Clinical Nutrition 61 (2026) 106645

Contents lists available at ScienceDirect

Clinical Nutrition

journal homepage: http://www.elsevier.com/locate/clnu

ESPEN Endorsed Recommendation

Sarcopenic obesity: Call to action and nutritional agenda from the international clinical nutrition community☆

Rocco Barazzoni [a,*], Maria D. Ballesteros [b], Stephan C. Bischoff [c], Renee Blaauw [d], Yves Boirie [e,f], Sharon Carey [g,h], Tommy Cederholm [i,j], Yadira Cortes [k], Cristina Cuerda [l,m], Nicolaas E.P. Deutz [n,o,p], Anna–Lena du Toit [q], Rafael Figueredo [r], Laurence Genton [s,t], Olga Gomez-Santa Maria [u], M. Cristina Gonzalez [v], Joseph Hartono [w], Darcy Holt [x,y], Deisuke Ichikawa [z], Anajanet Jaquez [aa], Stanislaw Klek [ab], Gustavo Kliger [ac,ad], Claudia Maza Moscoso [ae], Takeshi Naitoh [af], Doris Ng [ag], Veeradej Pisprasert [ah], Andres F. Sanchez [ai], Paola Sanchez-Corrales [aj], Stephane M. Schneider [ak,al], Han Ping Shi [am,an], Jahit Shukri [ao], Reynaldo Sinamban [ap,aq], Shilpa Varma [ar], Julianne Williams [as], Kremlin Wickramasinghe [as], Lorenzo M. Donini [at]

[a] Department of Medical, Surgical and Health Sciences, University of Trieste, Italy
[b] Department of Endocrinology and Nutrition, Complejo Asistencial Universitario de Leon, IBIOLEON, Spain
[c] Institute of Nutritional Medicine, University of Hohenheim, Stuttgart, Germany
[d] Division of Human Nutrition, Faculty of Medicine and Health Sciences, Stellenbosch University, Cape Town, South Africa
[e] Clinical Nutrition Department, University Hospital of Clermont-Ferrand, Clermont-Ferrand, France
[f] Human Nutrition Unit, Clermont Auvergne University, INRAe, Clermont-Ferrand, France
[g] Faculty of Medicine and Health, University of Sydney, Sydney, NSW, 2050, Australia
[h] Nutrition and Dietetics Department, Royal Prince Alfred Hospital, Sydney, NSW, 2050, Australia
[i] Clinical Nutrition and Metabolism, Department of Public Health and Caring Sciences, Uppsala University, Uppsala, Sweden
[j] Theme Inflammation and Aging, Karolinska University Hospital, Stockholm, Sweden
[k] Faculty of Science, Department of Nutrition and Biochemistry, Pontificia Universidad Javeriana, Bogotá, Colombia
[l] Department of Medicine, Universidad Complutense, Madrid, Spain
[m] Nutrition Unit. Endocrinology and Nutrition Service, Instituto de Investigacion Sanitaria, Hospital General Universitario Gregorio Maranon, Madrid, Spain
[n] Center for Translational Research in Aging & Longevity, College Station, TX, USA
[o] Dept. Kinesiology & Sport Management, School of Education & Human Development, College Station, TX, USA
[p] Dept. Primary Care & Rural Medicine, School of Medicine, Texas A&M University, College Station, TX, USA
[q] Dietetics Department, Groote Schuur Hospital, Cape Town, USA
[r] Faculty of Health Sciences, Catholic University, Asunción, Paraguay
[s] Clinical Nutrition, Geneva University Hospitals, Geneva, Switzerland
[t] Faculty of Medicine, University of Geneva, Geneva, Switzerland
[u] Center of Reaserch in Health Science (CICSA), Faculty of Health Science Anahuac University, Ciudad de Mexico, Mexico
[v] Postgraduate Program in Nutrition and Food, Federal University of Pelotas, Pelotas, Rio Grande do Sul, Brazil
[w] Indonesian Central Army Gatot Soebroto Hospital, Jakarta, Indonesia
[x] Monash University, Melbourne, Australia
[y] Clinical Nutrition and Metabolism Unit, Monash Health, Melbourne, Australia
[z] First Department of Surgery, Faculty of Medicine, University of Yamanashi, Yamanashi, Japan
[aa] Pontificia Universidad Catolica Madre y Maestra, Santiago, Dominican Republic
[ab] Surgical Oncology Clinic, Maria Skłodowska-Curie National Research Institute of Oncology-Krakow Branch, Krakow, Poland
[ac] Nutrition Service and Nutritional Support Unit, Austral University Hospital, Buenos Aires, Argentina
[ad] Faculty of Biomedical Sciences, Austral University in Pilar, Buenos Aires, Argentina
[ae] Division of Education and Research, Centro Médico Militar, Guatemala
[af] Department of Lower Gastrointestinal Surgery, Kitasato University School of Medicine, Sagamihara, Japan
[ag] Department of Gastroenterology and Hepatology, Tan Tock Seng Hospital, Singapore
[ah] Division of Clinical Nutrition, Department of Medicine, Faculty of Medicine, Khon Kaen University, Thailand
[ai] Center for Obesity and Metabolic Diseases, Bupa Santiago Clinic, Santiago, Chile
[aj] Nutritional Support Department, Hospital Dr. Rafael Ángel Calderón Guardia, San José, Costa Rica
[ak] Department of Gastroenterology and Nutrition, Centre Hospitalier Universitaire, Nice, France
[al] Adipo-Cible Research Study Group, Universite Cote d'Azur, Nice, France

☆ All affiliations and addresses provided for each author are present-permanent addresses. No author moved to other Institutions during preparation of the current work.

* Corresponding author.
E-mail address: barazzon@units.it (Y. Barazzoni).

https://doi.org/10.1016/j.clnu.2026.106645

[am] Department of Clinical Nutrition, Capital Medical Unversity, Beijing, China
[an] Center for Clinical Nutrition, The First Affiliated Hospital, Wenzhou Medical University, Wenzhou, China
[ao] National Cancer Institute, Putrajaya, Malaysia
[ap] Section of Metabolic and Surgical Nutrition, Makati Medical Center, Philippines
[aq] Division of Trauma and Critical Care and Division of Surgical Nutrition, St. Luke's Medical Center, Quezon City, Philippines
[ar] Department of Dietetics, BelleVue MultiSpeciality Hospital, Mumbai, India
[as] Prevention and Health Promotion (PHP), WHO Regional Office for Europe, Copenhagen, Denmark
[at] Department of Experimental Medicine – Sapienza University, Rome, Italy

ARTICLE INFO

Article history:
Received 16 December 2025
Accepted 23 March 2026

Keywords:
Obesity
Sarcopenia
Sarcopenic obesity
Skeletal muscle

SUMMARY

Sarcopenic obesity (SO; defined as obesity with low skeletal muscle function and mass) has been recently recognized as a relevant obesity phenotype, with substantial negative clinical impact. The prevalence of SO is likely increasing, due to the growing prevalence of major SO risk factors, including sedentary lifestyle, obesity-induced non-communicable diseases with muscle-catabolism, and a general global increase in the older adult population. Structured obesity management, including anti-obesity medications, also commonly leads to variable reductions in muscle mass with potential SO risk. Here, an international network of clinical nutrition Societies, including the European Society for Clinical Nutrition and Metabolism (ESPEN), the Latino American Federation of Nutritional Therapy, Clinical Nutrition and Metabolism (Federación Latinoamericana de Terapia Nutricional, Nutrición Clínica y Metabolismo - FELANPE), the Parenteral and Enteral Nutrition Society of Asia (PENSA), and the South African and Australasian Societies for Parenteral and Enteral Nutrition (SASPEN and AuSPEN) recognize SO as a research and clinical priority, with major implications for nutrition and nutritional care. They hereby promote a nutrition network focusing on SO nutritional implications, clinical nutrition research on SO, and implementation of SO diagnosis and treatment in routine clinical practice. The current paper includes statements on SO diagnosis, promoting simple tools based on the recent consensus-based Sarcopenic Obesity Global Leadership Initiative (SOGLI) diagnostic algorithm, and its relationship with malnutrition/undernutrition. Statements are also provided on nutritional prevention of SO through healthy diet and adequate protein and micronutrient provision, on the role of nutritional care in obesity with sarcopenia, and on nutrition-based strategies to minimize and monitor the risk of sarcopenia during obesity management. The relevance of multimodal treatment approaches including both optimal nutrition and exercise is also emphasized. This SOGLI Nutrition network hereby calls for coordinated action, including scientific, educational and advocacy initiatives, to raise awareness of SO and the key role of nutrition and nutritional care in its prevention and treatment, in order to reduce the burden of morbidity and mortality in the growing population of persons with obesity worldwide.

1. Introduction

1.1. Obesity with low skeletal muscle mass and strength - a sarcopenic obesity epidemic

The spread of obesity and overweight, defined as excess body fat with negative impact on health, has reached unprecedented epidemic proportions worldwide. Combined overweight and obesity affect more than half of the population in middle age and older age groups in several world regions [1,2], posing substantial challenges on healthcare resources and spending. Hence, this call for action is timely and critical. Although body mass index (BMI, kg/m^2) is commonly used to diagnose obesity in clinical practice, excess body fat is the obesity-defining derangement, and the cause of its negative health consequences, particularly in the presence of adipose tissue dysfunction [3]. Whereas high muscle and high lean mass (including muscle and non-muscle lean mass, i.e. visceral tissues) may be observed in obesity in parallel with high total body mass, it is increasingly clear that skeletal muscle mass and function may decline in persons with obesity, either in absolute terms or relative to body fat [4,5]. This is due to clustered obesity- and comorbidity-induced abnormalities leading to muscle protein catabolism, muscle anabolic resistance and impaired muscle ability to generate strength [4,5]. Factors leading to muscle-catabolic abnormalities include unhealthy dietary habits, sedentary lifestyle with lack of physical activity, clustered adiposity-induced metabolic derangements with oxidative stress, inflammation and insulin resistance [4,5]. A growingly recognized phenotype with high body fat and low muscle mass and strength has been defined “sarcopenic obesity” [4,5]. Its prevalence has been difficult to define due to low awareness, heterogeneity in definitions and diagnostic approaches, and technical limitations in defining and assessing muscle mass and its surrogate measures [4–6]. With regard to terminology, inconsistencies have also hampered comparative research and clinical implementation, and the current group supports the use of unified definitions in body composition such as those reported in recent expert consensus papers [7]. However, using recent consensus diagnostic algorithms for sarcopenic obesity, a general prevalence around 10% has been consistently reported in community-dwelling older adults living with obesity [8], and prevalence is expected to be even higher in patients with muscle-catabolic disease conditions regardless of age [9]. Most importantly, sarcopenic obesity is associated with higher morbidity and mortality in most clinical conditions as well as in the general population [4,5,8–14], suggesting a strong direct and negative clinical impact. Complications directly associated with sarcopenic obesity include insulin resistance and type 2 diabetes, low fitness which can worsen cardiorespiratory complications, frailty and disability, increased risk of hospitalization with prolonged hospital stay, increased need for intensive care and cost increase, lower quality of life, and reduced survival [4,5,8–14]. Also relevant, incidence and prevalence of sarcopenic obesity are likely

to increase globally in the near future, with higher life expectancy worldwide and higher prevalence of non-communicable diseases. Finally, and importantly, muscle mass loss occurs during obesity management [15–17]. Recent advances and growing utilization of incretin-based anti-obesity medications, while opening unprecedented perspectives for successful treatment and prevention of obesity and its complications [18,19], further highlight the need for accurate assessment and prevention of potential nutritional risk and sarcopenic obesity [16,17]. Based on the above considerations, diagnosing sarcopenic obesity and developing effective treatment strategies is a major unmet research and clinical need. Here, an international network of clinical nutrition Societies, including the European Society for Clinical Nutrition and Metabolism (ESPEN), the Latino American Federation of Nutritional Therapy, Clinical Nutrition and Metabolism (Federación Latinoamericana de Terapia Nutricional, Nutrición Clínica y Metabolismo - FELANPE), the Parenteral and Enteral Nutrition Society of Asia (PENSA), the South African Society for Parenteral and Enteral Nutrition (SASPEN) and the Australasian Society for Parenteral and Enteral Nutrition (AuSPEN) recognize SO as a research and clinical priority, with major implications for nutrition and nutritional care, and they propose a call to action for a nutritional agenda.

1.2. Sarcopenic obesity diagnosis: the SOGLI initiative and algorithm

As mentioned above, definition and diagnosis of SO have been elusive due to lack of consensus in both research and clinical fields [4–6], leading to high variability and difficulty in comparing research findings. In 2022, a global expert group proposed a consensus diagnostic algorithm under the auspices of the European Societies for Clinical Nutrition and Metabolism (ESPEN) and Obesity (EASO) [5]. The group also continued to hold regular meetings, discussing state of the art and new initiatives to address open research questions, including accurate assessment of body composition, physical activity and habitual dietary intake. In the meantime, the network has promoted the dissemination of the algorithm in the framework of the Sarcopenic Obesity Global Leadership Initiative (SOGLI) [8]. The diagnostic algorithm has been indeed implemented in clinical practice, with initial encouraging results supporting its ability to identify individuals at risk of negative clinical outcomes [8,9,12].

STATEMENTS on SO Diagnosis - The SOGLI clinical nutrition community:

1) supports utilization of the international consensus-based SOGLI algorithm for SO diagnosis, using ethnicity-specific thresholds, and promotes close collaboration to further develop a nutritional agenda and nutrition-based strategies for SO prevention and treatment;
2) supports the flexibility of the algorithm to facilitate implementation in all clinical settings, including low-resource settings and low-middle income countries at large. In particular, utilization of the simple handgrip and sit-to-stand tests to detect low muscle function is supported. Regarding muscle mass and body composition, along with techniques including dual x-ray absorptiometry (DXA), bioimpedance analysis (BIA) and computer tomography (CT) scans [5], the group advocates for further investigation and validation and potential future utilization of simple surrogate measures for skeletal muscle mass, e.g., based on recently-proposed adjusted anthropometric parameters [20];
3) as a global clinical nutrition community, the SOGLI network further underscores and promotes the importance of optimizing ethnicity-specific cut-offs, where needed, in order to further improve diagnostic and predictive ability.

1.3. Sarcopenic obesity and malnutrition (undernutrition)

Low skeletal muscle mass is a key component of malnutrition (undernutrition) [21,22]. Therefore, sarcopenic obesity and malnutrition share fundamental clinical and pathophysiological features, with a key role for low skeletal muscle mass and catabolic patterns of muscle protein metabolism [21–24]. Thus, malnutrition may be common in persons with sarcopenic obesity, independently of high BMI, particularly in the presence of older age and pro-inflammatory comorbidities that directly fulfill the GLIM inflammation-disease criterion [21–24].

STATEMENT on SO and malnutrition (undernutrition) - The SOGLI clinical nutrition community.

4) recommends that patients with sarcopenic obesity also undergo diagnostic assessment for malnutrition, using the consensus-based GLIM algorithm (GLIM) or other validated tools that include the same parameters. Conversely, we recommend that individuals with obesity and malnutrition undergo diagnostic procedures for sarcopenic obesity.

1.4. A 4-POINT sarcopenic obesity nutritional agenda

Since nutrition and nutritional care are key determinants of adipose and skeletal muscle mass and function, the SOGLI clinical nutrition community identifies the following key areas and priorities for a sarcopenic obesity nutritional agenda.

2. Sarcopenic obesity nutritional prevention (Fig. 1)

HEALTHY DIET: nutrition and its quality play an important role in the maintenance of health. Many studies have sought to identify nutritional patterns promoting maintenance of skeletal muscle mass and function, and therefore sarcopenia prevention. Unhealthy dietary patterns, high in saturated fat and glycemic load with high-glycemic index carbohydrates, as well as foods with higher levels of ultra-processed foods, play a well-known role in the spread of the obesity pandemic [25]. In addition, such diets may contribute to muscle-catabolic derangements, including inflammation, oxidative stress and insulin resistance [26–28]. On the other hand, healthy diets have the potential to reduce pro-inflammatory and pro-oxidative burdens, thereby limiting systemic and muscle contributors to sarcopenia and sarcopenic obesity [29–35]. These include healthy dietary recommendations at large, and traditional models such as the Mediterranean diet, which are generally enriched in non-processed foods, with high fiber content as well as fruit and vegetables and non-tropical vegetable oils, with fish and vegetables also representing relevant protein sources. Longitudinal studies have reported that adherence to a healthy lifestyle with healthy dietary patterns, including but not limited to traditional diets, and adequate physical activity, is associated with preserved lean body mass, preserved muscle strength, and preserved physical function [29–35]. Notably, the quality of available studies is at times limited, leading to weaker conclusions in systematic reviews and meta-analyses [36,37]. Also important, the number of available studies on dietary patterns and muscle health is highest for older adults [29–37] and it is substantially lower in persons with obesity [38]. Obesity-oriented research is therefore needed to provide optimized recommendations.

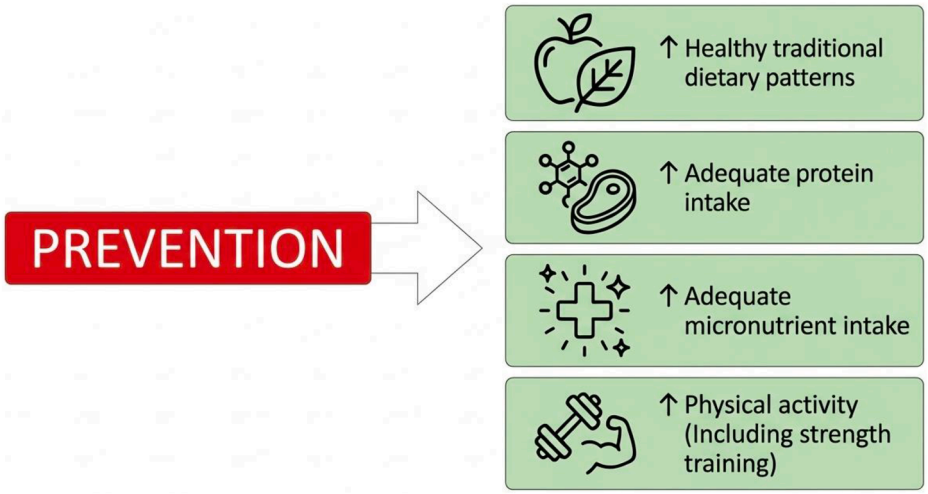

Fig. 1. Components of nutritional and lifestyle prevention of sarcopenic obesity.

PROTEIN INTAKE: dietary protein intake is an important determinant of muscle mass maintenance, with a protective role in maintaining adequate protein content and amino acid availability. It should be pointed out that anabolic resistance, i.e. reduced anabolic impact of protein and amino acids through activation of protein synthesis, may be common in obesity due to muscle catabolism associated with systemic inflammation and insulin resistance, potentially reducing effectiveness of dietary protein or medical protein supplementation to preserve or enhance skeletal muscle mass [4,5,39]. In this context, longitudinal population studies have demonstrated that protein intake higher than the general current recommendations for healthy adults (>1-1,2 as opposed to 0,8 g/kg·day) is associated with preservation of muscle mass in older individuals without obesity [40–46]. Indeed, for protein intake and allowance, a large majority of available studies was performed in geriatric cohorts [40–46], and optimal protein intake for muscle mass maintenance or recovery in persons with obesity remains incompletely defined, especially during weight loss [39,47]. High-quality studies should be designed to address these questions, along with the potential role of specific amino acids such as leucine and branched-chain amino acids, and optimization of protein quality [39].

MICRONUTRIENTS: micronutrients play key roles in the regulation of skeletal muscle mass and function [48]. Although an in-depth discussion of the role of micronutrient deficiencies in muscle and adipose tissue metabolic derangement is beyond the scope of this paper, coordinated activities have been described in the regulation of skeletal muscle metabolism and mass, particularly for, but not limited to, vitamin D [49] and omega-3 polyunsaturated fatty acids [49]. In pre-clinical studies, both may contribute to stimulate energy metabolism through enhanced mitophagy and mitochondrial function, and stimulated tissue repair potential and protein anabolism [50,51], with growing evidence also for clinical impact [51–55]. Additional interest comes from reported activities to modulate adipose tissue pathways involved in adipose responses to excess fat, including adipogenesis and stimulation of adipose tissue browning [50,56], consistent with synergistic body composition abnormalities in sarcopenic obesity.

STATEMENTS on nutritional prevention of SO - The SOGLI clinical nutrition community.

5) promotes general awareness on the relevance of healthy and traditional dietary patterns for general health and muscle preservation, and on the importance of avoiding unhealthy and ultra-processed food patterns, including public health campaigns specifically highlighting the role of healthy diets in maintenance of skeletal muscle mass and function, and therefore preventing sarcopenic obesity;
6) promotes general awareness on the importance of ensuring adequate protein intake [>1 g/kg adjusted body weight (ABW)/day], and micronutrient intake according to recommended dietary allowance in persons at risk for sarcopenic obesity, with particular regard to older adults and people living with comorbidities, taking into account local and regional traditional eating habits, as well as kidney function status; (see Statement 11 for ABW calculation);
7) in clinical practice, it promotes implementation of healthy dietary patterns, and adequate protein and micronutrient intake, as an integral component of the prevention of sarcopenic obesity, particularly in at-risk groups and individuals [37];
8) advocates for high-quality clinical research to establish optimal protein and micronutrient intakes in obesity, including specific patient groups based on age, sex and comorbidities.

3. Nutritional care in obesity with sarcopenia or malnutrition (undernutrition) (Fig. 2)

Obesity is a systemic disease and a risk factor for a number of diseases affecting virtually every organ and system in the body [57]. The onset of sarcopenic obesity may be gradual, in the context of aging and progressive obesity-associated metabolic derangements, or it may be accelerated by concomitant comorbidities, with negative synergistic impact on muscle mass, energy metabolism and function [5]. Chronic comorbidities in obesity include all major non-communicable diseases (NCDs). NCDs, as well as their acute complications, may worsen skeletal muscle catabolism, while also leading to lower physical activity, involuntary loss of appetite and body weight [4,57]. Hyper-catabolic states with a strong negative impact on nutritional status may require full nutritional care with medical nutrition

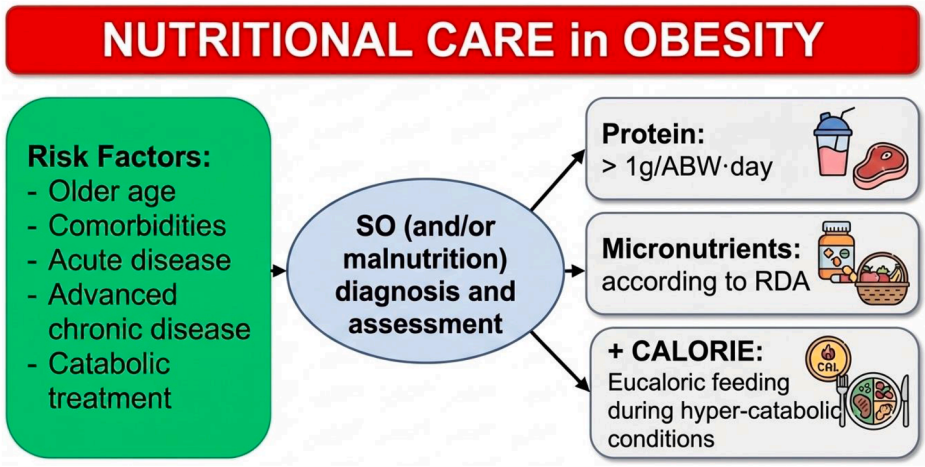

Fig. 2. Approaches for nutritional care in persons with obesity in the presence of sarcopenia and/or malnutrition.

implementation. Hyper-catabolic conditions at highest nutritional risk include a) acute disease with hospitalizations or ICU stays, that strongly enhance muscle loss and dysfunction [4,57–59], b) advanced chronic disease conditions including, but not limited to, chronic kidney disease on hemodialysis, advanced liver cirrhosis, heart failure or chronic obstructive pulmonary disease, advanced solid tumors [5,57] and c) catabolic treatments such as surgery or chemotherapy [58]. Under the above conditions, nutritional care may include diet optimization and, if appropriate, oral nutritional supplements, enteral tube feeding and parenteral nutrition. In hypercatabolic states, enhanced protein provision above 1–1.2 g/kg adjusted BW/day is commonly recommended. Nutritional treatment may also notably include eucaloric regimens also for persons with obesity, in order to minimize skeletal muscle loss [58–61].

STATEMENTS on nutritional care in patients with obesity and sarcopenia or malnutrition (undernutrition): The SOGLI clinical nutrition community:

9) promotes implementation of both sarcopenia and malnutrition screening and diagnostic assessment in patients with obesity and higher risk of either condition, including older adults and those with comorbidities, with particular regard to acute catabolic disease, advanced chronic disease conditions and hyper-catabolic treatments;
10) promotes awareness of the importance of nutritional care also in patients with obesity in the presence of sarcopenia or malnutrition, particularly under the above high-risk conditions. Nutritional care should include higher protein provision (above 1 g/kg ABW/day; see Statement 11 for ABW calculation) and adequate micronutrient intake according to RDA. Nutritional care may also aim at eucaloric energy intake to contribute to minimize muscle loss, particularly during hypercatabolic states. In the context of malnutrition diagnosis using the GLIM algorithm, specific criteria that may directly indicate the need for nutritional treatment include involuntary weight and muscle loss, involuntary reduction of appetite and food intake, and an inflammatory disease state.

3.1. Nutritional prescription in persons with obesity

When energy and protein requirements cannot be accurately measured (e.g. by indirect calorimetry or nitrogen balance), guidelines may suggest the use of predictive equations, which have however inherent limitations in accuracy [62], or weight-based prescription of calories and protein. In persons with obesity, using actual body weight may also lead to inaccurate provision with substantial overestimation and over-prescription. Alternatives to actual body weight in obesity include ideal body weight (IBW), which may be calculated using standard formulae [63,64]. However, IBW does not take into account metabolically active lean mass that contributes to excess body weight (EBW). Although an accurate assessment of the lean components of EBW is not available in clinical practice, estimates suggest that such components may represent approximately 25% [59–61,65]. Recent consensus proposals and guideline recommendations have indeed supported the use of adjusted body weight (ABW) to prescribe calories and protein in persons with obesity [5,59–61]. Importantly, available formulae for IBW calculation [63,64] lack widespread clinical validation and may introduce variability; proposing universal IBW and ABW calculation methods is however beyond the goals of the current paper. For clinical practice, it seems reasonable to accept the use of a practical approach indicating IBW as the theoretical body weight corresponding to a BMI of 25 kg/m^2 for Caucasian populations, 23 kg/m2 for East-Asian populations, or at the corresponding upper limit of normal BMI for any ethnicity.

STATEMENT on nutritional prescription in persons with obesity: The SOGLI clinical nutrition community.

11) supports prescription of calories and protein based on kg of adjusted body weight (ABW)/day in persons with obesity, when accurate measurement of energy and protein requirements, such as by indirect calorimetry or nitrogen balance, is not available. This approach is expected to minimize prescription errors and marked over- or under-estimation of nutritional needs. For practical purposes, individual ABW could be calculated as the body weight corresponding to a BMI of 25 + 25% of excess body weight (EBW) for Caucasian individuals, 23 + 25% for East-Asian individuals, or any appropriate ethnic-specific BMI cut-offs.

4. Management of obesity and related risk of sarcopenia (Fig. 3)

4.1. Minimizing muscle loss during obesity management

Although changes in total body weight are commonly the only monitored anthropometric parameter in routine clinical practice, fat loss should be the primary goal of obesity management. Excess

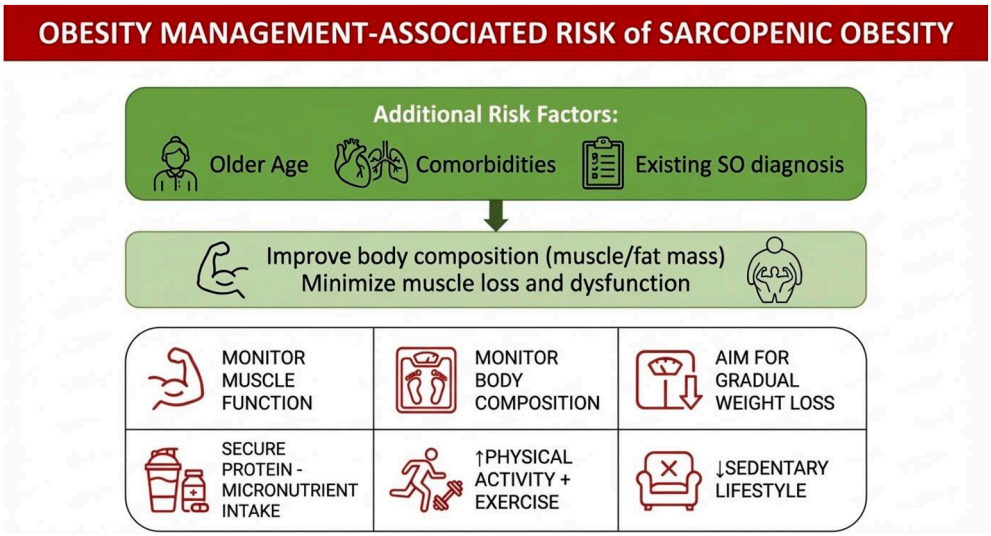

Fig. 3. Potential approaches for minimization of the risk of sarcopenia during obesity management.

fat is the defining derangement in obesity, and fat loss per se has a beneficial impact on body composition, potentially improving the muscle-fat balance and the sarcopenic obesity phenotype. However, therapeutic strategies to reduce body fat inevitably also lead to variable skeletal muscle loss, and the extent of muscle loss may limit the health benefits of fat loss, potentially causing or worsening sarcopenic obesity. The extent of muscle loss during obesity management is commonly lowest following lifestyle-based interventions [66], in the presence of less pronounced total weight loss, whereas substantial weight loss following metabolic and bariatric surgery (MBS) is associated with more pronounced lean mass loss, which may exceed 10% one year after intervention, partly depending on the type of surgery [67]. Also important, emerging incretin-based anti-obesity medications (AOM) lead to substantial weight loss above 15–20%, and increasing AOM utilization may increase the risk of sarcopenic obesity, with subgroup analyses showing that one-quarter to one-third of lost weight is attributable to lean tissues [15,16,18,19]. During obesity management in persons with or without sarcopenic obesity, loss of skeletal muscle mass should be therefore minimized and monitored through comprehensive multimodal strategies that include physical activity and exercise, with a key role for diet and nutrition, through preserved dietary protein intake (above 1 g/kg ABW/day) and adequate micronutrient provision [5], with potential adjustments according to comorbidities. In the current section, we will focus on nutritional approaches, as physical activity and exercise prescription will be explicitly addressed in a separate section.

4.2. Beyond muscle loss: body composition and muscle strength

Muscle loss should also be evaluated in the context of potential, concomitant favorable changes in both body composition and muscle composition as well as metabolism. First, as mentioned above, fat loss is commonly substantially larger than the loss of muscle mass, leading to higher percentage muscle mass and therefore potential improvement of the sarcopenic obesity phenotype [5]. Second, fat loss has been also associated with improved skeletal muscle strength or subjective functional parameters [18,19,68–70], likely reflecting metabolic and histological changes that include reduced muscle fat content and improved mitochondrial function and dynamics [71–74]. During obesity management, it is therefore essential to monitor body composition and muscle strength, as well as potentially other functional parameters, in order to determine the full impact of treatment on sarcopenic obesity.

Overall, the balance between fat and muscle loss during obesity management may lead to improvement or worsening of body composition, with variable changes of percent muscle mass and therefore of the sarcopenic obesity phenotype. Contributing factors may include initial body composition, age, sex and pre-existing or concomitant catabolic conditions. In addition, preclinical studies have suggested that incretin-mimetic molecules employed for obesity management may have an independent muscle-anabolic impact [75], which might need to be directly evaluated in future studies. In patients at risk for, or already affected by sarcopenic obesity, the clinical risk-benefit ratio associated with relative losses of body fat and muscle remains incompletely defined, and we advocate for future studies and clinical trials to directly address these issues, including assessment of body composition and muscle strength in pharmacological trials as well as other weight-loss management strategies. In current clinical practice, we suggest that in the presence of high risk or established SO, obesity management should not be automatically contraindicated, but a decision to treat obesity should be accompanied by nutritional and SO monitoring (including handgrip strength, sit-to-stand test and body composition assessment), and by all strategies to minimize loss of skeletal muscle mass and function. Finally, in all non-surgical obesity management strategies (and following MBS to a much smaller extent), treatment discontinuation commonly leads to variable weight regain, and weight cycling is commonly associated with unfavorable changes in body composition and muscle function [76,77]. Weight cycling should be therefore prevented by promoting effective long-term obesity management while implementing weight regain-preventing strategies, particularly in patients at high risk of sarcopenia and sarcopenic obesity.

STATEMENTS on minimizing muscle loss and risk of SO during obesity management: the SOGLI clinical nutrition community:

12) recommends initial assessment and subsequent monitoring of SO variables during obesity management, beyond muscle mass and including body composition and muscle function, particularly in at-risk patient groups such as older adults

and those with comorbidities. In the presence of SO, nutritional state should also be assessed, with diagnostic assessment based on the GLIM criteria. Strategies and goals for obesity management may need to be modified in case of onset or worsening of SO, or in case of worsening nutritional state;

13) recommends that gradual weight loss strategies should be preferred, to allow for timely identification of SO or nutritional worsening, with limited calorie restriction in patients at risk, or with SO (e.g., <20–25% reduction of estimated requirements);
14) emphasizes the importance of minimizing muscle loss through optimized nutrition, including adequate intake of protein (>1 g protein/kg ABW/day) and micronutrients, with particular regard to those identified as anabolic regulators of protein metabolism [48–56]. Nutritional supplements should be used if needed to reach nutritional targets, and in case of proven deficiency for micronutrients;
15) recommends that during weight loss treatment involving anti-obesity medications or MBS, care is taken to also implement appropriate lifestyle changes with optimized nutritional intake, that remains a key component of care;
16) recommends that nutritional strategies are implemented in the context of a multimodal approach including physical activity and exercise (see: Section 4);
17) recommends that weight cycling is prevented or minimized through thorough pre-treatment assessment, sustained-chronic obesity management and optimized weight regain prevention strategies.

5. Physical activity and exercise

Sedentariness and low physical activity are key pathogenetic drivers of sarcopenia and sarcopenic obesity, by enhancing muscle protein catabolism and reducing mitochondrial energy metabolism [5,38,71–75]. Reducing sedentary behavior, increasing physical activity and implementing exercise training including cardiac exercise and weight-bearing exercise are major components of prevention and treatment strategies for sarcopenic obesity [5,38,78–84]. Most importantly, they represent powerful tools to directly improve muscle protein anabolism and mitochondrial function [73], enhancing the effectiveness of nutritional care through anabolic nutrient utilization. Exercise programs are indeed recommended by sarcopenia guidelines [78,81], and by clinical nutrition guidelines as a measure for malnutrition (undernutrition) treatment in multimodal approach [58–61]. Resistance exercise should be included for its direct anabolic impact, and general approaches have been proposed for patients without obesity consisting of two exercise sessions per week, with a combination of upper- and lower-body exercises performed with a relatively high degree of effort for 1–3 sets of 6–12 repetitions [79]. Individualized programs should be implemented, with particular care for potential comorbidities and fitness level. Importantly, studies are needed to determine the optimal exercise prescription in persons with obesity. Whereas resistance exercise is primarily recommended due to its direct anabolic impact [78–84], metabolic benefits from aerobic exercise make physical activity at large, and aerobic training, also relevant.

STATEMENT on physical activity and exercise in obesity: The SOGLI clinical nutrition community.

18 recommends implementation of physical activity and exercise training programs throughout all steps of SO prevention, nutritional care and management; sedentary behavior should be reduced, and physical activity and exercise, including resistance training, should be implemented according to the patient conditions, to reduce muscle-catabolic derangements and favor muscle anabolism and energy metabolism.

6. Conclusion: a call for action

The international SOGLI clinical nutrition community, including the European Society for Clinical Nutrition and Metabolism (ESPEN), the Latino American Federation of Nutritional Therapy, Clinical Nutrition and Metabolism (Federación Latinoamericana de Terapia Nutricional, Nutrición Clínica y Metabolismo - FELANPE), the Parenteral and Enteral Nutrition Society of Asia (PENSA), the South African Society for Parenteral and Enteral Nutrition (SASPEN) and the Australasian Society for Parenteral and Enteral Nutrition (AuSPEN) recognize and indicate sarcopenic obesity as a relevant clinical challenge and clinical and research priority. They support the implementation of the SOGLI diagnostic algorithm while promoting research for further optimization of its components. The network commits to coordinated actions aimed at increasing awareness and promoting research and initiatives on sarcopenic obesity, with particular regard on a nutritional agenda including prevention strategies, optimization of nutritional state and obesity management focusing on both fat loss preservation of muscle mass and function in at-risk individuals. A multimodal prevention and treatment approach is considered essential, where nutritional components are associated with, and strengthened by, physical activity and exercise. Initiatives to be promoted may include workshops, congress sessions and educational activities, as well as advocacy to modify policies and favor healthier food patterns; research on treatment strategies remains a major goal. We are convinced that achievement of these goals has strong potential to reduce the burden of morbidity and mortality in the dramatically increasing population of persons with obesity in all medical specialties.

Authors contributions

RB conceptualized the paper, prepared the first draft and organized discussion rounds; LD contributed to prepare the first draft and participated in further discussion rounds; MDB, SCB, RBl, YB, SC, TC, YC, CC, NEPD, A-LdT, RF, LG, OG-SM, MCG, JH, DH, DI, AJ, SK, GK, CMM, TN, DN, VP, AFS, PS-C, SMS, HPS, JS, RS, SV, JW, KW all contributed to discussion rounds and critical revision with modifications and refinement of statements and comments reported in the paper. All authors approved the final submitted version. No one eligible for authorship has been excluded from the list of authors.

Data sharing statement

Not applicable; the paper does not contain any original data.

Ethical approval

Not applicable; no original studies were performed for this paper.

Disclaimer (JW, KW)

The authors alone are responsible for the views expressed in their publication and do not necessarily represent the decisions, policy, or views of WHO.

AI

The figures have been made with the help of Notebook LM, with the author's oversight and control, and authors have carefully reviewed and edited the result.

Funding statement

No funding sources of any kind were used for the preparation of this paper.

Conflict of interest

The authors declare the following potential conflicts of interest: **RB** received honoraria for lectures or session moderation from Eli-Lilly and Novo-Nordisk, and from Pfizer and Boehringer for Advisory board meetings; **MDB** received consulting fees from Abbott Nutrition, honoraria for lectures or moderation Abbott Nutrition, Eli-Lilly and Novo-Nordisk, and travel support from Eli-Lilly; **SCB** received grants for conduction of clinical trials from Heilerde-Gesellschaft Luvos Just GmbH & Co. KG, Symbiopharm GmbH and Lactopia GmbH, honoraria from Falk Foundation e. V., Eli-Lilly, German Society for Probiotic Medicine e. V. (DeProm), Takeda Pharma Vertrieb GmbH & Co. KG, Janssen-Cilag GmbH, and participated in Advisory boards for Nestlé Health Science and MEDIBIOM a member of MEDICE – The Health Family; **RBl** received consulting fees from Nestle, lecture fees from Fresenius Kabi and was Advisory Board member for Fresenius Kabi; **YB** received lecture fees from Novo Nordisk, Eli-Lilly and Fresenius Kabi; **TC** received lecture fees from Nestle, Nutricia and Fresenius Kabi, travel support from Nutricia and Fresenius Kabi, and is board member of the Stockholm Geriatric Hospital Stockholms Sjukhem; **NEPD** received grants from Abbott Nutrition, US Department of Defense, National Institutes of Health USA, honoraria from Abbott Nutrition; **A-LdT** received honoraria for lectures from Fresenius Kabi, BBraun and Nestle Nutrition Institute, travel support from Baxter and for Advisory board meetings from Fresenius Kabi South Africa; **LG** received grants from Nestle Health Science and Foundation Nutrition2000Plus, consulting fees from Fresenius Kabi and Eli-Lilly, lecture honoraria from Fresenius Kabi; **MCG** received grants from the National Council for Scientific and Technological Development, Brazil, honoraria for lectures from Abbott Nutrition, Nutricia and Nestle Health Science Brazil, and travel support from Nestle Health Science Brazil; **DH** received lectures honoraria from Takeda Pharmaceuticals; **AJ** received grants from FONDOCYT, lectures honoraria from Abbott Nutrition and Pharma Sem, travel support from Hospifar, Megalab, FINUT –ISA, and honoraria for Advisory board meetings from FINUT –ISA and Abbott Nutrition; **SK** received honoraria for lectures from Baxter, Braun, Fresenius, Nutricia, Nestle; **GK** received lectures honoraria from IFA CELTICS (México) and travel support from Fresenius Kabi and IFA CELTICS (México); **CMM** received lectures honoraria from Abbott Laboratories; **VP** received lectures honoraria from Abbott Nutrition, Baxter, Fresinius Kabi, Nestle, Novo Nordisk, Thai Otsuka, and travel support from Abbott, Novo Nordisk, ThaiOtsuka; **AFS** received lecture fees from Novo Nordisk, Abbott Nutrition, Fresenius Kabi, InBody, travel support from Baxter, Novo Nordisk, Megalabs and Advisory board fees from Adium and Baxter; **PS-C** received travel support from Abbott Nutrition; **SMS** received grants from VectivBio, Nestlé Health Science, Dr. Falk, Sanofi, Eli-Lilly, consulting fees from Pfizer and Axium mTech, lecture honoraria from Amgen, Fresenius-Kabi, MSD, Nestlé Health Science, Nutricia, Thermo Fisher, Eli-Lilly, EverPharma, Takeda, Coloplast, Theradial, Viatris; **SC, YC, CC, LD, RF, OG-SM, JH, DI, TN, DN, HPS, JS, RS, SV, JW, KW** have no conflicts of interest to discolse.

References

[1] NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. Lancet 2024;403:1027–50.
[2] Lingvay I, Cohen RV, Roux CWL, Sumithran P. Obesity in adults. Lancet 2024;404(10456):972–87.
[3] Branca F, Nikogosian H, Lobstein T, editors. The challenge of obesity in the WHO european region and the strategies for response: Summary. Copenhagen: WHO Regional Office for Europe; 2007.
[4] Barazzoni R, Gortan Cappellari G. Double burden of malnutrition in persons with obesity. Rev Endocr Metab Disord 2020;21(3):307–13.
[5] Donini LM, Busetto L, Bischoff SC, Cederholm T, Ballesteros-Pomar MD, Batsis JA, et al. Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement. Clin Nutr 2022;41(4):990–1000.
[6] Donini LM, Busetto L, Bauer JM, Bischoff SC, Boirie Y, Cederholm T, et al. Critical appraisal of definitions and diagnostic criteria for sarcopenic obesity based on a systematic review. Clin Nutr 2020;39(8):2368–88.
[7] Prado CM, Gonzalez MC, Norman K, Orsso CE, Barazzoni R, Cederholm T, et al. Methodological standards for body composition-an expert-endorsed guide for research and clinical applications: levels, models, and terminology. Am J Clin Nutr 2025;122(2):384–91.
[8] Gortan Cappellari G, Zanetti M, Donini LM, Barazzoni R. Detecting sarcopenia in obesity: emerging new approaches. Curr Opin Clin Nutr Metab Care 2024;27:402–9.
[9] Liu C, Liu T, Deng L, Zhang Q, Song M, Shi J, et al. Sarcopenic obesity and outcomes for patients with cancer. JAMA Netw Open 2024;7(6):e2417115.
[10] Gortan Cappellari G, Guillet C, Poggiogalle E, Ballesteros-Pomar MD, Batsis JA, Boirie Y, et al. Sarcopenic obesity research perspectives outlined by the sarcopenic obesity global leadership initiative (SOGLI) - proceedings from the SOGLI consortium meeting in rome November 2022. Clin Nutr 2023;42(5):687–99.
[11] Prado CM, Batsis JA, Donini LM, Gonzalez MC, Siervo M. Sarcopenic obesity in older adults: a clinical overview. Nat Rev Endocrinol 2024;20(5):261–77.
[12] Benz E, Pinel A, Guillet C, Capel F, Pereira B, De Antonio M, et al. Sarcopenia and sarcopenic obesity and mortality among older people. JAMA Netw Open 2024;7(3):e243604.
[13] Roh E, Mook K. Health consequences of sarcopenic obesity: a narrative review. Front Endocrinol 2020;11. https://doi.org/10.3389/fendo.2020.00332.
[14] Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, et al. Writing group for the european working group on sarcopenia in older people 2 (EWGSOP2), and the extended group for EWGSOP2. Sarcopenia: revised european consensus on definition and diagnosis. Age Ageing 2019;48(1):16–31.
[15] Conte C, Hall KD, Klein S. Is weight loss-induced muscle mass loss clinically relevant? JAMA 2024;332(1):9–10.
[16] Chen AS, Batsis JA. Treating sarcopenic obesity in the era of incretin therapies: perspectives and challenges. Diabetes 2025 Jul;11. https://doi.org/10.2337/dbi25-0004. dbi250004.
[17] Mechanick JI, Butsch WS, Christensen SM, Hamdy O, Li Z, Prado CM, et al. Strategies for minimizing muscle loss during use of incretin-mimetic drugs for treatment of obesity. Obes Rev 2025 Jan;26(1):e13841.
[18] Wilding JPH, Batterham RL, Calanna S, Davies M, Van Gaal LF, Lingvay I, et al., STEP 1 Study Group. Once-weekly semaglutide in adults with overweight or obesity. N Engl J Med 2021;384(11):989–1002.
[19] Jastreboff AM, Aronne LJ, Ahmad NN, Wharton S, Connery L, Alves B, et al. SURMOUNT-1 investigators. Tirzepatide once weekly for the treatment of obesity. N Engl J Med 2022;387(3):205–16.
[20] Gonzalez MC, Mehrnezhad A, Razaviarab N, Barbosa-Silva TG, Heymsfield SB. Calf circumference: cutoff values from the NHANES 1999-2006. Am J Clin Nutr 2021;113:1679–87.
[21] Cederholm T, Barazzoni R, Austin P, Ballmer P, Biolo G, Bischoff SC, et al. ESPEN guidelines on definitions and terminology of clinical nutrition. Clin Nutr 2017;36:49–64.
[22] Cederholm T, Jensen GL, Correia MITD, Gonzalez MC, Fukushima R, Higashiguchi T, et al., GLIM Core Leadership Committee, GLIM Working Group. GLIM criteria for the diagnosis of malnutrition - a consensus report from the global clinical nutrition community. Clin Nutr 2019;38(1):1–9.
[23] Cederholm T, Jensen GL, Ballesteros-Pomar MD, Blaauw R, Correia MITD, Cuerda C, et al. Guidance for assessment of the inflammation etiologic criterion for the GLIM diagnosis of malnutrition: a modified Delphi approach. Clin Nutr 2024;43(5):1025–32.
[24] Barazzoni R, Jensen GL, Correia MITD, Gonzalez MC, Higashiguchi T, Shi HP, et al. Guidance for assessment of the muscle mass phenotypic criterion for the global leadership initiative on malnutrition (GLIM) diagnosis of malnutrition. Clin Nutr 2022 Jun;41(6):1425–33.
[25] NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. Lancet 2024 16;403:1027–50.
[26] Wang YB, Shivappa N, Hébert JR, Page AJ, Gill TK, Melaku YA. Association between dietary inflammatory index, dietary patterns, plant-based dietary index and the risk of obesity. Nutrients 2021;13(5):1536.

[27] Granic A, Jagger C, Davies K, Adamson A, Kirkwood T, Hill TR, et al. Effect of dietary patterns on muscle strength and physical performance in the very old: findings from the Newcastle 85+study. PLoS One 2016;11(3):e0149699.
[28] Bian D, Xuan C, Li X, Zhou W, Lu Y, Ding T, et al. The association of dietary inflammatory potential with sarcopenia in Chinese community-dwelling older adults. BMC Geriatr 2023;23:281.
[29] Gopinath B, Russell J, Flood VM, Burlutsky G, Mitchell P. Adherence to dietary guidelines positively affects quality of life and functional status of older adults. J Acad Nutr Diet 2014;114(2):220–9.
[30] Gopinath B, Russell J, Kifley A, Flood VM, Mitchell P. Adherence to dietary guidelines and successful aging over 10 years. J Gerontol A Biol Sci Med Sci 2016;71(3):349–55.
[31] Isanejad M, Sirola J, Mursu J, Rikkonen T, Kröger H, Tuppurainen M, et al. Association of the Baltic sea and mediterranean diets with indices of sarcopenia in elderly women, OSPTRE-FPS study. Eur J Nutr 2018;57(4):1435–48.
[32] Frampton J, Murphy KG, Frost G, Chambers ES. Higher dietary fibre intake is associated with increased skeletal muscle mass and strength in adults aged 40 years and older. J Cachexia Sarcopenia Muscle 2021;12:2134–44. https://doi.org/10.1002/jcsm.12820.
[33] Davis JA, Mohebbi M, Collier F, Loughman A, Staudacher H, Shivappa N, et al. The role of diet quality and dietary patterns in predicting muscle mass and function in men over a 15-year period. Osteoporos Int 2021;32(11):2193–203.
[34] Karlsson M, Becker W, Cederholm T, Byberg L. A posteriori dietary patterns in 71-year-old Swedish men and the prevalence of sarcopenia 16 years later. Br J Nutr 2021;128(5):1–12.
[35] Bai A, Xu W, Liang Y, Jiang Y, Lin Z. Dietary patterns from mid-through later-life in relation to sarcopenia risk over 20 years among Chinese community-dwelling oldest old individuals. Clin Nutr 2023;42(12):2569–77.
[36] Wang YB, Shivappa N, Hebert JR, Page AJ, Gill TK, Melaku YA. Dietary patterns and the risk of sarcopenia: a systematic review and meta-analysis. Curr Dev Nutr 2022;6(5). nzac001.
[37] Robinson S, Granic A, Cruz-Jentoft AJ, Sayer AA. The role of nutrition in the prevention of sarcopenia. Am J Clin Nutr 2023;118:852–64. https://doi.org/10.1016/j.ajcnut.2023.08.015.
[38] Eglseer D, Traxler M, Schoufour JD, Weijs PJM, Voortman T, Boirie Y, et al., SO-NUTS Consortium. Nutritional and exercise interventions in individuals with sarcopenic obesity around retirement age: a systematic review and meta-analysis. Nutr Rev 2023;81(9):1077–90.
[39] Weijs PJM. Protein requirement in obesity. Curr Opin Clin Nutr Metab Care 2025 Jan 1;28(1):27–32.
[40] Castaneda C, Charnley JM, Evans WJ, Crim MC. Elderly women accommodate to a low-protein diet with losses of body cell mass, muscle function, and immune response. Am J Clin Nutr 1995;62(1):30–9.
[41] Houston DK, Nicklas BJ, Ding J, , et alHealth ABC Study. Dietary protein intake is associated with lean mass change in older, community-dwelling adults: the health, aging, and body composition (health ABC) study. Am J Clin Nutr 2008;87:150–5.
[42] Meng X, Zhu K, Devine A, Kerr DA, Binns CW, Prince RL. A 5-year cohort study of the effects of high protein intake on lean mass and BMC in elderly postmenopausal women. J Bone Miner Res 2009;24(11):1827–34.
[43] Deutz NEP, Bauer JM, Barazzoni R, Biolo G, Boirie Y, Bosy-Westphal A, et al. Protein intake and exercise for optimal muscle function with aging: recommendations from the ESPEN expert group. Clin Nutr 2014;33(6):929–36.
[44] Mangano KM, Sahni S, Kiel DP, Tucker KL, Dufour AB, Hannan MT. Dietary protein is associated with musculoskeletal health independently of dietary pattern: the Framingham third generation study. Am J Clin Nutr 2017;105(3):714–22.
[45] Mendonca N, Hengeveld LM, Visser M, Presse N, Canhão H, Simonsick EM, et al. Low protein intake, physical activity, and physical function in European and North American community-dwelling older adults: a pooled analysis of four longitudinal aging cohorts. Am J Clin Nutr 2021;114:29–41.
[46] Nunes EA, Colenso-Semple L, McKellar SR, Yau T, Usman Ali M, Fitzpatrick-Lewis D, et al. Systematic review and meta-analysis of protein intake to support muscle mass and function in healthy adults. J Cachexia Sarcopenia Muscle 2022 Apr;13(2):795–810.
[47] Boirie Y, Pinel A, Guillet C. Protein and amino acids in obesity: friends or foes? Curr Opin Clin Nutr Metab Care 2023;26(6):508–13.
[48] Berger MM, Shenkin A, Schweinlin A, Amrein K, Augsburger M, Biesalski HK, et al. ESPEN micronutrient guideline. Clin Nutr 2022;41:1357–424.
[49] Dzik KP, Kaczor JJ. Mechanisms of vitamin D on skeletal muscle function: oxidative stress, energy metabolism and anabolic state. Eur J Appl Physiol 2019;119(4):825e39.
[50] Monnard CR, Dulloo AG. Polyunsaturated fatty acids as modulators of fat mass and lean mass in human body composition regulation and cardiometabolic health. Obes Rev 2021;22(Suppl 2):e13197.
[51] Hirani V, Cumming RG, Naganathan V, Blyth F, Le Couteur DG, Hsu B, et al. Longitudinal associations between vitamin D metabolites and sarcopenia in older Australian men: the concord health and aging in men project. J Gerontol A Biol Sci Med Sci 2018;73:131e8.
[52] Gkekas NK, Anagnostis P, Paraschou V, Stamiris D, Dellis S, Kenanidis E, et al. The effect of vitamin D plus protein supplementation on sarcopenia: a systematic review and meta-analysis of randomized controlled trials. Maturitas 2021;145:56–63.
[53] Smith G, Julliand S, Reeds D, Sinacore DR, Klein S, Mittendorfer B. Fish oil–derived n−3 PUFA therapy increases muscle mass and function in healthy older adults. Am J Clin Nutr 2015;102(1):115e22.
[54] Buoite Stella A, Gortan Cappellari G, Barazzoni R, Zanetti M. Update on the impact of omega 3 fatty acids on inflammation, insulin resistance and sarcopenia: a review. Int J Mol Sci 2018 Jan 11;19(1):218. https://doi.org/10.3390/ijms19010218.
[55] Bird JK, Troesch B, Warnke I, Calder PC. The effect of long chain omega-3 polyunsaturated fatty acids on muscle mass and function in sarcopenia: a scoping systematic review and meta-analysis. Clin Nutr ESPEN 2021;46:73e86.
[56] Ding C, Gao D, Wilding J, Trayhurn P, Bing C. Vitamin D signalling in adipose tissue. Br J Nutr 2012;108(11):1915–23.
[57] Bischoff SC, Boirie Y, Cederholm T, Chourdakis M, Cuerda C, Delzenne NM, et al. Towards a multidisciplinary approach to understand and manage obesity and related diseases. Clin Nutr 2017;36:917–38.
[58] Weimann A, Braga M, Carli F, Higashiguchi T, Hübner M, Klek S, et al. ESPEN guideline: clinical nutrition in surgery. Clin Nutr 2017;36(3):623–50.
[59] Singer P, Blaser AR, Berger MM, Alhazzani W, Calder PC, Casaer MP, et al. ESPEN guideline on clinical nutrition in the intensive care unit. Clin Nutr 2019;38(1):48–79.
[60] Bischoff SC, Barazzoni R, Busetto L, Campmans-Kuijpers M, Cardinale V, Chermesh I, et al. European guideline on obesity care in patients with gastrointestinal and liver diseases - joint ESPEN/UEG guideline. Clin Nutr 2022;41(10):2364–405.
[61] Barazzoni R, Bischoff SC, Busetto L, Cederholm T, Chourdakis M, Cuerda C, et al., endorsed by the ESPEN Council. Nutritional management of individuals with obesity and COVID-19: ESPEN expert statements and practical guidance. Clin Nutr 2022;41(12):2869–86.
[62] Bendavid I, Lobo DN, Barazzoni R, Cederholm T, Coëffier M, de van der Schueren M, et al. The centenary of the Harris-Benedict equations: how to assess energy requirements best? Recommendations from the ESPEN expert group. Clin Nutr 2021;40(3):690–701.
[63] Pai MP, Paloucek FP. The origin of the “ideal” body weight equations. Ann Pharmacother 2000;34(9):1066–9.
[64] Peterson CM, Thomas DM, Blackburn GL, Heymsfield SB. Universal equation for estimating ideal body weight and body weight at any BMI. Am J Clin Nutr 2016;103(5):1197–203.
[65] Krenitsky J. Adjusted body weight, pro: evidence to support the use of adjusted body weight in calculating calorie requirements. Nutr Clin Pract 2005;20(4):468–73.
[66] Wycherley TP, Moran LJ, Clifton PM, Noakes M, Brinkworth GD. Effects of energy-restricted high-protein, low-fat compared with standard-protein, low-fat diets: a meta-analysis of randomized controlled trials. Am J Clin Nutr 2012;96(6):1281–98.
[67] Nuijten MAH, Eijsvogels TMH, Monpellier VM, Janssen IMC, Hazebroek EJ, Hopman MTE. The magnitude and progress of lean body mass, fat-free mass, and skeletal muscle mass loss following bariatric surgery: a systematic review and meta-analysis. Obes Rev 2022;23(1):e13370.
[68] Villareal DT, Aguirre L, Gurney AB, Waters DL, Sinacore DR, Colombo E, et al. Aerobic or resistance exercise, or both, in dieting obese older adults. N Engl J Med 2017;376(20):1943–55.
[69] Batsis JA, Villareal DT. Sarcopenic obesity in older adults: aetiology, epidemiology and treatment strategies. Nat Rev Endocrinol 2018;14(9):513–37.
[70] Kosiborod MN, Verma S, Borlaug BA, Butler J, Davies MJ, Jon Jensen T, et al., STEP-HFpEF Trial Committees and Investigators. Effects of semaglutide on symptoms, function, and quality of life in patients with heart failure with preserved ejection fraction and obesity: a prespecified analysis of the STEP-HFpEF trial. Circulation 2024;149(3):204–16.
[71] James DE, Stöckli J, Birnbaum MJ. The aetiology and molecular landscape of insulin resistance. Nat Rev Mol Cell Biol 2021;22(11):751–71.
[72] Pileggi CA, Parmar G, Harper ME. The lifecycle of skeletal muscle mitochondria in obesity. Obes Rev 2021;22(5):e13164.
[73] Pileggi CA, Hooks BG, McPherson R, Dent RRM, Harper ME. Targeting skeletal muscle mitochondrial health in obesity. Clin Sci (Lond) 2022;136(14):1081–110.
[74] Teng S, Huang P. The effect of type 2 diabetes mellitus and obesity on muscle progenitor cell function. Stem Cell Res Ther 2019;10(1):103.
[75] Ren Q, Chen S, Chen X, Niu S, Yue L, Pan X, et al. An effective glucagon-like Peptide-1 receptor agonists, semaglutide, improves sarcopenic obesity in Obese mice by modulating skeletal muscle metabolism. Drug Des Devel Ther 2022 Oct 25;16:3723–35. https://doi.org/10.2147/DDDT.S381546.
[76] Rossi AP, Rubele S, Calugi S, Caliari C, Pedelini F, Soave F, et al. Weight cycling as a risk factor for low muscle mass and strength in a population of males and females with obesity. Obesity 2019;27(7):1068–75.
[77] West S, Scragg J, Aveyard P, Oke JL, Willis L, Haffner SJP, et al. Weight regain after cessation of medication for weight management: systematic review and meta-analysis. BMJ 2026;392:e085304. https://doi.org/10.1136/bmj-2025-085304.
[78] Dent E, Morley JE, Cruz-Jentoft AJ, Arai H, Kritchevsky SB, Guralnik J, et al. International clinical practice guidelines for sarcopenia (ICFSR): screening, diagnosis and management. J Nutr Health Aging 2018;22(10):1148–61.
[79] Hurst C, Robinson SM, Witham MD, Dodds RM, Granic A, Buckland C, et al. Resistance exercise as a treatment for sarcopenia: prescription and delivery. Age Ageing 2022;51. https://doi.org/10.1093/ageing/afac003. afac003.

[80] Steffl M, Bohannon RW, Sontakova L, Tufano JJ, Shiells K, Holmerova I. Relationship between sarcopenia and physical activity in older people: a systematic review and meta-analysis. Clin Interv Aging 2017;12:835–45.
[81] Beudart C, Dawson A, Shaw SC, Harvey NC, Kanis JA, Binkley N, et al., IOF-ESCEO Sarcopenia Working Group. Nutrition and physical activity in the prevention and treatment of sarcopenia: systematic review. Osteoporos Int 2017;28(6):1817–33.
[82] Mo Y, Zhou Y, Chan H, Evans C, Maddocks M. The association between sedentary behaviour and sarcopenia in older adults: a systematic review and meta-analysis. BMC Geriatr 2023;23(1):877.
[83] Lim C, Nunes EA, Currier BS, McLeod JC, Thomas ACQ, Phillips SM. An evidence-based narrative review of mechanisms of resistance exercise-induced human skeletal muscle hypertrophy. Med Sci Sports Exerc 2022;54(9):1546–59.
[84] Nilsson MI, Xhuti D, de Maat NM, Hettinga BP, Tarnopolsky MA. Obesity and metabolic disease impair the anabolic response to protein supplementation and resistance exercise: a retrospective analysis of a randomized clinical trial with implications for aging, sarcopenic obesity, and weight management. Nutrients 2024;16(24):4407. https://doi.org/10.3390/nu16244407.