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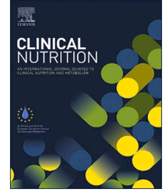

ESPEN Endorsed Recommendation

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

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S U M M A R Y

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.

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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.

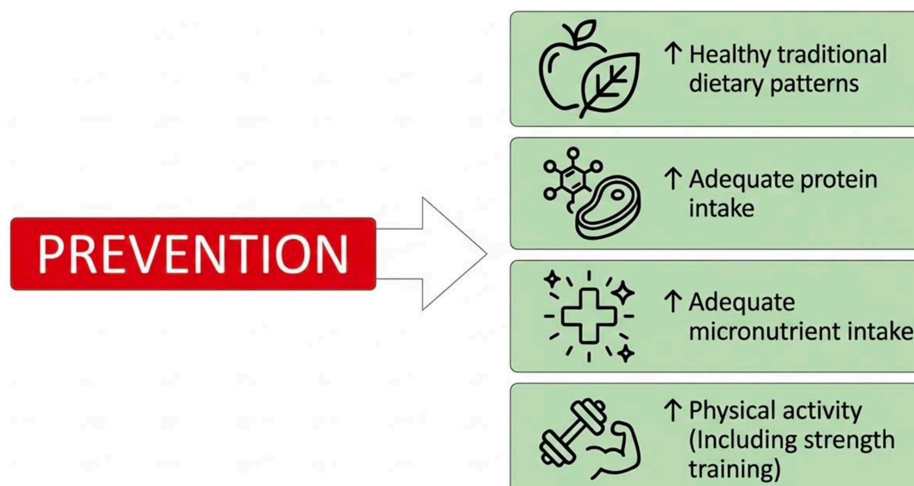

Fig. 1. Components of nutritional and lifestyle prevention of sarcopenic obesity.

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

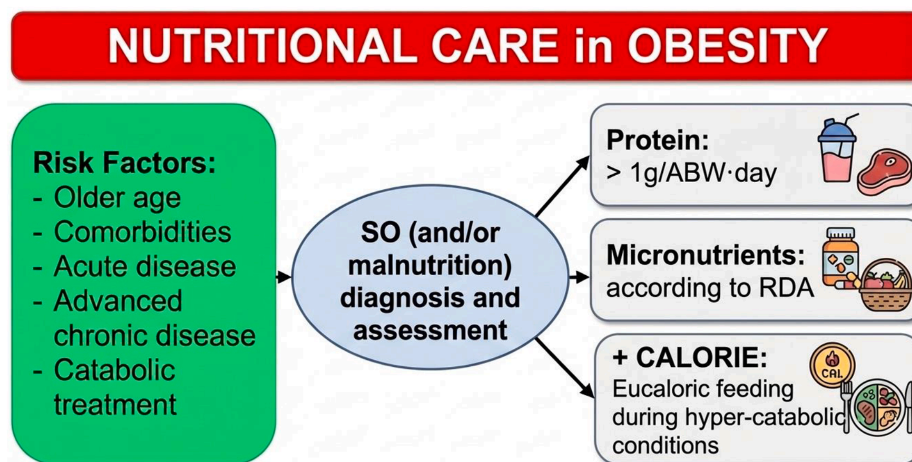

Fig. 2. Approaches for nutritional care in persons with obesity in the presence of sarcopenia and/or malnutrition.

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 underestimation 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

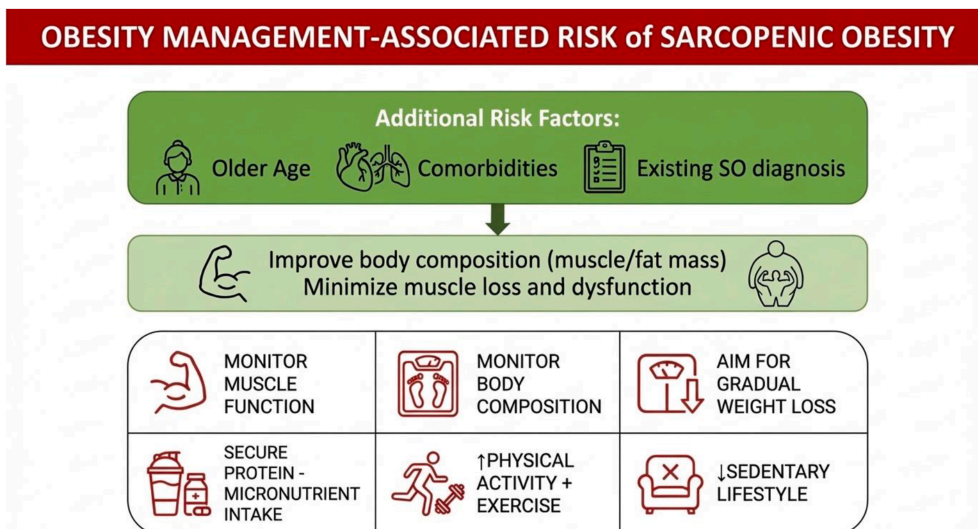

Fig. 3. Potential approaches for minimization of the risk of sarcopenia during obesity management.

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 preexisting 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.

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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.

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