RYCHLICA, Kacper, GAWIN, Konrad, ZAWIŚLAK, Wiktoria, IGNASIAK, Anita, CISOWSKI, Michał, DĄBROWSKA, Maria, CHOLEWIŃSKA-RYCHLICA, Jolanta, MADURA, Paulina, and MROZIK-GAŁECKA, Daria. Dietary Modulation and Clinical Outcomes During GLP-1 Receptor Agonist Therapy. A Literature Review. Quality in Sport. 2026;49:67649. eISSN 2450-3118.
https://doi.org/10.12775/QS.2026.49.67649
https://apcz.umk.pl/QS/article/view/67649

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 l.p. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 21.12.2025. Revised: 29.12.2025. Accepted: 16.01.2026. Published: 18.01.2026.

Dietary Modulation and Clinical Outcomes During GLP-1 Receptor Agonist Therapy.

A Literature Review

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ABSTRACT

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) represent a major advancement in the treatment of obesity, type 2 diabetes mellitus, and cardiometabolic disease. Large randomized controlled trials have demonstrated that semaglutide and tirzepatide induce substantial and sustained reductions in body weight, improve glycaemic control, and reduce cardiovascular risk. As their clinical use expands, increasing attention has been directed toward the interaction between pharmacological appetite modulation and dietary behavior. Although GLP-1 RAs

exert powerful effects on hunger, satiety, and gastric motility independent of dietary intervention, nutritional intake, meal structure, and food composition may influence treatment tolerability, nutrient adequacy, body composition, and long-term maintenance of weight loss. This narrative review synthesizes evidence from randomized trials, pharmacokinetic analyses, mechanistic reviews, and real-world studies to examine the physiological and clinical relevance of diet during GLP-1 receptor agonist therapy. Particular emphasis is placed on gastrointestinal tolerability, nutrient adequacy, preservation of lean body mass, pharmacokinetic constraints of oral formulations, cardiometabolic implications, and the challenges of long-term weight maintenance following treatment discontinuation.
Keywords: GLP-1 receptor agonists, obesity management, clinical nutrition, appetite regulation, metabolic health, weight loss therapy

Introduction

The introduction of glucagon-like peptide-1 receptor agonists has fundamentally altered the therapeutic landscape of obesity and type 2 diabetes mellitus. Semaglutide and tirzepatide, evaluated across multiple large randomized controlled trials, have demonstrated degrees of weight reduction that exceed those achieved with most prior pharmacotherapies and approach outcomes previously attainable primarily through bariatric surgery [1–6]. These reductions are accompanied by clinically meaningful improvements in glycaemic control, lipid metabolism, blood pressure, and markers of systemic inflammation. Cardiovascular outcome trials have further demonstrated reductions in major adverse cardiovascular events, supporting the role of GLP-1 receptor agonists as disease-modifying therapies in high-risk populations [7,23].

Despite their potent pharmacological effects, GLP-1 receptor agonists do not operate in isolation from behavioral and environmental determinants of energy balance. By directly modulating appetite, satiety, gastric emptying, and reward-related eating behavior, these agents profoundly alter patients' physiological responses to food [21]. Patients receiving GLP-1 therapy commonly report early satiety, diminished hunger, reduced preference for energy-dense foods, and altered meal patterns. While these changes facilitate caloric reduction and weight loss, they also introduce new clinical challenges related to dietary adequacy, gastrointestinal

tolerability, preservation of lean body mass, and sustainability of weight reduction once pharmacological treatment is discontinued.

Most GLP-1 clinical trials include general lifestyle advice encouraging reduced caloric intake and increased physical activity, yet they do not experimentally compare dietary patterns or macronutrient compositions. Consequently, evidence guiding dietary recommendations during GLP-1 therapy is indirect and must be inferred from mechanistic studies, pharmacokinetic data, observational analyses, and clinical experience. Given the rapid expansion of GLP-1 use in routine practice, a comprehensive synthesis of available evidence addressing diet–drug interactions is needed to inform clinical decision-making and identify priorities for future research.

Mechanistic Foundations Linking GLP-1 Physiology and Dietary Intake

The physiological actions of GLP-1 integrate gastrointestinal nutrient sensing with pancreatic endocrine responses and central nervous system regulation of appetite. Endogenous GLP-1 is secreted from enteroendocrine L-cells in response to nutrient exposure in the distal intestine, particularly carbohydrates and lipids, and enhances glucose-dependent insulin secretion while suppressing glucagon release [21]. Pharmacological GLP-1 receptor agonists amplify these effects by providing sustained receptor activation resistant to enzymatic degradation.

Delayed gastric emptying represents one of the most clinically relevant mechanisms linking GLP-1 therapy to dietary behavior. By prolonging gastric retention of food, GLP-1 receptor agonists increase sensations of fullness and reduce postprandial glycaemic excursions. However, this same mechanism predisposes patients to nausea, bloating, and abdominal discomfort when meal volume is large or dietary fat content is high. Dietary fat independently slows gastric emptying, and when combined with pharmacological inhibition of gastric motility, this effect becomes additive [10,12,13,15,16]. Clinical studies consistently demonstrate that high-fat meals exacerbate gastrointestinal symptoms during GLP-1 therapy, particularly during treatment initiation and dose escalation.

GLP-1 receptor agonists also modulate central reward pathways involved in food motivation. Reduced dopaminergic signaling diminishes the rewarding value of highly palatable foods, leading to spontaneous reductions in snacking and consumption of energy-dense items [14,17,18]. While this effect supports weight reduction, it may also reduce dietary diversity if appetite suppression is pronounced. Patients may consume fewer meals or select foods that are easier to tolerate in small quantities, which may inadvertently reduce intake of protein and micronutrients. Thus, appetite suppression alone does not guarantee nutritional adequacy.

Pharmacokinetic interactions further underscore the importance of diet during GLP-1 therapy. Oral semaglutide relies on an absorption enhancer to facilitate gastric uptake, and the presence of food markedly reduces its bioavailability. Controlled pharmacokinetic studies demonstrate that food ingestion during the absorption window substantially decreases systemic exposure, necessitating strict fasting administration protocols [11]. In this context, dietary timing becomes a direct determinant of therapeutic efficacy rather than merely a tolerability consideration.

Clinical Evidence Relevant to Dietary Interaction During GLP-1 Receptor Agonist Therapy

Randomized controlled trials evaluating GLP-1 receptor agonists consistently demonstrate substantial reductions in body weight that are primarily mediated through decreases in energy intake. Trials investigating semaglutide across diverse populations with obesity show that reductions in caloric intake closely parallel pharmacologically induced satiety and diminished hunger, with participants reporting earlier meal termination and reduced desire for energy-dense foods [1–4]. Although these trials incorporated general lifestyle counseling, they did not implement structured dietary interventions, indicating that observed changes in eating behavior were predominantly drug-driven rather than diet-prescribed. These findings provide indirect but clinically meaningful insight into how GLP-1–mediated appetite regulation interacts with habitual dietary patterns.

Trials examining tirzepatide demonstrate even greater reductions in energy intake and body weight compared with GLP-1 monotherapy [5]. Dual activation of GIP and GLP-1 receptors appears to exert synergistic effects on appetite-regulating neural circuits and gastrointestinal signaling, producing profound anorectic responses. While this pharmacological potency enhances weight reduction, it simultaneously raises concerns regarding nutrient adequacy, particularly in relation to protein intake and micronutrient density. Participants in tirzepatide trials frequently report marked suppression of hunger and reduced meal frequency, suggesting that spontaneous eating patterns may not always align with nutritional requirements.

Emerging evidence from studies of multi-agonist incretin therapies suggests that appetite suppression may intensify further with newer pharmacological formulations [6,8,9]. These agents act on multiple hormonal pathways involved in energy balance, leading to unprecedented degrees of weight loss. Although such effects represent therapeutic progress, they amplify the potential risk of excessive dietary restriction if nutritional intake is not actively monitored. As pharmacological efficacy increases, the margin for nutritional inadequacy may narrow, particularly in populations with pre-existing vulnerabilities such as older adults or individuals with low baseline muscle mass.

Gastrointestinal adverse events remain among the most frequently reported side effects of GLP-1 receptor agonist therapy and represent a major determinant of treatment adherence. Clinical trials and observational studies consistently demonstrate that nausea, early satiety, and abdominal discomfort are more pronounced in patients consuming large meals or meals with high fat content [10,12,13,15,16]. These findings align with the known physiological effects of GLP-1 on gastric motility and provide a mechanistic basis for dietary modulation of tolerability. Smaller meal volumes and moderation of dietary fat appear to reduce symptom burden and improve persistence with therapy, although randomized trials specifically evaluating these strategies are lacking.

Studies examining changes in diet quality during GLP-1 therapy suggest that patients often reduce consumption of highly processed foods, refined carbohydrates, and saturated fats, reflecting alterations in food preference mediated by central appetite pathways [14,17,18]. However, these spontaneous dietary shifts do not uniformly ensure adequate intake of essential nutrients. Reduced appetite may lead patients to prioritize foods that are easier to consume in small quantities, potentially limiting intake of protein, fiber, and micronutrients. Observational analyses indicate that such patterns may contribute to disproportionate losses of lean mass during rapid weight reduction, particularly in the absence of resistance exercise [14,19,20].

Weight-regain studies provide important insight into the long-term implications of diet–drug interactions. Discontinuation of GLP-1 receptor agonist therapy is frequently associated with re-emergence of hunger, increased caloric intake, and partial or complete weight regain [19,20]. These findings underscore the necessity of establishing sustainable dietary patterns during active treatment rather than relying exclusively on pharmacological appetite suppression. Individuals who develop structured eating habits and maintain nutritional adequacy during therapy appear more likely to preserve weight loss following discontinuation.

Cardiovascular outcome trials further contextualize the role of diet during GLP-1 therapy. GLP-1 receptor agonists reduce major adverse cardiovascular events through mechanisms that extend beyond weight loss, including improvements in lipid metabolism, endothelial function, and inflammatory signaling [7,23]. Diet independently influences these pathways, and observational evidence suggests that alignment with cardioprotective dietary patterns may augment pharmacological benefits. Although direct comparative evidence is lacking, mechanistic plausibility supports integrating dietary strategies known to reduce cardiovascular risk alongside GLP-1 therapy.

Dietary Modulation of Gastrointestinal Tolerability

Gastrointestinal tolerability represents one of the most clinically relevant interfaces between dietary behavior and GLP-1 receptor agonist therapy. Nausea, early satiety, abdominal fullness, and alterations in bowel habits are frequently reported during treatment initiation and dose escalation, reflecting predictable physiological consequences of delayed gastric emptying and central satiety signaling [10,12,13,15,16]. Although these adverse events are often transient, their severity varies considerably among individuals and may lead to dose reduction or premature discontinuation of therapy if not adequately managed.

Clinical and observational evidence consistently indicates that meal size plays a critical role in symptom expression. Large meals increase gastric distension in the context of pharmacologically delayed gastric emptying, intensifying sensations of fullness and nausea. Patients who habitually consume large portions or engage in rapid eating are therefore more susceptible to intolerable gastrointestinal symptoms. Conversely, smaller meal volumes allow gastric accommodation to occur within the altered physiological constraints imposed by GLP-1 receptor activation, reducing symptom burden and improving treatment persistence.

Dietary fat content further modulates gastrointestinal tolerability. Fat digestion requires prolonged gastric processing and stimulates hormonal pathways that independently slow gastric emptying. When combined with GLP-1–induced inhibition of gastric motility, high-fat meals exert an additive effect that prolongs gastric retention and exacerbates nausea and bloating. Multiple studies report higher rates of gastrointestinal adverse events following consumption of fatty or greasy foods during GLP-1 therapy, particularly during the early phases of treatment [12,13,16]. Many patients develop spontaneous aversions to such foods, which may represent an adaptive response to mitigate discomfort. While this aversion may facilitate adherence to moderate-fat dietary patterns, it does not ensure overall nutritional adequacy and may reduce dietary diversity if not addressed through structured counseling.

Eating pace and hydration status also influence tolerability. Rapid ingestion limits the stomach’s ability to accommodate delayed emptying, whereas slow, deliberate eating allows satiety signals to develop gradually and reduces mechanical stress on the gastric wall. Adequate hydration facilitates gastric transit and may reduce the sensation of fullness associated with dense or dry foods. Although these factors have not been systematically evaluated in randomized trials, their physiological plausibility and consistent observation in clinical practice support their incorporation into patient education.

The temporal relationship between dosing and food intake further affects tolerability. Some patients report increased gastrointestinal symptoms when large meals are consumed shortly

after administration of injectable GLP-1 receptor agonists, whereas spacing meals away from peak drug action may attenuate symptoms. These observations highlight the dynamic interplay between pharmacokinetics, gastric physiology, and dietary behavior, underscoring the need for individualized guidance rather than uniform recommendations.

Collectively, these findings suggest that dietary adaptation constitutes a primary non-pharmacological strategy for managing gastrointestinal adverse events during GLP-1 therapy. Early and proactive counseling focused on meal size, fat content, eating pace, and hydration may reduce symptom severity, improve adherence, and enable patients to achieve therapeutic doses more successfully.

Diet Quality, Nutrient Adequacy, and Preservation of Lean Body Mass

Beyond tolerability, dietary behavior during GLP-1 receptor agonist therapy has important implications for nutrient adequacy and body composition. Pharmacologically induced appetite suppression leads to substantial reductions in total energy intake, which, while beneficial for weight loss, may also reduce intake of essential macronutrients and micronutrients if dietary choices are not intentionally structured. Several observational studies indicate that although fat mass loss predominates during GLP-1 therapy, a clinically meaningful proportion of weight reduction may derive from lean tissue, particularly in individuals with inadequate protein intake or limited engagement in resistance exercise [14,19,20].

Preservation of lean body mass is critical for maintaining resting metabolic rate, physical function, and glycaemic stability. Lean tissue constitutes the primary determinant of basal energy expenditure, and excessive loss during weight reduction may exacerbate metabolic adaptation and increase susceptibility to weight regain. Protein intake plays a central role in mitigating lean mass loss by supporting muscle protein synthesis and attenuating catabolic responses to caloric restriction. However, appetite suppression during GLP-1 therapy may lead patients to consume fewer protein-rich foods, especially if such foods are perceived as less palatable or more difficult to consume in small quantities.

Older adults and individuals with low baseline muscle mass are particularly vulnerable to sarcopenia under conditions of rapid weight loss. In these populations, insufficient protein intake combined with reduced mechanical loading of muscle tissue may accelerate functional decline. Although randomized trials evaluating protein-optimized diets in conjunction with GLP-1 therapy are lacking, physiological principles and observational data strongly support the prioritization of protein adequacy as a component of comprehensive care.

Micronutrient adequacy represents an additional consideration. Reduced total food intake may lower consumption of vitamins and minerals such as iron, calcium, vitamin D, folate, and

dietary fiber. Patients who adopt simplified eating routines or skip meals are at heightened risk of such deficiencies. While clinically significant micronutrient deficiencies have not been systematically documented in GLP-1 trials, the potential risk warrants attention, particularly during long-term therapy. Nutritional assessment and targeted supplementation may be appropriate in selected patients to ensure adequacy.

Diet quality, defined by nutrient density rather than caloric content alone, therefore remains a critical determinant of health outcomes during GLP-1 therapy. Diets emphasizing whole foods, adequate protein, fiber-rich carbohydrates, and unsaturated fats may support metabolic health, preserve lean mass, and reduce the risk of nutrient deficiencies. In contrast, overly restrictive or monotonous diets may undermine these goals despite successful weight loss.

Pharmacokinetics, Meal Timing, and Practical Dietary Constraints

Pharmacokinetic considerations constitute a distinct yet clinically important dimension of diet–drug interaction during GLP-1 receptor agonist therapy. Oral semaglutide, in particular, exhibits marked sensitivity to food intake, with substantially reduced bioavailability when administered in proximity to meals [11]. Controlled pharmacokinetic studies and clinical trial data demonstrate that fasting administration with minimal water is required to achieve consistent systemic exposure, and deviation from these instructions may result in subtherapeutic drug levels. In this context, dietary timing becomes a determinant of pharmacological efficacy rather than merely a factor influencing tolerability. Injectable GLP-1 receptor agonists do not share this degree of food-dependent absorption; however, meal timing may still influence gastrointestinal tolerability due to the interaction between peak drug activity and gastric motility.

Adherence to dosing instructions presents practical challenges for some patients, particularly those with irregular schedules or early-morning obligations. Failure to comply with fasting requirements may lead to variable clinical response and patient frustration. In such cases, transitioning to injectable formulations may represent a pragmatic solution. These considerations underscore the need for individualized therapeutic decisions that account not only for pharmacological properties but also for patients' daily routines and dietary habits.

Long-Term Weight Maintenance and Post-Treatment Physiology

Long-term weight maintenance remains one of the most challenging aspects of obesity management. Although GLP-1 receptor agonists produce substantial weight loss during active treatment, discontinuation is frequently followed by partial or complete weight regain [19,20]. This phenomenon reflects the reactivation of homeostatic mechanisms that defend pre-treatment body weight, including increased hunger, enhanced reward-driven eating, and

reductions in energy expenditure. The removal of pharmacological appetite suppression exposes patients to these biological pressures, often resulting in a rapid increase in caloric intake. Dietary behavior plays a central role in determining whether weight regain occurs. Patients who rely predominantly on pharmacological appetite suppression without developing structured eating patterns during treatment are particularly vulnerable to relapse. In contrast, those who establish sustainable dietary habits that align caloric intake with metabolic requirements appear more likely to maintain weight loss after discontinuation. Protein adequacy and lean mass preservation are especially important in this context, as reductions in muscle mass lower resting metabolic rate and exacerbate metabolic adaptation, increasing susceptibility to weight regain.

Psychological and behavioral factors further influence post-treatment outcomes. During GLP-1 therapy, patients may experience a profound reduction in food-related preoccupation, which can be perceived as liberation from chronic hunger or cravings. Upon discontinuation, the return of hunger may be distressing and misinterpreted as pathological rather than physiological. Behavioral support aimed at reframing hunger signals and reinforcing adaptive eating behaviors may therefore improve long-term success and reduce emotional distress associated with weight maintenance.

Cardiometabolic Implications and Diet–Drug Synergy

GLP-1 receptor agonists improve cardiometabolic outcomes through mechanisms that extend beyond weight loss, including favorable effects on lipid metabolism, endothelial function, and inflammatory signaling [7,22–24]. Dietary patterns independently influence these same pathways, suggesting the potential for additive or synergistic effects. Diets rich in fiber, unsaturated fats, and minimally processed foods support insulin sensitivity, reduce atherogenic lipid profiles, and attenuate systemic inflammation. Although randomized trials have not directly assessed the interaction between GLP-1 therapy and specific dietary patterns, mechanistic plausibility and observational evidence support alignment with cardioprotective dietary recommendations during treatment.

Conversely, diets characterized by high intake of saturated fats and refined carbohydrates may blunt some cardiometabolic benefits by promoting postprandial lipotoxicity and glycaemic variability. Extremely restrictive diets may also compromise nutrient adequacy in the context of already reduced appetite. Thus, balanced, nutrient-dense dietary approaches are preferable to rigid or excessively restrictive regimens during GLP-1 therapy.

Discussion

The evidence synthesized in this narrative review highlights diet as a critical, modifiable determinant of outcomes during GLP-1 receptor agonist therapy. Pharmacological modulation of appetite and gastric motility creates a distinct physiological context in which dietary behavior exerts amplified effects on tolerability, nutrient adequacy, body composition, and long-term weight stability. While GLP-1 receptor agonists effectively address biological drivers of obesity, they do not eliminate the need for intentional dietary practices.

Several consistent themes emerge across mechanistic studies, clinical trials, and real-world observations. Gastrointestinal tolerability is strongly influenced by meal size, fat content, and eating pace. Nutrient adequacy, particularly protein intake, is essential for preserving lean mass and metabolic rate during rapid weight loss. Pharmacokinetic constraints necessitate strict adherence to fasting administration for oral formulations. Long-term success depends on early establishment of sustainable dietary patterns that persist beyond active pharmacological treatment.

Despite these insights, the current evidence base has notable limitations. The absence of randomized controlled trials comparing dietary interventions during GLP-1 therapy represents a major gap. Future research should prioritize controlled dietary trials with robust assessment of body composition, metabolic outcomes, and long-term maintenance. Additional studies are needed to evaluate micronutrient status, optimize dietary strategies for vulnerable populations, and identify approaches that mitigate weight regain following treatment discontinuation.

Conclusions

Dietary behavior remains an essential component of effective GLP-1 receptor agonist therapy. Although these agents produce powerful reductions in appetite and body weight, diet continues to influence tolerability, nutritional adequacy, preservation of lean body mass, and durability of weight loss. Mechanistic and clinical evidence supports the integration of nutrient-dense dietary patterns, adequate protein intake, moderation of dietary fat during treatment initiation, adherence to pharmacokinetic requirements for oral formulations, and early planning for long-term weight maintenance. The development of evidence-based nutritional guidelines will require rigorously designed trials integrating dietary interventions into GLP-1 treatment paradigms. Until such data are available, clinicians should adopt individualized, mechanistically informed dietary strategies within multidisciplinary care frameworks to maximize therapeutic benefit.

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All authors have read and agreed with published version of the manuscript.

Financing statement:

This research received no external funding.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Not applicable.

Data Availability Statement:

Not applicable.

Conflict of interest:

The authors deny any conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies the writing process.

While preparing this manuscript, the authors used the ChatGPT tool to enhance language quality and readability. After using the tool, the authors thoroughly reviewed and edited the text as necessary and take full responsibility for the scientific content of the publication.

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