-analysis. *Am J Geriatr Psychiatry* 2024; **32:** 117–27.

123 Breit S, Hubl D. The effect of GLP-1RAs on mental health and psychotropics-induced metabolic disorders: a systematic review. *Psychoneuroendocrinology* 2025; **176:** 107415.

124 Plenn E, Amin D, Henry J, Leavitt G, Walker J, Soleymani T. A qualitative analysis of patient experiences using semaglutide 2·4 mg for weight loss. *Obes Sci Pract* 2025; **11:** e70085.

125 Brown A, Flint SW. 'My words would have more weight': exploring weight stigma in UK dietetic practice and dietitian's lived experiences of weight stigma. *J Hum Nutr Diet* 2024; **37:** 1143–58.

126 Lawrence BJ, Kerr D, Pollard CM, et al. Weight bias among health care professionals: a systematic review and meta-analysis. *Obesity* 2021; **29:** 1802–12.

127 Post SM, Stock ML, Persky S. Comparing the impact of GLP-1 agonists *vs* lifestyle interventions and weight controllability information on stigma and weight-related cognitions. *Int J Behav Med* 2025; **32:** 528–40.

128 Naveed M, Perez C, Ahmad E, Russell L, Lees Z, Maybury C. GLP-1 medication and weight loss: Barriers and motivators among 1659 participants managed in a virtual setting. *Diabetes Obes Metab* 2025; **27:** 3780–88.

129 Jung FUCE, Luck-Sikorski C, Wiemers N, Riedel-Heller SG. Dietitians and nutritionists: stigma in the context of obesity. a systematic review. *PLoS One* 2015; **10:** e0140276.

130 Hanson P, Shuttlewood E, Halder L, et al. Application of mindfulness in a tier 3 obesity service improves eating behavior and facilitates successful weight loss. *J Clin Endocrinol Metab* 2019; **104:** 793–800.

131 Kudlek L, Eustachio Colombo P, Mueller J, et al. Individual participant data meta-analysis of eating behaviour traits as effect modifiers in acceptance and commitment therapy-based weight management interventions. *Int J Obes* 2025; **49:** 1142–52.

132 Post SM, Persky S. The effect of GLP-1 receptor agonist use on negative evaluations of women with higher and lower body weight. *Int J Obes* 2024; **48:** 1019–26.

133 Despain D, Hoffman BL. Optimizing nutrition, diet, and lifestyle communication in GLP-1 medication therapy for weight management: a qualitative research study with registered dietitians. *Obes Pillars* 2024; **12:** 100143.

134 Osborne D, Abdelgadir E. Mental health outcomes in obesity interventions with GLP-1 receptor agonists: is it similar to other obesity interventions? A narrative review with systematic evidence synthesis. *Int J Obes* 2026; published online Jan 5. https://doi.org/10.1038/s41366-025-02002-1.

135 Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire: a new screening tool for eating disorders. *West J Med* 2000; **172:** 164–65.

136 Pierret ACS, Mizuno Y, Saunders P, et al. Glucagon-like peptide 1 receptor agonists and mental health: a systematic review and meta-analysis. *JAMA Psychiatry* 2025; **82:** 643–53.

137 West S, Scragg J, Aveyard P, et al. Weight regain after cessation of medication for weight management: systematic review and meta-analysis. *BMJ* 2026; **392:** e085304.

138 Kenkre JS, Gesell S, Keller A, Milani RM, Scholtz S, Barley EA. Alcohol misuse post metabolic and bariatric surgery: a systematic review of longer-term studies with focus on new onset alcohol use disorder and differences between surgery types. *Curr Obes Rep* 2024; **13:** 596–616.

139 Hendershot CS, Bremmer MP, Paladino MB, et al. Once-weekly semaglutide in adults with alcohol use disorder: a randomized clinical trial. *JAMA Psychiatry* 2025; **82:** 395–405.

140 Klausen MK, Jensen ME, Møller M, et al. Exenatide once weekly for alcohol use disorder investigated in a randomized, placebo-controlled clinical trial. *JCI Insight* 2022; **7:** e159863.

141 Arillotta D, Floresta G, Papanti Pelletier GD, et al. Exploring the potential impact of GLP-1 receptor agonists on substance use, compulsive behavior, and libido: insights from social media using a mixed-methods approach. *Brain Sci* 2024; **14:** 617.

142 Schoretsanitis G, Weiler S, Barbui C, Raschi E, Gastaldon C. Disproportionality analysis from World Health Organization data on semaglutide, liraglutide, and suicidality. *JAMA Netw Open* 2024; **7:** e2423385.

143 McIntyre RS, Mansur RB, Rosenblat JD, et al. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) and suicidality: a replication study using reports to the World Health Organization pharmacovigilance database (VigiBase). *J Affect Disord* 2025; **369:** 922–27.

144 Tang H, Lu Y, Donahoo WT, et al. Glucagon-like peptide-1 receptor agonists and risk for suicidal ideation and behaviors in US older adults with type 2 diabetes : a target trial emulation study. *Ann Intern Med* 2024; **177:** 1004–15.

145 van Reekum EA, Darcy M, Drage J, et al. Psychiatric and cognitive functioning after metabolic and bariatric surgery: a systematic review and meta-analysis. *Obes Rev* 2025; **26:** e13968.

146 Ganeshalingam AA, Uhrenholt N, Arnfred S, et al. Semaglutide treatment of antipsychotic-treated patients with schizophrenia, prediabetes, and obesity: the HISTORI Randomized Clinical Trial. *JAMA Psychiatry* 2025; **82:** 1065–74.

147 Herman BK, Deal LS, DiBenedetti DB, Nelson L, Fehnel SE, Brown TM. Development of the 7-Item Binge-Eating Disorder Screener (BEDS-7). *Prim Care Companion CNS Disord* 2016; **18**: 15m01896

148 De R, Smith ECC, Navagnanavel J, et al. The impact of weight gain on antipsychotic nonadherence or discontinuation: a systematic review and meta-analysis. *Acta Psychiatr Scand* 2025; **151:** 109–26.

149 Treasure J, Duarte TA, Schmidt U. Eating disorders. *Lancet* 2020; **395:** 899–911.

150 Castellini G, Graffigna G. "Food is more than just a source of nutrients": a qualitative phenomenological study on food involvement. *Appetite* 2022; **178:** 106179.

151 Linge J, Petersson M, Forsgren MF, Sanyal AJ, Dahlqvist Leinhard O. Adverse muscle composition predicts all-cause mortality in the UK Biobank imaging study. *J Cachexia Sarcopenia Muscle* 2021; **12:** 1513–26.

152 Landré B, Czernichow S, Goldberg M, Zins M, Ankri J, Herr M. Association between life-course obesity and frailty in older adults: findings in the GAZEL cohort. *Obesity* 2020; **28:** 388–96.

153 Caturano A, Amaro A, Berra CC, Conte C. Sarcopenic obesity and weight loss-induced muscle mass loss. *Curr Opin Clin Nutr Metab Care* 2025; **28:** 339–50.

154 Beavers KM, Cortes TM, Foy CM, et al. GLP1Ra-based therapies and DXA-acquired musculoskeletal health outcomes: a focused meta-analysis of placebo-controlled trials. *Obesity* 2025; **33:** 225–37.

155 Karakasis P, Patoulias D, Fragakis N, Mantzoros CS. Effect of glucagon-like peptide-1 receptor agonists and co-agonists on body composition: systematic review and network meta-analysis. *Metabolism* 2025; **164:** 156113.

156 Dash S. Opportunities to optimize lifestyle interventions in combination with glucagon-like peptide-1-based therapy. *Diabetes Obes Metab* 2024; **26** (suppl 4)**:** 3–15.

157 Sanchis-Gomar F, Neeland IJ, Lavie CJ. Balancing weight and muscle loss in GLP1 receptor agonist therapy. *Nat Rev Endocrinol* 2025; **21:** 584–85.

158 Nevill AM, Lang JJ, Niemz M, Tomkinson GR. How should youth handgrip strength be normalized? New insights using 3-D allometry with "generalizable" norm-referenced values, data from NHANES. *Sports Med* 2025; **55:** 2303–12.

159 Pouget M, Pinel A, Miolanne M, et al. Improving the functional detection of sarcopenic obesity: prevalence and handgrip scoring in the OBESAR cohort. *Obesity* 2024; **32:** 2237–45.

160 Abdalla PP, Bohn L, Dos Santos AP, et al. Adjusting grip strength to body size: analyses from 6 countries. *J Am Med Dir Assoc* 2022; **23:** 903.e13–21.

161 Locatelli JC, Costa JG, Haynes A, et al. Incretin-based weight loss pharmacotherapy: can resistance exercise optimize changes in body composition? *Diabetes Care* 2024; **47:** 1718–30.

162 Neeland IJ, Linge J, Birkenfeld AL. Changes in lean body mass with glucagon-like peptide-1-based therapies and mitigation strategies. *Diabetes Obes Metab* 2024; **26** (suppl 4)**:** 16–27.

163 Holmes CJ, Racette SB. The utility of body composition assessment in nutrition and clinical practice: an overview of current methodology. *Nutrients* 2021; **13:** 2493.

164 Silveira EA, Castro MCR, Rezende ATO, et al. Body composition assessment in individuals with class II/III obesity: a narrative review. *BMC Nutr* 2024; **10:** 142.

165 Prado CM, Landi F, Chew STH, et al. Advances in muscle health and nutrition: a toolkit for healthcare professionals. *Clin Nutr* 2022; **41:** 2244–63.

166 Norman K, Stobäus N, Gonzalez MC, Schulzke JD, Pirlich M. Hand grip strength: outcome predictor and marker of nutritional status. *Clin Nutr* 2011; **30:** 135–42.

167 Busetto L, Dicker D, Frühbeck G, et al. A new framework for the diagnosis, staging and management of obesity in adults. *Nat Med* 2024; **30:** 2395–99.

168 Wu Q, Ling Y, Yuan G, et al. Association between lipid accumulation products and relative handgrip strength: a large population-based study. *Sci Rep* 2025; **15:** 11562.

169 Boyle LD, Akbas F, Yazıcı D, McGowan BM, Yumuk V. Pharmacotherapy for older people with obesity. *Eur J Intern Med* 2024; **130:** 33–37.

170 Alsaqaaby MS, Cooney S, le Roux CW, Pournaras DJ. Sex, race, and BMI in clinical trials of medications for obesity over the past three decades: a systematic review. *Lancet Diabetes Endocrinol* 2024; **12:** 414–21.

171 Rossi AP, Rubele S, Calugi S, et al. Weight cycling as a risk factor for low muscle mass and strength in a population of males and females with obesity. *Obesity* 2019; **27:** 1068–75.

172 Turicchi J, O'Driscoll R, Finlayson G, Beaulieu K, Deighton K, Stubbs RJ. Associations between the rate, amount, and composition of weight loss as predictors of spontaneous weight regain in adults achieving clinically significant weight loss: a systematic review and meta-regression. *Obes Rev* 2019; **20:** 935–46.

173 Beavers KM, Lyles MF, Davis CC, Wang X, Beavers DP, Nicklas BJ. Is lost lean mass from intentional weight loss recovered during weight regain in postmenopausal women? *Am J Clin Nutr* 2011; **94:** 767–74.

174 Oppert JM, Ciangura C, Bellicha A. Health-enhancing physical activity in obesity management: the need to (seriously) go beyond weight loss. *Int J Obes* 2025; **49:** 211–13.

175 Bellicha A, van Baak MA, Battista F, et al. Effect of exercise training on weight loss, body composition changes, and weight maintenance in adults with overweight or obesity: an overview of 12 systematic reviews and 149 studies. *Obes Rev* 2021; **22** (suppl 4)**:** e13256.

176 Piercy KL, Troiano RP, Ballard RM, et al. The Physical Activity Guidelines for Americans. *JAMA* 2018; **320:** 2020–28.

177 Cuthbertson DJ, Dobbie LJ. Role of exercise and physical activity in promoting weight loss and weight loss maintenance. In: Kopelman PG, Caterson ID, Dietz WH, Armstrong S, Sweeting AN, Wilding JPH, eds. Clinical obesity in adults and children, 4th edn. Wiley, 2022: 262–78.

178 Wadden TA, Bailey TS, Billings LK, et al. Effect of subcutaneous semaglutide *vs* placebo as an adjunct to intensive behavioral therapy on body weight in adults with overweight or obesity: the STEP 3 randomized clinical trial. *JAMA* 2021; **325:** 1403–13.

179 Oppert JM, Bellicha A, van Baak MA, et al. Exercise training in the management of overweight and obesity in adults: synthesis of the evidence and recommendations from the European Association for the Study of Obesity Physical Activity Working Group. *Obes Rev* 2021; **22** (suppl 4)**:** e13273.

180 Oppert JM, Ciangura C, Bellicha A. Physical activity and exercise for weight loss and maintenance in people living with obesity. *Rev Endocr Metab Disord* 2023; **24:** 937–49.

181 Bushman BA. Developing the P (for progression) in a FITT-VP exercise prescription. *ACSM Health Fit J* 2018; **22:** 6–9.

182 Chang YH, Chou YC, Chang YC, Tan KH, Wu MH. The effects of high-intensity power training versus traditional resistance training on exercise performance. *Int J Environ Res Public Health* 2022; **19:** 9400.

183 Fassio A, Idolazzi L, Rossini M, et al. The obesity paradox and osteoporosis. *Eat Weight Disord* 2018; **23:** 293–302.

184 Gandham A, Zengin A, Bonham MP, et al. Incidence and predictors of fractures in older adults with and without obesity defined by body mass index versus body fat percentage. *Bone* 2020; **140:** 115546.

185 Papageorgiou M, Kerschan-Schindl K, Sathyapalan T, Pietschmann P. Is weight loss harmful for skeletal health in obese older adults? *Gerontology* 2020; **66:** 2–14.

186 Johnson KC, Bray GA, Cheskin LJ, et al. The effect of intentional weight loss on fracture risk in persons with diabetes: results from the Look AHEAD randomized clinical trial. *J Bone Miner Res* 2017; **32:** 2278–87.

187 Paccou J, Compston JE. Bone health in adults with obesity before and after interventions to promote weight loss. *Lancet Diabetes Endocrinol* 2024; **12:** 748–60.
188 Paccou J, Tsourdi E, Meier C, et al. Bariatric surgery and skeletal health: a narrative review and position statement for management by the European Calcified Tissue Society (ECTS). *Bone* 2022; **154:** 116236.
189 Hansen MS, Wölfel EM, Jeromdesella S, et al. Once-weekly semaglutide versus placebo in adults with increased fracture risk: a randomised, double-blinded, two-centre, phase 2 trial. *EClinicalMedicine* 2024; **72:** 102624.
190 Karam L, Mabilleau G, Paccou J. Effects of glucagon-like peptide-1 receptor agonists on bone health in people living with obesity. *Osteoporos Int* 2025; **36:** 2115–26.
191 Nicholson WK, Silverstein M, Wong JB, et al. Screening for osteoporosis to prevent fractures: US Preventive Services Task Force Recommendation Statement. *JAMA* 2025; **333:** 498–508.
192 Palermo A, Tsourdi E, Yavropoulou MP, et al. The effects of obesity and weight loss interventions on bone health: a narrative review. *Curr Diab Rep* 2025; **25:** 52.
193 Waterhouse M, Ebeling PR, McLeod DSA, et al. The effect of monthly vitamin D supplementation on fractures: a tertiary outcome from the population-based, double-blind, randomised, placebo-controlled D-Health trial. *Lancet Diabetes Endocrinol* 2023; **11:** 324–32.
194 Chevalley T, Brandi ML, Cashman KD, et al. Role of vitamin D supplementation in the management of musculoskeletal diseases: update from an European Society of Clinical and Economical Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (ESCEO) working group. *Aging Clin Exp Res* 2022; **34:** 2603–23.
195 Harvey NC, Biver E, Kaufman JM, et al. The role of calcium supplementation in healthy musculoskeletal ageing: an expert consensus meeting of the European Society for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (ESCEO) and the International Foundation for Osteoporosis (IOF). *Osteoporos Int* 2017; **28:** 447–62.
196 Paccou J, Gagnon C, Yu EW, Rosen CJ. Effects of weight-loss interventions on bone health in people living with obesity. *J Bone Miner Res* 2025; **40:** 1319–31.
197 Kumar S, Smith C, Clifton-Bligh RJ, Beck BR, Girgis CM. Exercise for postmenopausal bone health—can we raise the bar? *Curr Osteoporos Rep* 2025; **23:** 20.
198 Jensen SBK, Sørensen V, Sandsdal RM, et al. Bone health after exercise alone, GLP-1 receptor agonist treatment, or combination treatment: a secondary analysis of a randomized clinical trial. *JAMA Netw Open* 2024; **7:** e2416775.
199 Coulman KD, Margelyte R, Jones T, et al. Access to publicly funded weight management services in England using routine data from primary and secondary care (2007–2020): an observational cohort study. *PLoS Med* 2023; **20:** e1004282.
200 Mekonnen A, Vasilevski V, Chapman A, et al. Barriers and enablers to effective weight management for people living with overweight and obesity: a rapid scoping review. *Obes Rev* 2025; **26:** e13858.
201 Kendrick KN, Bode Padron KJ, Bomani NZ, et al. Equity in obesity review. *Endocrinol Metab Clin North Am* 2023; **52:** 617–27.
202 Karagiannis T, Bekiari E, Tsapas A. Socioeconomic aspects of incretin-based therapy. *Diabetologia* 2023; **66:** 1859–68.
203 Waldrop SW, Johnson VR, Stanford FC. Inequalities in the provision of GLP-1 receptor agonists for the treatment of obesity. *Nat Med* 2024; **30:** 22–25.
204 Food and Agriculture Organization of the UN. Changing policy concepts of food security. Food and Agriculture Organization of the United Nations. June 30, 2006. https://reliefweb.int/report/world/policy-brief-food-security-issue-2-june-2006 (accessed Nov 20, 2025).
205 Johnstone A, Lonnie M, FIO-Food project team. The cost-of-living crisis is feeding the paradox of obesity and food insecurities in the UK. *Obesity* 2023; **31:** 1461–62.
206 Stone RA, Brown A, Douglas F, et al. The impact of the cost of living crisis and food insecurity on food purchasing behaviours and food preparation practices in people living with obesity. *Appetite* 2024; **196:** 107255.
207 Craig H, Sharib JR, Ridberg R, et al. Development, implementation, and validation of 2-item nutrition security screener. *Curr Dev Nutr* 2024; **8:** 103151.
208 Czernichow S, Rassy N, Carette C, Shoung N, Hu FB, Rives-Lange C. Nutritional and functional outcomes in trials of nutrient-stimulated hormone-based therapy—a systematic mapping review. *Obes Rev* 2025; **26:** e13890.
209 Tinsley GM, Heymsfield SB. Fundamental body composition principles provide context for fat-free and skeletal muscle loss with GLP-1 RA treatments. *J Endocr Soc* 2024; **8:** bvae164.

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