NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ

**Journal of Education, Health and Sport. eISSN 2391-8306.**

**Journal Home Page**
**https://apcz.umk.pl/JEHS/index**

**BABIK, Karolina, BARTKOWSKA, Oliwia, BANATKIEWICZ, Joanna, DEKA, Emilia, DOMIŃCZAK, Jan, CZARNECKA, Sofia, ŁUCZYŃSKA, Gabriela Ewa, DOBOSZ, Adam, WOJNOWSKI, Antoni, and BOJANOWSKA, Hanna Maria. Metabolic Adaptation and Health Regain Phenomenon After Cessation of GLP-1 Analogue Pharmacotherapy – A Review of Preventive Strategies. Journal of Education, Health and Sport. 2026;90:70450. eISSN 2391-8306. https://doi.org/10.12775/JEHS.2026.90.70450**

**The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).**
**Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).** 
**The authors declare that there is no conflict of interests regarding the publication of this paper.**
**Received: 31.03.2026. Revised: 12.04.2026. Accepted: 16.04.2026. Published: 19.04.2026.**

# Metabolic Adaptation and Weight Regain Phenomenon After Cessation of GLP-1 Analogue Pharmacotherapy – A Review of Preventive Strategies

Corresponding Author: **Karolina Babik**

ORCID: https://orcid.org/0009-0001-0824-286X

E-mail: karolinababik02@gmail.com

Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Oliwia Bartkowska**

ORCID: https://orcid.org/0009-0007-5665-5638

E-mail: oliwiabartkowska2@gmail.com

Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Joanna Banatkiewicz**
ORCID: https://orcid.org/0009-0007-9884-4656
E-mail: banatkiewiczj@gmail.com
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Emilia Deka**
ORCID: https://orcid.org/0009-0001-1283-8084
E-mail: emdeka00@gmail.com
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Jan Domińczak**
ORCID: https://orcid.org/0009-0002-8072-8191
E-mail: Dominczak.j@gmail.com
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Sofia Czarnecka**
ORCID: https://orcid.org/0009-0001-5899-770X
E-mail: czarneckasofia@gmail.com
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Gabriela Ewa Łuczyńska**
ORCID: https://orcid.org/0009-0002-7112-1291
E-mail: g.luczynska@interia.pl
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Adam Dobosz**
ORCID: https://orcid.org/0009-0002-8863-9361
E-mail: a.dobosz086@gmail.com
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Antoni Wojnowski**
ORCID: https://orcid.org/0009-0000-3339-848X
E-mail: antek.wojnowski@gmail.com
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**Hanna Maria Bojanowska**
ORCID: https://orcid.org/0009-0004-3414-2960
E-mail: haniabojanowska@wp.pl
Warszawski Uniwersytet Medyczny, Warsaw, Poland

**ABSTRACT**

Obesity is currently recognized as a chronic, relapsing and progressive disease, constituting one of the most serious public health challenges of the 21st century. The introduction of glucagon-like peptide-1 receptor agonists (GLP-1 RAs), such as semaglutide and tirzepatide, has led to unprecedented therapeutic success, enabling weight reduction comparable to that achieved by bariatric surgery. However, despite their high efficacy, discontinuation of these agents is associated with rapid and clinically significant weight regain.

The aim of this paper is to analyze the physiological mechanisms responsible for post-treatment weight regain following GLP-1 analogue withdrawal and to evaluate the effectiveness of selected strategies aimed at limiting this phenomenon, with particular emphasis on resistance training, nutritional interventions and pharmacological deprescribing models.

Available clinical and experimental data indicate that weight regain is driven primarily by metabolic adaptation, including a disproportionate reduction in resting metabolic rate, loss of lean body mass and dysregulation of appetite-controlling hormones. Evidence from major clinical trials (STEP, SURMOUNT) confirms that up to two-thirds of the lost body weight may be regained within one year after cessation of therapy.

The findings highlight the necessity of treating obesity pharmacotherapy as part of a long-term management strategy. Integration of pharmacological treatment with resistance training, high-protein dietary patterns and gradual dose reduction protocols appears essential for sustaining therapeutic effects and preventing the rebound phenomenon.

## 1.Introduction

Contemporary medicine defines obesity not merely as an excess of body weight, but as a chronic, relapsing, and progressive disease, representing one of the most significant public health challenges of the 21st century (1). Recent years have witnessed a radical shift in the treatment paradigm, driven by the introduction of next-generation pharmacotherapy: glucagon-like peptide-1 receptor agonists (GLP-1 RAs), such as semaglutide and tirzepatide. Clinical trials have confirmed their high efficacy, documenting weight reduction in the range of 15–20%—results that begin to rival those achieved through bariatric surgery (2,3). These outcomes are striking.

However, despite these impressive therapeutic results, maintaining them poses a major clinical hurdle. Data from extension studies, notably the STEP-1 extension, reveal that stopping pharmacotherapy is linked to rapid weight regain. Estimates suggest that patients recover, on average, two-thirds of their lost weight within a year of discontinuing the medication (4). This raises an important clinical question. The phenomenon is rooted in potent mechanisms of metabolic adaptation, including a suppression of resting metabolic rate and a dysregulation of hunger and satiety hormones, all of which drive the body to return to its original weight, consistent with the set-point theory (5). In practice, this means that weight regain is not merely behavioral-it is biological.

With the surging popularity of GLP-1 analogues, developing strategies for managing patients during the drug withdrawal phase (deprescribing) has become crucial. The aim of this review is to examine the current literature regarding the physiological mechanisms behind this "rebound effect" and to analyze the efficacy of non-pharmacological interventions-specifically resistance training and nutritional education in preventing the recurrence of obesity. These observations are clinically relevant and increasingly urgent.

Importantly, current clinical guidelines still provide limited guidance regarding the management of patients after pharmacological weight loss has been achieved. Most recommendations focus on initiation and titration of therapy, while the post-treatment phase remains largely under-addressed. This gap in knowledge exposes patients to a high risk of therapeutic failure despite initial success. Consequently, defining evidence-based exit strategies from GLP-1-based therapy represents one of the most pressing challenges in modern obesity medicine.

## 2. Methodology

This study was conducted as a narrative review of the literature focusing on the phenomenon of weight regain after discontinuation of GLP-1 receptor agonists and on strategies aimed at limiting this effect.

A structured search was performed in the PubMed, Scopus and Web of Science databases using the following keywords and their combinations: “GLP-1 receptor agonists”, “semaglutide”, “tirzepatide”, “weight regain”, “withdrawal”, “metabolic adaptation”, “resting metabolic rate”, “lean body mass”, “resistance training”, “diet”, “deprescribing”.

The analysis included articles published in English addressing contemporary approaches to obesity pharmacotherapy and lifestyle interventions.

Eligible publications comprised randomized controlled trials, extension phases of major obesity trials (including the STEP and SURMOUNT programs), systematic reviews, meta-analyses, and observational studies evaluating body weight dynamics after treatment cessation. Animal studies, case reports and publications lacking post-withdrawal follow-up data were excluded.

From the selected studies, data were extracted regarding the magnitude and rate of weight regain, changes in resting metabolic rate and body composition, hormonal adaptations influencing appetite regulation, and the effectiveness of resistance training, dietary interventions and pharmacological tapering strategies.

The collected evidence was analyzed qualitatively in order to integrate physiological mechanisms with clinical outcomes and to formulate practical recommendations for long-term obesity management.

### 2.1. Clinical relevance and rationale of the review

The clinical relevance of addressing weight regain after GLP-1 receptor agonist discontinuation has increased substantially with the widespread use of these agents in routine obesity management. While their short-term efficacy is well established, real-world clinical practice increasingly reveals that discontinuation is frequent due to economic constraints, adverse effects, or limited access to long-term therapy.

From a public health perspective, failure to maintain achieved weight loss undermines the cost-effectiveness of pharmacological obesity treatment and may contribute to patient discouragement and therapeutic non-adherence. Moreover, repeated cycles of weight loss and regain are associated with adverse cardiometabolic consequences, including increased insulin resistance, visceral adiposity, and endothelial dysfunction.

Therefore, understanding post-treatment physiology and developing structured withdrawal strategies is not only a matter of weight control but also of preventing secondary metabolic

deterioration. This review seeks to fill this gap by integrating physiological mechanisms with pragmatic clinical solutions.

## 3. Physiological determinants of weight regain – the mechanism of metabolic adaptation

Pharmacologically induced weight loss through GLP-1 analogues activates a series of counter-regulatory mechanisms that evolved as protection against starvation. This phenomenon- adaptive thermogenesis- is one of the primary physiological barriers to long-term weight maintenance following treatment cessation (5).

A central feature of this adaptive response is the disproportionate reduction in resting metabolic rate (RMR). Although lower body mass naturally decreases energy expenditure, research shows that RMR drops more sharply than would be expected based on body-composition changes alone. The organism shifts into an energy-conserving mode, demonstrated by increased mitochondrial efficiency and reduced thermogenesis in skeletal muscle (6).

An additional challenge during GLP-1 therapy is the potential loss of fat-free mass (FFM). Rapid weight loss, particularly when driven by caloric restriction without sufficient exercise stimulus, may promote sarcopenia. Given that muscle mass is a major determinant of RMR, reductions in FFM further aggravate the metabolic decline and make post-treatment weight maintenance more difficult (7).

Hormonal dysregulation also plays a central role. GLP-1 analogues enhance satiety through exogenous stimulation of the gut-brain axis. When therapy stops, endogenous satiety signals (leptin, PYY) remain suppressed-reflecting reduced adipose stores—while ghrelin levels rise sharply. This imbalance creates an “energy gap,” in which the biological drive to eat exceeds the actual caloric needs of the body, accelerating weight regain (8).

## 4. Pharmacotherapy efficacy and withdrawal effect – evidence from clinical trials

Large-scale, multicenter randomized clinical trials (RCTs) confirm the theoretical concerns regarding metabolic adaptation. Major research programs, specifically STEP (semaglutide) (4,9) and SURMOUNT (tirzepatide) (10), provided unequivocal evidence in their extension phases that the therapeutic effect is transient once pharmacological intervention ends.

These studies utilized rigorous protocols where, following an initial weight reduction phase, patients were randomized to either continue the active treatment or switch to a placebo (simulating drug withdrawal). Table 1 summarizes the key findings regarding body weight dynamics in the post-treatment phase.

Table 1. Summary of key clinical trials evaluating weight regain after GLP-1 agonist withdrawal.

| Study (Author, Year) | Active Substance | Treatment Phase (weeks) | Follow-up / Withdrawal Phase (weeks) | Outcomes in Withdrawal Group (Placebo) |
|---|---|---|---|---|
| STEP-1 Extension | Semaglutide 2.4 mg | 68 | 52 (1 year) | Patients regained on average 2/3 (11.6%) of prior weight loss; cardiometabolic risk factors returned to baseline. |
| STEP-4 | Semaglutide 2.4 mg | 20 (run-in) | 48 | Placebo switch group regained 6.9% body weight; continued treatment group lost an additional 7.9%. |
| SURMOUNT-4 | Tirzepatide (MTD*) | 36 (run-in) | 52 | Discontinuation led to 14% weight regain; continued therapy resulted in an additional 5.5% reduction. |

As the data indicates, discontinuing the medication leads to an immediate reversal of the weight loss trend. In the STEP-1 Extension study, participants weighed only 5.6% less than their baseline one year after stopping therapy, effectively losing the majority of the benefits gained during the 68-week treatment (4). Similarly, the STEP-4 trial demonstrated the critical role of continued therapy; patients who switched to placebo after the run-in phase regained 6.9% of their weight, standing in sharp contrast to those who continued the drug and achieved further

weight loss (9). The results were even more pronounced in the SURMOUNT-4 study involving tirzepatide (a dual GLP-1/GIP agonist), where the weight regain curve following withdrawal was nearly linear (10).

These findings suggest that obesity requires a chronic treatment model, analogous to the management of hypertension. Removing the pharmacological agent allows the disease's pathophysiology to return. This underscores the necessity of implementing aggressive behavioral strategies—specifically resistance training and high-protein diets—during the deprescribing period to counteract the physiological drive toward adipose tissue restoration.

## 5. Resistance training as a tool for restoring Resting Metabolic Rate (RMR) after pharmacological intervention

The primary driver of weight regain following GLP-1 withdrawal is the reduction in Resting Metabolic Rate (RMR), a consequence largely attributed to the loss of muscle tissue during the rapid weight reduction phase. Evidence shows that patients treated with semaglutide lose not only adipose tissue but also a significant amount of Lean Body Mass (LBM), directly lowering their daily energy expenditure (11). Consequently, upon drug discontinuation, the patient faces a suppressed RMR combined with a resurgent appetite—creating the perfect storm for rapid weight regain.

In this context, physical activity—specifically resistance training—shifts from being merely a tool for burning calories to an essential component of "metabolic rehabilitation." Systematic reviews demonstrate that resistance training is the only non-pharmacological intervention capable of effectively counteracting the decline in RMR by stimulating muscle hypertrophy (12,13). Unlike aerobic exercise, resistance training induces a prolonged increase in muscle protein synthesis and enhances tissue insulin sensitivity, which is vital for patients whose glycemic control often deteriorates after stopping incretin drugs (14).

Another significant aspect is the impact of exercise on appetite regulation. Research suggests that intense physical exertion can modulate levels of acylated ghrelin (the hunger hormone), assisting patients in managing the "hyperphagia" typical of the withdrawal phase (15). Thus, the role of "Sport" in this context refers to the strategic application of exercise to close the energy gap created by the removal of the drug.

In addition to its metabolic effects, resistance training contributes significantly to long-term functional and clinical outcomes in patients recovering from pharmacologically induced weight loss. Improvements in muscular strength and endurance translate into enhanced physical

autonomy, reduced fatigue and increased spontaneous physical activity, all of which further support energy balance.

Importantly, resistance-based exercise has also been shown to exert beneficial effects on mental health, including reduction of depressive symptoms, improved self-efficacy and body image. These psychological factors are recognized predictors of sustained adherence to lifestyle interventions and weight maintenance, underscoring the multidimensional role of resistance training in obesity relapse prevention.

## 6. Nutritional and educational strategies buffering the "rebound" appetite effect (Post-drug Hyperphagia)

Stopping pharmacotherapy results in the abrupt loss of the drug's anorectic effect. Patients who have felt satisfied with small portions for months are suddenly confronted with a biological compulsion to eat. Nutritional education during this critical window must focus on strategies that increase meal volume without drastically raising caloric content (volumetrics) (16).

The cornerstone of the post-treatment diet should be protein intake. Among macronutrients, protein exhibits the highest satiety index and generates the greatest Thermic Effect of Food (TEF), thereby supporting metabolism (17). Studies indicate that a high-protein diet (1.2-1.6 g/kg BW) helps preserve the lean body mass recovered through training (as described in Chapter 3) and mitigates glycemic fluctuations after discontinuing insulinotropic agents (18).

Equally important is the restructuring of eating patterns rather than focusing solely on macronutrient composition or caloric intake. Emphasis on regular meal timing, reduced consumption of ultra-processed foods, and prioritization of low-energy-density products contributes to stabilization of postprandial glycemic and insulinemic responses, which are often exaggerated after discontinuation of incretin-based therapy.

Furthermore, nutritional literacy empowers patients to recognize early signs of relapse and intervene before clinically significant weight regain occurs, transforming patients from passive recipients of care into active participants in long-term disease management.

A key educational component is preparing the patient for the reality of metabolic adaptation. Implementing drug tapering protocols (slow withdrawal) rather than abrupt cessation, combined with diligent self-monitoring, allows for a gentler adaptation of the hunger and satiety centers (19). Research by Wing and Phelan on successful weight maintainers (NWCR) highlights that regular body weight monitoring and having an immediate "action plan" for weight spikes are essential to prevent the yo-yo effect in the absence of pharmacological support (20,21).

## 7. Pharmacological exit strategies – tapering and establishing the lowest effective dose

Given the strong evidence supporting rapid weight regain after abrupt therapy discontinuation, clinical practice is shifting from a binary on/off strategy toward gradual dose reduction models. Although product labels for semaglutide and tirzepatide assume long-term use at maximal therapeutic doses, real-world contexts- such as side effects or treatment cost-often necessitate alternative approaches (22).

Tapering typically involves stepwise dose reductions in four-week intervals (essentially the reverse of titration). The goal is to identify the lowest effective dose that prevents weight regain, even if it no longer produces additional weight loss (23). This more gradual reduction also gives patients time to intensify behavioral strategies such as exercise and dietary modification.

Evidence from STEP-4 strongly supports this approach. Participants who continued semaglutide maintained their weight loss, whereas those switched to placebo experienced prompt regain. These findings suggest that for many individuals, maintenance therapy using reduced doses-or potentially extended dosing intervals, such as every 10-14 days-may be preferable to complete discontinuation, although such regimens require further evaluation (9).

Another potential strategy is drug rotation. When GLP-1 injectables must be discontinued, transitional use of oral agents (e.g., bupropion/naltrexone or low-dose liraglutide) may help blunt the hormonal rebound associated with rising ghrelin levels (24).

Another emerging concept is the use of combination therapy at lower doses rather than monotherapy at maximal doses. Low-dose GLP-1 analogues combined with agents acting on complementary pathways, such as SGLT2 inhibitors or centrally acting appetite suppressants, may provide metabolic stability with improved tolerability and reduced cost.

Although evidence in this area remains limited, such strategies may represent a pragmatic compromise between complete withdrawal and indefinite full-dose maintenance, particularly in healthcare systems with restricted reimbursement.

## 8. Summary and Conclusions

Current evidence demonstrates that while GLP-1 analogues represent a major breakthrough in obesity treatment, they do not ensure durable results once therapy ends. Rapid metabolic adaptation and weight regain highlight the need to view obesity pharmacotherapy not as a temporary intervention but as part of a long-term management framework.

Resistance training emerges as a crucial component for preserving muscle mass and resting metabolic rate, both of which are central to weight-maintenance physiology. Additionally,

deprescribing strategies should emphasize gradual tapering and high-protein dietary approaches to help control appetite during the withdrawal phase.

In summary, effective long-term obesity management requires an integrated model combining pharmacotherapy, structured lifestyle interventions, and continuous patient education.

## Author Contribution

Conceptualization, S.C. and J.B.; methodology, G.Ł., E.D. and J.D.; formal analysis, A.D.,K.B. and O.B.; investigation, K.B.and H.B.; resources, A.W.,S.C. and E.D.; data curation, H.B.,G.Ł. and J.D.; writing, S.C.,J.B. and E.D.; review and editing, A.D. and O.B.; supervision, S.C.; project administration, S.C and A.W.;

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

## Funding Statement

This research received no external funding.

## Institutional Review Board Statement

Not applicable. This is a literature review based on published studies and does not involve primary data collection from human subjects.

## Informed Consent Statement

Not applicable. This is a literature review that does not involve human subjects or patient data.

## Data Availability Statement

Not applicable. This literature review is based on publicly available published literature. All sources are cited in the References section.

## Conflicts of Interest

The authors declare no conflicts of interest.

## AI

During the preparation of this work, the authors used SciSpace for the purpose of basic data analysis and verification of bibliographic styles. After using this tool/service, the authorsreviewed and edited the content as needed and take full responsibility for the substantive content of the publication.

## References


1. Blüher M. Obesity: global epidemiology and pathogenesis. Nat Rev Endocrinol. 2019 May;15(5):288–98.
2. Wilding JPH, Batterham RL, Calanna S, Davies M, Van Gaal LF, Lingvay I, et al. Once-Weekly Semaglutide in Adults with Overweight or Obesity. N Engl J Med. 2021 Mar 18;384(11):989–1002.
3. Jastreboff AM, Aronne LJ, Ahmad NN, Wharton S, Connery L, Alves B, et al. Tirzepatide Once Weekly for the Treatment of Obesity. N Engl J Med. 2022 July 21;387(3):205–16.
4. Wilding JPH, Batterham RL, Davies M, Van Gaal LF, Kandler K, Konakli K, et al. Weight regain and cardiometabolic effects after withdrawal of semaglutide: The STEP 1 trial extension. Diabetes Obes Metab. 2022 Aug;24(8):1553–64.
5. Müller MJ, Geisler C. From the past to future: from energy expenditure to energy intake to energy expenditure. Eur J Clin Nutr. 2017 Mar;71(3):358–64.
6. Rosenbaum M, Leibel RL. Adaptive thermogenesis in humans. Int J Obes 2005. 2010 Oct;34 Suppl 1(0 1):S47-55.
7. Sargeant JA, Henson J, King JA, Yates T, Khunti K, Davies MJ. A Review of the Effects of Glucagon-Like Peptide-1 Receptor Agonists and Sodium-Glucose Cotransporter 2 Inhibitors on Lean Body Mass in Humans. Endocrinol Metab Seoul Korea. 2019 Sept;34(3):247–62.
8. Sumithran P, Prendergast LA, Delbridge E, Purcell K, Shulkes A, Kriketos A, et al. Long-term persistence of hormonal adaptations to weight loss. N Engl J Med. 2011 Oct 27;365(17):1597–604.
9. Rubino D, Abrahamsson N, Davies M, Hesse D, Greenway FL, Jensen C, 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 Apr 13;325(14):1414–25.
10. Aronne LJ, Sattar N, Horn DB, Bays HE, Wharton S, Lin WY, et al. Continued Treatment With Tirzepatide for Maintenance of Weight Reduction in Adults With Obesity: The SURMOUNT-4 Randomized Clinical Trial. JAMA. 2024 Jan 2;331(1):38–48.
11. McCarthy D, Berg A. Weight Loss Strategies and the Risk of Skeletal Muscle Mass Loss. Nutrients. 2021 July 20;13(7):2473.
12. Hunter GR, Byrne NM, Sirikul B, Fernández JR, Zuckerman PA, Darnell BE, et al. Resistance training conserves fat-free mass and resting energy expenditure following weight loss. Obes Silver Spring Md. 2008 May;16(5):1045–51.

13. Westcott WL. Resistance training is medicine: effects of strength training on health. Curr Sports Med Rep. 2012;11(4):209–16.
14. Codella R, Luzi L, Terruzzi I. Exercise has the guts: How physical activity may positively modulate gut microbiota in chronic and immune-based diseases. Dig Liver Dis Off J Ital Soc Gastroenterol Ital Assoc Study Liver. 2018 Apr;50(4):331–41.
15. Dorling J, Broom DR, Burns SF, Clayton DJ, Deighton K, James LJ, et al. Acute and Chronic Effects of Exercise on Appetite, Energy Intake, and Appetite-Related Hormones: The Modulating Effect of Adiposity, Sex, and Habitual Physical Activity. Nutrients. 2018 Aug 22;10(9):1140.
16. Rolls BJ. The relationship between dietary energy density and energy intake. Physiol Behav. 2009 July 14;97(5):609–15.
17. Westerterp-Plantenga MS. Protein intake and energy balance. Regul Pept. 2008 Aug 7;149(1–3):67–9.
18. Leidy HJ, Clifton PM, Astrup A, Wycherley TP, Westerterp-Plantenga MS, Luscombe-Marsh ND, et al. The role of protein in weight loss and maintenance. Am J Clin Nutr. 2015 June;101(6):1320S-1329S.
19. Kahan S, Manson JE. Obesity Treatment, Beyond the Guidelines: Practical Suggestions for Clinical Practice. JAMA. 2019 Apr 9;321(14):1349–50.
20. Wing RR, Phelan S. Long-term weight loss maintenance. Am J Clin Nutr. 2005 July;82(1 Suppl):222S-225S.
21. Butryn ML, Webb V, Wadden TA. Behavioral treatment of obesity. Psychiatr Clin North Am. 2011 Dec;34(4):841–59.
22. Nauck MA, Quast DR, Wefers J, Meier JJ. GLP-1 receptor agonists in the treatment of type 2 diabetes - state-of-the-art. Mol Metab. 2021 Apr;46:101102.
23. Yumuk V, Tsigos C, Fried M, Schindler K, Busetto L, Micic D, et al. European Guidelines for Obesity Management in Adults. Obes Facts. 2015;8(6):402–24.
24. Pichichero ME, Gonzalez E, Cox A, Thayer TC, Bajorski P. Variability in Vaccine Response and Trajectory in Early Childhood and Association With Demographic Variables, Antibiotic Exposure, and Infection Proneness. J Infect Dis. 2025 July 30;232(1):170–80.