

Nutritional Strategies for Sarcopenic Obesity: A Systematic Literature Review

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Abstract

Background: Sarcopenic obesity (SO) is the coexistence of excess adiposity with low skeletal muscle mass and impaired muscle function. It is increasingly recognised as a distinct clinical entity with compounded metabolic, functional and mortality risk. Nutritional management is difficult because the central intervention for obesity, energy restriction, can accelerate the loss of the very tissue whose preservation is the therapeutic priority.

Objective: To synthesise and critically appraise evidence on nutritional strategies for the prevention and management of SO, to compare findings across related reviews and primary trials, and to derive a practical, evidence-graded framework.

Methods: A PRISMA 2020-guided structured review was designed, covering consensus statements, systematic reviews, meta-analyses, randomised controlled trials and mechanistic studies. Evidence was organised by nutritional domain: energy balance, protein quantity and distribution, amino acid quality, micronutrients and fatty acids, dietary patterns, pharmacotherapy interfaces and combined exercise. A multi-perspective analytical approach (clinical, physiological, methodological, public-health, equity and digital) was applied.

Results: Across the evidence base, a moderate energy deficit combined with protein intake of roughly 1.0–1.5 g/kg/day, evenly distributed across meals, and with supervised resistance-based exercise, yields the most consistent preservation of lean mass and improvement of physical function. Leucine-enriched whey protein, vitamin D repletion and omega-3 fatty acids show supportive but heterogeneous evidence. Intermittent fasting and incretin-based weight-loss therapies demand explicit muscle-protection strategies. Definitional heterogeneity remains the dominant barrier to evidence synthesis.

Conclusion: Nutritional strategies for SO should be multicomponent, protein-adequate, deficit-moderate and exercise-anchored, and delivered through equitable, culturally adapted care models. Standardised diagnostic criteria and trials using SO-specific endpoints are the priority research needs.

Keywords: sarcopenic obesity; nutrition; protein; resistance exercise; weight loss; lean mass; PRISMA; systematic review

1. Introduction

Two demographic and metabolic transitions are converging in contemporary populations: rising life expectancy and rising adiposity. When age-related loss of skeletal muscle meets a surplus of body fat, the result is a phenotype first described as the "confluence of two epidemics" [1]. This phenotype is now termed sarcopenic obesity. In the recent ESPEN and EASO consensus, SO is defined as the coexistence of excess adiposity and reduced skeletal muscle mass and function [2]. The concept is not new. The term entered the geriatric literature at the turn of the millennium, when body-composition research showed that fat gain and muscle loss could proceed simultaneously with little change in body weight [3]. Early cohort evidence linked the combination to disability in instrumental activities of daily living beyond what either condition predicted alone [4].

The clinical significance of SO derives from synergy. Excess adipose tissue, especially visceral and intramuscular depots, produces a pro-inflammatory, insulin-resistant milieu that is hostile to muscle maintenance. Reduced muscle mass in turn lowers resting metabolic rate, glucose disposal capacity and physical activity, which promotes further fat accumulation [5,6]. This bidirectional loop makes SO more than the sum of its parts, and it makes nutritional intervention uniquely delicate. A critical appraisal of

the evidence concluded that, although biologically coherent, SO research was undermined by inconsistent operational definitions and a lack of trials designed around SO-specific endpoints [7].

The public-health relevance is considerable. Sarcopenia is a recognised driver of falls, fractures, hospitalisation and loss of independence [8]. When it coexists with obesity, health systems face compounded demands from cardiometabolic disease and disability. Prevalence estimates vary widely, depending on definition, measurement technique, age group and setting [9]. In rapidly transitioning economies, including India, a shifting disease burden toward non-communicable conditions suggests that the clinical population at risk is expanding [10,11]. Pooled analyses indicate that SO is associated with an increased risk of all-cause mortality, although the magnitude differs between definitions and settings [12–14].

Despite this importance, clinicians seeking nutritional guidance face an apparent contradiction. Obesity guidelines emphasise weight reduction through energy restriction, whereas sarcopenia guidelines emphasise adequate energy and protein to protect muscle. Resolving the contradiction is the core challenge of SO nutrition. This review therefore asks four questions:

- What nutritional strategies have been evaluated in people with, or at risk of, SO?
- How consistent is the evidence across trials, meta-analyses and related reviews?
- How do different perspectives (clinical, physiological, methodological, equity-oriented and digital) alter the interpretation of that evidence?
- What practical, evidence-graded framework follows from the synthesis?

2. Methods

2.1 Review design and reporting standards

This review was designed according to the PRISMA 2020 statement [15], which updates the original PRISMA guidance [16]. Because the literature on SO nutrition is heterogeneous in design, population and outcomes, the review combines a systematic search logic with a structured narrative synthesis, in line with the transparency principles of the PRISMA extension for scoping reviews [17]. Methodological commentaries on applying PRISMA-ScR to mapping research landscapes were consulted for structuring the charting process [18,19]. Risk of bias in randomised trials was appraised conceptually using the revised Cochrane tool [20], and the certainty of conclusions was graded according to GRADE principles [21].

2.2 Eligibility criteria

The eligibility framework is summarised in Table 1.

Table 1. Eligibility criteria and protocol elements (PICOS framework)

Element	Specification
Population	Adults (predominantly ≥ 50 years) with sarcopenic obesity, or obesity with sarcopenia risk; studies in sarcopenia or obesity alone were included where findings were mechanistically or clinically transferable
Interventions	Dietary energy modification; protein/amino acid strategies; micronutrient, fatty acid and ergogenic supplementation; dietary patterns; nutrition combined with exercise; nutrition during pharmacological weight loss
Comparators	Usual diet, hypocaloric diet alone, placebo, exercise alone, alternative nutritional regimens
Outcomes	Lean/appendicular mass, fat mass, muscle strength, physical performance, metabolic markers, inflammation, mortality, adverse events
Study designs	Consensus statements, systematic reviews, meta-analyses, RCTs, large prospective cohorts, key mechanistic studies
Language/period	English-language, peer-reviewed; 1994 to present for foundational protein-requirement work, with emphasis on 2000 onward
Information sources	PubMed/MEDLINE, Embase, Scopus, Web of Science, Cochrane CENTRAL; reference-list screening

Example search string	("sarcopenic obesity" OR "sarcopenia" AND "obesity") AND (nutrition OR diet OR protein OR "amino acid" OR "vitamin D" OR "weight loss")
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2.3 Study selection and data charting

Records identified through database searching and citation tracking were de-duplicated, screened on title and abstract, and assessed in full text against Table 1. The numerical PRISMA flow should be reported as follows: records identified (n = [n]); duplicates removed (n = [n]); records screened (n = [n]); full texts assessed (n = [n]); studies excluded with reasons (n = [n]); studies included in the synthesis (n = [n]). Charted variables were author, year, design, sample, intervention, comparator, duration, body-composition and functional outcomes, and principal conclusion.

2.4 Synthesis approach

Because outcome definitions, assessment tools (dual-energy X-ray absorptiometry versus bioimpedance), populations and intervention doses were heterogeneous, a meta-analytic pooling of effect sizes was not undertaken. Instead, a thematic and comparative synthesis was performed. Findings were grouped by nutritional domain, then triangulated across related reviews and primary trials, and finally examined from multiple analytical perspectives.

2.5 Research integrity

Originality was safeguarded by paraphrasing all sources and verifying similarity using detection tools, in line with published guidance on plagiarism detection and prevention in scholarly writing [22–24]. Ethical principles for secondary research were respected throughout [25]. No new data from human participants were collected.

3. Evidence Synthesis I: Definitions, Epidemiology and Consequences

3.1 The definitional problem

The most persistent obstacle to a coherent SO literature is the absence, until recently, of a unified definition. Sarcopenia criteria themselves have evolved. The revised European consensus placed muscle strength at the centre of case-finding, with low muscle quantity or quality confirming the diagnosis and poor physical performance indicating severity [26]. An earlier international working group emphasised lean mass and gait speed [27]. The Foundation for the National Institutes of Health project advanced cut-points for appendicular lean mass adjusted for body mass index [28], an approach that matters for obesity because absolute lean mass is typically higher in heavier individuals. The Asian Working Group for Sarcopenia updated population-specific thresholds [29], and the Sarcopenia Definition and Outcomes Consortium found that weak grip strength and slow gait were the strongest predictors of adverse outcomes [30].

In people with obesity, these frameworks diverge sharply. Adjusting lean mass for body weight, for body mass index or for height squared classifies different individuals as sarcopenic, which has been shown to mis-assign muscle depletion across body-mass-index groups [31]. This matters because the choice of denominator can flip a person from "normal" to "sarcopenic obese". Reviews of the field described the resulting prevalence range as too wide to guide policy [7,32,33], and population analyses applying several established definitions to one cohort demonstrated that the same individuals are classified inconsistently [34].

The ESPEN/EASO consensus [2] was developed to resolve this. It proposes a two-step logic. First, screening uses body mass index or waist circumference together with markers of suspected sarcopenia. Second, diagnosis requires altered muscle function (such as handgrip strength or chair-stand performance) and altered body composition (such as reduced appendicular lean mass relative to body weight). Stage I (no complications) and stage II (at least one complication attributable to altered body composition) are then distinguished. Complementary reviews describe the same conceptual architecture [35–37]. For nutritional research, the consensus is a turning point. It supplies a common eligibility standard against which future trials can be compared.

3.2 Prevalence and burden

Systematic evidence on sarcopenia prevalence in ageing adults has shown estimates ranging from a few percent to nearly one-third, depending on the setting and criteria used [38], and the public-health burden

attributable to sarcopenia is well described [39]. For SO specifically, meta-analytic estimates of prevalence differ by definition and region [9]. Because the denominator for SO is people with obesity and the numerator requires muscle depletion, any population ageing with a rising adiposity burden is predicted to produce more cases. National analyses of India's disease profile document a continuing shift toward non-communicable disease alongside a persisting infectious burden [10,11]. This implies that nutritional programmes for SO will increasingly need to operate within double-burden health systems.

3.3 Consequences: function, cardiometabolic risk and mortality

Outcome evidence is consistent in direction, if variable in magnitude. Analyses of population surveys show that associations between sarcopenia, SO and mortality depend on the definition applied [40]. In older men, SO has been examined as a determinant of cardiovascular disease and death [41], and weight-change trajectories in sarcopenic obese adults have been linked to survival in a national ageing cohort, which raises the question of whether weight loss in SO is uniformly beneficial [42]. Prospective studies associate SO and its sibling "dynapenic obesity" with falls [43], and with frailty, disability and institutionalisation [44].

Metabolic outcomes follow a similar logic. Low relative muscle mass exacerbates obesity-associated insulin resistance and dysglycaemia [45], and relative muscle mass is inversely associated with insulin resistance and prediabetes [46]. SO has been associated with higher estimated cardiovascular risk [47], and sarcopenia is highly prevalent in type 2 diabetes [48]. Oxidative stress is raised in sarcopenia and relates to cardiovascular risk in the sarcopenic obese [49]. The disease-burden perspective is reinforced by socio-legal analyses documenting structural health inequities in the Punjab region, which suggest that SO risk is unlikely to be randomly distributed across social groups [50].

4. Evidence Synthesis II: Pathophysiology Relevant to Nutrition

Understanding mechanisms is necessary because nutritional strategies act on them. Four mechanistic clusters are especially relevant.

4.1 Chronic low-grade inflammation and adipose–muscle crosstalk

Dysfunctional adipose tissue releases inflammatory mediators that promote catabolic signalling in muscle. Mechanistic reviews describe how age-associated adipose and muscle inflammation interact as a principal pathogenic route to SO [51]. Cohort evidence from the InCHIANTI study associated SO with elevated inflammatory markers [52], and earlier work linked interleukin-6 and tumour necrosis factor- α to lower muscle mass and strength [53]. Muscle is also an endocrine organ. Contracting muscle releases myokines with metabolic and anti-inflammatory effects, so loss of muscle removes a protective signalling source [54]. The practical implication is that nutritional strategies with anti-inflammatory properties, such as long-chain omega-3 fatty acids and vegetable- and fibre-rich dietary patterns, are mechanistically plausible adjuncts.

4.2 Myosteatosis and anabolic resistance

Fat does not only accumulate beside muscle but also within it. Intramuscular and intermuscular fat infiltration reduces muscle quality, defined as strength per unit mass. This has been documented in large ageing cohorts [57,58] and reviewed as a distinct epidemiological entity [59]. Molecular reviews propose that lipid-induced cellular stress contributes to the particular pathogenesis of SO [55]. Experimental work in obese sarcopenic old rats shows that ectopic fat deposition contributes to anabolic resistance, through activation of the integrated stress response [56]. Anabolic resistance, the blunted muscle protein synthesis response to amino acids, is also demonstrated in ageing human muscle through impaired anabolic signalling [60], and it has been reviewed as a central feature of age-related muscle loss [61,62]. Clinically, anabolic resistance explains why a given dose of protein may be less effective in an older person with obesity, and why dose, distribution and amino acid composition are important design variables.

4.3 Hormonal and micronutrient axes

Vitamin D has attracted particular attention. Low vitamin D and high parathyroid hormone were found to predict loss of muscle strength and mass in older adults [64], and obesity is associated with a

markedly higher prevalence of vitamin D deficiency [66], partly because the vitamin is sequestered in adipose tissue, reducing bioavailability [65]. A synthesis of definition, epidemiology and pathophysiology further situates these hormonal and nutritional influences in the broader aetiology of sarcopenia [67], and a recent integrative review of epidemiology, pathophysiology, cardiovascular disease, mortality and management of SO unites these themes [63]. These mechanisms justify a nutritional strategy that addresses inflammation, anabolic signalling and micronutrient status simultaneously, rather than relying on any single lever.

5. Evidence Synthesis III: Nutritional Strategies

5.1 Energy balance: the central tension

Weight loss is generally indicated in SO, but weight loss has a body-composition cost. A systematic review of energy restriction and exercise in middle-aged and older adults concluded that adding exercise to a hypocaloric diet attenuates the loss of fat-free mass and explicitly drew implications for SO [68]. Weight-loss reviews show that a considerable proportion of weight lost can be fat-free mass [69], although the long-quoted rule that roughly one quarter of lost weight is lean tissue has been criticised, since the proportion varies with sex, baseline adiposity and the speed and mode of weight loss [70].

The flagship demonstration of the paradox came from a one-year randomised trial in 107 frail, obese older adults. Diet-induced weight loss improved physical function, but diet alone reduced lean mass and bone density, whereas the combination of diet and exercise gave the greatest functional improvement while attenuating these losses [71]. The same trial reported favourable cardiometabolic changes with combined intervention [72]. Findings from an 18-month intervention in overweight and obese older adults with cardiovascular or metabolic risk likewise indicated that weight loss combined with physical activity yields more favourable body-composition outcomes than weight loss alone [73], and the associated community-based programme showed improvement in mobility outcomes [74].

These data support a clear principle: **a moderate, not aggressive, energy deficit** (commonly in the range of 500–750 kcal below requirements) is preferable in SO, with targeted weight loss of approximately 5–10% of body weight over several months. Reviews of lean-mass retention during weight loss recommend slower loss, adequate protein and resistance exercise as the core protective trio [75,76].

Intermittent fasting and time-restricted eating are popular but under-studied in SO. A randomised trial of 16:8 time-restricted eating in adults with overweight or obesity found no superiority over consistent meal timing for weight loss, and secondary body-composition findings prompted concern regarding lean mass [77]. Until trials in older sarcopenic obese populations show lean-mass safety, time-restricted regimens should be considered investigational in SO, and, if used, paired with high-quality protein at each eating occasion and resistance training.

5.2 Protein quantity

Protein intake is the most thoroughly examined nutritional lever. The PROT-AGE Study Group recommended that older people consume on average 1.0–1.2 g of protein per kilogram of body weight per day, and 1.2–1.5 g/kg/day for those with acute or chronic disease, with individuals having severe kidney disease being a special case [78]. The ESPEN expert group reached a similar conclusion regarding protein and exercise for muscle function in ageing [79], and nutritional recommendations for sarcopenia echo this [80]. These figures exceed the traditional recommended dietary allowance of 0.8 g/kg/day, which reassessments of nitrogen-balance data had already suggested was too low for older people [91].

Observational data support the rationale. In the Health ABC cohort, higher protein intake was associated with smaller loss of lean mass over three years [81], and reviews of dietary protein and sarcopenia reach a consistent conclusion [82,83]. Reviews on protein "requirements" beyond the RDA have argued that optimal rather than minimal intake should guide older adults [84], and a biological-to-clinical review of protein and muscle health in old age confirms plausibility across levels of evidence [85].

The reason older adults need more protein per meal is explained by anabolic resistance. Older men required a higher relative protein dose than younger men to maximally stimulate myofibrillar protein

synthesis [86], consistent with dose-response data in younger men after resistance exercise [87]. Interventions that counter anabolic resistance are therefore reviewed as central [88].

For SO specifically, the weight-loss context is key. In overweight older adults on energy restriction, lean-mass preservation depended on protein intake [89], and a meta-analysis of weight-loss trials in older adults found that higher dietary protein attenuated lean-mass loss [90]. A practical target in SO is **1.0–1.5 g/kg/day**, calculated on adjusted or ideal body weight when obesity is marked, to avoid unrealistic absolute intakes. Renal function should be assessed beforehand. Healthy older people can tolerate these intakes, but chronic kidney disease requires individualised limits [78].

5.3 Protein distribution and meal timing

Total daily protein is only part of the answer. Distributing protein evenly across three meals (about 30 g each) produced higher 24-hour muscle protein synthesis than a skewed pattern concentrating protein at dinner in healthy adults [93], and an even mealtime distribution of protein was associated with greater muscle strength in free-living older adults [94]. Because muscle underpins whole-body metabolism, these results interlock with the broader argument that muscle is an under-appreciated determinant of health and disease [95]. Practically, an SO diet plan should anchor approximately 25–35 g of high-quality protein at each main meal, rather than a protein-poor breakfast and protein-heavy dinner, which is the typical pattern in many diets.

5.4 Protein quality and leucine

Not all protein is equal in anabolic potency. Leucine acts as a signal for muscle protein synthesis through the mTOR pathway, first shown for translation initiation in skeletal muscle [96]. In older adults, a mixture with a high proportion of leucine restored the muscle protein synthesis response [97], essential amino acids were primarily responsible for anabolic stimulation [98], and leucine supplementation improved synthesis independent of the plasma amino acid concentration [99]. Whey protein, being rapidly digested and rich in leucine, has become the standard supplement vehicle.

Randomised evidence is encouraging. In frail elderly people, protein supplementation during prolonged resistance training increased muscle mass gain [100]. A meta-analysis found that protein supplementation augments the adaptive response of muscle to resistance exercise [101], and a large meta-analysis of healthy adults found that benefits plateau at approximately 1.6 g/kg/day, with the benefit reduced as age increases [102]. Reviews of nutritional supplements in support of resistance exercise reach comparable conclusions [103]. In sarcopenic older adults, a vitamin D and leucine-enriched whey supplement improved muscle mass and chair-stand performance in the PROVIDE trial [104], and a whey–amino acid–vitamin D supplementation with physical activity increased fat-free mass and strength and reduced inflammation in sarcopenic elderly [105].

The SO-specific bridge study is a randomised trial in obese older adults on a hypocaloric diet. A whey-protein, leucine and vitamin D-enriched supplement preserved muscle mass during intentional weight loss [92]. Together, these studies support leucine-rich, high-quality protein (about 2.5–3 g leucine per meal) as a rational component of SO management, though it is worth noting that the PROVIDE and Rondanelli populations were not obese, which limits direct transfer.

5.5 Vitamin D and calcium

A meta-analysis of randomised trials found that vitamin D supplementation improved muscle strength, with the clearest benefit in those with low baseline levels [106], and vitamin D, in supplemental and active forms, reduced falls in a meta-analysis of trials [107]. Considering the strong link between obesity and vitamin D deficiency [65,66], assessment and correction of vitamin D status is a low-risk, plausible element of the SO nutritional strategy. Obese individuals often require higher doses than lean individuals to reach the same serum level, and dosing should be guided by measured 25-hydroxyvitamin D and local clinical guidance.

5.6 Long-chain omega-3 fatty acids

Omega-3 fatty acids are of interest because of their anti-inflammatory and anabolic-sensitising effects. In older adults, fish oil-derived omega-3 therapy increased thigh muscle volume and grip strength in a six-month randomised trial [108], and omega-3 augmented the muscle protein anabolic response to

amino acid and insulin infusion in younger and middle-aged adults [109]. A review and a meta-analysis of omega-3 in the elderly describe supportive but heterogeneous effects on muscle mass, strength and performance [110,111]. Reviews of dietary factors and low-grade inflammation in overweight and obesity provide the broader inflammatory rationale [112]. Because omega-3 trials were not conducted in SO specifically, the strength of this recommendation is conditional. Nonetheless, with a favourable safety profile, a dietary emphasis on oily fish, or supplementation at doses studied in trials, is reasonable.

5.7 Creatine and HMB

Creatine combined with resistance training increased lean tissue mass and upper-body strength in older adults in a meta-analysis [113]. Beta-hydroxy-beta-methylbutyrate (HMB) reduced muscle loss in older adults in one meta-analysis [114], while a later review of clinical practice reported improvements in muscle mass with more variable effects on function [115]. Both agents are best regarded as adjuncts rather than substitutes for protein adequacy and exercise, and their role in SO requires dedicated trials.

5.8 Gut microbiota and the gut–muscle axis

An emerging concept links the ageing gut microbiota with frailty and sarcopenia via inflammation and nutrient handling [116]. Because obesity also alters microbiota composition, this axis is conceptually attractive for SO, supporting dietary fibre, fermented foods and polyphenol-rich plants as supportive strategies. Direct interventional evidence in SO is, however, currently lacking, so this remains a research frontier rather than a clinical recommendation.

5.9 Dietary patterns

Dietary-pattern research complements nutrient-focused work. A review of dietary patterns, skeletal muscle health and sarcopenia in older adults concluded that diets rich in vegetables, fruit, whole grains, fish and legumes, resembling Mediterranean-style eating, are associated with better muscle outcomes [117]. A systematic review of observational studies found Mediterranean diet adherence to be associated with better musculoskeletal-functional outcomes in community-dwelling older people [118]. The cardiometabolic credentials of the pattern are supported by a major randomised trial of Mediterranean diets supplemented with olive oil or nuts [119], which is relevant because SO patients are at high cardiometabolic risk.

A critical limitation is that most pattern evidence is observational and that plant-forward patterns can be low in leucine and total protein if not planned deliberately. This is directly relevant to vegetarian populations. Traditional Indian dietary principles, such as the Sattvic diet, are plant-forward and have been reviewed in light of modern nutritional science [120]. Integrative frameworks that combine Ayurvedic constitutional concepts with lifestyle practice [121] may help cultural acceptability, though their evidence base for body-composition outcomes remains limited. For vegetarians, strategies include combining legumes and cereals, using dairy proteins (paneer, curd, whey) and soya, and planning protein by meal. Consumer trends in organic and health-oriented food markets show growing interest in "clean" foods [122], which could be harnessed for adherence, although food-marketing claims should never replace protein targets.

5.10 The pharmacotherapy interface: incretin-based weight loss

Pharmacological weight loss has transformed obesity care. Once-weekly semaglutide achieved substantial weight reduction in adults with overweight or obesity [123], and tirzepatide produced reductions that, at higher doses, exceeded 20% of body weight [124]. Such efficacy raises the stakes for lean-mass protection, since greater weight loss may mean greater absolute lean loss. A review of lean-mass changes with incretin-based therapies and mitigation strategies concluded that reductions in lean mass broadly track the degree of weight loss and emphasised protein adequacy and resistance training as mitigation, while noting that the clinical significance of lean-mass decline, particularly in older and sarcopenic people, needs prospective evaluation [125]. For SO, therefore, drug-assisted weight loss should be accompanied by a structured nutrition and exercise protocol, and older patients with established sarcopenia warrant particularly cautious risk–benefit discussion.

5.11 Nutrition combined with exercise

Every strand of evidence converges on the same conclusion: nutrition works best when combined with resistance-based exercise. In a trial of 160 older adults with obesity, aerobic exercise, resistance exercise and their combination, each added to a diet-induced weight-loss programme, all improved function, with the combination producing the greatest gain [126]. A comprehensive review of exercise and nutrition strategies against SO recommended energy restriction with high protein intake and exercise, particularly resistance training [127].

SO-specific trials are smaller but consistent in direction. Comparing exercise types in older adults with SO, combined resistance and aerobic training improved body composition and strength [128]. A trial in Japanese women with SO combining exercise and amino acid supplementation reported improvements in body composition and physical function [129]. Progressive resistance exercise with a high-speed component improved physical function in older women with SO [130], resistance training reduced a sarcopenic obesity index in older women [131], and whole-body electromyostimulation improved body composition in community-dwelling women at risk of SO [132]. A five-ingredient nutritional supplement combined with home-based resistance exercise improved lean mass and strength in free-living older adults [133].

Meta-analytic and review evidence confirms that resistance training yields substantial strength gains in older adults [134], and reviews of exercise for sarcopenia reach the same conclusion [135]. Systematic reviews of nutrition and physical activity for sarcopenia, and of nutrition's role in prevention and management, favour exercise as the primary intervention, with nutrition (protein, vitamin D) as an important but secondary contributor [136,137]. A review of nutrition's effect on muscle in older adults supports this [138], and international clinical practice guidelines for sarcopenia endorse resistance-type exercise as a foundation [139].

5.12 Complementary lifestyle approaches

Mind-body practices are widely used in South Asia and are sometimes proposed as complementary strategies. A meta-analytic review of yoga and cardiovascular health [140] is relevant to the cardiometabolic comorbidity of SO, and biomechanical analysis of yoga postures concerning alignment and injury prevention [141] is pertinent to the joint-loading concerns that limit exercise in people with obesity. Work examining oxygen and carbon dioxide balance during yogic breathing [142] addresses physiological effects of breath practice. Exploratory reviews address cellular ageing and telomere length [143], psychoneuroimmunology and immune function [144] and parasympathetic activation via the vagus nerve [145], and yoga-based approaches to depression [146]. Because depression and low mood commonly accompany obesity and can undermine dietary adherence, this last domain is not trivial. These sources are, however, largely narrative or exploratory with respect to SO, and no evidence has been presented that yoga alone reverses sarcopenia. They are best positioned as low-impact adjuncts that may support adherence, flexibility, balance and stress regulation, not as replacements for protein-adequate nutrition and progressive resistance training.

6. Related Research Reviews, Outputs and Comparative Analysis

6.1 Summary of related reviews

Table 2 summarises key reviews and consensus documents, their principal outputs and how each bears on nutritional strategy.

Table 2. Related reviews and consensus documents: outputs and relevance

Source (ref.)	Type	Principal output	Relevance to nutritional strategy
Prado et al. [7]	Critical appraisal	Definitional heterogeneity undermined conclusions; consensus needed	Explains inconsistent trial eligibility
Batsis & Villareal [5]	Narrative review	Lifestyle intervention (diet plus exercise) is the	Anchors the multicomponent model

		cornerstone; pharmacology limited	
Barazzoni et al. [6]	Position paper	Called for definition, screening and integrated management	Precursor to the consensus
Donini et al. [2]	Consensus statement	Two-step screening/diagnosis; stages I and II	Provides common inclusion standard
Gao et al. [9]	Systematic review/meta-analysis	Prevalence varies strongly by definition	Shows scale and uncertainty
Weinheimer et al. [68]	Systematic review	Exercise attenuates fat-free mass loss during energy restriction	Supports exercise pairing
Kim et al. [90]	Meta-analysis	Higher protein attenuates lean loss in weight-loss trials	Supports protein targets
Cermak et al. [101]	Meta-analysis	Protein supplementation augments gains from resistance training	Supports supplement use
Morton et al. [102]	Meta-analysis	Benefit plateaus near 1.6 g/kg/day in healthy adults	Sets upper reference point
Trouwborst et al. [127]	Review	Energy restriction + high protein + resistance exercise	Closest SO-specific synthesis
Beaudart et al. [136]	Systematic review	Exercise is primary; nutrition adds modest benefit	Hierarchy of interventions
Robinson et al. [137]	Review	Nutrition (protein, vitamin D) plays a supportive role	Supports multinutrient approach
Neeland et al. [125]	Review	Lean mass declines broadly track weight loss with incretin therapy	Defines pharmacotherapy risk
Wei et al. [63]	Narrative review	Integrates epidemiology, mechanisms, mortality, management	Contextual overview

6.2 Comparative analysis of primary trials

Table 3 compares selected randomised trials that inform SO nutrition, with notes on population and transferability.

Table 3. Selected randomised trials relevant to nutritional strategies in SO

Trial (ref.)	Population	Intervention	Main findings (summary)	Transferability to SO
Villareal 2011 [71]	n = 107; obese older adults, frail	1 year: diet, exercise, both, or control	Combined arm best for function; diet alone lost lean mass	High (obese, older)
Villareal 2017 [126]	n = 160; obese older adults	6 months: diet + aerobic, resistance, or both	All improved function; combination greatest	High
Verreijen 2015 [92]	n = 80; obese older adults	13 weeks: hypocaloric diet with whey/leucine/vitamin D vs control, plus exercise	Supplement preserved muscle during weight loss	High

Kim 2016 [129]	Older Japanese women with SO	Exercise ± amino acid supplement	Improved body composition and function	High
Rondanelli 2016 [105]	n = 130; sarcopenic older adults	Whey + amino acids + vitamin D with activity	Higher fat-free mass, lower inflammation	Moderate (not obese)
Bauer 2015 (PROVIDE) [104]	n = 380; sarcopenic older adults	Vitamin D + leucine-whey supplement	Improved chair-stand and muscle mass	Moderate (not obese)
Smith 2015 [108]	n = 60; older adults	6 months omega-3 vs placebo	Increased thigh muscle volume and grip strength	Low–moderate
Lowe 2020 [77]	n = 116; overweight/obese adults	12 weeks time-restricted eating	No weight-loss advantage; lean mass concern	Moderate (younger)

Participant numbers are those reported in the original trials and should be cross-checked against the primary publications before submission.

6.3 Convergences, divergences and interpretation

Convergences. Four findings recur across reviews and trials. First, combined interventions outperform nutrition-only or exercise-only approaches for function [71,126,127]. Second, protein supplementation or higher intake attenuates lean-mass loss during energy restriction [89,90,92]. Third, resistance-type exercise is the most reliably effective single element [134–136,139]. Fourth, trial heterogeneity and small samples limit effect-size precision [7,127].

Divergences. Several tensions deserve emphasis. The first is the degree to which supplements add benefit once protein intake and exercise are adequate. Meta-analyses show benefits of protein supplementation [101,102], but the most comprehensive sarcopenia reviews rank exercise first and nutrition second [136,137]. The second is the safety of weight loss in older adults. Weight-loss trials demonstrate functional benefit [71,126], while cohort data linking weight change to mortality in SO raise caution [42]. The two bodies of evidence are reconcilable if the key variable is **intentional, protein-supported, exercise-accompanied** weight loss versus unintentional loss. The third divergence concerns population transfer. Many nutritional trials enrolled sarcopenic but non-obese adults [104,105], and many weight-loss trials enrolled obese adults without confirmed sarcopenia [73,123]. Few enrolled the true intersection, so conclusions rely on inference.

Interpretive thought. A reasonable inference from the combined evidence is that SO should be managed as a *body-composition* disorder, not a weight disorder. The primary therapeutic target is a favourable change in the ratio of muscle to fat, and body weight is only a proxy. This reframing has practical consequences. Scales are insufficient, and monitoring should include strength (grip, chair-stand) and, where available, body-composition assessment.

7. Analysis from Different Perspectives

7.1 Clinical perspective

Clinicians need actionable, low-risk rules. The evidence supports a staged approach: screen, confirm, treat the nutritional deficits, prescribe progressive resistance exercise, and monitor function. The ESPEN/EASO framework supports using stage to prioritise intensity of intervention [2]. Stage II patients, who already have complications, warrant faster multidisciplinary referral. Medication review is also important, because polypharmacy and drug-induced appetite change can affect protein adequacy.

7.2 Physiological and mechanistic perspective

From a mechanistic viewpoint, the efficacy of the multicomponent approach is predicted rather than surprising. Resistance exercise and amino acids converge on muscle protein synthesis signalling, and exercise increases the sensitivity of muscle to amino acids, partially offsetting anabolic resistance [60,88,96]. Weight loss reduces the inflammatory burden [51,53], while omega-3 and diet quality may add anti-inflammatory benefit [108,112]. Vitamin D repletion addresses a prevalent deficiency [65,66]. Mechanistic coherence strengthens confidence in the strategy but does not substitute for outcome data.

7.3 Methodological and critical perspective

Methodologically, three weaknesses threaten the evidence base. The first is the definition problem already noted [7,31,34]. The second is measurement. Dual-energy X-ray absorptiometry and bioimpedance differ in accuracy, and hydration changes during weight loss can distort lean-mass estimates. The third is endpoint selection. Lean mass is a surrogate, and muscle strength and physical performance relate more directly to patient-important outcomes, as recognised by consortium analyses [30]. Risk of bias is also relevant. Supplement trials are frequently industry-supported, and blinding of exercise interventions is impossible [20]. Following GRADE principles, certainty for most nutritional recommendations in SO is best rated as low to moderate, with higher certainty reserved for the combined diet-and-exercise principle [21].

7.4 Public health, equity and social perspective

SO risk is shaped by social determinants. Evidence on structural health inequities, including socio-legal analyses of health disparities in Punjab, indicates that access to protein-rich foods, safe exercise opportunities and health services is unequal [50]. National disease-pattern analyses highlight the epidemiological transition and public-health challenges that frame this problem [10,11]. For nutritional strategy this means affordability must be considered. Cost-effective protein sources (legumes, eggs, dairy, soya, fish where available) can often meet targets without commercial supplements, and recommendations that rely on expensive products risk widening gaps.

7.5 Digital health and artificial intelligence perspective

Digital delivery offers a means of scaling dietitian-led, exercise-supported programmes. Evaluations of telemedicine across socioeconomic groups caution that access and outcomes are not uniform [147], which implies that remote SO programmes must be designed with low-bandwidth, low-literacy and language-adapted options. Artificial intelligence is increasingly used in diagnostic medicine [148], and could in principle support body-composition estimation from imaging or routine data and automate dietary assessment. Analyses of AI-driven patient engagement emphasise that system design shapes user experience [149]. A parallel caution is that algorithmic systems can reproduce or amplify social inequalities [150]. Any AI tool used for SO screening must therefore be validated across ethnicities, body sizes and age groups, because body-composition cut-points already differ between populations [29].

7.6 Policy and legal perspective

Policy influences the food environment and access to care. Reviews of health legislation emphasise the role of a policy-legal framework in strengthening national health systems [151]. Relevant levers for SO include front-of-pack labelling that makes protein content transparent, public-procurement standards for institutional (hospital and care-home) meals that ensure adequate protein for older residents, and reimbursement for dietitian-led care. These are inferences from the health-system literature rather than SO-specific evidence, and should be treated as hypotheses for policy evaluation.

7.7 Patient-centred and educational perspective

Adherence determines real-world effectiveness. Research on evidence-based design and educational interventions in healthcare indicates that environment and education can influence outcomes [152]. For SO this suggests that patient education should explain why "losing weight" and "losing muscle" are not the same, set expectations that the scale may move slowly while strength improves, and respect cultural food practices. Strategies that build on familiar diets, such as protein-enriched traditional meals, are more sustainable than prescriptive imported regimens [120,121].

8. A Practical Evidence-Graded Framework

The synthesis can be translated into a stepwise framework. The certainty ratings are the author's judgement on the basis of the evidence reviewed and should be read as indicative.

Step 1: Identify and stage. Screen older adults with obesity for muscle weakness (grip strength, chair-stand), then confirm with body composition where available, following the consensus logic [2]. Certainty of the diagnostic framework: moderate.

Step 2: Set a moderate energy deficit. Aim for roughly 500–750 kcal/day below requirements, with weight loss of about 5–10% over 3–6 months, avoiding rapid loss [68,71,75]. Certainty: moderate.

Step 3: Prescribe protein for muscle. Target 1.0–1.5 g/kg/day (adjusted body weight in marked obesity), with approximately 25–35 g per main meal and a leucine-rich source at each [78,79,86,93,97]. Check renal function. Certainty: moderate.

Step 4: Correct micronutrient status. Measure and correct vitamin D; consider calcium adequacy [64,66,106]. Certainty: moderate for deficient individuals.

Step 5: Consider adjuncts. Omega-3 fatty acids (dietary or supplemental) and, selectively, creatine or HMB may be considered [108,111,113,114]. Certainty: low to moderate.

Step 6: Choose an anti-inflammatory dietary pattern. Mediterranean-style or planned plant-forward eating, with protein targets explicitly met [117–120]. Certainty: low to moderate (largely observational).

Step 7: Anchor with exercise. Progressive resistance training at least twice weekly, ideally combined with aerobic and balance work, supervised initially [126,127,134,139]. Certainty: high that exercise adds benefit.

Step 8: Manage drug-assisted weight loss carefully. If incretin-based therapy is used, mandate protein and resistance protocols and monitor strength and lean mass [123–125]. Certainty: low (emerging).

Step 9: Monitor and adapt. Track strength, performance, body composition, and metabolic markers, and use equitable delivery modes, including adapted digital options [147,150]. Certainty: expert consensus.

9. Limitations and Research Gaps

This review has several limitations. First, as a structured narrative synthesis rather than a quantitative meta-analysis, it cannot provide pooled effect sizes, and the PRISMA counts must be completed from the final search export. Second, definitional heterogeneity affects every cited source [7,31,34], and results obtained under one definition may not apply under another. Third, most direct SO trials are small, short and conducted in specific populations (frequently older women in East Asia or Europe), limiting generalisability to South Asian and other populations with different body-composition profiles [29]. Fourth, several secondary sources used to contextualise complementary practice and health-system issues [140–152] are narrative or exploratory and were used for contextual, hypothesis-generating purposes only. Fifth, publication bias and industry sponsorship of supplement trials may overstate benefit [20].

Priority research needs are as follows:

- Adequately powered randomised trials enrolling participants diagnosed under the unified consensus criteria [2].
- Trials with functional and patient-important endpoints, not only lean mass [30].
- Evaluation of protein dose-response specifically in obesity with sarcopenia, including vegetarian populations.
- Long-term safety and lean-mass outcomes of time-restricted eating and incretin therapy in older adults [77,125].
- Cost-effectiveness and equity-focused implementation studies, including digital delivery [50,147].
- Validation of AI-assisted screening across diverse populations [148,150].

10. Conclusion

Sarcopenic obesity forces nutrition science to reconcile two goals that appear to conflict: reducing fat and preserving muscle. The reviewed evidence indicates that the conflict is resolvable when interventions are designed around body composition rather than body weight. A moderate energy deficit, adequate and well-distributed high-quality protein, correction of vitamin D status, consideration of omega-3 fatty acids, an anti-inflammatory, culturally adapted dietary pattern and, above all, progressive resistance exercise together provide the most consistent route to preserving lean mass and improving function [2,71,92,126,127]. Emerging weight-loss pharmacotherapy and intermittent fasting amplify the need for explicit muscle-protection protocols [77,125]. The evidence base remains constrained by inconsistent definitions, small trials and limited representation of diverse populations,

and future research should use the consensus criteria and patient-important outcomes. Finally, nutritional guidance will only improve population health if it is affordable, culturally relevant and accessible across socioeconomic groups [50,147].

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