

Sarcopenic Obesity and Falls in Older Adults: A Narrative Review

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Abstract

Sarcopenic obesity (SO) is a pathological condition in which two problems show up together: skeletal muscle mass and function go down, while adipose tissue goes up. A double hit, in other words. As populations age and obesity rates rise, SO has become a growing public health concern. Existing epidemiological evidence suggests that older adults with SO may be at higher risk of falls than those with sarcopenia or obesity alone. But the findings are inconsistent across studies, and because no diagnostic criteria are universally accepted, it is hard to synthesize what's out there. That remains a real obstacle. This narrative review offers an overview of how definitions of SO have evolved and how prevalence estimates vary. It then summarizes the epidemiological evidence linking SO to falls, discusses the pathophysiological mechanisms that may be involved, and evaluates current interventions—exercise, nutrition, and pharmacotherapy. Throughout, we pay particular attention to differences in diagnostic criteria, study populations, and effect sizes. We also offer some preliminary, evidence-based suggestions for clinical identification and management, with the aim of informing future research and clinical practice.

Keywords

Sarcopenic Obesity, Falls, Older Adults, Sarcopenia, Obesity

1. Introduction

As the global population grows older—and fast—and obesity keeps climbing, sarcopenic obesity (SO) is becoming an increasingly heavy burden on older adults' health. SO basically means sarcopenia and obesity existing side by side. A dual condition like this may affect physical function in additive—or maybe even synergistic—ways [1]. Falls? They're among the most common accidental events in the elderly. Roughly 30% of community-dwelling adults aged 65 and older fall

each year, and 23% to 40% of fall-related injuries end up having serious clinical consequences [2]. Sarcopenia is already well established as an independent risk factor for falls [3]. Obesity, though, is trickier. It may raise fracture risk by amplifying the impact force during a fall, but the higher bone mineral density often seen in obese individuals might offer some protection too [4]. So when SO combines declining muscle function with excess adiposity, its influence on fall risk may be even more intricate. And the relative contributions of its individual components—low muscle mass, reduced muscle strength, and abnormal fat distribution—are still very much up for debate [5].

This review is meant to take a systematic look at what we currently know about the link between SO and falls. The main question we care about is why studies don't always agree—could differences in diagnostic criteria, study populations, or effect estimates help explain the discrepancies? We also sum up the possible pathophysiological pathways involved and go over the clinical intervention strategies that are available. In doing so, we hope to offer something useful for clinical practice and to point toward where future research should head.

Review Approach

For this narrative review, we brought together evidence from peer-reviewed original studies, systematic reviews, meta-analyses, and international consensus statements—English-language sources only. Searches ran through PubMed, Embase, and Web of Science, using core terms like “sarcopenic obesity,” “falls,” “older adults,” “sarcopenia,” and “obesity.” We included population-based cohort studies, cross-sectional studies, case-control studies, and randomized controlled trials, as long as they looked at SO in relation to fall outcomes in adults aged 50 and older. Studies were chosen for how relevant they were to our aims, with priority given to recent high-quality evidence, large-scale epidemiological studies, and official consensus guidelines.

2. Definition and Epidemiology of Sarcopenic Obesity

2.1. Evolution of Diagnostic Criteria

So far, no globally accepted diagnostic criteria for SO exist. And that's really the core problem: the proposed definitions vary so much that comparing results across studies head-to-head is difficult [6]. The earliest definitions were pretty simple—just low muscle mass combined with high adiposity. Back in 2000, Baumgartner first put forward the concept of SO, using DXA-derived appendicular skeletal muscle mass index (ASMI) together with percent body fat, and setting the obesity cutoff at the 60th percentile of the study population [7].

Over time, though, diagnostic frameworks have gotten more comprehensive. In 2010, the European Working Group on Sarcopenia in Older People (EWGSOP) put forward a comprehensive diagnostic algorithm for sarcopenia—one that brought muscle strength into the picture and highlighted gait speed as a key functional measure [8]. After that, several other international organizations came up

with their own algorithms. The core differences? They mostly come down to three things: 1) whether functional measures like handgrip strength and gait speed are included, or whether muscle mass alone is used; 2) whether BMI, percent body fat, or waist circumference is chosen as the obesity indicator; and 3) what specific cutoff values are picked for each indicator. Then, in 2022, the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) jointly published a consensus statement. It divides SO diagnosis into three levels—screening, diagnosis, and staging—and puts functional assessment first, recommending handgrip strength and/or the five-times sit-to-stand test as initial screening tools. That consensus is an important foundation for a unified diagnostic framework. Still, how well it works in clinical practice needs further validation [6].

2.2. Variability in Prevalence

Prevalence estimates for SO are all over the map—and they depend heavily on which diagnostic criteria you use, who you’re studying, and where they live. A meta-analysis of 86,285 participants put the global prevalence of SO among adults aged 60 and older at roughly 11% [9]. But apply different definitions to that same population, and the numbers can swing from as low as 0.1% to over 85% [10]. That’s an enormous range. It’s a clear warning to be careful when interpreting study findings—and it also shows why consistent definitions matter so much.

Regional differences are substantial too. Population-based epidemiological data put SO prevalence among community-dwelling older adults at about 6.0% in China and somewhere between 7.6% and 9.1% in Korea [9] [11]. In the United States, NHANES data show 12.6% in men and 33.5% in women [12]. Part of this variation may come from genetic background, lifestyle, and body composition differences across populations. But the main driver is probably still the lack of uniform diagnostic criteria. It’s also worth noting that SO prevalence is considerably higher in hospitalized or rehabilitative populations than in community settings. That suggests targeted screening in high-risk clinical groups is warranted [9].

3. Epidemiological Evidence on the Association between SO and Falls

3.1. Systematic Reviews and Meta-Analyses

Back in 2024, Veronese and colleagues published an umbrella review that pulled together data from nine meta-analyses—roughly 384,710 participants in total—to systematically assess how strongly SO is linked to various health outcomes. In older adults, having SO was associated with a 30% higher risk of falls compared with not having SO (RR = 1.30, 95% CI: 1.10 - 1.54). That said, the certainty of the evidence was rated as low. And when the reference group was narrowed to obese individuals without SO, the risk increase dropped to 17% (RR = 1.17, 95% CI: 1.008 - 1.36). The authors themselves cautioned that definitions varied so much

across the included studies that those pooled estimates should be interpreted carefully [5].

There's also a systematic review by Gandham and colleagues, registered in PROSPERO (CRD42019133773), that focused on SO in relation to falls, fractures, and bone mineral density. They planned to compare SO groups against non-SO obese groups and non-SO non-obese groups. And they included studies using all sorts of different SO definitions—specifically to explore how that definitional heterogeneity might shape the effect estimates. What did they find? Fall risk was clearly elevated in the SO group. But the size of that effect varied widely across the definitional subgroups [13].

Put the two syntheses together, and the pooled evidence points in the same direction: SO is linked to a higher risk of falls. The numbers from the meta-analyses behind them—one pooling 26 studies (37,124 participants), the other 17 studies (31,540 participants)—suggest that people with SO had about a 30% higher fall risk than healthy controls. That's around 17% higher than those with obesity alone, and higher than those with sarcopenia alone too. In other words, the combined effect of SO may be greater than what you'd see with either condition by itself.

3.2. Evidence from Prospective Cohort Studies (Incident Falls)

The choice of diagnostic criteria has a substantial impact on the estimated association between SO and falls. Scott and colleagues (2017) followed 1486 Australian community-dwelling men aged 70 years and older for a median of 6.1 years within the Concord Health and Ageing in Men Project (CHAMP). Using both the EWGSOP and the Foundation for the National Institutes of Health (FNIH) definitions of sarcopenia, with obesity defined as percent body fat $\geq 30\%$, they found that SO defined by EWGSOP was significantly associated with higher fall rates over the first 2 years of follow-up (incidence rate ratio IRR = 1.66, 95% CI: 1.16 - 2.37), whereas SO defined by FNIH showed no significant association (IRR = 1.01, 95% CI: 0.63 - 1.60). Falls were ascertained via biennial self-report, and recurrent fall events were modeled directly using IRR to account for multiple falls per participant. This discrepancy may be attributable to the absence of gait speed in the FNIH definition—indeed, gait speed was the only SO component that independently predicted falls, with each 0.1 m/s increase in gait speed corresponding to a 7% reduction in fall risk [14]. This strongly suggests that functional indicators (gait speed, handgrip strength) may have greater predictive value for falls than muscle mass measures alone.

Follis and colleagues, back in 2018, turned to data from the Women's Health Initiative (WHI)—9924 postmenopausal women aged 50 - 79. They used DXA to assess body composition, defined obesity as percent body fat $\geq 42\%$, and defined sarcopenia as the lowest 20% of appendicular lean mass. Over 7 years of follow-up, SO was significantly associated with fall risk. For women aged 50 - 64, the relative risk was 1.35 (95% CI: 1.17 - 1.56); for those aged 65 - 79, it was 1.21 (95%

CI: 1.05 - 1.39). Falls were captured through annual self-report questionnaires, and the relative risks were estimated for any fall occurring during follow-up. In ethnic subgroup analyses, something striking emerged: Hispanic women had the highest SO-related fall risk (RR = 2.40, 95% CI: 1.56 - 3.67), while non-Hispanic Black and White women had relatively lower risks [15]. These findings hint at possible racial/ethnic differences in the clinical phenotype and risk profile of SO—an area that clearly deserves further investigation.

Dowling and colleagues (2023) dug into UK Biobank data—16,147 women, aged 60 to 82. They defined dynapenia as the lowest tertile of handgrip strength (≤ 21 kg), and obesity using BMI. What did they find? Those with dynapenic obesity had a significantly higher fall risk. And dynapenia seemed to offset the protective effect that obesity usually offers against fractures at certain sites [16]. Falls were identified by linking to hospital inpatient records and self-report, and the analyses focused on first fall events. All of this underscores just how critical muscle function is when it comes to fall risk—it may even matter more than obesity itself.

Evidence from Asian populations? Still pretty scarce, honestly. One large national Chinese cohort—CHARLS, the China Health and Retirement Longitudinal Study—did use the Asian Working Group for Sarcopenia (AWGS) criteria. It showed that sarcopenic obesity was independently associated with a higher fall risk among Chinese middle-aged and older adults (adjusted HR = 1.25, 95% CI: 1.00 - 1.56, $P = 0.048$). That finding really highlights the ethnic differences in the SO-fall relationship. Falls were identified through self-report during follow-up, and the hazard ratios came from time-to-event models [17].

Differential Contributions of SO Components to Fall Risk

Not all components of SO are equal when it comes to fall risk—and that distinction really matters for designing targeted interventions. Take the TASOAC study. Scott and colleagues (2014) enrolled 674 middle-aged and older adults (50 - 79 years) and defined sarcopenia two different ways: once by muscle mass (DXA lean mass) and once by muscle strength (lower-limb power). Over 5 years of follow-up, here's what stood out. When obesity was defined by low strength—what you'd call dynapenic obesity—it predicted a higher fall-risk score ($\beta = 0.33$ for men, $\beta = 0.46$ for women). But when obesity was defined by low muscle mass—sarcopenic obesity—the association wasn't significant. Fall risk itself was estimated using a validated composite score that combined self-reported fall history with physical function measures. The takeaway? Loss of muscle function, rather than loss of muscle mass per se, may be the real driver of fall risk in SO [18].

Danielewicz and colleagues (2025) pushed this idea further—this time in a cohort of 90 severely obese older patients who were hospitalized. They used the ESPEN/EASO consensus framework and compared handgrip strength with the five-times sit-to-stand test as functional measures. Here's what came out: SO was significantly associated with falls only when the definition included handgrip assessment (OR = 3.61 - 6.03). When the definition relied on the sit-to-stand test instead, the association didn't reach statistical significance. Falls were defined by

self-reported history of falls in the prior 12 months—so a retrospective, cross-sectional assessment. The authors argued that handgrip strength, being a reliable indicator of global muscle strength, may better capture overall physiological reserve and functional vulnerability than a lower-limb-specific test [19]. On that basis, they recommended prioritizing handgrip assessment in clinical screening.

3.3. Evidence from Cross-Sectional and Retrospective Studies (Recalled Falls)

Cross-sectional and retrospective studies assess fall history by self-reported recall of prior falls, and typically compare prevalence or frequency of falls between groups. These designs cannot establish temporal sequence but provide supportive evidence for the association.

Despite variations in effect sizes, most studies consistently support a positive association between SO and fall risk. Montalvão-Sousa and colleagues (2024) applied the ESPEN/EASO criteria to 232 Brazilian older women and found that the prevalence of falls in the previous year was highest in the SO group (53.5%), compared with 38.9% in the obesity-only group and 27.0% in the normal group. The between-group difference in recalled fall prevalence was statistically significant ($p = 0.004$); no adjusted effect estimate was reported for this comparison [20]. Huo and colleagues (2016) studied 680 Australian older outpatients with a history of falls and reported that those with SO had a mean of 4.43 self-reported falls over 6 months, versus 3.24 in the non-SO non-obese group and 2.75 in the non-SO obese group. Fall frequency was assessed by 6-month retrospective self-report, and analyses compared mean fall counts across groups [21].

Overall, the direction of the SO-falls association appears consistent across studies, although the causal strength and independent contribution of SO still need to be confirmed by more high-quality prospective studies.

4. Pathophysiological Mechanisms

4.1. Chronic Inflammation

Chronic low-grade inflammation is widely seen as one of the core mechanisms tying SO to a higher risk of falls. In obesity, visceral fat piles up, and that leads to adipocyte hypertrophy. This, in turn, recruits macrophages and pushes the secretion of pro-inflammatory cytokines—TNF- α , IL-6, CRP, the usual suspects. These inflammatory mediators are toxic to muscle cells. They activate the NF- κ B and ubiquitin-proteasome pathways, which suppress protein synthesis and speed up protein degradation. The result? Less muscle mass, and worse muscle function. As muscle mass drops, basal metabolic rate falls and physical activity tends to decline too—which only encourages more fat accumulation. A vicious cycle, basically.

On top of that, chronic inflammation can directly affect the central nervous system and peripheral nerves. That can prolong reaction times and impair balance control, making falls more likely [22]-[24].

4.2. Insulin Resistance

Skeletal muscle is the body's largest insulin-sensitive organ—it handles roughly 80% of insulin-mediated glucose uptake [25]. In SO, though, that muscle mass is shrinking, and insulin signalling often goes haywire at the same time. Lipid metabolites like diacylglycerols and ceramides start to accumulate, which activates serine/threonine kinases. That leads to phosphorylation of insulin receptor substrate (IRS)—and once that happens, insulin signal transduction gets disrupted. The fallout doesn't stop there. This insulin resistance further suppresses the mTOR signalling pathway, so the expression of key enzymes needed for protein synthesis drops. Which, in turn, makes muscle loss and weakness even worse [23] [26]. And there's more: insulin resistance is frequently tied to hyperglycaemia, and the build-up of advanced glycation end-products can impair the elasticity and contractile properties of muscle tissue. That compromises postural control and gait stability even further [27].

4.3. Biomechanical Factors

Abdominal obesity does something pretty straightforward: it pushes the body's center of mass forward. That means the ankle joints end up dealing with more torque. And at the same time, muscle weakness cuts down the capacity for a quick neuromuscular response. So when balance gets disturbed, generating enough compensatory torque becomes a real challenge. This whole “forward-shifted center of mass plus weak muscles” situation? It markedly increases postural sway—and the odds of falling [28].

On top of that, even though obese individuals carry more overall fat mass, losing muscle means losing the protective “cushioning” effect that muscle normally gives to the skeleton. Combine that with the higher impact forces during a fall, and you may be setting the stage for lower-extremity fractures [29]. Dowling and colleagues pointed out that reduced soft-tissue padding in obese individuals, together with high-impact falls, might push fracture risk beyond what you'd see with obesity alone or sarcopenia alone [16].

4.4. The Mediating Role of Diabetes-Related Complications

Chen and colleagues (2026) took a look at 580 patients with type 2 diabetes. What they found was pretty striking: when SO was defined by visceral fat area, fall risk was 4.33 times higher (HR = 4.33, 95% CI: 2.70 - 6.94). And for those with early-onset diabetes, the risk went up even more [30]. What's behind this? Several things seem to be at play. Diabetic peripheral neuropathy messes with sensory input from the lower limbs. Retinopathy interferes with how visual information gets taken in. And hyperglycaemia-induced decline in central information processing doesn't help either [31]. Together, these factors disrupt the precise integration that postural control depends on. So in diabetic patients, the SO-falls link may be especially strong. Bottom line: this comorbidity really deserves extra attention during clinical assessment.

5. Clinical Interventions

5.1. Exercise Interventions

When it comes to managing SO, resistance training plus aerobic exercise is basically the cornerstone [32]. The American College of Sports Medicine recommends that older adults do strength training at least twice a week, on non-consecutive days, covering 8 - 10 major muscle groups, with 8 - 12 reps per set and a gradual increase in load [33]. Meta-analyses back this up: combined exercise programmes beat single-modality training when it comes to lowering BMI and percent body fat—and they also help build muscle mass and improve gait speed [32]. Villareal and colleagues ran a randomized controlled trial showing that combined resistance and aerobic training worked better than either one alone for physical function in obese older adults—things like the Timed Up and Go test and stair-climbing ability [34]. And balance training—Tai Chi, standing weight-shift exercises, that kind of thing—should be added as an adjunct, specifically to help bring fall incidence down.

5.2. Nutritional Interventions

Getting enough protein really matters—plain and simple—if you want to protect muscle mass in older adults [35]. Current guidelines point to about 1.2 - 1.6 g/kg body weight per day, with a particular nod to leucine-rich whey protein. Why leucine? It's a potent activator of the mTOR pathway and does a solid job stimulating muscle protein synthesis [36]. And for older adults with documented vitamin D deficiency—or another clear clinical indication—vitamin D supplementation (800 - 1000 IU daily) has independent benefits for improving muscle strength and reducing fall risk, which lines up with international clinical guidelines for fall prevention in older populations [37]. Caloric restriction? It can help with weight loss, sure. But it has to be done cautiously, because it can make muscle loss worse. A daily energy deficit of 200 - 700 kcal is often suggested, with a target weight loss not exceeding 5% - 8% of initial body weight [38]. Importantly, while cutting calories, people still need adequate protein intake and resistance training—otherwise preserving muscle mass becomes a lot harder.

5.3. Pharmacotherapy

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are highly effective for weight reduction, but they carry the risk of concomitant muscle loss. One 68-week randomized controlled trial reported that the GLP-1RA group lost 10.4 kg of fat but also 6.9 kg of lean mass, a proportion that warrants caution [39]. Notably, this trial evaluated weight and body composition changes rather than fall-prevention outcomes, and did not specifically enroll participants with SO. For patients with SO, if GLP-1RAs are prescribed, simultaneous reinforcement of exercise is crucial to protect muscle function [40]. Currently, no drugs are specifically approved for SO. Some studies have explored myostatin inhibitors and selective androgen re-

ceptor modulators, but these agents remain in early developmental stages and are not yet ready for clinical recommendation.

5.4. Integrated Management Strategy

Clinically, we'd argue that SO should be treated as a multidimensional syndrome—one that really calls for an integrated approach, bringing together exercise, nutrition, and medication adjustments. First thing's first: screen the high-risk folks. Simple measures like handgrip strength and waist circumference (or BMI) are a good starting point. If an older adult has low grip strength (men < 27 kg, women < 16 kg) and a larger waist circumference (men $\geq$ 102 cm, women $\geq$ 88 cm), then it's time to go further—DXA or bioelectrical impedance analysis [6]. Once diagnosed, put together an individualized, multicomponent intervention plan. And experts recommend reassessing muscle function and fall risk regularly—every 3 - 6 months—so the treatment strategy can be adjusted as things change.

6. Conclusions and Future Perspectives

To wrap things up, this review has pulled together what we currently know about sarcopenic obesity and falls in older adults. The observational evidence keeps pointing in the same direction: SO is tied to a higher fall risk. And the strength of that association may be greater than what you'd see with sarcopenia or obesity alone. This comes through most clearly when diagnostic criteria include functional measures—handgrip strength, gait speed, that sort of thing. That said, we can't ignore the caveats. Existing observational studies leave room for residual confounding from comorbidities, medications, prior falls, physical activity levels—the list goes on. So we can't say definitively that SO is an independent causal risk factor for falls. And then there's the definitional mess: because SO definitions vary so much, effect sizes swing widely across studies. That really hampers evidence synthesis and makes clinical translation tricky. The ESPEN/EASO 2022 consensus does give us a useful standardized framework—no question. But whether it holds up in practice? That still needs confirmation from larger, multi-centre prospective studies.

In the clinic, we'd suggest working simple functional indicators—handgrip strength and waist circumference—into routine fall-risk checks for older adults. That way, people at high risk of SO can be flagged early. As for intervention, combining resistance and aerobic exercise with optimized protein and vitamin D intake is probably the most reliable non-drug strategy we have. And if medication comes into play, it has to strike a careful balance—weight reduction on one side, muscle preservation on the other.

Future research should focus on: 1) promoting the development of globally unified diagnostic criteria for SO to ensure comparability of data across studies; 2) conducting well-designed, large-scale prospective cohort studies to clarify the independent and interactive effects of SO components (muscle strength, muscle

mass, and fat distribution) on falls, and to quantify their relative weights; 3) exploring the specific biological pathways through which SO increases fall risk, such as the inflammation-neural-muscle axis, and identifying actionable molecular targets; and 4) carrying out randomized controlled trials to evaluate the long-term effectiveness and cost-effectiveness of combined exercise and nutritional interventions in reducing fall risk in SO patients. As global population aging accelerates, early recognition and comprehensive management of SO will remain a major challenge in geriatric medicine and public health.

Author Contributions

Concept and design: Bo Zhou, Yan Wei. Acquisition, analysis, or interpretation of data: Yan Wei, Bo Zhou. Drafting of the manuscript: Yan Wei. Critical revision of the manuscript for important intellectual content: All authors. Statistical analysis: Yan Wei. Supervision: Bo Zhou.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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