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Effects of semaglutide on anthropometric measures, diet, and eating behaviors in obese women: a retrospective observational study

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Abstract

Background. Semaglutide is a glucagon-like peptide-1 (GLP-1) receptor agonist primarily used to improve glycemic control in patients with type 2 diabetes mellitus (T2DM). It is also recommended as part of the therapeutic strategy for individuals with metabolic syndrome or obesity. This study aimed to evaluate the effects of semaglutide administration on body weight reduction, eating behaviors, energy intake, and appetite control in women with diagnosed obesity and insulin resistance.

Material and methods. The study included 104 women diagnosed with obesity and insulin resistance who tolerated semaglutide therapy without adverse effects.

Results. Participants using semaglutide demonstrated a mean decrease in daily energy intake of 103 kcal (p=0.034; d=-0.21) compared with baseline. A significant reduction in body weight (mean -2.85 kg) was observed, along with decreases in body fat content, Body Mass Index (BMI), and waist-to-hip ratio (WHR).

Conclusions. Semaglutide use was associated with appetite suppression, reduced snacking frequency, and a subsequent decrease in body weight and body fat percentage, indicating its beneficial role in weight management among women with obesity and insulin resistance.

Keywords: obesity therapy, GLP-1 analogue, appetite control, semaglutide, pharmacotherapy

Introduction

Semaglutide is a chemical compound that functions as a glucagon-like peptide-1 (GLP-1) receptor agonist. GLP-1 is an incretin hormone secreted by the gut, and a portion of circulating GLP-1 binds directly to pancreatic receptors, modulating insulin secretion [1]. Additionally, GLP-1 interacts with the vagus nerve, which mediates stimulatory or inhibitory signals to the pancreas, influencing insulin release [1,2]. Due to its effect on glycemic control, semaglutide is primarily indicated for patients with type 2 diabetes mellitus (T2DM) [3]. It is also recommended for the management of metabolic syndrome and obesity. The metabolic effects of semaglutide include glucose-dependent stimulation of insulin secretion [4], delayed gastric emptying, appetite suppression, reduced food intake, and increased natriuresis and diuresis [5]. GLP-1 also exerts cardioprotective and neuroprotective effects [6]. Therapeutic GLP-1 analogues are predominantly administered orally or via injection. Each formulation has specific indications, dosing regimens, and preparations, which should be tailored to the patient's individual health status [7]. The U.S. Food and Drug Administration (FDA) highlights that these agents are particularly effective in improving glycemic control in T2DM patients. Injectable

formulations further reduce the risk of adverse cardiovascular events in obese patients with T2DM and have demonstrated efficacy in promoting weight loss among individuals with overweight or obesity [3,4]. In overweight and obese patients, semaglutide is indicated not only for glycemic regulation but also as an adjunct to non-pharmacological interventions such as calorie-restricted diets and regular physical activity [8]. By reducing hunger and enhancing satiety, semaglutide may improve dietary adherence and eating behaviors, although the precise mechanisms remain under investigation. Importantly, GLP-1 analogue therapy should always be combined with lifestyle modifications, including dietary changes and consistent physical activity [9].

Despite its beneficial effects on glycemic control and appetite regulation, semaglutide is associated with adverse events that may limit long-term use and dietary adherence in some patients [4]. The most commonly reported side effects include gastrointestinal disturbances, nausea, vomiting, diarrhea, and abdominal pain, which may lead to treatment discontinuation or difficulty maintaining dietary and exercise routines. Other potential complications include dehydration, hypoglycemia, renal impairment, and cholelithiasis [5]. Consequently, not all patients are suitable candidates for semaglutide therapy.

While the pharmacological profile of semaglutide suggests benefits for patients with T2DM and obesity, its clinical indications, potential adverse effects, and efficacy in obesity management warrant further research. Current evidence suggests that, beyond glycemic regulation, semaglutide may influence appetite and promote weight loss. However, its effects on eating patterns and long-term dietary behavior modification remain incompletely understood [8]. Semaglutide is currently included in international guidelines for the management of obesity and T2DM, based on large multicenter randomized controlled trials demonstrating its efficacy and safety [1,3]. However, real-world observational data focusing on eating behaviors, spontaneous energy intake, and short-term anthropometric changes in routine clinical practice remain limited. Therefore, the present study was designed to complement existing randomized evidence by providing practice-based data from a homogeneous age group of women treated with semaglutide under standard outpatient conditions.

Aim of the work

Based on the aforementioned rationale, the aim of this study was to evaluate the effects of semaglutide administration on body weight reduction, eating behaviors, energy intake, and appetite control in women diagnosed with obesity and insulin resistance.

Material and methods

Study population

The study included adult women with overweight and/or obesity who were prescribed semaglutide following medical consultation. None of the participants had a diagnosis of T2DM or cardiovascular diseases. Semaglutide therapy was recommended due to abdominal obesity, insulin resistance, and elevated blood pressure. Medical history interviews indicated that participants reported a family history of overweight or obesity, cardiovascular diseases, T2DM, metabolic syndrome, and cancer. Inclusion criteria comprised women aged 20-39 years with Body Mass Index (BMI) ≥27 kg/m² accompanied by insulin resistance and abdominal obesity. Consequently, the study group included individuals with both overweight and obesity according to the WHO classification, which was considered acceptable given the metabolic indication for pharmacotherapy.

Study design and setting

The study was a retrospective observational analysis. Semaglutide treatment duration was three months in all participants, corresponding to routine clinical follow-up after dose stabilization. This retrospective observation enrolled 103 adult women who initiated pharmacological treatment with semaglutide. Participants were recruited consecutively and met the inclusion criteria: age 20-39 years, absence of significant chronic diseases (except obesity and related metabolic complications), and written informed consent. The mean age was 34.1±2.7 years. The sample was relatively homogeneous regarding age and anthropometric indices, minimizing confounding effects due to population heterogeneity and allowing for more precise interpretation of the results without extensive multivariate analyses.

Data collection and procedures

Data were collected at two time points: baseline – prior to initiation of semaglutide therapy and follow-up – after a predefined period of treatment, once therapy stabilization was achieved. Patients reporting clinically significant adverse gastrointestinal effects leading to treatment discontinuation were not included in the final analysis. As a result, adverse events were not systematically analyzed in the present study.

At each time point, comprehensive anthropometric assessments were performed, including:

- body weight (kg), body fat percentage, and lean mass measured using a calibrated electronic scale;
- BMI (kg/m²) calculated from height and weight;
- waist-to-hip ratio (WHR), determined using standard anthropometric tape measurements in accordance with the WHO guidelines.

Dietary intake was assessed in detail at both time points, including total energy intake (kcal/day), macronutrient composition (percentage of energy from protein, fat, and carbohydrates), absolute macronutrient intake (g/day), dietary fiber intake (g/day), and water intake from food and beverages (mL/day). Dietary records were analyzed using standard nutrient tables and specialized dietary analysis software.

Participants also reported subjective appetite perceptions and the frequency of specific eating behaviors, such as snacking between meals, consumption of sweets and salty snacks, and fast-food intake. All the participants received unified general dietary recommendations consistent with standard hypocaloric guidelines for obesity management, as well as advice to maintain regular moderate physical activity. No individualized calorie targets were imposed. Dietary intake was assessed using self-reported food diaries analyzed with nutritional software. Standardized questionnaires assessing eating behavior were not applied, which constitutes a methodological limitation. Fasting plasma glucose and fasting insulin concentrations were assessed using standard laboratory methods. Insulin resistance was estimated using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR). Changes in these parameters were not the primary endpoints of the present analysis and are therefore not reported in detail.

Statistical analysis

Quantitative variables were described using mean ± standard deviation, median, interquartile range, and minimum-maximum values. Paired-sample Student's t-tests were used to compare baseline and follow-up measurements; in cases of non-normal distribution, the Wilcoxon signed-rank test was applied. 95% confidence intervals (CIs) and effect sizes (Cohen's d for paired samples) were calculated to assess clinical significance. Categorical variables were analyzed using frequency distributions and the chi-square test. All statistical tests were two-sided, with a significance level set at $\alpha=0.05$.

Results

Anthropometric measurements

At baseline, the mean body weight of the participants was 84.38±9.60 kg. Following the treatment period, a statistically significant reduction in body weight was observed, with the mean value decreasing to 81.52±9.29 kg, corresponding to an average weight loss of -2.85 kg (95% CI: -3.22 to -2.49). The Wilcoxon signed-rank test confirmed the high statistical significance of this change (Z=93.0; p<0.0001). The effect size, calculated as Cohen's d_z, was -1.52, indicating a large and clinically meaningful effect. The median weight reduction was -2.8 kg, with an interquartile range (IQR) of -3.8 to -1.5 kg. Notably, 20.4% of participants (n=21) achieved a weight loss of ≥5% relative to baseline, whereas no participant reached a reduction of ≥10% (Figure 1).

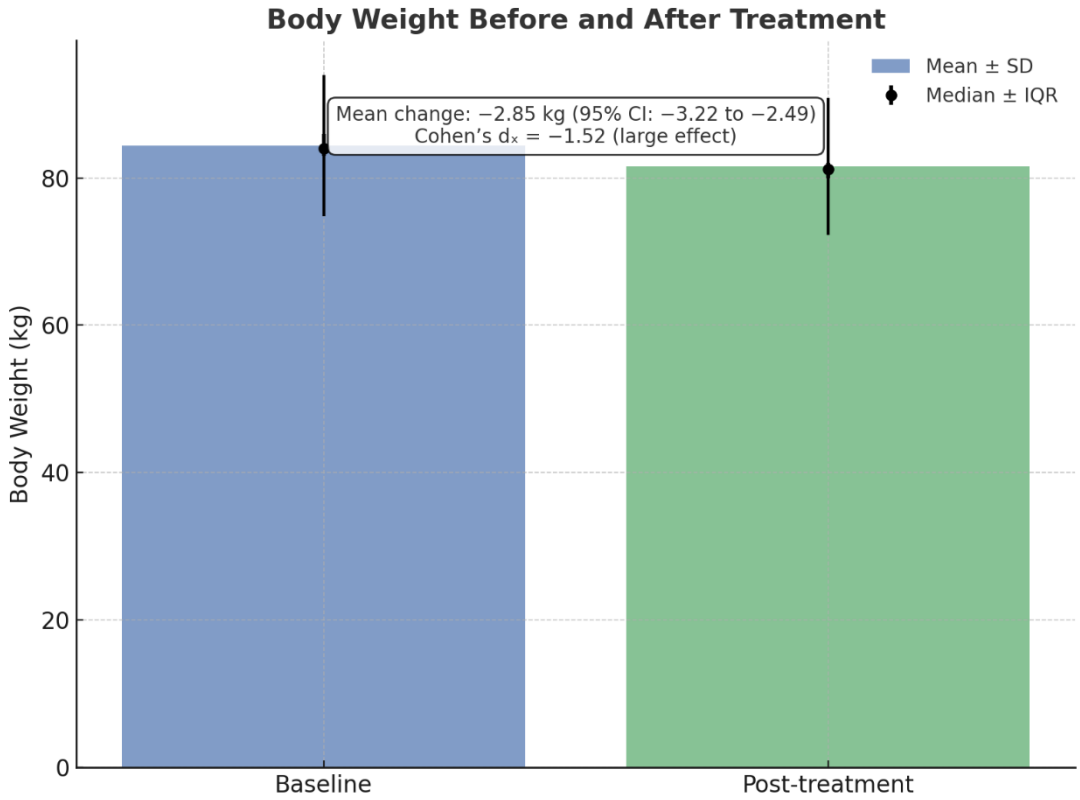

Figure 1. Distribution of body weight reduction among participants undergoing semaglutide treatment

Body fat percentage decreased by an average of 1.95%, from an initial value of 37.43±4.07% to 35.48±5.27%. Baseline BMI averaged 30.66±3.15 kg/m², corresponding to WHO-defined Class I obesity. After treatment, the mean BMI decreased to 29.63±3.10 kg/m²,

representing a reduction of -1.03 units (95% CI: -1.17 to -0.90). This change was statistically significant (Wilcoxon Z=96.0; $p<0.0001$), with a large effect size (d_z=-1.49). The median BMI reduction was -0.92 units, and the IQR ranged from -1.38 to -0.59. The mean WHR declined from 0.861±0.15 to 0.847±0.15, corresponding to an average change of -0.013 units (95% CI: -0.027 to -0.0005). This difference was statistically significant (Wilcoxon Z=1496.5; $p=0.026$), though the effect size (d_z=-0.19) suggests a modest practical significance.

Dietary composition

Analysis of daily dietary intake revealed a modest but statistically significant reduction in total energy intake, from 1,953±293 kcal/day at baseline to approximately 1,850 kcal/day post-treatment, corresponding to an average decrease of 103 kcal/day ($p=0.034$; d=-0.21). The relative contribution of protein to total energy intake increased from 13.7±4.2% to approximately 15% ($p=0.047$), which in absolute terms equated to an increase of 3.5 g/day ($p=0.048$). Conversely, the proportion of dietary fat decreased from 30.7±6.6% to around 28% of total energy intake, with an absolute reduction of 9 g/day ($p=0.032$). Carbohydrate intake remained stable (55.6% vs. 56.0%; $p>0.05$). No statistically significant changes were observed in fiber or dietary water intake ($p>0.05$).

Eating behaviors

Following semaglutide therapy, 84.5% of participants (87/103) reported a marked decrease in appetite, whereas 15.5% continued to experience hunger, indicating a strong anorexigenic effect of the drug. Analysis of the frequency of consumption of specific food categories demonstrated that sweets were still consumed by 62.1% of participants, salty snacks by 62.1%, fast-food meals by 55.3%, and between-meal snacking by 70.1%. A statistically significant association was observed between appetite suppression and reduced fast-food consumption ($\chi^2=6.42$; df=1; $p=0.011$). Differences in the consumption of sweets and salty snacks did not reach statistical significance ($p>0.05$).

Anorexigenic effect of semaglutide

The mean age of the participants was 34.1±2.7 years (range: 23-39 years). In the subgroup reporting appetite suppression, the mean age was 34.0±2.6 years, whereas in

participants maintaining hunger, the mean age was 34.4±2.9 years. This difference was not statistically significant (t=-0.64; *p*=0.52; d=-0.13). Among participants experiencing appetite suppression (n=87), the mean BMI reduction was -1.12±0.44 units, whereas in those maintaining hunger (n=16), the reduction was markedly smaller at -0.54±0.37 units. The difference between the two groups was statistically significant (t=-4.28; *p*<0.001), with a large effect size (d=0.88). Regarding WHR, differences between the appetite-suppressed and hunger-maintaining subgroups were less pronounced. The mean reduction in WHR in the appetite-suppressed group was -0.015±0.042, compared with -0.007±0.039 in the hunger-maintaining group; this difference was not statistically significant (*p*=0.27) and demonstrated a small effect size (d=0.19) (Table 1).

Table 1. Changes in the variables in the studied women between baseline and month 3 of pharmacology intervention

Variable	Participants N=103 (M±SD)	
	Pre-intervention	Post-intervention
	M±SD	M±SD
BMI [kg/m^2]	30.66±3.15	29.62±3.1
BW [kg]	84.38±9.60	81.52±9.29
BF [%]	37.43±4.07	35.48±5.27
BF [kg]	31.68±5.76	29.05±5.99
LBM [%]	62.56±4.07	64.51±5.27
LBM kg]	52.69±6.10	52.46±6.21
WHR	0.86±0.14	0.84±0.14

Notes: BF – Body Fat Percentage, BF – Body Fat Mass, BMI – Body Mass Index, BW – Body Weight, LBM – Lean Body Mass Percentage, LBM – Lean Body Mass, M – Mean, SD – Standard Deviation, WHR – Waist-to-Hip Ratio.

Discussion

Semaglutide is increasingly recognized as a pharmacological intervention for patients with T2DM and for weight reduction, despite the potential for adverse effects [10]. Large-scale analyses, such as the study by Castellana and Chiappetta [11], which included 5,310 cases (of which 73.35% were women) indicate that women are more likely than men to use semaglutide and may also be more susceptible to adverse events. The frequent use of semaglutide beyond clinical indications has been attributed to heightened social and cultural pressures on women to

maintain body weight. In the present study, the participants were women diagnosed with obesity and insulin resistance, which constituted the primary clinical indication for semaglutide therapy. Only participants without common gastrointestinal side effects, such as nausea, vomiting, or diarrhea, were included. The study demonstrated significant reductions in body weight, BMI, and WHR, alongside measurable effects on appetite and eating behaviors.

Effects of semaglutide on body weight and BMI

Our findings demonstrated a significant reduction in body weight after three months of semaglutide therapy, with an average decrease of -2.85 kg. This was accompanied by decreases in BMI, WHR, and body fat percentage, from 37.43±4.07% at baseline to 35.48±5.27% post-treatment. These results are consistent with those reported by Wilding et al. [12], in which 140 participants received either semaglutide injections or placebo over 60 weeks. In that study, semaglutide induced a mean body weight reduction of -15.0% compared to -3.6% in the placebo group, with reductions in total body fat (-19.3%) and visceral fat mass (-27.4%). Similarly, Phillips and Clements observed dose-dependent weight loss in a cohort of 5,000 adults with overweight or obesity, with a greater proportion of participants achieving 5-10% body weight reduction when treated with subcutaneous semaglutide compared with oral therapies [13]. Therefore, the magnitude of weight loss observed in this study was lower than that reported in large randomized controlled trials such as the STEP program. This discrepancy can be explained by the substantially shorter treatment duration (3 months vs. ≥68 weeks), real-world dosing strategies, and the observational nature of the study, which did not include intensive behavioral interventions [14].

While semaglutide generally promotes favorable changes in body composition, particularly through the reduction of fat mass, the effect on lean body mass (LBM) remains less well understood. Bikou et al. [15], in a review of randomized controlled trials and observational studies, reported that semaglutide-induced weight loss predominantly reflects fat mass reduction, with maintenance of LBM. However, in some cases (especially in larger populations), significant reductions in lean mass were observed, with losses of up to 40% of total weight loss, highlighting the need for caution regarding potential adverse effects on muscle mass and physical function [16,17]. These findings underscore the importance of combining semaglutide therapy with lifestyle interventions, including tailored dietary strategies and structured physical activity, to preserve lean mass and optimize long-term health outcomes.

Effects on energy intake

The observed weight reduction in our cohort was accompanied by a modest but significant decrease in overall energy intake, averaging 103 kcal/day (p=0.034; d=-0.21). These findings align with previous studies, such as the study by Blundell et al. [8], who reported a significant reduction in ad libitum energy intake after 12 weeks of semaglutide therapy compared to placebo. Similarly, Gibbons et al. demonstrated decreased caloric consumption and improved dietary patterns after 12 weeks in patients with diabetes [18]. In our study, women also reported decreased frequency of between-meal snacking, suggesting that semaglutide may promote both quantitative and qualitative improvements in dietary intake.

Effects on appetite and eating behaviors

Semaglutide exerted a pronounced anorexigenic effect in the majority of participants (84.5%), consistent with previous findings [10,19]. Participants reported reduced hunger and lower frequency of high-energy food consumption, particularly fast-food items. While intake of sweets and salty snacks did not significantly decrease, the overall improvement in appetite control and satiety contributed to reductions in total caloric intake and body weight. The mechanisms underlying these effects likely involve neurohormonal modulation, including GLP-1-mediated signaling, rather than alterations in gastric motility, as no significant changes in gastric emptying were observed [19,20].

Clinical implications and safety considerations

Despite its efficacy in reducing body weight and improving appetite regulation, semaglutide therapy carries potential risks, particularly concerning the loss of lean body mass, which may compromise muscle strength, physical performance, bone density, and overall quality of life [21]. Regulatory guidelines from the FDA recommend combining pharmacotherapy with lifestyle modifications, including dietary optimization and regular physical activity, to mitigate these risks and promote sustainable improvements in body composition [8,17]. The integration of semaglutide into obesity management should therefore be individualized, taking into account baseline health status, potential adverse effects, and patient-specific goals for weight loss and metabolic improvement.

Limitations and future directions

Although semaglutide has been approved for clinical use since 2017, data regarding its long-term impact on appetite regulation, eating behavior, and the balance between fat and lean mass remain limited. Further studies, particularly randomized trials with active comparators and diverse populations, are warranted to fully elucidate the efficacy, safety, and mechanistic pathways of semaglutide therapy. Such research is critical for optimizing treatment strategies in individuals with overweight, obesity, and insulin resistance, while minimizing adverse outcomes. The main limitations of this study include its retrospective design, lack of a control group, short follow-up period, heterogeneous BMI range, absence of standardized questionnaires assessing eating behavior, and exclusion of patients with significant adverse events. Therefore, the findings should be interpreted as exploratory and hypothesis-generating rather than confirmatory.

Conclusions

Semaglutide represents an effective adjunct to lifestyle interventions in adult women with obesity and insulin resistance, demonstrating significant and clinically meaningful reductions in body weight and BMI. While overall fat loss was evident, changes in WHR were modest, suggesting that adipose tissue redistribution may occur at a slower pace than total weight reduction. The pronounced anorexigenic effect observed in the majority of participants appears to be the primary mechanism driving these outcomes, contributing to decreased caloric intake and reduced consumption of high-energy foods. Therapy was also associated with beneficial modifications in dietary patterns, including lower total energy intake, reduced fat consumption, and a higher proportion of protein, reflecting improved nutritional behavior alongside pharmacological intervention. These findings highlight the dual benefits of semaglutide, both in directly influencing anthropometric parameters and indirectly supporting healthier eating behaviors. For optimal long-term outcomes, careful monitoring of lean body mass and sustained lifestyle modifications, including diet and physical activity, remain essential.

Disclosures and acknowledgements

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The study was conducted in accordance with the Declaration of Helsinki and good clinical practice guidelines. The protocol was approved by the Bioethics Committee of the Medical University of Silesia in Katowice (Poland) (approval number: BNW/NWN/0052/KB/261/25). All the participants were fully informed about the study objectives and procedures and provided written informed consent. Anonymity and confidentiality were strictly maintained, and the participants were free to withdraw at any stage without providing a reason.

Artificial intelligence (AI) was not used in the creation of the manuscript.

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