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SPECIAL REPORT

Sarcopenic obesity: pathogenesis, epidemiology and management in older adults

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ABSTRACT

Introduction: Sarcopenic obesity is the confluence of low skeletal muscle mass and function with excess adiposity. Sarcopenic obesity is becoming increasingly prevalent among older adults and may contribute to greater risk of functional decline, falls, fractures and mortality than sarcopenia or obesity alone.

Areas covered: This narrative expert review, based on targeted literature searches and the authors' personal libraries, outlines the current understanding of sarcopenic obesity, including its multifactorial pathophysiology. We also describe the current operational definition and estimated prevalence in older populations, and its impact on musculoskeletal and cardiometabolic health. Evidence from interventional studies exploring the use of targeted multimodal lifestyle behavior interventions, with a focus on the primary role of exercise and caloric restriction, to address sarcopenic obesity and its consequences is presented. Finally, we discuss recommendations for clinical practice and future research aimed at optimizing body composition and physical function in older adults.

Expert opinion: Despite a lack of consistent evidence on its prevalence, it is clear that sarcopenic obesity conveys serious health consequences. Further research is required to determine the optimal approaches to its diagnosis and management, but this should not act as a barrier to assessment and intervention in clinical settings.

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Sarcopenic obesity; ageing; falls; fractures; cardiometabolic health; exercise; nutrition

1. Introduction

'Sarcopenic obesity' describes the confluence of insufficient skeletal muscle mass and function (sarcopenia), and excessive adiposity (obesity) [1]. Given both sarcopenia and obesity are most common among older adults, the prevalence of sarcopenic obesity can be expected to increase as populations internationally continue to age [2,3].

Older adults with sarcopenic obesity may have a greater risk of undesirable health outcomes compared to individuals with obesity, sarcopenia or neither condition [4–6]. Despite growing recognition of the condition's clinical importance over the past ~20 years, there has historically been no universally accepted diagnostic definition of sarcopenic obesity [7]. The Sarcopenic Obesity Global Leadership Initiative (SOGLI), comprising members of the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO), recently published a consensus statement that provides an opportunity for standardizing efforts to understand and address sarcopenic obesity's health impacts globally [8]. SOGLI defines sarcopenic obesity based on the presence of poor skeletal muscle function, low skeletal muscle mass and excess adiposity [8]. Targeted exercise alongside dietary restriction has shown promise in improving body composition and functional capacity, though weight loss must be approached cautiously to minimize the loss of lean mass and concomitant declines in bone mineral density (BMD) [9–13].

This expert review outlines the current understanding of sarcopenic obesity and evidence-based approaches to exercise and dietary interventions to mitigate the adverse outcomes of sarcopenic obesity in older adults. The narrative review was informed by a combination of targeted searches and expert knowledge of the field. Searches were conducted in PubMed, Scopus, and Google Scholar to identify relevant peer-reviewed literature. The search strategy used combinations of key terms related to sarcopenic obesity, musculoskeletal and cardiometabolic health, and lifestyle interventions, and was refined iteratively to identify pertinent studies. Additional articles were identified through manual searching of reference lists and inclusion of relevant publications from the authors' personal libraries. Given the narrative nature of this review, inclusion was based on the relevance, originality, and quality of the studies.

2. Pathophysiology

Sarcopenic obesity is driven by physiological, metabolic and behavioral mechanisms, many of which may be amplified by advancing age and comorbidities [14,15]. As demonstrated in Figure 1, key contributors to sarcopenic obesity include malnutrition, inflammation, hormonal dysregulation, mitochondrial dysfunction and changes in muscle and adipose tissue [14].

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Article highlights

- Sarcopenic obesity is the confluence of low muscle mass and function, and excess adiposity, primarily affecting older adults.
- Sarcopenic obesity may confer a greater risk of falls, fractures, poor cardiometabolic health and mortality than sarcopenia or obesity alone.
- The pathogenesis of sarcopenic obesity is multifactorial and heterogeneity in operational definitions contributes to inconsistent prevalence estimates.
- Multimodal lifestyle interventions, combining resistance training, caloric restriction and nutritional supplementation, demonstrate promise in improving body composition, muscle function and metabolic health in older adults with sarcopenic obesity.
- Education and training for health professionals is necessary to ensure they appropriately assess and manage sarcopenic obesity in clinical settings.

With aging, muscle mass may progressively decline due to a reduction in the size and number of muscle fibers (particularly type II fibers), which leads to decreased muscle mass, impaired muscle strength and power and a greater risk of sarcopenia [16]. Declines in muscle parameters are accelerated by a sedentary lifestyle, poor nutrition and chronic disease [17–19]. Indeed, malnutrition, resulting in inadequate protein and caloric intake alongside micronutrient deficiencies, has been proposed as an important contributor to the development and exacerbation of sarcopenic obesity [20]. Nutritional deficits may exacerbate muscle catabolism and impair protein synthesis, even in people with excess adiposity. While the relationship between sarcopenic obesity and malnutrition is inconclusive [21], nutritional inadequacies may contribute to the broader metabolic disturbances that characterize this condition.

Increased adipose tissue promotes the secretion of pro-inflammatory cytokines (e.g. IL-6, TNF-α), which can cause chronic low-grade inflammation and insulin resistance [22]. These metabolic disturbances impact muscle protein synthesis and enhance degradation, perpetuating a cycle of muscle wasting and fat gain, commonly seen in sarcopenia and obesity, respectively [15,23]. Oxidative stress is another factor that contributes to sarcopenic obesity [24]. Elevated reactive oxygen species (ROS) production alongside disruption to mitochondrial and endoplasmic reticulum function results in imbalances to the pathways responsible for muscle mass regulation [24,25]. Additionally, both age- and obesity-related hormonal changes contribute to the development of sarcopenic obesity. Declining levels of anabolic hormones such as testosterone, growth hormone, and insulin-like growth factor (IGF-1) reduce protein synthesis, impair cellular activation and dysregulate the promotion of myogenesis [26–28]. Concurrently, increases in cortisol and alterations in adipokines promote visceral adiposity, exacerbate insulin resistance, and contribute to a pro-inflammatory state [27]. These endocrine disturbances interact with inflammatory and metabolic pathways, further compounding muscle catabolism and adipose accumulation [24,26–28].

Collectively, these mechanisms may contribute to a self-sustaining and potentially accelerating cycle of reduced muscle mass and function, and increased adiposity, resulting in the development of sarcopenic obesity.

3. Epidemiology

The reported prevalence of sarcopenic obesity amongst older adults varies considerably [29]. This is due to heterogeneity in the measurements and diagnostic criteria that have been applied across different studies as a result of the lack of a consensus on its definition, and also due to inherent differences in studied populations including age, gender and ethnicity. A recent systematic review and meta-analysis which included 50 studies in older adults (aged ≥60 years) reported the estimated pooled prevalence of sarcopenic obesity was 11% (95% CI: 11%-13%) [30]. However, this estimate should be interpreted with caution due to high heterogeneity (I^2 = 99.5%; $p < 0.001$) among included studies, with the prevalence of sarcopenic obesity ranging from < 1% to over 40% [30]. This was reported to be influenced by varying approaches to

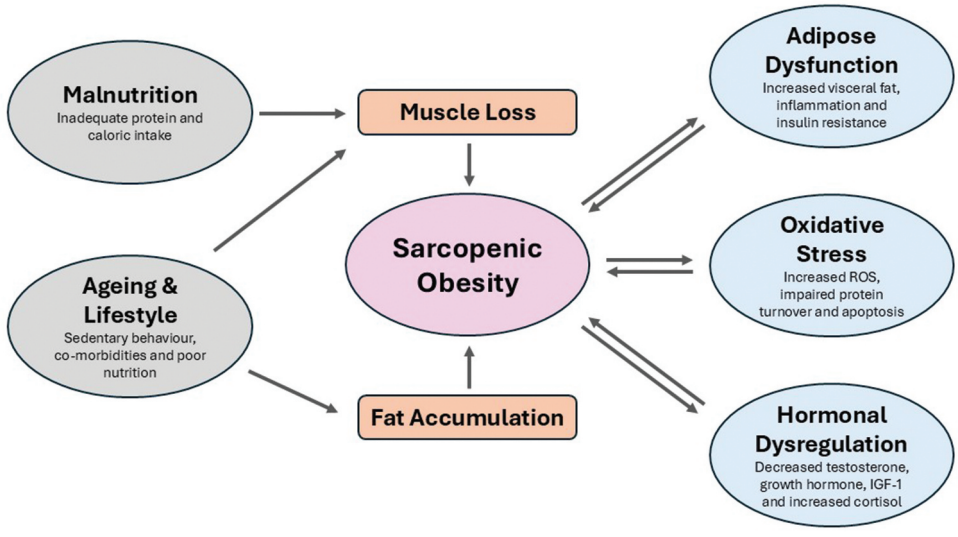

Figure 1. Age-related pathological mechanisms contributing to development of sarcopenic obesity.

measuring muscle health and body composition as well as thresholds for sarcopenia and obesity, and differences in population characteristics [30].

A cross-sectional analysis of the National Health and Nutrition Examination Survey (NHANES) data compared eight definitions for sarcopenia and obesity, reporting up to a 26-fold difference in sarcopenic obesity prevalence across these definitions among older adults [31]. This stems in part from the historic lack of consensus on operational definitions of sarcopenia, which has been assessed based on low muscle mass alone, or more recently, low muscle mass combined with poor muscle strength and/or physical function [32]. Sarcopenic obesity definitions relying solely on low appendicular lean mass often yield higher prevalence estimates than those incorporating muscle function measures, and the latter may better identify individuals with poor physical function [33]. Similarly, obesity defined by body mass index (BMI) versus body fat percentage (e.g. using dual X-ray absorptiometry [DXA]) may result in substantially different estimates of prevalence [34].

SOGLI recently presented the first consensus operational definition of sarcopenic obesity [8]. As demonstrated in Table 1, the SOGLI definition includes three steps for assessing sarcopenic obesity: screening, diagnosis and staging [8]. In the diagnosis step, the sarcopenia and obesity characteristics are referred to as 'altered skeletal muscle function' and 'altered body composition,' respectively. Once sarcopenic obesity is identified, clinicians are encouraged to further classify as Stage I or II based on the absence or presence of related comorbidities [8].

Recent studies have quantified the prevalence of sarcopenic obesity using the SOGLI consensus criteria, though estimates vary [43–46]. In the Concord Health and Aging in Men Project (CHAMP) [43], sarcopenic obesity prevalence in men aged ≥70 years was 9.6% when operationalized using the SOGLI criteria, while in a cohort from the Rotterdam study (mean±SD age 70 ± 9 years), the prevalence of sarcopenic obesity was only 0.8% [44]. In another prospective study using NHANES data for over 2000 older adults (aged 50–75 years), the prevalence of sarcopenic obesity was 15% [45]. Collectively, these findings illustrate that even with the availability of a consensus operational definition, the prevalence of sarcopenic obesity varies substantially according to cohort demographics, assessment methods and diagnostic thresholds.

Regardless of the operational definition applied, sarcopenic obesity is consistently associated with adverse health outcomes. A study in 2,303 community-dwelling older adults (aged 70–84 years) reported that handgrip strength was significantly lower in older adults with sarcopenic obesity (males:

Table 1. Characteristics and diagnostic procedures for the identification of sarcopenic obesity based on the SOGLI definition.

Process	Component	Criteria	Method/Tool	Cut-Points	Requirements
1) Screening	Obesity indicator	High BMI or WC, based on ethnicity specific cut-points	Anthropometry	**BMI**: ≥30 kg/m^2 [35]; ≥27.5 kg/m^2 (Asian) [36] **WC**: 2 levels; I: ≥90cm for M, ≥80cm for F; II: ≥102cm for M, ≥88cm for F [37]; 2 levels (Asian-Indian); I: ≥78cm for M, ≥72cm for F; II: ≥90cm for M, ≥80cm for F [38]	Must be present to proceed to the next phase
	Sarcopenia indicator	Screening parameters: clinical symptoms, suspicion of sarcopenic obesity or validated instrument (e.g. SARC-F)	Clinical judgement or validated instruments	N/A	Must be present to proceed to the next phase
2) Diagnosis	Step 1: Altered skeletal muscle function	Evidence of impaired muscle function	Muscle function measures (e.g. HGS)	**HGS**: <27 kg for M, <16 kg for F (Caucasian) [39]; <28 kg for M, <16 kg for F (Asian) [40]	Must be present to proceed to the body composition assessments
	Step 2: Altered body composition	Increased percentage fat mass alongside reduced skeletal muscle mass	**DXA**: FM% and ALM/W or **BIA**: SMM/W	**FM%**: 20-39y: >39% for F, >26% for M (Caucasian); >40% for F, >28% for M (Asian); >38% for F, >26% for M (African-American); 40-59y: >41% for F, >29% for M (Caucasian, Asian); >39% for F, >27% for M (African-American); 60-79y: >43 for F, >31 for M (Caucasian); >41% for F, >29% for M (Asian, African-American) [41] **ALM/W**: <25.7% for M, <19.4% for F (Mixed-ethnicity) [31] **SMM/W**: Class I of Sarcopenia (1–2 SD): 31.5–37% for M, 22.1–27.6% for F; Class II of Sarcopenia (<2 SD): <31.5% for M, <22.1% for F [42]	Both FM% and skeletal muscle mass must be altered to confirm the diagnosis of sarcopenic obesity
3) Staging	Stage I: No complications	Absence of complications that arise from low skeletal muscle mass and high fat mass	Clinical assessment	N/A	Indicates early stage or uncomplicated sarcopenic obesity
	Stage II: With complications	Presence of ≥ 1 complications related to sarcopenic obesity (e.g. metabolic disease, functional disabilities, cardiovascular conditions, etc)	Clinical assessment	N/A	Indicates advanced stage or complicated sarcopenic obesity

ALM/W: appendicular lean mass-to-weight ratio; BIA: Bioelectrical Impedance Analysis; BMI: body mass index; DXA: Dual-Energy X-ray Absorptiometry; FM%: fat mass percentage; F: Female; HGS: handgrip strength; M: Male; SMM/W: skeletal muscle mass-to-weight ratio; WC: waist circumference.

27.9 ± 5.1 kg, $p < 0.05$; females: 19.2 ± 3.5 kg, $p < 0.001$) compared to their counterparts with obesity alone (males: 32.0 ± 5.9 kg; females: 21.0 ± 4.2 kg) or with no condition (males: 32.2 ± 5.8 kg; females: 20.9 ± 3.8 kg) [5]. This functional decline likely contributes to an increased risk of falls and fractures. In the CHAMP study, older men (aged ≥70 years) with sarcopenic obesity (sarcopenia defined by the original European Working Group on Sarcopenia in Older People [EWGSOP] definition) had significantly elevated risk of falls (IRR: 1.66; 95% CI: 1.16–2.37) over a two-year period [47]. Furthermore, older men with sarcopenic obesity (sarcopenia defined by the Foundation for the National Institutes of Health [FNIH] definition) demonstrated an increased risk of fractures (HR: 2.38; 95% CI: 1.29–4.36) over a six-year period when compared to their non-sarcopenic, non-obese counterparts [47]. A meta-analysis of eight studies subsequently demonstrated that sarcopenic obesity conferred a 30% greater risk of falls compared with counterparts with neither condition and a 17% greater risk of falls than those with obesity alone [4]. Furthermore, older adults with sarcopenic obesity had an 88% greater rate of non-vertebral fractures than counterparts with sarcopenia alone, although this was based on data from only two studies [4].

The coexistence of reduced muscle mass and excess adiposity contributes to a pro-inflammatory state, insulin resistance and metabolic dysregulation which likely impacts cardiometabolic health [48]. A recent meta-analysis encompassing 106 studies of over 160,000 older adults demonstrated that sarcopenic obesity, compared with neither condition, was associated with a greater risk of cardiovascular events (OR: 1.97; 95% CI: 1.25–3.11) and cardiovascular-related mortality (HR: 1.63; 95% CI: 1.01–2.62) than sarcopenia (OR: 1.51; 95% CI: 1.00–2.29, HR: 1.38; 95% CI: 1.19–1.60, respectively) or obesity alone (OR: 1.19; 95% CI: 0.4–1.98, HR: 1.15; 95% CI: 0.78–1.69, respectively) [49]. Additionally, sarcopenic obesity was associated with a greater likelihood for type 2 diabetes mellitus (T2DM) (OR: 2.02; 95% CI: 1.39–2.93) than sarcopenia (OR: 1.14; 95% CI: 0.93–1.38) and obesity (OR: 1.44; 95% CI: 0.94–2.20) alone, as well as a notably greater risk of metabolic syndrome (OR: 4.31; 95% CI: 2.23–8.35) [49]. These findings highlight the importance of early identification and targeted management of sarcopenic obesity to mitigate its deleterious effects among aging populations. Furthermore, given that cluster-based analyses incorporating anthropometry, metabolic and genetic characteristics are being explored to improve poor cardiometabolic health prediction and prevention strategies [50], sarcopenic obesity may present a clinically meaningful subphenotype for future risk stratification models.

4. Management

Given the multi-dimensional phenotype and multifactorial pathogenesis of sarcopenic obesity, a multimodal approach to interventions is warranted to mitigate its effects on musculoskeletal and cardiometabolic health. Interventions targeting sarcopenic obesity should aim to address both sarcopenia and obesity, by preserving or improving skeletal muscle mass and function while simultaneously reducing excess adipose tissue. Combined exercise and dietary interventions appear most appropriate for achieving these goals [51–58].

Resistance training consistently results in improvements to muscle mass, strength, and body composition in older adults [59,60]. According to the National Strength and Conditioning Association, older adults should complete resistance training programs that are personalized and periodic [53]. These programs should consist of 1–2 multi-joint exercises per major muscle group and aim for 2–3 sets at a 70–85% one repetition maximum intensity, performed 2–3 times per week on nonconsecutive days [53]. This approach aims to improve muscle mass and strength, while promoting reductions in adipose tissue. Progressive resistance training, when performed as little as two days per week has been shown to improve functional measures and body composition in older adults [61–64]. A review of 121 studies including 6,700 participants investigated the effects of progressive resistance training on physical function in older adults (aged ≥60 years) reporting significant improvements to gait speed, chair stand time and muscle strength [61]. Performance of resistance training at high intensity may increase its benefits in older adults and elicit significant increases in muscle mass, function and body composition compared to low-intensity training [65–69].

Relatively few studies have investigated the effects of resistance training in those with sarcopenic obesity. A study compared resistance training, aerobic training and no training, conducted twice weekly over 8 weeks in 60 older adults (aged 65–75 years) with sarcopenic obesity [70]. Participants in the resistance training group exhibited greater improvements in handgrip strength than the other groups [70]. A 15-week RCT compared high-intensity circuit resistance training with regular strength and hypertrophy resistance training in 17 community-dwelling older adults (aged 60–89 years) with sarcopenic obesity, and observed high-intensity training had significantly greater beneficial effects on lower limb power (mean difference = 158W, 95% CI: 2–315) [68].

Caloric restriction interventions are effective for obesity management, however in older adults with sarcopenic obesity they must be utilized with caution as it may result in declines in lean muscle mass [71]. For example, a study in 107 frail older adults (aged ≥65 years) with obesity demonstrated that a hypocaloric diet (500-750 kcal daily deficit) resulted in a significant reduction to lean muscle mass (−3.2 ± 2.0 kg; $p < 0.001$) over 12 months when compared with controls [72]. Resistance training is likely to minimize the decline in lean mass observed with caloric restriction, as well as optimize functional benefits. In a trial where 24 older adults (aged 50–70 years) with sarcopenic obesity underwent a very low-calorie ketogenic diet (< 800 kcal/day) with 12 of those participants also completing interval training, both groups had significant reductions in body mass and fat mass [73]. However, fat-free mass was significantly reduced in the no exercise group (mean difference±SD: −2.3 ± 1.3 kg; $p < 0.001$) but was preserved in the exercise group (0.3 ± 1.0 kg; $p = 0.329$) [73]. We also completed an RCT of 60 older adults with obesity who were randomized to either a 12-week supervised gym-based high-intensity resistance and impact training program, or a home-based aerobic program, with both groups undergoing caloric restriction [65]. We found that resistance

and impact training improved gait speed compared with aerobic training and also resulted in within-group improvements in handgrip strength and Short Physical Performance Battery scores [65].

Maintaining a high protein intake (1.2–1.5 g/kg of body weight) during caloric restriction may also attenuate losses in muscle mass and function in older adults with sarcopenic obesity [74,75]. In 104 older women (aged > 65 years) with sarcopenic obesity who underwent a 12-week hypocaloric diet receiving either 0.8 g/kg or 1.2 g/kg of protein, estimated muscle mass (according to the Janssen equation) decreased in the low protein group and increased in the high protein group [75]. However, evidence remains limited, and achieving such high protein intakes may be challenging for this population due to the large absolute quantities required relative to body mass. Calcium and vitamin D intakes should also be monitored during weight loss due to their roles in maintaining bone health [76], especially in older adults with sarcopenic obesity who are at an increased risk of both falls and fractures [4,47]. Current guidelines recommend a calcium intake of approximately 1,000–-1,300 mg/day through diet, with supplementation if dietary intake is inadequate [77]. Similarly, vitamin D supplementation of 800–1,000 IU/day is advised, particularly in individuals with low serum 25-hydroxyvitamin D concentrations or limited sun exposure [77]. While recent meta-analyses suggest minimal effects of supplementation on muscle strength and function [78,79], adequate intake is important to mitigate bone loss during energy restriction and expenditure, supporting fracture prevention in this vulnerable population [76].

Further studies are required to develop a better understanding of the role of nutritional supplementation in the management of sarcopenic obesity. A study in 139 older women (aged ≥70 years) with sarcopenic obesity investigated the effects of a 12-week resistance training and nutrition intervention (essential amino acid supplementation and tea fortified with catechins) on physical function and body composition [80]. They reported that fat mass significantly decreased in the exercise and nutrition group compared with controls [80]. Furthermore, the exercise and nutrition group was 3.7-fold more likely to improve muscle strength (handgrip + knee extensor strength) than the control group [80]. Another study investigated the effects of a resistance training and dietary intervention with either 35 g of whey protein or placebo daily in 26 older women (aged ≥60 years) with sarcopenic obesity [81]. The whey protein group demonstrated increased appendicular lean soft tissue (6.0% vs 2.5%; $p < 0.05$) and decreased total fat mass (−3.3% vs − 0.3%; $p < 0.05$) compared with placebo [81]. These findings highlight the potential benefits of integrating resistance training with targeted nutritional strategies, however, as will be discussed in the 'Expert Opinion and Future Directions' section, further studies investigating the effects of combined caloric restriction, nutritional supplementation and resistance training in older adults with sarcopenic obesity are required to establish optimal prescriptions for this population.

5. Conclusion

Societies internationally face a growing burden from sarcopenic obesity, due to declines in independence, and increased risk of falls, fractures and poor cardiometabolic health. Although the prevalence of sarcopenic obesity is difficult to determine based on the heterogenous studies completed to date, the concurrently rising prevalence of both obesity and sarcopenia suggests that sarcopenic obesity will become increasingly prominent in countries and regions with aging populations. Evidence supports the implementation of multimodal tailored lifestyle interventions to address the unique pathophysiology and adverse outcomes of sarcopenic obesity. While caloric restriction effectively reduces adiposity and improves cardiometabolic health, it may exacerbate lean muscle loss and should be prescribed with caution in this vulnerable population. However, resistance training is an effective intervention to improve muscle mass and function and can offset weight loss-associated losses of muscle tissue. The optimal combination of resistance training, caloric restriction and potential nutritional supplementation must be investigated further in this population, but in the near term, recommendations that follow current best practice for management of sarcopenia and obesity are likely to effectively support health, independence and longevity for all older adults, and particularly those with sarcopenic obesity.

6. Expert opinion & future directions

Despite a significant body of research demonstrating the impact of sarcopenic obesity on older adult health, there is little evidence that these learnings have been translated into clinical practice. A major barrier has been the absence of a unified diagnostic framework, and the recent SOGLI consensus definition represents a critical step forward by offering a structured, three-step algorithm that integrates functional assessment with measures of body composition and clinical staging. The proposed measurement techniques within the SOGLI definition are generally clinically feasible, although in some lower-resourced settings, assessment of fat and lean mass using techniques such as DXA may not be possible. In such circumstances, clinicians may hesitate to make a diagnosis, thereby delaying intervention. We propose a pragmatic approach in such settings where, in the absence of body composition data, individuals with evidence of impaired muscle function (e.g. low grip strength or slow gait speed) and high BMI or waist circumference should be considered for management of sarcopenic obesity. Similarly, while the third and final step of the sarcopenic obesity diagnosis algorithm recommends staging based on the presence of comorbidities, we would argue that all patients should be treated, regardless of whether they are classified as having Stage 1 or Stage 2 sarcopenic obesity. Early-stage sarcopenic obesity may progress rapidly and confers increased risk of poor health outcomes, and so timely intervention is warranted even in Stage 1 sarcopenic obesity. This philosophy aligns with the preventive paradigm increasingly emphasized in geriatric medicine.

As described previously, the optimal approach to management of sarcopenic obesity remains to be determined, but as for diagnosis, this should not serve as a barrier to offering interventions to older adults who are likely to benefit. There is a wealth of evidence that weight loss can improve health in

individuals with obesity, even those who are older and therefore at increased risk of consequences from associated losses of muscle and bone mass. This risk appears to be satisfactorily mitigated by interventions to minimize these losses, particularly progressive resistance training, supported by adequate intake of key nutrients such as protein and calcium. A sample exercise, caloric restriction and nutritional prescription which is likely to be effective for optimizing health in older adults with sarcopenic obesity is provided in Table 2.

Despite growing evidence, widespread adoption of treatment protocols remains limited. Contributing factors include a lack of awareness among healthcare professionals, limited training in prescribing exercise and nutrition interventions, and healthcare system constraints. Additionally, existing interventions may be considered overly resource-intensive and not scalable to real-world settings. While the SOGLI consensus definition provides an important framework for diagnosis, future research must prioritize the development and validation of pragmatic, scalable models of care for the identification and management of sarcopenic obesity in clinical and community settings. It is possible that technological advances in the years to come will result in affordable point-of-care devices supporting accurate estimates of body composition, such as portable and low-cost, and perhaps even wearable, ultrasound or BIA systems. Such devices would substantially increase the capacity for sarcopenic obesity diagnosis. In the meantime, there is a need for simplified screening tools that can be applied in resource-limited settings without compromising diagnostic accuracy. The integration of such tools into routine clinical care needs to be supported with education and training for healthcare professionals with a focus on identifying patients who are at an increased risk of sarcopenic obesity. An example of such a population that will likely increase in future may include older patients receiving glucagon-like peptide-1 receptor agonist therapy. Further research is required to determine how long-term treatment with these drugs, and particularly multiple cycles of treatment, impact the incidence of sarcopenic obesity.

Interventional research should focus on large, hybrid effectiveness and implementation randomized controlled trials that evaluate long-term efficacy, cost-effectiveness, and implementational potential of combined exercise and dietary interventions for older adults with sarcopenic obesity. Particular

Table 2. Example of weekly exercise, caloric restriction and nutrition prescription.

Exercise	Protocol	Diet & Nutrition
Progressive Resistance Training [82]	**Example Exercises**: Squat/leg press, leg curls, chest press, seated row, pull down **Volume**: 4–8 exercises, 2–3 times per week 2–3 sets of 5–12 repetitions 60–120 seconds rest between sets and at least 48 hours rest between sessions **Progression**: Incrementally increase load or volume every 1–2 weeks as acceptable, progressing to 75–85% of 1RM*	**Weight Loss** A caloric deficit of 450–500 kcal/day is generally optimal for most to target a 5–10% reduction in total body weight over a 6-month period [83] Very-low-calorie diets (consumption of <800 kcal/day) and low-calorie diets (consumption of 800–1200 kcal/day) are not recommended due to risks of nutrient deficiencies and exacerbated loss of lean muscle [73] **Nutrition** *Protein Intake*: Aim for ≥1.0 g/kg/day during caloric restriction, even without exercise If feasible aim for ≥1.2 g/kg/day when combined with exercise to preserve and enhance muscle mass Distribute protein intake evenly across 3–4 meals per day (20-30g protein per meal) and supplement with whey protein shakes if necessary [74,75] *Calcium Intake*: Aim for 1000–1,300 mg/day; prioritise food-based sources (e.g. dairy, leafy greens); supplement only if dietary intake is insufficient [76] *Vitamin D Intake*: Aim for 800–1,000 IU/day through 15–30 minutes of midday sun exposures, most days of the week; supplement as necessary [77]
Balance Training [82]	**Example Exercises**: Single leg stance, tandem stance, tandem walk, backwards walk, figure 8 walk **Volume**: 2–4 exercises, ≥3 sessions per week 20–30 minutes per session, rest as needed **Progression**: Begin at a 3 and progress to 4 on the 5-point global rating scale of balance effect**	
Impact Training [82]	**Example Exercises**: Foot stomping, heel drops, jumping, hopping, skipping, drop jumps **Volume**: 2–4 exercises, ≥3 sessions per week 2–4 sets of 15–20 repetitions 30–90 seconds rest between sets and at least 48 hours rest between sessions **Progression**: Begin at low impact (body weight) and progress by increasing impact and changing direction or speed of movements Increase to a moderate impact (>2–4 times body weight) as tolerated	
Aerobic Activity [82]	**Example Exercises**: Walking, swimming, cycling, aerobics/water aerobics, yard and garden work, golf (no cart) **Volume**: ≥150 minutes per week 10–30 minutes, 1–2 sessions per day **Progression**: Begin with 10 minutes per day and gradually increase in 5-minute increments	

*1RM: 1 repetition maximum.

**5-point global rating scale of balance effect: How hard did you have to work to keep your balance/mobility during this task? It was … 1 = No effort at all; 2 = A little effort; 3 = Some effort; 4 = A lot of effort; 5 = Maximal effort.

attention should be paid to identifying the most effective components and delivery modes of these interventions, including remote and/or digitally delivered programs, which may enhance access and adherence among certain populations with sarcopenic obesity. Importantly, interventions should be co-developed with older adults themselves, health professionals and policymakers. This will ensure that interventions address the preferences and needs of key stakeholders, increasing the likelihood of wide-scale and long-term implementation

Ultimately, we must move toward integrated, multidisciplinary models of care that recognize sarcopenic obesity as a distinct and treatable clinical condition. This will require experts in this field to lobby relevant stakeholders to include sarcopenic obesity in clinical guidelines, health professional education and training, and preventive health care policy.

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