

Sarcopenic obesity in older adults: the dual burden of muscle loss and fat gain. Clinical consequences, diagnostic challenges, and integrated management strategies

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Abstract

Sarcopenic obesity is an emerging geriatric syndrome defined by the simultaneous presence of reduced skeletal muscle mass and function alongside excess adiposity. As populations age and obesity prevalence rises, this condition has grown in clinical importance, carrying risks of frailty, falls, disability, metabolic syndrome, cardiovascular disease, poor quality of life, and premature mortality that exceed those associated with either condition independently.

This narrative review synthesizes current evidence on the epidemiology, pathophysiology, clinical consequences, diagnostic challenges, and management of sarcopenic obesity in older adults. Its development involves a complex interplay of age-related muscle atrophy, adipose tissue dysfunction, chronic low-grade inflammation, insulin resistance, hormonal alterations, mitochondrial impairment, and diminished regenerative capacity, mechanisms that collectively reinforce one another in a self-perpetuating cycle.

Diagnosis is hampered by the absence of a universally accepted definition; current frameworks differ in their choice of muscle mass indices, strength thresholds, performance measures, and adiposity cut-offs. Recent international consensus statements have proposed stepwise approaches to improve clinical recognition.

Management centers on progressive resistance exercise combined with optimized protein intake and multidisciplinary care. Emerging pharmacological options, notably glucagon-like peptide-1 receptor agonists, offer important metabolic benefits but require careful use in older adults because of the risk of lean mass loss. Early detection, standardized diagnostic frameworks, and integrated lifestyle interventions are essential to mitigate the functional and metabolic burden of sarcopenic obesity.

Key words: sarcopenic obesity, older adults, sarcopenia, obesity, frailty, body composition, diagnosis, management.

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Introduction

Population aging is one of the defining demographic shifts of the 21st century. Advances in healthcare, nutrition, and living standards have extended life expectancy worldwide, creating a rapidly expanding older population and new priorities for geriatric medicine.¹ Aging brings progressive changes in body composition, most notably a decline in skeletal muscle mass and strength alongside an accumulation of adipose tissue, changes that reduce mobility, limit functional capacity, and heighten susceptibility to chronic disease.^{2,3}

Sarcopenia, defined as the age-related loss of muscle mass and function, is a principal driver of physical decline in later life. Concurrently, obesity has become increasingly prevalent among older adults. When these two states coexist, they constitute sarcopenic obesity, a complex clinical entity characterized by excess

adiposity combined with low muscle mass or impaired muscle performance.⁴⁻⁶

Older adults with sarcopenic obesity face a substantially greater burden than those with either condition in isolation, because the coexistence of muscle deficit and adiposity excess creates compounding physiological harm.⁷⁻⁹ Specifically, reduced muscle strength and poor physical performance curtail daily function, while surplus fat mass sustains chronic inflammation, drives insulin resistance, and amplifies metabolic dysregulation.^{10,11} Together, these processes fuel a progressive cycle of muscle loss and fat gain that hastens functional decline.

Given its multifactorial nature and rising clinical relevance, early recognition and effective management of sarcopenic obesity are urgent priorities. Integrated strategies combining exercise, nutritional optimization, and multidisciplinary care can help preserve

muscle function, curtail adiposity, and improve outcomes.^{12,13} Despite the existence of several narrative reviews on this topic, meaningful gaps persist in the published literature. Most prior reviews predate the 2025 Asia-Oceania consensus diagnostic framework and do not critically evaluate the emerging role of glucagon-like peptide-1 (GLP-1) receptor agonists in the context of sarcopenic obesity.¹⁴ Furthermore, few have explicitly stratified management recommendations according to the strength of available evidence or distinguished investigational therapies from those supported by robust trial data. The present review addresses these gaps by providing an updated, evidence-graded synthesis that integrates the most recent diagnostic criteria and discusses novel pharmacological therapies with specific attention to their implications for muscle preservation in older adults.¹⁵

Methods

This narrative review was conducted to summarize and critically appraise current evidence on sarcopenic obesity in older adults. A systematic literature search was performed in four databases, PubMed, Scopus, Web of Science, and Google Scholar, covering publications from January 2000 to December 2025. The following Boolean search strings were applied: (i) (“sarcopenic obesity” OR “sarcopenia” OR “muscle wasting”) AND (“obesity” OR “adiposity” OR “body composition”) AND (“older adults” OR “elderly” OR “aging” OR “ageing” OR “geriatric”); (ii) (“sarcopenic obesity”) AND (“frailty” OR “falls” OR “disability” OR “metabolic syndrome” OR “cardiovascular disease” OR “mortality”); (iii) (“sarcopenic obesity”) AND (“diagnosis” OR “screening” OR “criteria” OR “DXA” OR “BIA”); (iv) (“sarcopenic obesity”) AND (“exercise” OR “resistance training” OR “nutrition” OR “protein” OR “pharmacotherapy” OR “GLP-1” OR “myostatin”).

Studies were included if they: (i) were original research articles, systematic reviews, meta-analyses, narrative reviews, or international consensus statements; (ii) involved adults aged 60 years or older; (iii) addressed the epidemiology, pathophysiology, clinical consequences, diagnosis, or management of sarcopenic obesity; and (iv) were published in English. Studies were excluded if they were case reports, conference abstracts, or editorials without original data, if they focused exclusively on pediatric or adolescent populations, if they were unrelated to body composition or aging, or if they were published in languages other than English. Articles were assessed by title, then abstract, then full text. Approximately 320 records were initially identified; after removal of duplicates and staged screening, 85 articles were selected for inclusion. Evidence was synthesized narratively to reflect the current state of knowledge and highlight areas of clinical and research priority.^{16,17}

Epidemiology and global burden

Sarcopenic obesity is increasingly recognized as a major public health concern among older populations. The age-related decline in skeletal muscle mass and strength, occurring alongside a rise in fat mass, particularly visceral fat, underpins its growing prevalence in later life. Reported rates vary substantially depending on the diagnostic framework, adiposity assessment method, and population studied. A meta-analysis of 50 studies including 86,285 older adults estimated a pooled global prevalence of 11%, rising to 23% in those aged 75 years or older and to 16% in hospitalized populations.¹⁸

Regional differences are also pronounced. In Asian populations, metabolic risk often emerges at lower body mass index (BMI) values, with central adiposity serving as a more sensitive indicator than total body fat. This has prompted the development of region-specific diagnostic cut-offs that better reflect the body composition characteristics of these groups.¹⁹

Beyond prevalence statistics, sarcopenic obesity carries a substantial functional and economic burden. Affected older adults experience higher rates of frailty, impaired mobility, falls, disability, metabolic disease, and healthcare utilization than those with only obesity or only sarcopenia.²⁰ As the global older population continues to expand, the clinical and economic impact of sarcopenic obesity is expected to grow, making early detection and preventive strategies increasingly important within geriatric care systems.^{18,19}

Pathophysiology of sarcopenic obesity

Age-related muscle loss

The progressive decline in skeletal muscle mass, strength, and quality that characterizes sarcopenia begins as early as the fourth decade of life, with losses of approximately 3–8% per decade accelerating thereafter.²¹ This atrophy is both quantitative and structural: aging selectively reduces the number and cross-sectional area of type II fast-twitch fibers, which are critical for rapid force generation, postural correction, and functional mobility.²¹ Satellite cell activity, the principal mechanism for muscle repair and regeneration, also declines with age, limiting the capacity to recover from injury or disuse.²⁰

At the molecular level, aging disrupts the equilibrium between muscle protein synthesis and degradation. Activation of the ubiquitin-proteasome system (UPS) is upregulated through muscle-specific E3 ubiquitin ligases, particularly MuRF1 (muscle RING-finger protein-1) and atrogin-1, which target myofibrillar proteins for proteolytic degradation.^{20,21} Simultaneously, anabolic signaling through the mammalian target of rapamycin (mTOR) pathway becomes progressively blunted, a phenomenon termed anabolic resistance, reducing the synthetic response to amino acids, mechanical loading, and insulin stimulation.²¹ Age-related denervation of fast-twitch motor units, with subsequent reinnervation by slow-twitch neurons, further contributes to fiber-type shifts, loss of explosive strength, and heightened fatigability.²⁰

Adipose tissue dysfunction

In sarcopenic obesity, expanded adipose tissue, especially visceral and ectopic depots, functions as an active endocrine and inflammatory organ rather than a passive energy store. Visceral fat releases pro-inflammatory cytokines, including interleukin-6 and tumor necrosis factor- α , which stimulate proteolytic pathways in skeletal muscle, suppress protein synthesis, and accelerate net muscle catabolism.²⁰ Adipokine signaling is also disrupted: leptin resistance develops while beneficial adipokine effects diminish, further impairing metabolic homeostasis.^{20,22} The deposition of lipid within muscle fibers (myosteatosis) reduces contractile quality and worsens insulin sensitivity.

At the cellular level, adipose macrophages shift from an anti-inflammatory M2 phenotype to a pro-inflammatory M1 phenotype, amplifying cytokine release and activating the nuclear factor- κ B pathway in skeletal muscle.²³ This cascade upregulates proteolytic enzyme expression while suppressing myogenesis. Adipose-derived exosomes carrying pro-inflammatory microRNAs have additionally

been shown to modulate muscle metabolism through paracrine signalling, representing a novel adipose–muscle crosstalk mechanism.²⁰ Intramyocellular lipid accumulation promotes the synthesis of diacylglycerols and ceramides, which interfere with insulin receptor substrate-1 phosphorylation and downstream Akt/mTOR signalling, thereby blunting anabolic responses to both nutrients and physical activity.^{22,23}

Hormonal alterations

Age-related endocrine changes create a hormonal milieu that favors fat accumulation over muscle maintenance. Declining testosterone, growth hormone (GH), and insulin-like growth factor-1 (IGF-1) collectively reduce muscle protein synthesis, impair satellite cell activation, and promote adipogenesis.^{21,22} Testosterone deficiency is directly associated with reduced grip strength and increased visceral fat mass.

The GH/IGF-1 axis decline specifically attenuates PI3K/Akt signaling, which is required for mTOR-mediated protein synthesis and for suppressing FoxO transcription factors that drive atrogenic expression (atrogin-1, MuRF1).^{21,22} In women, the menopausal withdrawal of estrogen redistributes adipose tissue toward visceral depots, heightens systemic inflammation, and accelerates loss of muscle quality through sex-specific pathogenic pathways.²² Elevated cortisol, which tends to rise with chronic stress and advancing age, activates glucocorticoid receptors in skeletal muscle, suppressing mTOR-mediated synthesis while simultaneously engaging the UPS to promote protein breakdown, thereby reinforcing the catabolic state.^{20,22}

Mitochondrial dysfunction

Mitochondrial impairment connects aging, obesity, and sarcopenia at the cellular level. Reduced oxidative phosphorylation capacity lowers adenosine triphosphate production and diminishes the energetic efficiency of muscle contraction, while excess reactive oxygen species (ROS) generation promotes oxidative damage to contractile proteins and cellular membranes.^{20,24} Mitochondrial dysfunction additionally compromises satellite cell activation and proliferation, limiting the regenerative response to muscle damage.²⁰

Mechanistically, aging reduces the activity of peroxisome proliferator-activated receptor gamma coactivator 1- α , the master regulator of mitochondrial biogenesis, resulting in fewer and functionally inferior mitochondria.²⁴ Impaired mitophagy allows dysfunctional organelles to accumulate, perpetuating ROS production and triggering pro-apoptotic cascades that contribute to myonuclear loss and fiber atrophy.^{20,24} In sarcopenic obesity, free fatty acid overload within muscle cells further compromises electron transport chain function at complexes I and III, establishing a feed-forward loop of lipotoxicity, oxidative stress, and worsening metabolic dysfunction.²⁴

The vicious cycle of sarcopenic obesity

Sarcopenic obesity is best conceptualized as a self-reinforcing cycle in which obesity and muscle loss mutually amplify one another. Excess adiposity drives inflammation, insulin resistance, hormonal dysregulation, and myosteatosis, all of which accelerate muscle catabolism. Conversely, the reduction in muscle mass lowers basal metabolic rate, diminishes spontaneous physical activity, and impairs glucose disposal, promoting further fat deposition.^{20,23} This bidirectional interaction creates a pathological state qualitatively dis-

tinct from either obesity or sarcopenia in isolation, one in which the functional and metabolic consequences of each condition are substantially magnified by the other, and explains why early, targeted intervention is so clinically important.

Figure 1 illustrates the key pathophysiological mechanisms underlying sarcopenic obesity, depicting how age-related muscle atrophy, adipose tissue dysfunction, hormonal changes, insulin resistance, and mitochondrial impairment interact to sustain a self-reinforcing cycle of progressive muscle loss and fat gain. This is an original schematic figure prepared by the authors based on published mechanisms described in references.²⁰⁻²⁴

Clinical consequences

The clinical significance of sarcopenic obesity extends well beyond body composition abnormalities. By combining the mechanical and metabolic harm of excess adiposity with the functional consequences of low muscle mass, it confers a synergistic risk that exceeds the additive burden of its component conditions.^{25,26}

Frailty, loss of strength, and impaired mobility represent its most prevalent functional consequences. Reduced grip strength, impaired balance, slowed gait, and diminished physiological reserve render older adults vulnerable to frailty and progressive dependence in daily life. Population-based studies have confirmed that sarcopenic obesity is significantly associated with both prefrailty and established frailty, reinforcing its standing as a high-risk geriatric phenotype.²⁶ Weakness and poor performance further translate into difficulty rising from a chair, ascending stairs, and managing other routine tasks that underpin independent living.²⁵

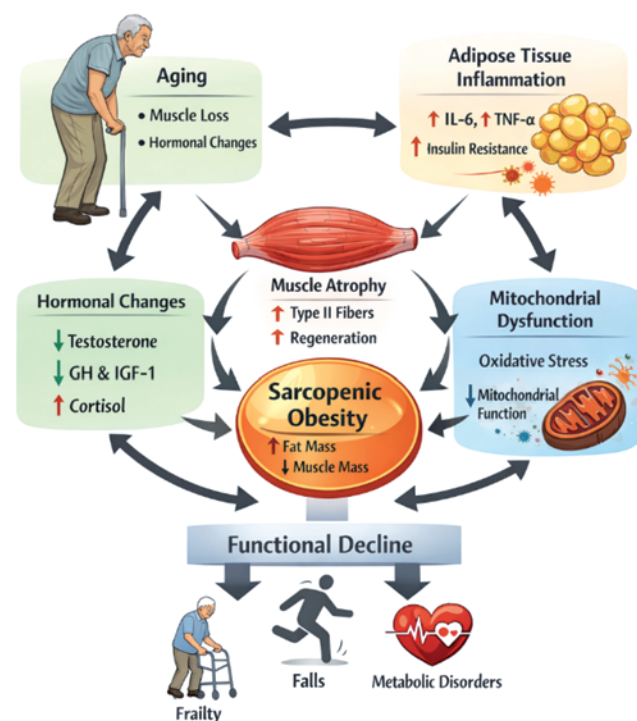


Figure 1. Pathophysiological mechanisms of sarcopenic obesity in older adults. IL-6, interleukin-6; TNF, tumor necrosis factor; GH, growth hormone; IGF-1, insulin-like growth factor-1.

Falls represent another major and potentially preventable consequence. The combination of weak musculature and excess body mass impairs postural stability, reaction time, and balance recovery after perturbation. A recent population-based cohort study demonstrated a significantly elevated fall risk among older adults with sarcopenic obesity relative to those without this phenotype.²⁷

Metabolic complications are equally prominent. Excess visceral fat drives chronic inflammation and insulin resistance, while reduced muscle mass curtails glucose disposal, together generating a high cardiometabolic burden that encompasses metabolic syndrome, type 2 diabetes, and atherosclerotic cardiovascular disease.^{28,29} The risk of major cardiovascular events is independently elevated in sarcopenic obesity even after adjustment for traditional risk factors.²⁹

Disability and reduced quality of life are frequent sequelae. Chronic weakness and impaired mobility restrict participation in social and daily activities, increase the need for long-term care, and are associated with poorer overall physical and mental health ratings.^{25,30}

The mortality implications of sarcopenic obesity are also well established. Large longitudinal cohort analyses indicate that older adults with concurrent muscle and fat abnormalities have inferior long-term survival compared with those who are free of both.³¹ Collectively, these findings confirm that sarcopenic obesity is a high-risk clinical condition demanding systematic identification and targeted intervention.

Figure 2 illustrates the major clinical outcomes associated with sarcopenic obesity in older adults. Sarcopenic obesity contributes to frailty, mobility impairment, and metabolic complications, which increase the risk of falls, hospitalization, and mortality. The figure also highlights important metabolic consequences, including metabolic syndrome, type 2 diabetes mellitus, insulin resistance, and cardiovascular disease. Overall, it demonstrates the progression of sarcopenic obesity from a body composition

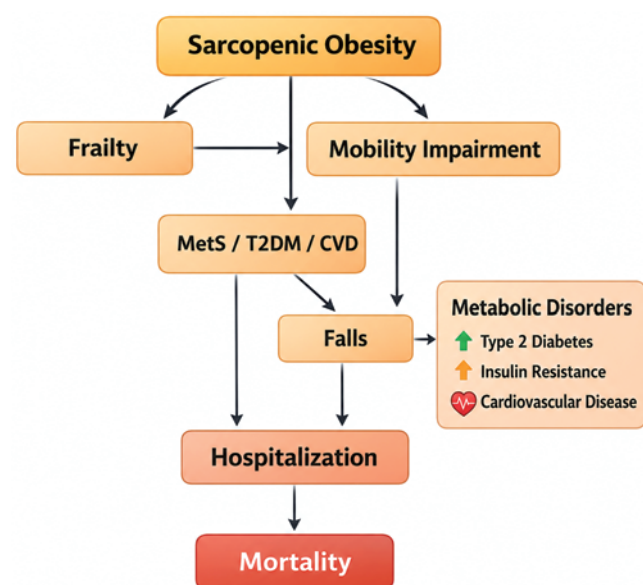


Figure 2. Clinical outcomes associated with sarcopenic obesity. MetS, metabolic syndrome; T2DM, type 2 diabetes mellitus; CVD, cardiovascular disease.

abnormality to significant functional decline and adverse health outcomes. This is an original schematic figure prepared by the authors based on clinical outcome evidence summarized in references.²⁵⁻³¹

Diagnostic criteria and challenges

The diagnosis of sarcopenic obesity remains one of the most challenging aspects of its clinical management, largely because no single universally accepted definition exists. Different expert groups have used varying combinations of muscle mass indices, strength thresholds, physical performance measures, and adiposity cut-offs, producing marked heterogeneity in reported prevalence and limiting cross-study comparisons.¹⁷

In practice, evaluation typically encompasses four domains: muscle mass, muscle strength, physical performance, and adiposity. Muscle mass is most commonly estimated by dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA), which offer acceptable clinical practicality. Computed tomography and magnetic resonance imaging provide superior precision regarding muscle quantity and quality but remain confined to research settings because of cost and limited availability.^{6,14} In older adults with obesity, accurate assessment is further complicated because excess fat mass can mask underlying low lean mass, particularly when BMI is used as a proxy.

Muscle strength and physical performance have gained increasing prominence in recent diagnostic frameworks because they reflect functional decline more sensitively than tissue quantity alone. Handgrip dynamometry is the most widely adopted and validated measure of muscle strength, whereas gait speed and the Short Physical Performance Battery (SPPB) are standard tools for assessing physical function.¹⁶ Both domains are strongly predictive of frailty, hospitalization, and mortality in older populations.¹⁶

Table 1 provides a structured comparison of the diagnostic criteria recommended by five major international consortia for sarcopenic obesity, including specific cut-off values for each component.

The 2025 Asia-Oceania consensus has introduced a practical three-step clinical pathway for screening, confirmation, and intervention that provides actionable guidance for community and hospital settings.¹⁹ Recommended screening tools include the Strength, Assistance with walking, Rise from a chair, Climb stairs, and Falls (SARC-F) questionnaire, calf circumference measurement, and the Finger Ring test, combined with assessment of adiposity using BMI and waist circumference. Confirmatory diagnosis requires evaluation of muscle mass by DXA or BIA together with grip strength and physical performance, with central obesity carrying particular weight.¹⁹

Persistent challenges include the limited discriminatory validity of BMI in older adults (in whom normal BMI may conceal low lean mass), ethnic variation in body composition independent of adiposity level, and inadequate clinical awareness among non-specialist practitioners. Greater harmonization of diagnostic standards is needed to enable meaningful comparisons across studies and to support routine clinical application.

Figure 3 depicts the stepwise diagnostic pathway for sarcopenic obesity, beginning with population screening using SARC-F, calf circumference, the Finger Ring test, BMI, and waist circumference, followed by confirmatory assessment of handgrip

strength, gait speed, SPPB, and muscle mass by DXA or BIA, culminating in a definitive clinical diagnosis. This figure is adapted conceptually from international sarcopenia and sarcopenic obesity diagnostic frameworks, particularly references.^{16,19}

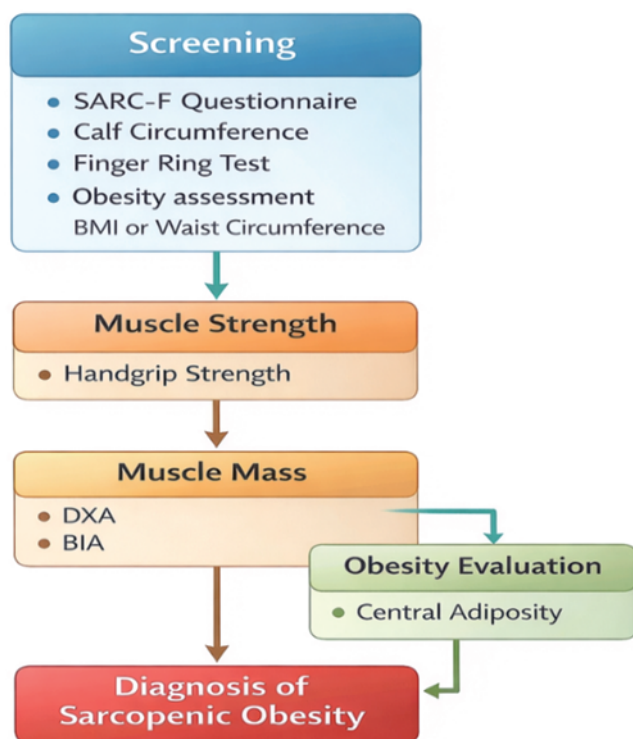


Figure 3. Screening and diagnostic approach for sarcopenic obesity in older adults. SARC-F, Strength, Assistance with walking, Rise from a chair, Climb stairs, and Falls; DXA, dual-energy X-ray absorptiometry; BIA, bioelectrical impedance analysis.

Integrated management strategies

The interventions discussed below are stratified by the current strength of supporting evidence. Recommendations are classified as supported by high-quality evidence (consistent findings from randomized controlled trials and meta-analyses), moderate evidence (findings from smaller or heterogeneous trials), or emerging/investigational (preliminary data from early-phase studies or mechanistic research).

Exercise interventions: high-quality evidence

Resistance training is the most thoroughly evidenced intervention for sarcopenic obesity and is considered first-line therapy.^{7,11} Progressive loading directly stimulates muscle protein synthesis, promotes hypertrophy, augments grip strength and lower-limb power, and enhances balance and physical performance in older adults.⁷ Sustained resistance exercise also improves insulin sensitivity and functional independence, making it the single most important modifiable determinant of outcome in this population. Multiple systematic reviews and meta-analyses consistently support resistance training as a Level I intervention for improving strength, physical performance, and body composition in older adults with sarcopenic obesity.^{7,11,25}

Combining resistance exercise with aerobic activity can provide complementary benefits by supporting fat oxidation and cardiovascular health; however, preservation of lean mass should remain the primary objective whenever weight reduction is pursued.^{11,25} Programs should be individualized, supervised where feasible, and progressively adjusted to maintain physiological challenge.

Nutritional therapy: moderate-to-high-quality evidence

Nutritional management is integral to the treatment of sarcopenic obesity because many older adults have inadequate muscle-supportive nutrition despite calorically sufficient or excessive diets. A higher dietary protein intake, ideally 1.2–1.6 g/kg body weight per day, distributed evenly across meals, is recommended to overcome

Table 1. Comparison of diagnostic criteria proposed by major international consortia for sarcopenic obesity.

Consortium/guideline	Muscle mass criterion	Muscle strength criterion	Physical performance criterion	Obesity criterion
EWGSP2 (2019) [Cruz-Jentoft <i>et al.</i>]	ASM/height ² <7.0 kg/m ² (M) <5.5 kg/m ² (F) - DXA	Handgrip <27 kg (M) <16 kg (F)	Gait speed ≤0.8 m/s; SPPB ≤8; TUG ≥20 s	Combined with obesity assessment per local criteria
FNIH Sarcopenia Project [Studenski <i>et al.</i>]	ASM/BMI <0.789 (M) <0.512 (F)	Handgrip <26 kg (M) <16 kg (F)	Gait speed ≤0.8 m/s	Not separately specified; BMI or WC thresholds used contextually
ESPEN/EASO (2022) [Barazzoni <i>et al.</i>]	Reduced ASM by DXA or BIA (sex/ethnicity-specific cut-offs)	Reduced handgrip or impaired chair-stand	Gait speed, SPPB, or TUG (population-specific thresholds)	BMI ≥30 kg/m ² or WC ≥88 cm (F) / >102 cm (M)
Asia-Oceania Consensus (2025) [Chen <i>et al.</i>]	ASM/height ² or ASM/weight (population-specific cut-offs)	Handgrip <28 kg (M) <18 kg (F)	Gait speed <1.0 m/s; SPPB ≤9	BMI ≥25 kg/m ² (Asian) or WC ≥90 cm (M) / ≥80 cm (F)
AWGS 2019 [Chen <i>et al.</i>]	ASM/height ² <7.0 kg/m ² (M) <5.4 kg/m ² (F) - DXA	Handgrip <28 kg (M) <18 kg (F)	6-m walk <1.0 m/s; SPPB ≤9; 5-chair stands ≥12 s	Combined with regional BMI/WC thresholds

ASM, appendicular skeletal muscle mass; DXA, dual-energy X-ray absorptiometry; BIA, bioelectrical impedance analysis; BMI, body mass index; WC, waist circumference; SPPB, Short Physical Performance Battery; TUG, Timed Up-and-Go test; M, male; F, female. Cut-off values are provided as published in the respective source documents.^{16–19}

age-related anabolic resistance and maintain muscle mass during any phase of weight management.^{17,25}

Leucine supplementation may further enhance muscle protein synthesis, particularly when used alongside resistance exercise; however, the supporting evidence is derived primarily from short-term trials with moderate sample sizes, and data on long-term functional outcomes remain limited.^{20,24} Vitamin D repletion is important in deficient individuals, given its role in neuromuscular function, and is supported by moderate evidence. Omega-3 fatty acids have shown anti-inflammatory and anabolic potential in some clinical investigations, though findings across studies have been inconsistent and long-term benefit has not been established.²⁴ Dietary consultation should be a routine component of care, with nutritional plans designed to achieve body recomposition, reducing fat while preserving or increasing lean mass rather than weight loss alone.^{17,19}

Glucagon-like peptide-1 receptor agonists: emerging evidence and the muscle-loss concern

GLP-1 receptor agonists, including semaglutide and tirzepatide, have established a prominent role in obesity and type 2 diabetes management, producing clinically significant weight reductions of 15-22% in pivotal randomized trials.^{32,33} Their metabolic benefits extend to improved glycemic control, reduced cardiovascular risk, and attenuated systemic inflammation, making them attractive candidates for consideration in older adults with cardiometabolic comorbidities.³²

A critical concern specific to older adults with sarcopenic obesity, however, is the composition of weight lost. Evidence from large clinical trials indicates that approximately 25-40% of the total mass reduction achieved with GLP-1 receptor agonists consists of lean body tissue, including skeletal muscle.³³ In older individuals who already have diminished muscle reserve, such lean mass loss can precipitate or worsen sarcopenia, further impairing strength, mobility, balance, and fall resilience. This risk has been described as pharmacologically induced or weight-loss-associated sarcopenia and is of particular concern where the margin between functional independence and disability is narrow.^{25,32}

Current expert recommendations advise that, if GLP-1 receptor agonists are prescribed to older adults with sarcopenic obesity, they should be accompanied by structured progressive resistance exercise and protein-optimized dietary support (≥ 1.2 g/kg/day) to attenuate lean mass reduction.^{17,25,33} Ongoing monitoring of body composition by DXA or BIA, alongside serial assessments of handgrip strength and physical performance, is advisable to detect early signs of muscle decline. The decision to initiate GLP-1 receptor agonist therapy in this population requires careful individual risk-benefit evaluation and should involve multidisciplinary input from geriatricians, endocrinologists, and dietitians.^{19,32}

Other pharmacological therapies investigational

Myostatin inhibitors represent a mechanistically promising but currently investigational approach. By blocking myostatin, a potent negative regulator of skeletal muscle growth, these agents may theoretically promote muscle hypertrophy; however, clinical trials in older adults have yielded inconsistent results, and no myostatin inhibitor has received regulatory approval for sarcopenia or sarcopenic obesity.²⁰ Testosterone replacement may benefit selected older men with documented hypogonadism, improving lean mass and muscle strength, though routine use is not recommended because of cardiovascular and prostate safety concerns that require

careful case-by-case assessment.²² At present, no pharmacological agent can substitute for lifestyle-based management, and any drug therapy should be viewed as complementary rather than primary treatment.³⁰

Multidisciplinary geriatric care

Effective management of sarcopenic obesity demands a coordinated, team-based approach. Physiotherapists design safe and progressive exercise programs tailored to individual capacity and comorbidities. Dietitians address protein intake, micronutrient adequacy, and the risk of nutritional inadequacy during caloric restriction. Endocrinologists manage obesity, diabetes, and hormonal disturbances, while geriatricians provide overarching coordination of functional goals, polypharmacy review, and long-term care planning.²⁵ This integrated model has been shown to be effective in improving muscle strength, mobility, metabolic control, and quality of life in older adults with complex body composition disorders.³¹

Figure 4 demonstrates the integrated management framework for sarcopenic obesity, presenting exercise, nutritional optimization, pharmacological support, and coordinated geriatric care as complementary pillars directed toward the shared goals of preserving muscle mass, reducing adiposity, and restoring functional capacity in older adults. This is an original schematic figure prepared by the authors based on management recommendations and evidence summarized in references.^{7,11,17,25,31}

Future directions and research gaps

Several important research gaps continue to limit progress in sarcopenic obesity. The most pressing need is a globally standardized definition that integrates muscle mass, strength, physical performance, and adiposity into clinically actionable criteria applicable across diverse ethnic groups. Population-specific cut-offs are particularly needed for Asian populations, where central adiposity confers

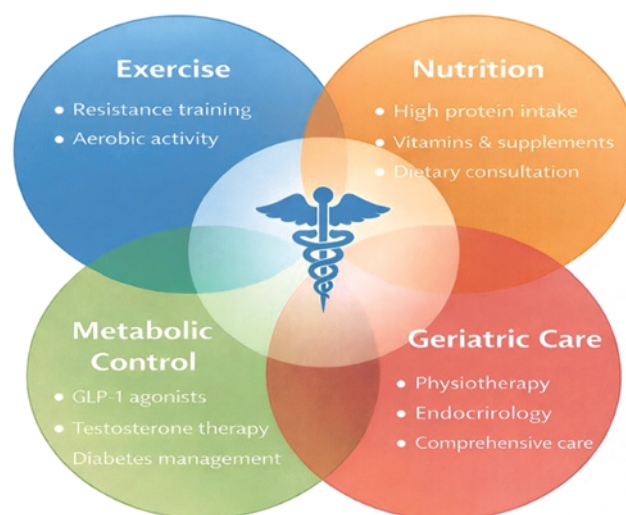


Figure 4. Multidisciplinary management strategy for sarcopenic obesity. GLP-1, glucagon-like peptide-1.

metabolic risk at body mass index values below conventional obesity thresholds.

The predominance of cross-sectional study designs in the current literature restricts understanding of disease progression, risk trajectories, and the impact of early intervention. Long-term longitudinal cohorts with standardized outcome measures are essential to address these limitations. In the pharmacological domain, further adequately powered trials are needed to evaluate the long-term efficacy and safety of novel agents, including myostatin inhibitors and GLP-1 receptor agonists, specifically in older adults with sarcopenic obesity, with pre-specified assessment of body composition as a primary endpoint.

Finally, advancing towards a personalized medicine approach in which treatment is individually tailored based on age, comorbidity burden, hormonal status, physical function profile, and body composition phenotype represents an important and achievable future direction for this field.

Conclusions

Sarcopenic obesity is a clinically important geriatric syndrome in which muscle loss and excess adiposity coexist and mutually intensify one another, resulting in frailty, falls, disability, metabolic dysfunction, impaired quality of life, and increased mortality. Its pathogenesis involves an interconnected network of age-related muscle atrophy, adipose tissue dysfunction, hormonal imbalance, insulin resistance, and mitochondrial impairment. Although diagnosis is complicated by the absence of uniform criteria, early identification guided by the most recent international consensus frameworks is feasible and essential. Current evidence most strongly supports integrated management centered on progressive resistance exercise and protein-optimized nutrition, delivered through a multidisciplinary team. Emerging pharmacological agents, particularly GLP-1 receptor agonists, hold promise but must be used with caution in older adults because of the risk of lean mass loss. As aging populations continue to grow, standardized diagnostic tools and individually tailored interventions will be indispensable for improving functional, metabolic, and survival outcomes in this high-risk group.

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