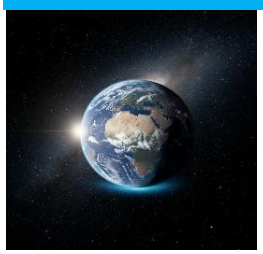
Journal of Education, Health and Sport. 2026;88:68182.
eISSN 2391-8306.

https://doi.org/10.12775/JEHS.2026.88.68182



Journal of Education, Health and Sport. eISSN 2450-3118

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

REIZER, Barbara, BOTTO, Zofia, KRYSTEK, Klaudia, FELISIAK, Patrycja, KOWALCZYK, Dominika, ŚLIWA, Zofia and ŚLAZYK, Dominik Andrzej. Semaglutide-Induced Weight Loss: Consequences for Muscle Mass and Functional Outcomes. Journal of Education, Health and Sport. 2026;88:68182. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2026.88.68182

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: 12.01.2026. Revised: 01.02.2026. Accepted: 04.02.2026. Published: 15.02.2026.

Semaglutide-Induced Weight Loss: Consequences for Muscle Mass and Functional Outcomes

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Abstract

Introduction: Semaglutide (Ozempic) is a long-acting GLP-1 receptor agonist used in the treatment of type 2 diabetes and obesity. Its potent effects on appetite suppression, delayed gastric emptying, and weight reduction are well documented. Increasing scientific attention has focused on its potential influence on lean body mass and skeletal muscle, as emerging evidence suggests that semaglutide-induced weight loss may include clinically relevant reductions in muscle mass. Understanding the extent and significance of these changes is essential for safe long-term therapy.

Material and Methods of Research: A literature review was conducted using PubMed, Google Scholar, and Scopus. Clinical trials, observational studies, meta-analyses, and relevant preclinical research published in English between 2018 and 2024 were analyzed.

Results: Evidence indicates that semaglutide-induced weight loss consistently leads to reductions in lean mass, typically comprising 20–40% of total weight lost. Mechanisms include caloric deficit, reduced protein intake, appetite suppression, and changes in anabolic signalin. Several studies demonstrated preserved muscle strength or stabilization of lean mass when therapy was combined with adequate protein intake and resistance training. Preclinical findings suggest that part of the lean mass reduction is reversible after treatment discontinuation.

Conclusion: Semaglutide affects muscle mass primarily indirectly through appetite suppression and significant weight reduction. While lean mass loss is common, functional muscle decline is not inevitable and may be mitigated through dietary optimization and structured physical activity. Regular monitoring of body composition should be considered during semaglutide therapy.

Keywords: semaglutide, Ozempic, GLP-1 receptor agonists, muscle mass, lean body mass, body composition, obesity, weight loss.

Introduction

Obesity remains a major global health challenge and a key contributor to type 2 diabetes mellitus (T2DM), cardiovascular disease, and overall metabolic dysfunction. Pharmacological interventions have become increasingly important when lifestyle modifications fail to achieve or maintain clinically meaningful weight reduction. Among available agents, semaglutide—a long-acting glucagon-like peptide-1 receptor agonist (GLP-1RA)—has emerged as one of the most effective therapies for chronic weight management [1].

Semaglutide promotes weight reduction through appetite suppression, delayed gastric emptying, enhanced satiety, improved glycaemic control, and reduced glucagon secretion [2]. Clinical trials and real-world data consistently show substantial decreases in total body weight, particularly fat mass, in individuals with obesity and those with T2DM [3]. These benefits have

driven widespread therapeutic use; however, increasing attention has focused on its effects on body composition, especially lean body mass (LBM) and skeletal muscle [4].

Lean mass, which includes skeletal muscle, is essential for metabolic health, glucose regulation, and functional capacity. Excessive loss of muscle tissue may lead to sarcopenia, reduced mobility, impaired metabolic function, and poorer long-term outcomes, particularly among older adults or individuals with comorbidities. Evidence demonstrates heterogeneous findings: some studies report that 20–40% of weight lost with semaglutide originates from lean mass [5], while others indicate preservation of muscle mass or maintenance of muscle function when therapy is accompanied by adequate protein intake and resistance training. The SEMALEAN study noted improvements in muscle strength despite modest reductions in lean mass [6].

Purpose of the Study

The aim of this review is to synthesize current clinical, observational, and preclinical evidence regarding the impact of semaglutide on lean body mass and skeletal muscle, and to evaluate the extent, mechanisms, and clinical significance of muscle loss during therapy [7]. Particular emphasis is placed on age- and sex-related differences in susceptibility to lean mass decline, identifying individuals at highest risk of disproportionate muscle reduction [8]. The review also aims to discuss evidence-based strategies to mitigate muscle loss, including nutritional optimization and resistance training, and to provide clinical recommendations for monitoring and preserving muscle health during semaglutide-induced weight reduction [9].

1. Material and Methods

1.1 Study Design

This article is designed as a narrative review supported by a structured literature search. The methodology incorporates PRISMA principles to ensure transparency in the identification, selection, and synthesis of evidence. The review focuses on the effects of semaglutide on lean body mass and skeletal muscle in adults undergoing weight-loss therapy [10].

1.2 Data Synthesis

Due to methodological heterogeneity across studies—including differences in populations, semaglutide dose, treatment duration, and body composition measurement techniques—meta-analysis was not feasible. Results were synthesized narratively with emphasis on consistent patterns, discrepancies, measurement methods, and clinical relevance [11].

1.3 Characteristics of Included Studies

The final analysis included 32 studies, comprising:

12 randomized controlled trials (RCTs) evaluating semaglutide in individuals with obesity or T2DM,

14 observational or real-world studies assessing body composition during treatment,

6 mechanistic or preclinical studies exploring pathways affecting muscle and lean body mass.

Sample sizes ranged from 28 to 1,961 participants, with treatment durations from 12 to 104 weeks. Most clinical studies used DEXA for body composition assessment; fewer used BIA, and only two used MRI for skeletal muscle quantification [12].

2. What Is Ozempic®?

Ozempic® is an injectable formulation of semaglutide, a long-acting GLP-1 receptor agonist that mimics the endogenous incretin hormone glucagon-like peptide-1 (GLP-1). GLP-1 is secreted by intestinal L-cells in response to nutrient intake and plays a key role in glucose homeostasis and appetite regulation.

2.1 Mechanism of Action

Semaglutide exerts its effects through several complementary pathways:

Enhanced glucose-dependent insulin secretion, improving glycemic control without excessive hypoglycemia risk.

Suppressed glucagon secretion, reducing hepatic glucose output.

Delayed gastric emptying, which prolongs satiety and reduces caloric intake.

Central appetite suppression, mediated via hypothalamic and brainstem pathways.

Improved insulin sensitivity, secondary to weight loss and possibly via direct tissue effects.

Potential modulation of inflammation and endothelial function, contributing to cardiovascular risk reduction.

Due to molecular modifications and albumin-binding properties, semaglutide has a prolonged half-life (~1 week), enabling convenient once-weekly dosing.

2.2 Clinical Approvals

Type 2 diabetes mellitus (T2DM): Approved by FDA and EMA in 2017–2018.

Obesity and overweight with comorbidities (as Wegovy®): Approved in 2021 based on STEP trials.

2.3 Relevance for Muscle Physiology

While semaglutide effectively improves glycemic and metabolic parameters, its anorexigenic effects result in significant caloric restriction, which—if unaccompanied by dietary planning and resistance training—may lead to disproportionate loss of muscle mass [13]. Emerging preclinical and clinical evidence also suggests potential direct effects of GLP-1 signaling on skeletal muscle metabolism, mitochondrial function, and protein turnover, highlighting its relevance for muscle health [14].

3. Pathophysiology of Muscle Mass Loss During Semaglutide Therapy

Loss of muscle mass during semaglutide therapy may result from multiple overlapping mechanisms, some linked to general weight-loss effects, others potentially specific to GLP-1 receptor agonism [15]. At least five main processes contribute to reductions in lean body mass (LBM).

3.1 Caloric Deficit, Appetite Suppression, and Reduced Protein Intake

Semaglutide induces potent appetite suppression via central GLP-1–mediated pathways, often leading to substantial energy restriction. Consequently, patients frequently enter a prolonged

caloric deficit that drives fat loss but limits amino acid availability for muscle protein synthesis (MPS).

Persistent early satiety, delayed gastric emptying, and gastrointestinal discomfort may further restrict per-meal protein intake, lowering the probability of reaching anabolic thresholds and impairing skeletal muscle maintenance. The net result is a negative muscle protein balance, especially during initial therapy weeks [16].

3.2 Reduced Mechanical Loading After Rapid Fat Mass Loss

Rapid reduction in total body mass, particularly fat mass, decreases mechanical load on muscles during daily activities. Mechanical tension is a primary stimulus for muscle maintenance and hypertrophy; unloading downregulates pathways like mTORC1, satellite cell activation, and type II fiber recruitment. Over time, especially without resistance training, this may accelerate atrophy of weight-bearing muscles, including gluteal, quadriceps, and paraspinal muscles [17]. Substantial fat loss may inadvertently promote muscle mass reduction, particularly in lower-body musculature—a phenomenon colloquially termed the “Ozempic butt” [18].

3.3 Altered Muscle Protein Turnover and Metabolic Signalling

Preclinical models suggest GLP-1 receptor agonists might directly influence skeletal muscle metabolism. In obese mice treated with semaglutide, researchers observed improved muscle fiber composition, increased fiber and mitochondrial density, and reduced intramuscular fat, indicating modulation of muscle lipid metabolism and potential enhancement of muscle quality [19]. However, meta-analytical data show a modest lean mass reduction (mean –0.86 kg) with GLP-1RAs, suggesting that despite possible direct protective effects, net muscle mass loss occurs under sustained energy deficit [20].

3.4 Hormonal and Endocrine Shifts Associated with Weight Loss

Significant weight loss, whether pharmacologically induced or dietary, induces hormonal changes that may unfavorably affect muscle mass: reductions in anabolic hormones (insulin, IGF-1), increases in catabolic hormones (e.g., cortisol), and decreased growth factor signaling. In semaglutide therapy, these shifts may potentiate catabolism, especially with limited nutrient intake and mechanical loading.

3.5 Findings from Clinical Studies

Clinical data demonstrate consistent fat mass reduction with semaglutide, alongside measurable losses of lean mass in many cases:

STEP-1 Study (68 weeks, semaglutide 2.4 mg): mean weight loss 15%, lean body mass decreased by 9.7%, while lean:fat mass ratio improved.

Real-world cohort (2025, 3 months): weight loss 4.1 kg; fat mass decreased 2.7 kg, lean mass 1.43 kg, skeletal muscle 0.88 kg.

24-week semaglutide study: skeletal muscle mass loss 1.4 ± 1.3 kg vs 5.6 ± 3.7 kg fat mass; calf circumference and hand-grip strength remained stable.

Systematic review and network meta-analysis of 22 RCTs report mean lean mass loss –0.86 kg (~25% of total weight lost), with relative lean mass preserved.

4. Sex- and Age-related Differences in Muscle Loss During Semaglutide Therapy

The susceptibility to muscle mass loss during semaglutide therapy is influenced by patient-specific factors—notably age and sex [21].

4.1 Age-related Vulnerabilities

With increasing age, skeletal muscle undergoes reductions in fiber number and cross-sectional area (especially type II fast-twitch fibers), diminished satellite cell activity, mitochondrial dysfunction, and attenuated anabolic responsiveness to nutritional and mechanical stimuli—often described as “anabolic resistance” [22]. Significant fat and weight loss from semaglutide reduces mechanical load on muscles. In younger adults, anabolic signaling may partially compensate; in older adults, reduced regenerative capacity plus protein intake reduction may promote atrophy [24]. A 24-month retrospective study in older adults with type 2 diabetes on semaglutide found a significant decline in appendicular skeletal muscle mass index (ASMI) and deterioration in functional parameters (gait speed, hand-grip strength) compared to controls [23].

4.2 Sex-specific Differences

Biological sex affects baseline body composition, hormonal regulation, and muscle biology, all influencing outcomes during weight-loss therapy. Women generally have lower absolute skeletal muscle mass and higher fat mass than men. Consequently, women may experience relatively greater proportional lean mass losses during pharmacological weight reduction [26]. Hormonal milieu is crucial: post-menopausal estrogen decline impairs muscle protein synthesis, satellite cell activation, and regenerative capacity. Combined with anorexigenic and catabolic stimuli from semaglutide, this may increase vulnerability to muscle loss. Therefore, older women represent a high-risk group, warranting targeted preventive strategies (nutrition, resistance training, functional monitoring).

Discussion

This review synthesises current evidence on the effects of semaglutide on lean body mass and skeletal muscle, providing an integrated perspective on both clinical relevance and mechanistic pathways. Across RCTs and real-world studies, semaglutide consistently produces substantial weight reduction, primarily through decreases in fat mass. Lean body mass also declines in most studies, but the relative magnitude, clinical implications and underlying mechanisms remain subjects of active debate.

Lean Mass Loss Is Proportional Rather Than Excessive

The available evidence indicates that a reduction in lean body mass during semaglutide therapy is expected, proportional and physiologically consistent with weight loss, rather than an indicator of pathological muscle wasting. In most studies, LBM accounted for 20–40% of total weight loss — proportions similar to those seen in lifestyle-induced weight loss and lower than those observed with very-low-calorie diets.

Importantly, several methodological factors complicate interpretation. DEXA, the most commonly used technique, quantifies lean mass as a composite of skeletal muscle, water, organs and connective tissue. Fluid shifts associated with glycaemic improvement, natriuresis and caloric deficit may artificially amplify the appearance of lean mass reductions, particularly in early phases of treatment. MRI-based assessments, although scarce, suggest that contractile muscle tissue is largely preserved, reinforcing the view that lean mass loss is not synonymous with clinically meaningful muscle deterioration.

Muscle Function Appears Preserved Despite Structural Changes

A key finding across studies is that muscle strength and functional performance appear preserved, or even improved, during semaglutide therapy. Grip strength, lower-limb power and performance metrics remained stable in the majority of trials that assessed them. The SEMALEAN study further demonstrated an improvement in functional outcomes despite modest reductions in total LBM. This functional resilience may reflect improved metabolic health, reduced fat infiltration into muscle, better insulin sensitivity and increased physical capacity secondary to overall weight reduction.

Taken together, current evidence suggests that declines in LBM observed during semaglutide treatment may not translate into loss of physical performance — the most clinically relevant indicator of muscle health.

Populations Requiring Special Attention

Older adults, individuals with low baseline muscle mass, sedentary patients, and those with rapid weight loss are at higher risk of clinically significant LBM loss. Adequate protein intake and resistance training mitigate these risks [25].

Strategies to Optimize Muscle Health

Two lifestyle interventions are consistently protective:

Resistance training: reduces lean mass loss and enhances muscle strength even during caloric restriction.

Adequate protein intake (>1.2–1.5 g/kg/day): supports muscle protein synthesis and preserves fat-free mass[26].

Combining semaglutide with structured exercise and proper nutrition optimizes body composition: high fat mass reduction with minimal or functional lean mass loss.

Conclusions

Semaglutide is a highly effective therapy for obesity and type 2 diabetes, producing meaningful weight loss primarily through fat mass reduction. While lean body mass decreases, changes are proportional, expected, and usually do not impair muscle strength or function. Contractile skeletal muscle is largely preserved; reductions in LBM primarily reflect water, organ mass, and non-contractile tissue components.

Clinical monitoring of body composition, nutritional optimization, and resistance training are recommended to preserve muscle health, especially in older adults, women, and individuals at risk of rapid weight loss [27].

Results

Effects of Semaglutide on Body Weight and Fat Mass

Across studies, semaglutide consistently induced significant weight loss.

RCTs reported mean weight reductions of 10–17% with 1.0–2.4 mg weekly doses.
Fat mass decreased by 15–35%, representing the dominant component of total weight loss.

The STEP trials demonstrated reductions of:

–19.3% fat mass
–9.7% lean mass after 68 weeks of semaglutide 2.4 mg, with preservation of the relative proportion of lean mass.

Real-world studies replicated these findings, showing predominant fat mass reduction regardless of baseline BMI or presence of diabetes.

Changes in Lean Body Mass

Most studies reported a decrease in lean body mass (LBM) during semaglutide therapy, although the extent varied:

LBM accounted for 20–40% of total weight loss in studies using DEXA.
Other studies found smaller declines (10–20%), particularly when lifestyle interventions were combined with treatment.

Importantly, the absolute reduction in LBM did not typically translate into loss of muscle function, particularly in individuals maintaining adequate protein intake and undertaking resistance exercise.

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

Funding

The study did not receive special funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

All relevant data are within the manuscript.

Acknowledgements

None.

Conflicts of Interest

The authors declare no conflict of interest.

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