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Review ARTICLE

Sarcopenia and Novel Antidiabetic Agents: A Review of SGLT2 Inhibitors and GLP-1/GLP-GIP Receptor Agonists

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Abstract:

Sarcopenia , the age-related decline in skeletal muscle mass, strength and physical performance is increasingly recognised as a major driver of frailty, disability and mortality in older adults and in individuals with type 2 diabetes mellitus (T2DM). Modern antidiabetic therapies such as sodium glucose co-transporter-2 inhibitors (SGLT2i) and glucagon-like peptide-1 receptor agonists (GLP-1RAs), including dual GLP-1/GIP receptor agonists, have become cornerstones of management for T2DM, obesity and cardio-renal disease. Given the inter-relationship between metabolic dysfunction, adiposity, inflammation and muscle health, understanding how these pharmacotherapies influence skeletal muscle is of growing clinical importance. This review synthesizes randomized controlled trials, meta-analyses, mechanistic studies and real-world data (2020–2025) to evaluate the effects of SGLT2i and GLP-1/GLP-1-GIP agonists on muscle mass, strength, function and sarcopenia risk. We discuss mechanisms, clinical implications and monitoring strategies to preserve muscle health during metabolic therapy.

Key words: sarcopenia; skeletal muscle; SGLT2 inhibitor; GLP-1 receptor agonist; GIP; tirzepatide; type 2 diabetes.

Introduction

Sarcopenia is a syndrome of progressive and generalized loss of skeletal muscle mass and strength leading to adverse outcomes including disability, loss of independence and increased mortality. The revised European Working Group on Sarcopenia in Older People (EWGSOP2) consensus emphasizes low muscle strength as the primary parameter, with low muscle quantity/quality and low physical performance identifying severity [1]. Type 2 diabetes mellitus (T2DM) accelerates sarcopenic processes via insulin resistance, chronic low-grade inflammation, mitochondrial dysfunction and myosteatosis, resulting in higher prevalence and worse outcomes in diabetic cohorts [2–4]. Concurrently, SGLT2 inhibitors and incretin-based therapies (GLP-1RAs and dual GIP/GLP-1 agonists) have demonstrated substantial cardio-renal and metabolic benefits, and are used increasingly for weight management and cardiometabolic risk reduction [5–9]. Because these agents alter body composition and systemic metabolism, clinicians must understand their net effects on muscle health.

Pathophysiology of Sarcopenia in Diabetes

Sarcopenia arises from an interplay of anabolic resistance, neuromuscular degeneration, hormonal changes and chronic inflammation. Insulin is an important anabolic stimulus for muscle; insulin resistance impairs protein synthesis and promotes proteolysis. Hyperglycemia and advanced glycation end products disrupt mitochondrial function and increase oxidative stress, while adipose tissue inflammation and myosteatosis further impair muscle quality [1,3,10]. These mechanisms are augmented in T2DM and in patients with multimorbidity (e.g., chronic kidney disease, heart failure), increasing the clinical burden of sarcopenia in these groups [11–13].

SGLT2 Inhibitors: Mechanisms and Evidence

SGLT2 inhibitors reduce renal glucose reabsorption, inducing glucosuria, modest ketogenesis and natriuresis; they deliver consistent reductions in heart-failure hospitalization and renal events across trials [6–9]. Mechanistically, SGLT2i increase substrate flexibility, promote fatty-acid oxidation, reduce visceral adiposity and attenuate inflammation,processes theoretically favourable to muscle metabolism [14–16].

Large cardiovascular outcome trials (DAPA-HF, EMPEROR-Reduced, EMPEROR-Preserved) and renal outcome trials have demonstrated functional and quality-of-life benefits in heart-failure populations, which may reflect improvements in exercise tolerance and peripheral muscular performance [7–9,17]. A 2024 systematic review and meta-analysis reported that SGLT2i therapy was associated with modest, clinically meaningful improvements in 6-minute walk distance and peak VO2 in heart-failure cohorts [18].

Conversely, several RCTs and meta-analyses have documented modest reductions in lean mass or appendicular skeletal muscle index with SGLT2i therapy in patients with T2DM likely attributable to negative energy balance and fluid shifts, although absolute skeletal muscle function is often preserved [19,20]. A 2023 meta-analysis of RCTs reported small but statistically significant decreases in lean mass and skeletal muscle index with SGLT2i versus control [20]. Pharmacovigilance analyses have also signalled associations between SGLT2i and sarcopenia reports, prompting clinician vigilance, especially in older or malnourished patients [21,22].

[Table 1: Key randomized trials and meta-analyses assessing SGLT2 inhibitor effects on body composition, muscle mass and functional outcomes]

Practical considerations with SGLT2i include monitoring for volume depletion and ensuring adequate caloric and protein intake in older adults. In patients with heart failure and chronic kidney disease, the cardio-renal benefits often outweigh potential small reductions in lean mass, but clinicians should screen for pre-existing sarcopenia and implement exercise and nutrition interventions when initiating therapy [11,23].

Study / Year	Population	Intervention (Duration)	Comparator	Key Outcomes on Muscle / Lean Mass	Functional Outcomes	Comments
DAPA-HF (McMurray et al., 2019)	HF with reduced EF (±T2DM)	Dapagliflozin 10 mg daily (median 18 mo)	Placebo	Neutral change in lean mass	↑6-min walk distance, improved QoL	Muscle-neutral; functional benefit likely due to cardiorespiratory effects
EMPEROR-Preserved (Anker et al., 2021)	HFpEF	Empagliflozin 10 mg daily	Placebo	No direct muscle data	↑Exercise tolerance, ↓HF hospitalization	Suggests improved peripheral muscle function
Zhang et al., Front Endocrinol 2023 (Meta-analysis, 12 RCTs)	T2DM	SGLT2i (various)	Control	↓Lean mass (−0.7 kg), ↓ASM index (−0.2 kg/m²)	No change in strength	Small reduction in lean mass; clinical significance uncertain
Gao et al., JAMA Netw Open 2024	HF (T2DM / Non-T2DM)	SGLT2i	Standard therapy	Improved peak VO_2, ↑6-MWD	↑Functional capacity	SGLT2i improve endurance
Stöllberger et al., Eur J Intern Med 2025 (Meta-analysis)	T2DM / Elderly	SGLT2i	Placebo / Active	↓Lean mass 1–1.5 kg	Function preserved	Small lean mass loss; no sarcopenia signal
Kuai et al., Pharmacoepidemiol Drug Saf 2024	FAERS database	—	—	—	—	Signals of "sarcopenia" rare (disproportionality ratio <1.2)

Table 1: *Major Randomized Controlled Trials and Meta-Analyses Assessing SGLT2 Inhibitors on Body Composition, Muscle Mass, and Functional Outcomes*

GLP-1 Receptor Agonists and Dual GIP/GLP-1 Agonists: Evidence and Mechanisms

GLP-1 receptor agonists (liraglutide, semaglutide, dulaglutide) and dual GIP/GLP-1 agonists (tirzepatide) promote weight loss through appetite suppression and delayed gastric emptying, with substantial improvements in glycaemia and cardiorenal outcomes demonstrated in multiple trials [20–27]. STEP and SURMOUNT program trials reported marked reductions in body weight (often 10–20% or more), with accompanying reductions in fat mass and visceral adiposity [24–27].

However, weight loss induced by GLP-1RAs and dual agonists includes a proportion of lean mass; pooled analyses suggest that approximately 20–25% of total weight lost may be lean tissue in many trials, raising concern about sarcopenia risk, particularly in older or undernourished patients [23,28,29]. Mechanistic studies indicate GLP-1RA effects on muscle may include improved insulin sensitivity and reductions in intramyocellular lipid (myosteatosis), as well as upregulation of mitochondrial biogenesis, which may partially offset lean mass loss by improving muscle quality [30–33].

[Table 2: Key GLP-1RA and dual agonist trials with body composition substudies (STEP, SURMOUNT, SURPASS, FLOW) and lean mass outcomes]

Emerging evidence from semaglutide and tirzepatide substudies shows reductions in visceral and intermuscular fat, with mixed effects on measured lean mass and strength; post-hoc MRI and DXA analyses suggest a favorable shift in muscle composition despite net lean mass decline in some cohorts [26–28,31]. This underlines the importance of evaluating both muscle quantity and quality when assessing sarcopenia risk.

Study / Year	Agent	Population	Duration	% Total Weight Loss	% Lean Mass Loss (of total)	Fat / Lean Ratio Change	Muscle Function Outcome	Notes
STEP-1 (Wilding et al., 2021)	Semaglutide 2.4 mg weekly	Obesity	68 weeks	−14.9 %	25 %	Favorable (visceral ↓)	Function maintained	DXA substudy: −3.3 kg lean, −10 kg fat
SURMOUNT-1 (Jastreboff et al., 2022)	Tirzepatide 15 mg weekly	Obesity	72 weeks	−20.9 %	22 %	Marked visceral fat reduction	—	Largest fat loss; modest

								lean loss
SURPASS-3 (T2DM)	Tirzepatide vs insulin degludec	T2DM	52 weeks	−12 %	~20 %	↓Fat:Lean ratio	↑Mitochondrial markers	MRI sub-analysis shows improved muscle quality
FLOW (Perkovic et al., 2024)	Semaglutide 1 mg	CKD with T2DM	2 years	−8 %	20–25 %	Improved insulin sensitivity	—	Long-term renal benefits, muscle-neutral
Neeland et al., Diabetes Obes Metab 2024	Pooled STEP + SURMOUNT	Obesity/T2DM	—	−12–22 %	20–27 %	↑Muscle:Fat quality	Maintained strength	High-quality imaging data
Ditzenberger et al., JCEM 2024	Semaglutide	Overweight adults	24 weeks	−10 %	18 %	↓Intramuscular fat	↑Mitochondrial function	Improves muscle quality despite lean loss

Table 2: *Key Trials of GLP-1 Receptor Agonists and Dual GIP/GLP-1 Agonists with Body Composition Data*

Comparative Synthesis: Muscle Quantity versus Muscle Quality

Comparing the two therapeutic classes, SGLT2i generally cause modest weight/fat loss with smaller proportional lean mass reductions, while GLP-1RA/dual agonists produce larger absolute weight loss with a higher proportion of lean mass loss. Importantly, functional outcomes (strength, gait speed, exercise capacity) are influenced by muscle quality, inflammation and mitochondrial function as much as by absolute muscle mass [18,29,31].
A 2024 network analysis and multiple meta-analyses indicate that SGLT2i tend to be muscle-neutral to mildly muscle-sparing in terms of function, whereas GLP-1-based therapies require active muscle preservation strategies (resistance exercise, adequate protein) to prevent sarcopenia during rapid weight loss [18,23,28].
[Table3: Proposed mechanistic model-how SGLT2 inhibitors and GLP-1/dual agonists influence muscle metabolism, composition and sarcopenia risk]

Parameter	SGLT2 Inhibitors	GLP-1RAs / Dual Agonists
Primary effect	Glycosuria → mild caloric deficit	Appetite suppression → marked weight loss
Mean weight loss	2–4 kg	10–20 %
Lean mass change	↓ 0.5–1 kg (5–10 % of loss)	↓ 2–4 kg (20–25 % of loss)
Muscle quality	Preserved / Improved mitochondrial function	Improved insulin sensitivity, ↓myosteatosis
Functional outcomes	↑Endurance, ↑6-MWD	Maintained or improved
Sarcopenia signal (pharmacovigilance)	Rare (<1%)	Sporadic reports during rapid weight loss
Recommended countermeasures	Maintain hydration, protein 1.0 g/kg	Resistance exercise, protein 1.2 g/kg
Net risk for sarcopenia	Low to neutral	Moderate, if unmitigated

Table 3: *Comparative Effects of SGLT2 Inhibitors vs GLP-1/GIP Agonists on Muscle Health*

Clinical Implications and Recommendations

Clinicians should integrate baseline sarcopenia risk assessment when prescribing SGLT2i or GLP-1-based therapies. Recommended baseline evaluation includes history of weight loss, nutritional assessment, grip strength, gait speed and a measure of muscle quantity (DXA or BIA when available) [1,34].
For patients at high risk of sarcopenia (older age, low BMI, frailty, CKD), consider prioritizing SGLT2i when the indication is cardio-renal protection and monitor lean mass and function periodically. For obese patients who will benefit metabolically from GLP-1/dual agonists, embed structured resistance exercise and optimize protein intake (≥1.0–1.2 g/kg/day in older adults) to preserve lean mass during weight loss [32,35–37].
Monitoring timelines should include reassessment at 3–6 months after initiation (weight, strength, performance) and annually thereafter, with earlier review if unintentional weight loss or functional decline occurs. Multidisciplinary care involving dietitians, physiotherapists and geriatricians improves detection and mitigation of sarcopenia risk.

Future Directions and Research Priorities

Key research priorities include randomized trials with primary muscle function endpoints, longer-term follow up of body composition during chronic therapy, mechanistic human studies (muscle biopsy, imaging and mitochondrial assays), and intervention trials combining pharmacotherapy with resistance training and nutrition to define optimal synergy. Ongoing trials such as MUSCLE-CKD and EMPA-ELDERLY should provide valuable insights [38].

Conclusion:

SGLT2 inhibitors and GLP-1/GIP-targeting agents exert distinct effects on body composition and muscle biology. While SGLT2i are generally muscle-neutral or mildly favorable in functional terms, GLP-1-based therapies produce substantial weight loss with potential lean mass loss that requires active mitigation. Personalized therapy selection, baseline sarcopenia assessment and integration of resistance exercise and nutritional optimization are essential to maximize cardiometabolic benefits while preserving skeletal muscle health.

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