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Sarcopenic Obesity in Metabolic and Bariatric Surgery: A Scoping Review

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ABSTRACT

The risk of sarcopenic obesity (SO), characterized by the coexistence of excess adiposity and low muscle mass and function, may be increased in metabolic and bariatric surgery (MBS). There is a possibility of SO development after surgery, but also aggravation of pre-existing SO, a hidden condition associated with poor health-related outcomes. This scoping review synthesizes existing literature on SO in MBS, with a thorough discussion of diagnostic criteria and assessment methods, investigation of SO prevalence (presurgery and postsurgery), incidence postsurgery, and impact on clinical outcomes. SO prevalence in MBS is highly heterogeneous, depending on the applied diagnostic criteria and body composition/physical function assessments. Following appropriate diagnostic criteria, one of four individuals both before and post-MBS seems to have SO, thus requiring targeted interventions. SO may be associated with lower weight loss and quality of life, increased risk of gastric leak, prolonged operation time, and hospital stay. Increased awareness of postsurgery SO is recommended, especially with aging. Standardization of SO diagnosis is urgently needed to improve identification and enable comparisons among studies and associations with clinical outcomes. This is important for developing effective policies, guidelines, and interventions to better address and manage this condition.

1 | Introduction

In the context of metabolic and bariatric surgery (MBS), there is an increased risk of development or aggravation of sarcopenic obesity (SO), a hidden condition characterized by the coexistence of excess fat mass (FM) with sarcopenia (i.e., low muscle mass and low muscle function) [1, 2]. This highlights the importance of muscle as a functional and metabolic organ [3]. SO is associated with even worse health-related outcomes than sarcopenia or obesity alone, including lower quality of life and increased morbidity and mortality [4–6].

This is an important area of study in an era of rapidly increasing prevalence of MBS worldwide [7]. Patients undergoing MBS require close monitoring of their nutritional and physical activity statuses, focused not only on weight loss but also on body composition and physical function, including the risk for SO. In this scoping review, we initially provide a background discussion to highlight key points for the reader about obesity, MBS, and SO. Then we review the evidence regarding SO in the context of MBS, including definitions, prevalence, incidence after surgery, and impact on clinical outcomes.

2 | Background

2.1 | Obesity and MBS

Obesity is defined as an excessive accumulation of fat, with or without adipose tissue abnormal function or distribution, with a multifactorial etiology. Obesity can be further divided into preclinical obesity, a state of excess adiposity with the preserved function of other organs and tissues, and clinical obesity, when alterations due to excess adiposity are identified in the function of organs, tissues, or the whole individual, potentially leading to noncommunicable diseases and severe end organ damage [8]. The rates are increasing globally; in 2022, there were 2.5 billion adults affected by overweight, and of these, 890 million had obesity classified by a body mass index (BMI) greater than 30 kg/m^2 [9].

MBS has strong evidence of safety and efficacy in terms of weight loss and remission of comorbidities [10]. MBS works through multiple mechanisms, including a reduction in appetite and energy intake, and alterations in gastrointestinal neurohormones (e.g., ghrelin, glucagon-like peptide-1 [GLP-1], and peptide YY [PYY]) and gut microbiota [11]. The most commonly performed procedures are sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB) [12]. Since 2012, SG has replaced RYGB as the most commonly performed MBS procedure in the United States, with similar trends worldwide [7, 13] and globally accounts for approximately 63% of current MBS [12]. SG involves the removal of the greater curvature of the stomach to create a smaller, tubular-shaped stomach [14]. RYGB (29% of MBS worldwide) [12] is a more complex procedure that involves creating a small stomach pouch and connecting it to the middle of the small intestine, bypassing the majority of the stomach, the duodenum, and part of the jejunum [14].

2.2 | SO Etiology in the Context of MBS

There are several mechanisms implicated in the development of SO, which typically arises from a combination of chronic inflammation and hormonal dysregulation compounded by reduced physical activity and a low protein and/or pro-inflammatory diet [6]. Figure 1 illustrates potential risk factors for SO in the context of MBS throughout the postoperative period, which are discussed below. MBS reduces appetite and overall energy consumption, including protein [15, 16]. This leads to a rapid and substantial weight loss of not only FM but also lean soft tissue (LST, commonly known as lean mass). At 1 year postoperatively, the average MBS recipient loses 8.1 kg of LST (approximately 23% of total weight loss) [17], increasing the risk of body composition abnormalities such as low muscle mass and sarcopenia. Over time, a combination of factors, including lifestyle and

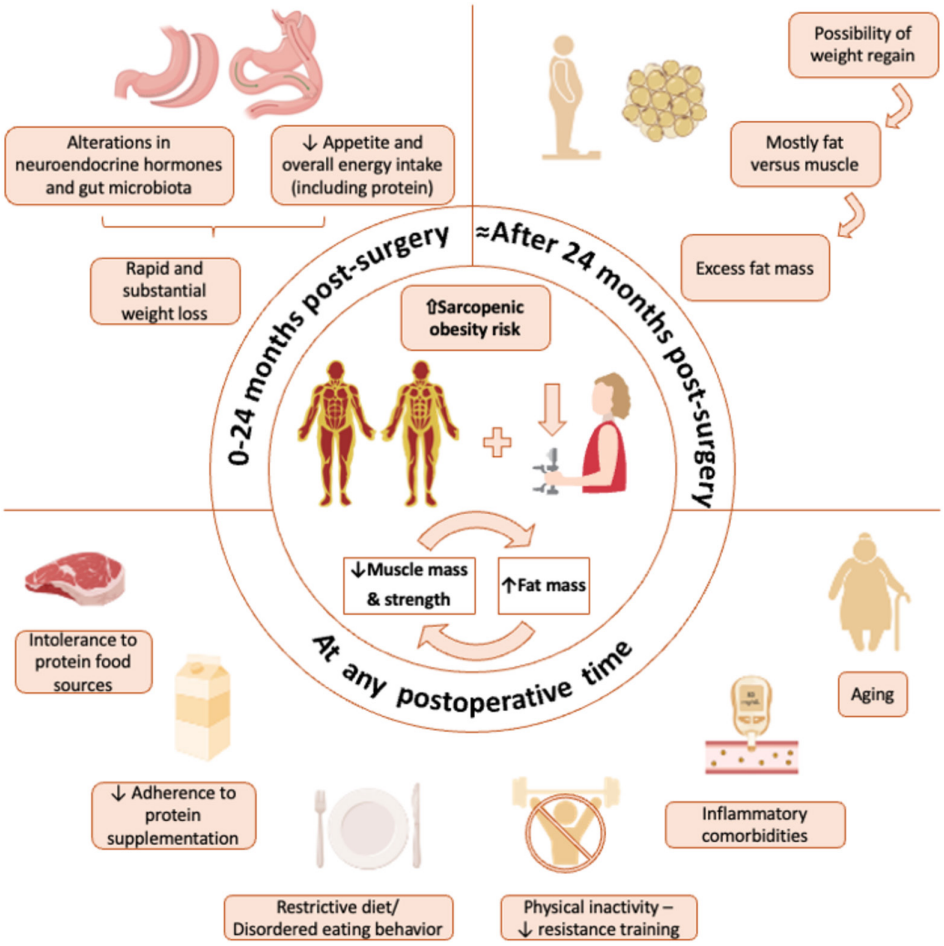

FIGURE 1 | Increased sarcopenic obesity risk post-metabolic and bariatric surgery.

physiological changes, can allow for significant weight regain, often derived from FM [18]. There is no consensus on weight regain criteria, and it could consider increases in weight (absolute or percentage) from the postoperative weight nadir or a decrease in the total weight loss percentage, which is a more clinically meaningful approach. Weight regain may be followed by re-emergence of inflammatory comorbidities [19] and perpetuation of low muscle mass alongside high FM, with elevated risk for SO [6, 20, 21].

Protein intake and exercise are chief anabolic stimuli for muscle tissue and are of high importance to optimal MBS postoperative care. However, the protein requirements post-MBS vary relative to the postoperative time based on nitrogen balance analysis, independently of surgery type, and most individuals do not reach the required protein amount [22]. Additionally, food intolerance and poor food adaptation to protein-based solid textures such as red meat are often reported post-MBS [23]. Protein supplementation is highly recommended postoperatively to account for these barriers to adequate protein intake; however, low adherence to protein supplementation [24] and unsupervised restrictive diets are common postoperatively. MBS involves important changes in food preferences and may impose a risk factor for disorganized eating patterns [25]. Onset or recurrence of disordered eating behaviors (e.g., feeding and eating disorders) can occur after surgery. This may lead to insufficient intake of micronutrients and macronutrients (especially protein) even if the energy intake is in excess [26]. Furthermore, sedentary behavior is still a concern post-MBS, especially the lack of resistance training, as surgery appears to only trivially impact moderate-to-vigorous physical exercise [27]. This can exacerbate SO as reduced muscle function leads to further reductions in physical activity, creating another self-perpetuating cycle that underlies SO. Physical exercise is required to help maintain and promote gains in LST and physical function in the postoperative period [28]. Post-MBS, resistance training stimulates improvements both in muscle function and physical performance [29] and, when combined with protein supplementation, also generates gains in muscle mass [30], which helps prevent or treat SO.

Chronic inflammation appears to be both a result of and a cause of obesity. In obesity, there is an expansion of adipose tissue subcutaneously, around viscera, and ectopic fat within muscle. Excess adipose tissue creates a pro-inflammatory environment with infiltration by immune cells, including mast cells, pro-inflammatory macrophages, and T lymphocytes. Increased levels of leptin in obesity upregulate pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) and reduce the anabolic insulin-like growth factor-1 (IGF-1) [31]. Pro-inflammatory cytokines recruit further inflammatory cells for adipose tissue and muscle infiltration [32]. Within muscle, inflammatory cells promote muscle cell atrophy, which is replaced by fibrosis and further intramuscular adipose tissue. Individuals undergoing MBS are at significant risk for SO because they have severe obesity and usually at least one associated condition linked to chronic inflammation. Insulin is a chief promoter of muscle anabolism, and insulin resistance, common in individuals with obesity, has been shown to result in both a reduction in protein synthesis and an increase in protein degradation. With advancing sarcopenia, insulin resistance also worsens further as there is a progressive loss of insulin-sensitive myocytes [32]. Chronic inflammation and insulin resistance thus create a self-fulfilling cycle propagating SO. Physical exercise also appears as a potential modulator of inflammation post-MBS, promoting reductions in leptin and, in individuals with higher visceral fat area, reduction in C-reactive protein [33].

Globally, the median age of MBS recipients varies from 31 to 45 years, depending on the country. However, many countries report that 25% of MBS is performed in individuals older than the age of 50 [12]. With increasing postoperative time, the impact of aging is also worth noting. Aging is well established as a cause of sarcopenia, sometimes referred to as primary sarcopenia, in contrast to secondary sarcopenia related to diseases like SO. Muscle protein in the body is continuously undergoing turnover, but after the age of 30, the balance progressively moves toward protein degradation rather than synthesis [31, 34]. There is a reduction in the diameter and number of muscle fibers, and muscle cells are less sensitive to anabolic stimuli; the loss of total LST decreases resting energy expenditure, which increases the risk of obesity [34]. Additionally, there are also alterations in adipose tissue, with a change in composition and redistribution of deposits, in combination with senescent cell accumulation and adipocyte progenitor functional decline [35]. Older individuals undergoing MBS may therefore be at increased risk of baseline SO because of the combined effects of primary and secondary sarcopenia, and the aging MBS recipient may also be at increased risk for SO development. Although aging typically results in the loss of muscle mass and strength and FM accumulation, not everyone will develop SO. These abnormalities occur at vastly different rates in different individuals, which reflects multiple additional mechanisms that influence SO development.

2.3 | SO Definition, Screening, and Assessment

Historically, sarcopenia has often been defined as low muscle mass or LST, irrespective of muscle function. This was particularly common when defining sarcopenia in the setting of obesity [36]. However, with time, there has been increasing emphasis on concurrent assessment of physical function (i.e., muscle function [e.g., strength] and physical performance), particularly championed within the aging literature. Several groups have previously published operational criteria to define sarcopenia. The most used is the European Working Group on Sarcopenia in Older People (EWGSOP), revised in 2019 (EWGSOP2), which defines sarcopenia as the concurrent presence of low muscle mass and reduced muscle function, either in terms of strength or performance [37]. Similarly, the Asian Working Group for Sarcopenia (AWGS) acknowledges the concurrent presence of both low muscle mass and low muscle function for the diagnosis of sarcopenia in Asian people [38]. The Foundation for the National Institutes of Health (FNIH) proposes the use of low muscle mass and weakness (low handgrip strength [HGS]) to define sarcopenia and, alternatively, the use of BMI-adjusted cutoff points [39]. Lastly, the Sarcopenia Definition and Outcomes Consortium (SDOC) defines sarcopenia as reduced HGS and slowness, indicated by diminished gait speed. The SDOC definition does not include low muscle mass and highlights the importance of reduced muscle strength as a primary parameter, as it is a more

consistent predictor of adverse outcomes than muscle mass decline alone [40]. The recent and ongoing efforts of the Global Leadership Initiative on Sarcopenia (GLIS) aim to consolidate these definitions and propose an international consensus [41].

For sarcopenia, the most used assessment of low muscle mass includes appendicular LST (sum of LST of both arms and legs) lower than two standard deviations compared to reference data, or the lowest two quintiles for relative muscle mass (ALST/height^2 or ALST/body weight × 100) of the specific population [36, 42, 43]. Obesity definitions are also variable. Excess adiposity has often been defined using BMI (kg/m^2), %FM, or assessments of height-adjusted FM index (FMI). A comprehensive list of how these conditions have been combined in the diagnosis of SO has been summarized in a previous publication [36]. A major challenge in understanding its prevalence and health outcomes has been the lack of a unanimous consensus on diagnostic criteria. To address the lack of common criteria, the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) launched the first specific diagnostic criteria for SO [1, 2]. The Sarcopenic Obesity Global Leadership Initiative (SOGLI) group proposed that SO should be defined as the coexistence of excess adiposity and low muscle mass and function [1, 2]. They have also set an agenda for research and policy priorities to advocate for the identification and treatment of this condition [44].

2.3.1 | SO: Screening and Assessment

The first step in identifying SO is screening. Ideal screening methods should be quick, practical, and cost-effective without requiring specialized knowledge [45]. The SOGLI expert group recommended high BMI, an elevated waist circumference using sex and ethnic-specific thresholds, and surrogate markers of sarcopenia as screening tools [3, 21, 44]. Of note, the use of BMI may miss hidden excess adiposity [46]. These surrogate markers include clinical signs or risk factors, including post-MBS, older age (> 70 years), presence of chronic disease, recent acute disease, history of fatigue, and functional limitation. Validated questionnaires like the Strength, Assistance with walking, Rising from a chair, Climbing stairs, and Falls (SARC-F) questionnaire (for older adults) [47], used alone or combined with calf circumference (SARC-Calf; for patients of all ages) [48], can also be applied for SO screening.

If screening results are positive, the next step involves the diagnostic process, emphasizing the evaluation of both muscle function and body composition. To conclude the process, the staging of SO is determined based on the presence of complications such as metabolic disease, functional disability, or cardiovascular or respiratory disease. Those without complications are categorized as Stage 1, whereas those with complications related to body composition or skeletal muscle functionality are classified as Stage 2 [1, 2, 44].

Regarding physical function and body composition methods used in defining SO, several modalities have been applied, each with its strengths and drawbacks. For muscle function, HGS is a simple, widely accepted method, although it only represents upper-body strength [49]. Similarly to body composition, more research is needed on adjusting muscle strength, especially as benchmarks for HGS are not well-established. The chair stand test (CST, 30 seconds or five repetitions) evaluates lower body strength and performance but can be influenced by factors like balance and the presence of osteoarthritis [50]. For physical performance, the 6-minute walk distance test (6MWD) measures the distance that an individual can quickly walk on a flat, hard surface in 6 min. It assesses aerobic capacity and endurance, but results can be affected by nonmuscular factors. The timed-up and go test (TUG), the duration to stand up, walk a distance of 3 m, turn, walk back, and sit down again, evaluates mobility, but its performance can be influenced by cognitive factors. The short physical performance battery (SPPB) assesses balance, gait speed, and lower extremity function/performance, offering a comprehensive measure of physical function but requiring special training for administration and lacking validation in adults [51].

For body composition assessment [52], dual-energy x-ray absorptiometry (DXA) is considered the reference standard in clinical settings, providing a more comprehensive analysis of body composition, but it is not always available and skeletal muscle mass (SM) is not directly assessed; instead, muscle is estimated using ALST. Bioelectrical impedance analysis (BIA) is noninvasive, affordable, and easy to use. However, it is less accurate for individuals with obesity and only estimates body composition measures. More sophisticated techniques, such as computerized tomography (CT) scans, offer precise measurements but are costly and come with radiation risks. Similarly, magnetic resonance imaging (MRI) analysis, due to its high cost and time-consuming nature, is currently limited to research settings. Ultrasound, an emerging imaging technique, is portable and free of radiation. Yet, it is operator-dependent and may be less reliable in assessing adipose tissue [52]. The reader is referred to additional publications summarizing the pros and cons of these techniques [45, 53].

A significant limitation is the absence of specific protocols and cutoff points to define abnormalities in body composition. These differ based on the evaluation technique, the target population, and the criteria used to characterize unusual levels of muscle and fat. Another important consideration is how body composition data are expressed. Adjusting for body weight is essential when obesity is present. Typically, as mentioned above, measures of low muscle mass are adjusted for height, often squared, but this might not accurately reflect the muscle mass in individuals with excess adiposity. Even with seeming muscle reduction, they might match or exceed the muscle mass of people without excess adiposity. The concept of "relative adequate muscle" is emerging but needs further research. Although some suggest using BMI as a measure, its reliability is questionable [1, 2]. Understanding the definitions, operationalizations, and evaluation methods of SO is crucial for its diagnosis, management, and research.

3 | Materials and Methods

The PI(E)COs strategy used for study inclusion in this scoping review is described in Table 1. The following exclusion criteria

TABLE 1 | PI(E)CO(s) strategy to explore sarcopenic obesity in the context of metabolic and bariatric surgery.

Population	Humans, ≥18 years old, males and females
Intervention/exposure	Metabolic and bariatric surgery of any kind (i.e., Roux-en-Y gastric bypass, sleeve gastrectomy, gastric band, duodenal switch, biliopancreatic diversion with duodenal switch, mini gastric bypass, and one anastomosis gastric bypass)
Comparator	Nonoperated peers, individuals post-metabolic and bariatric surgery without sarcopenia/sarcopenic obesity, and pre- and post-metabolic and bariatric surgery
Outcomes	Sarcopenia/sarcopenic obesity, assessed by any diagnostic criteria and assessment technique (i.e., EWGSOP, ESPEN/EASO, AWGS, SDOC, FNIH, body composition alone [e.g., muscle and surrogates], physical function alone [e.g., strength and physical function tests], and body composition plus physical function)
Studies	Original studies (observational and intervention), with no restrictions on date and language

Abbreviations: AWGS: Asian Working Group on Sarcopenia; ESPEN/EASO: Sarcopenic Obesity Consensus by The European Society for Clinical Nutrition and Metabolism and The European Association for the Study of Obesity; EWGSOP: European Working Group on Sarcopenia in Older People; FNIH: Foundation for the National Institutes of Health Sarcopenia Project; SDOC: Sarcopenia Definitions and Outcomes Consortium.

were applied: (1) conference and abstract publications; (2) studies that evaluated body composition and physical function post-MBS but did not diagnose SO/sarcopenia; and (3) studies involving individuals who underwent placement of intragastric balloon. The full search strategy for all databases is described in Appendix S1. This review protocol was not registered.

We aimed to locate both published and unpublished studies and reported results according to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines, extension for scoping reviews (Appendix S2) [54]. An initial limited search of MEDLINE (1946 to present via Ovid) was undertaken to identify articles on the topic. The text words contained in the titles and abstracts of relevant articles, and the index terms used to describe the articles were used to develop a full search strategy for MEDLINE (1946 to present via Ovid), following the Peer Review of Electronic Search Strategies (PRESS) guidelines [55]. The search employs both controlled vocabularies, such as MeSH, and keywords representing concepts related to the topic. The search strategy, including all identified keywords and index terms, was adapted for each database, and reference lists of articles selected for full-text review were used to screen for additional papers. The search strategy did not include any limiters.

Searched databases included MEDLINE (1946 to present via Ovid), CINAHL Plus with Full Text (EBSCO), Embase (1974 to present via Ovid), Cochrane Library (Wiley Online Library), and Web of Science-All Databases (Clarivate Analytics), which in itself includes the following: Web of Science Core Collection, BIOSIS Citation Index, BIOSIS Previews, CABI: CAB Abstracts, Current Contents Connect, Derwent Innovations Index, KCI-Korean Journal Database, Russian Science Citation Index, SciELO Citation Index, and Zoological Record. Sources of unpublished studies and gray literature were searched through the database: Dissertations and Theses Global (ProQuest).

All databases were searched, and the results were exported on December 5, 2022. The search was later updated on July 12, 2024, rerunning in each database to add results from 2023 to 2024. Collated citations were exported to Covidence (2021, Melbourne, Australia), and duplicates were removed. The remaining citations were screened by two independent reviewers for assessment against the inclusion criteria for the review (titles, abstracts, and full texts). Missing data were requested by email from the authors of the included studies. If no answer was received after 15 days, another email was sent, and if not successful, only the available data were included. The data were summarized narratively, followed by a descriptive quantitative summary.

4 | Results

The results of the search strategy and selection process are presented in Figure 2. We identified 22 studies [56–77] that discussed SO in the context of MBS: 10 in MBS candidates and 12 post-MBS, with eight of them also reporting prevalence before the surgery. Characteristics of the included studies are described in Table 2. Most studies were in patients post-RYGB (50.0%, $n=11$), followed by SG (36.4%, $n=8$), and no studies involved patients with other surgery types (e.g., duodenal switch or gastric banding). Most studies were in adults, and only two included individuals ≥65 years old in their sample [70, 72]. Race/ethnicity was poorly reported and, when reported, included mainly Caucasians. Information regarding SO diagnosis and associated outcomes appears in Table 3. Assessment of SO across the literature is illustrated in Figure 3, whereas the prevalence of SO at various time points is illustrated in Figure 4.

4.1 | How Has the Literature Defined SO in the Context of MBS?

The majority of the studies (59.1%, $n=13$) used body composition alone to identify SO [56, 57, 61, 63, 66–72, 74, 77]. SO was then defined as muscle mass derivative below a specified cutoff [56, 57, 61, 63, 66, 67, 70, 72, 77] or as the lowest tertile or quintile of muscle mass within the study cohort [68, 69, 71, 74].

Eight studies used a consensus definition combining the assessments of body composition and physical function, whereas

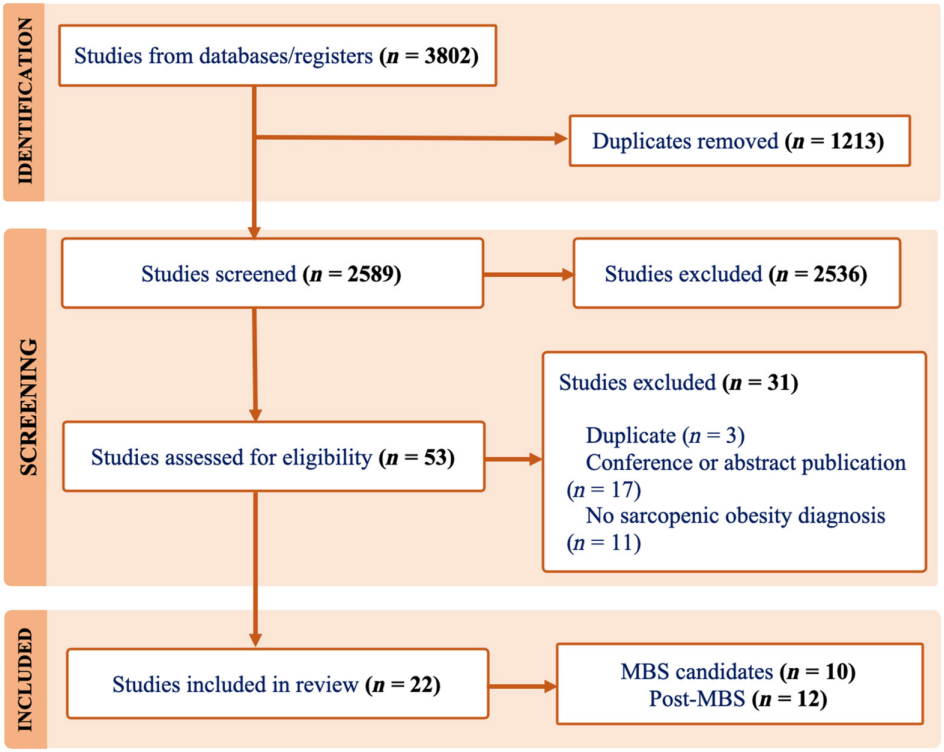

FIGURE 2 | Flow diagram of the literature search about sarcopenic obesity in the context of metabolic and bariatric surgery (MBS).

Crispim Carvalho et al. [75] used body composition or physical function for SO definition. The EWGSOP2 was the most commonly used diagnostic criteria, with different modalities to define either low muscle mass or low muscle strength/performance [58–60, 62, 64, 65, 73]. Two studies used the FNIH 2014 diagnostic criteria while also applying the EWGSOP2 [62, 73]. Two studies used the ESPEN/EASO 2022 [64, 76] and only Vieira et al. [64] used the SDOC 2020, alongside other diagnostic criteria.

Most studies (90.9%, $n = 20$) assumed the excess adiposity ("obesity") component upon eligibility criteria based on elevated BMI or post-MBS. Only the two studies that used the ESPEN/EASO 2022 consensus confirmed excess adiposity by analysis of body composition by DXA [64, 76]. Vieira et al. [64] highlighted that 30% of their sample with a median postoperative time of 6.8 years after MBS would not be considered as having excess adiposity if only BMI was used as an evaluation criterion.

4.2 | How Are Muscle Mass and Function Evaluated in the Context of MBS?

There were several techniques by which muscle mass and its derivatives were evaluated. The most commonly used method and the one used in the largest studies was BIA [58, 61, 64, 68, 70–76]. There was significant variability in how the BIA was used, with the most common approach using the Jansen 1985 equation to calculate the SM adjusted for height squared [61, 68, 72, 74], followed by adjusted ALST [58, 71, 73, 75], and one study used the ratio between FMI and fat-free mass index (FFMI) [70]. There were only three studies where BIA was adjusted for weight or BMI, using ALST or SM [64, 71, 73, 75, 76]. All studies separated analysis by sex, when applicable, but few considered and adjusted for age.

The next most common modality for muscle mass evaluation was DXA [56, 59, 60, 62, 64–67, 76]. Studies using DXA all used ALST, but there was again significant variability in how the value was adjusted. From the DXA studies, 55.6% ($n = 5$) adjusted ALST for weight or BMI [62, 64, 66, 67, 76], whereas the remaining four were adjusted by height squared. Vieira et al. [64] compared the performance of DXA and BIA in mid- to long-term post-MBS, and agreement was poor to moderate (kappa of 0.3–0.43 depending on the SO definition used). In contrast, Arnaiz et al. [76] found a kappa of 0.977 when comparing DXA and BIA in MBS candidates; however, the prevalence of low muscle mass in their sample was 88%–100%.

There were three studies that defined low muscle mass using skeletal muscle index (SMI) at the level of the third lumbar vertebra, two from CT [57, 69], and one using MRI [63]. An additional study using CT defined SO by the ratio between FM and FFM estimated by predictive equations involving skeletal muscle area and adipose tissue area [77]. Finally, one pilot study compared BIA to thigh muscle thickness by ultrasound and proposed cut-offs to predict low SMI [74]. However, low SMI was defined as the lowest tertile within the cohort, and there was no external validation.

Despite deficits in physical function in addition to low muscle mass being central to the diagnosis of SO, only nine out of 22 studies evaluated muscle strength and/or physical performance

TABLE 2 | Characteristics of the included studies assessing sarcopenic obesity in individuals in the context of metabolic and bariatric surgery.

Reference, year, country	Objective	Study design	Population/sample	Surgery-related time	Surgery type
Post-metabolic and bariatric surgery ($n=12$)					
Vaurs et al. 2015 [54], France	1. To describe MM and lean body mass changes after obesity surgery (RYGB and SG) 2. To analyze the factors that make a major contribution to changes in MM during weight loss	Prospective cohort	n: 114 (93♀, 21♂) Age: mean 39.6 ± 11.7 years BMI: mean 43.3 ± 5.4 kg/m^2 Race/ethnicity: NA	Baseline vs. 3 and 12 months	RYGB ($n=70$), SG ($n=44$)
Voican et al. 2018 [55], France	To define a predictive score of sarcopenia occurrence in a large observational prospective cohort of patients with severe obesity 1 year after SG	Prospective cohort	n: 184 (146♀, 38♂) Age: mean 42.0 ± 0.9 years BMI: mean 43.2 ± 0.5 kg/m^2 Race/ethnicity: NA	Baseline vs. 1 year	SG
Coral et al. 2021 [56], Brazil	To evaluate the correlation between sarcopenia diagnosis criteria and metabolic repercussions during the first 6 months following MBS	Prospective cohort	n: 62 (52♀, 10♂) Age: mean 38.4 ± 10.8 years BMI: mean 42.2 ± 5.4 kg/m^2 Race/ethnicity: NA	Baseline vs. 6 months	RYGB ($n=21$), SG ($n=41$)
Baad et al. 2022 [57], Brazil	To evaluate the body composition of patients undergoing SG and RYGB surgery and to correlate it with metabolic and physical performance outcomes	Cross-sectional	n: 71 (65♀, 6♂) Age: mean 41.9 ± 6.5 years BMI: mean 31.2 ± 5.4 kg/m^2 Race/ethnicity: NA	> 1 year mean 4.6 ± 3.4 years	RYGB ($n=29$) SG ($n=42$)
Buzza et al. 2022 [58], Brazil	To investigate sarcopenia incidence during the post-MBS at stable-weight period in a group of women subjected to RYGB, whose findings were compared to those of the control group, which comprised nonoperated matched women with obesity	Cross-sectional	n: 120♀ Age: mean 50.0 ± 9.7 years BMI: mean 30.2 ± 4.8 kg/m^2 Race/ethnicity: 66.7% Caucasian	≥ 2 years mean 6.0 ± 3.8 years	RYGB
Molero et al. 2022 [59], Spain	(1) To prospectively evaluate the prevalence of low-SM prior to and up to 5 years after MBS; (2) to evaluate whether presurgical low-SM is an independent risk factor for the presence of low-SM after MBS; (3) to evaluate whether low-SM is associated with weight changes after MBS; and (4) to evaluate whether the presence of low-SM prior to or up to 5 years after MBS is associated with systemic inflammation, decreased pre-albumin or lower 25 (OH) vitamin D levels.	Prospective cohort	n: 952 (713♀, 239♂) Age: mean 45.0 ± 10.9 years BMI: mean 46.0 ± 5.7 kg/m^2 Race/ethnicity: NA	Baseline vs. 1 year and 5 years	RYGB ($n=508$), SG ($n=444$)

(Continues)

TABLE 2 | (Continued)

Reference, year, country	Objective	Study design	Population/sample	Surgery-related time	Surgery type
Ruthes et al. 2022 [60], Brazil	To verify the effect of RYGB on lean mass in women prior to and 1 year of surgery, comparing protocols of both FNIH and EWGSOP2	Prospective cohort	*n*: 28♀ Age: mean 40.5 ± 9.8 years BMI: ≥ 35.0 kg/m^2; mean 42.7 ± 0.05 kg/m^2 Race/ethnicity: NA	Baseline vs. 6 months and 1 year	RYGB
Vassilev et al. 2022 [61], Germany	To investigate if the BIA as a common technique for estimating the body composition is still robust in comparison with the SMI measured by MRI in a cohort of patients undergoing RYGB	Prospective cohort	*n*: 17 (13♀, 4♂) Age: mean 41.9 ± 11.1 years BMI: mean 42.96 ± 4.5 kg/m^2 Race/ethnicity: NA	Baseline vs. 1.5, 3, and 6 months	RYGB
Vieira et al. 2022 [62], Brazil	To apply and explore the ESPEN/EASO SO consensus criteria to identify SO in adults mid to long-term post-MBS using both DXA and BIA and to further compare it with commonly used sarcopenia diagnostic criteria	Cross-sectional	*n*: 186 (169♀, 17♂) Age: 43.6 years (37.0–51.0)* BMI: 30.6 kg/m^2 (27.6–34.6)* Race/ethnicity: NA *Median (IQR)	≥ 2 years median 6.8 years (IQR: 4.1–9.5)	RYGB
Florencio et al. 2023 [63], Brazil	To assess the phase angle and sarcopenia in young individuals in the preoperative and late postoperative periods of RYGB	Cross-sectional	*n*: 69 (46♀, 23♂) Age: mean 38.1 ± 8.8 years (pre-MBS) Mean 39.1 ± 7.2 years (post-MBS) BMI: mean 44.2 ± 5.5 kg/m^2 Mean 27.1 ± 3.5 kg/m^2 Race/ethnicity: NA	45.1 ± 9.5mo	RYGB
Maïmoun et al. 2023 [64], France	To analyze the fat mass, LST, and visceral adipose tissue changes from the acute phase of body weight loss (1 month) until a recognized phase of body weight stabilization (12 and 24 months) following SG.	Prospective cohort	*n*: 83 (63♀, 20♂) Age: mean 40.9 ± 12.3 years BMI: mean 40.7 ± 4.2 kg/m^2 Weight > 190 kg excluded Race/ethnicity: Caucasian	Baseline vs. 1, 12, and 24 months	SG
Rodrigues et al. 2024 [65], Portugal	To assess the effects of MBS on SO outcomes within a 1-year follow-up period in women, considering their metabolic parameters, the remission of multiple comorbidities, and the possible impact of physical exercise in SO's management	Prospective cohort	*n*: 140♀ Age: mean 50.9 ± 7.0 years *BMI: mean 42.1 ± 8.4 kg/m^2 Weight > 140 kg excluded Race/ethnicity: NA	Baseline vs. 12 months	RYGB (*n* = 100), SG (*n* = 40)
Metabolic and bariatric surgery candidates (*n* = 10)					

(Continues)

TABLE 2 | (Continued)

Reference, year, country	Objective	Study design	Population/sample	Surgery-related time	Surgery type
Mastino et al. 2016[a] [66], France	To determine the influence of SO on RYGB and SG results regarding weight loss and comorbidities resolution at 3, 6, and 12 months	Retrospective cohort	*n*: 69 (41♀, 28♂) Age: mean 44.0 ± 2.0 years BMI*: mean 41.1 ± 0.7 kg/m^2 *BMI > 48 kg/m^2 excluded Race/ethnicity: Caucasian	Baseline* vs. 3, 6, and 12 months *SO only assessed at baseline	RYGB (*n* = 52), SG (*n* = 17)
Gaillard et al. 2018[a] [67], France	To prospectively assess the usefulness of preoperative CT scan-determined SO to predict the occurrence of staple-line leak after SG	Retrospective cohort	*n*: 205 (174♀, 31♂) Age: mean 39.0 ± 12.0 years BMI*: median 40.8 kg/m^2 *BMI ≥ 50 kg/m^2 excluded Race/ethnicity: NA	Baseline* vs. 3 months, 1 year, and 2 years *SO only assessed at baseline	SG
Xiao et al. 2018 [68], United States	To investigate the prevalence of SO and its association with health outcomes in patients seeking weight loss treatment from a bariatric center	Retrospective cohort	*n*: 144 (99♀, 45♂) Age: mean 55.6 ± 11.5 years BMI: mean 46.6 ± 8.4 kg/m^2 Race/ethnicity: Caucasian	Surgery candidates	NA
Crispim Carvalho et al. 2019 [69], Brazil	(1) Identify low SM in women with recommendation for MBS through two MM indexes (ALST/wt × 100 and ALST/BMI). (2) Compare obesity with low MM (OLMM) group to obesity with normal MM (ONMM) group regarding age, blood pressure, anthropometric measurements, body composition, HGS, 6MWD, metabolic profile, and bone mineral density for each MM index. (3) Verify any correlation between the two MM indexes and the studied variables.	Cross-sectional	*n*: 62♀ Age: mean 39.53 ± 8.99 years BMI: mean 42.6 ± 4.6 kg/m^2 Race/ethnicity: NA	Surgery candidates	NA
Molero et al. 2020 [70], Spain	To evaluate the impact of age on the prevalence of SO in MBS candidates	Cross-sectional	*n*: 1370 (1019♀, 351♂) Age: ≥ 18 to ≥ 60 years BMI: mean 46.1 ± 5.2 kg/m^2 Race/ethnicity: Caucasian	Surgery candidates	NA
Bacelar et al. 2022 [71], Brazil	To compare the prevalence of SO by different methods and the accordance between them in a group of patients with obesity referred for MBS	Cross-sectional	*n*: 189 (139♀, 50♂) Age: mean 38.1 ± 9.8 years BMI: mean 40.8 ± 4.6 kg/m^2 Race/ethnicity: NA	Surgery candidates	NA
Simo-Servat et al. 2022 [72], Spain	To compare the thigh muscle thickness measurement obtained by musculoskeletal ultrasound with that obtained by using BIA as a conventional method	Cross-sectional	*n*: 122 (89♀, 33♂) Age: mean 51.2 ± 9.75 years BMI: mean 44.22 ± 5.0 kg/m^2 Race/ethnicity: NA	Surgery candidates	NA

(Continues)

TABLE 2 | (Continued)

Reference, year, country	Objective	Study design	Population/sample	Surgery-related time	Surgery type
Crispim Carvalho et al. 2023[a] [73], Brazil	To evaluate anthropometric, metabolic, and musculoskeletal outcomes in females with low muscle mass and/or strength who underwent MBS for a 1-year follow-up	Prospective cohort	*n*: 36♀ Age: mean 39.0 ± 11.2 years BMI: mean 44.0 ± 4.4 kg/m^2 Race/ethnicity: NA	Baseline* vs. 1 year *SO only assessed at baseline	RYGB (*n* = 27), SG (*n* = 9)
Arnaiz et al. 2024 [74], Spain	To apply the ESPEN/EASO consensus criteria for the identification of SO in adult candidates for bariatric surgery by DXA and BIA according to different cutoff points	Retrospective cohort	*n*: 124 (89♀, 35♂) Age: mean 42.6 ± 8.9 years BMI: mean 45.9 ± 5.2 kg/m^2 Race/ethnicity: NA	Surgery candidates	NA
Shang-Guan et al. 2024 [75][a], China	To evaluate disparities in weight loss and conduct a quality of life (QOL) evaluation for patients with SO vs. non-SO post-SG.	Retrospective cohort	*n*: 245 (187♀, 58♂) Age: mean 29.7 ± 7.7 years BMI: mean 37.4 ± 8.2 kg/m^2 Race/ethnicity: NA	Baseline* vs. 1, 3, 6, 9, and 12 months *SO only assessed at baseline	SG

Abbreviations: 6MWD, 6-min walk distance; ALST: appendicular lean soft tissue; BIA, bioelectrical impedance analysis; BMI, body mass index; CT, computed tomography; DXA, dual-energy x-ray absorptiometry; EASO, European Association for the Study of Obesity; ESPEN, European Society for Clinical Nutrition and Metabolism; EWGSOP2, The Revised European Working Group on Sarcopenia in Older People; FNIH, Foundation of the National Institutes of Health; HGS, handgrip strength; IQR, interquartile range; MBS, metabolic and bariatric surgery; MM, muscle mass; NA, not available/not applicable; OLMM, obesity with low muscle mass; ONMM, obesity with normal muscle mass; RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy; SM, skeletal muscle mass; SMI, skeletal muscle index; SO, sarcopenic obesity.

[a]A study focused on post-MBS but only assessed SO at baseline.

[58–60, 62, 64, 65, 73, 75, 76]. HGS was done in 100% of these studies either as the lone functional measure [62, 73, 75, 76] or in addition to other measures [58–60, 64, 65], and only one of them adjusted for body size [64]. The five studies that evaluated a mixture of functional measurements [58–60, 64, 65] included HGS, as well as CST (five repetitions or 30 seconds), gait speed, 6MWD, TUG, and/or SPPB.

4.3 | What Is the Prevalence of SO in MBS Candidates?

We identified 14 studies that reported the prevalence of SO prior to MBS, including eight studies that also provided data post-surgery. SO prevalence ranged from 0% to 89.3%, depending on the diagnostic criteria used [57, 58, 60–63, 65–67, 70, 72, 73, 76, 77]. Eight of these studies used body composition alone [57, 61, 63, 66, 67, 70, 72, 77], and six used body composition in combination with physical performance [58, 60, 62, 65, 73, 76]. Of note, five additional studies defined SO based on the lowest tertile, quartile, or quintile within their study cohort; thus, they were not considered as prevalence measures for this review [68, 69, 71, 74, 75].

In the studies using body composition alone to define SO, there were three that used gold standard cross-sectional imaging assessment at the L3 vertebra level, reporting a preoperative prevalence of 8%–40.4% using CT [57, 77], and 12% using MRI [63]. In two large studies using BIA with sex-specific regression of the BMI vs. SMI relationship versus a reference group, Molero et al. reported pre-surgical SO prevalence of 20.2% and 22.9% [61, 72]. Xiao et al. reported a prevalence of SO at 50.7% in MBS candidates, using BIA FMI/FFMI above the 95th percentile compared to age, sex, and BMI-specific values from NHNES [70]. The remaining two studies used DXA and applied cutoff points proposed by consensus diagnostic criteria; however, they did not include any physical function evaluation [66, 67]. Maïmoun et al. [66] tested different muscle mass adjustments and only found SO when ALST was relative to BMI, not height squared, whereas Rodrigues et al. [67] found the highest SO prevalence among all studies involving MBS candidates with 89.3%.

The prevalence of SO was noticeably lower in studies using body composition in combination with physical function compared to body composition alone. Four studies reported 0% prevalence, all using the EWGSOP2 definition with muscle mass derivative adjusted for height squared and unadjusted HGS [58, 62, 65, 73]. Buzza et al. also used the EWGSOP2 definition, but they reported a 16.6% prevalence of SO in a nonoperated control group [60]. In this study, CST, not unadjusted HGS, was the main defining feature for low muscle strength, as nearly all individuals had HGS above the EWGSOP2 cutoffs [60]. Two studies used FNIH, with one finding a 4.1% prevalence of SO [73] and the other reported 100% pre-sarcopenia (i.e., low muscle mass by ALM/BMI) but 0% SO prevalence when unadjusted HGS was combined [62]. Only Arnaiz et al. [76] applied the ESPEN/EASO 2022 diagnostic criteria and found prevalences ranging from 12.9% to 23.4% overall, depending on the body composition method and cutoff used for HGS.

TABLE 3 | Sarcopenic obesity diagnostic criteria and prevalence in individuals in the context of metabolic and bariatric surgery.

Reference, year, country	Sarcopenic obesity diagnostic criteria	Cutoff points	Prevalence	Outcome/additional analyses
Post-metabolic and bariatric surgery ($n=12$)				
Vaurs et al. 2015 [54], France	Only body composition (DXA) ↓ALST, change over time	MM loss ≥ 15% of total weight loss* *Identified via cluster analysis within the study	3 months: 54% 12 months: 29.3% RYGB vs. Sleeve: no difference in MM change	3 months to predict excess MM loss: (1) FM loss (OR: 0.89, 95% CI: 0.81–0.97; $p=0.007$); (2) baseline TSH (OR: 0.49, 95% CI: 0.28–0.86; $p=0.01$); (3) changes in glycemia at 3 months (OR: 0.52, 95% CI: 0.28–0.95; $p=0.03$); 12 months to predict excess MM loss: (4) changes in glycemia between 3 and 12 months (OR: 0.18, 95% CI: 0.03–0.97; $p=0.046$)
Voican et al. 2018 [55], France	Only body composition (CT at 3rd lumbar) SMI (cm^2/m^2) = SMA/ht^2	SMI: ♀ < 38.5 cm^2/m^2, ♂ < 52.4 cm^2/m^2	Baseline: 8.0% vs. 1 year 32.0%, $p<0.001$	Sarcopenia vs. no sarcopenia at 1 year: (1) Male 40.7% vs. 11.2%, $p<0.001$ (2) %wt loss: 30.2 ± 1.2 vs. 26.9 ± 0.8, $p<0.05$ (3) Predictive score for sarcopenia at 1 year: 19.34 + 6.67 × (male = 1; female = 0) − 0.41 × SMI at baseline; Proposed cutoff point = 0.55
Coral et al. 2021 [56], Brazil	EWGSOP2, 2019: Sarcopenia: ↓MM (BIA: ALST/ht^2) + ↓MS (HGS) Severe sarcopenia: Sarcopenia + ↓PF (↓GS or ↑TUG)	ALST/ht^2: ♀ < 5.5 kg/m^2; ♂ < 7.0 kg/m^2 HGS: ♀ < 16 kg, ♂ < 30 kg; GS: ≤0.8 m/s TUG: ≥ 20 s	Baseline: 0% 6 months: 0% RYGB vs. Sleeve: no difference	Baseline vs. 6 months postoperative (1) ALST: 24.4 ± 0.6 vs. 20.9 ± 0.5, $p<0.001$ (2) ALST/ht^2: 9.0 ± 0.1 vs. 7.7 ± 0.1, $p<0.001$ (3) ALST/BMI: 0.58 ± 0.01 vs. 0.69 ± 0.02, $p<0.001$ (4) HGS: No difference (5) TUG: 9.9 ± 0.2 vs. 8.9 ± 0.2, $p<0.001$ (6) GS: 0.62 ± 0.01 vs. 0.69 ± 0.001, $p<0.001$
Baad et al. 2022 [57], Brazil	EWGSOP2, 2019: Sarcopenia): ↓MM (DXA: ALST/ht^2) ↓MS (HGS) SPPB	ALST/ht^2: ♀ < 5.5 kg/m^2; ♂ < 7.0 kg/m^2 HGS: ♀ < 16 kg, ♂ < 27 kg; ALST/BMI: ♀ < 0.512 kg/m^2; ♂ < 0.789 kg/m^2 SPPB: not specified	22.5% (SPPB) RYGB vs. Sleeve: no difference	SG vs. RYGB (1) %wt loss: 33.8 ± 8.2 vs. 40.2 ± 9.2, $p=0.004$ (2) ALST/ht^2: 7.9 ± 1.3 vs. 7.38 ± 1.3, $p=0.069$ (3) ALST/BMI: 0.65 ± 0.14 vs. 0.65 ± 0.13, $p=0.931$ (4) HGS: 27.3 ± 6.1 vs. 25.2 ± 7.0, $p=0.194$ (5) SPPB: 9.5 ± 1.7 vs. 9.3 ± 1.3, $p=0.293$

(Continues)

TABLE 3 | (Continued)

Reference, year, country	Sarcopenic obesity diagnostic criteria	Cutoff points	Prevalence	Outcome/additional analyses
Buzza et al. 2022 [58], Brazil	EWGSOP2, 2019: Sarcopenia: ↓MM (DXA: ALST/ht^2) + ↓MS (HGS or 5-CST) Severe sarcopenia: Sarcopenia + ↓PF (↓GS or ↓SPPB)	ALST/ht^2: < 5.5 kg/m^2 HGS: < 16 kg 5-CST: > 15 s GS: ≤ 0.8 m/s SPPB: ≤ 8 points	MBS vs. nonoperated (1) Sarcopenia: 28.3% vs. 16.6%, $p = 0.12$ (no difference) (2) Low MM: 35% vs. 18%, $p = 0.04$ Severe sarcopenia NA	Sarcopenia vs. non-sarcopenia post-MBS (1) Postoperative time: no difference (2) %wt loss: 34.3 ± 8.3% vs. 39.8 ± 8.6%, $p = 0.01$
Molero et al. 2022 [59], Spain	Only body composition (BIA): SM from Jansen equation, 1985[a]: SMI (kg/m^2) = SM/ht^2	Class I: SMI between −1 and −2 SD Class II: SMI < −2 SD Gender-specific regression line of the BMI versus the SMI relationship in a reference group	Baseline vs. 1 year vs. 5 years Class I: 15.6%, 5.3%, 16.6% Class II: 4.6%, 1.4%, 6.3%	%wt loss: Low MM vs. normal MM (1) 12 months: 40.7 ± 12.3 vs. 43.0 ± 13.5, $p < 0.05$ (2) 60 months: 32.4 ± 14.1 vs. 35.6 ± 15.0, $p < 0.05$ Baseline age independently predicted (3) ↓SMI at 1 year (HR: 1.05; 95% CI 1.02–1.08) and 5 years (HR: 1.04; 95%CI 1.02–1.07) Baseline ↓SMI independently predicted (4) ↓SMI at 1 year (HR: 10.72; 95%CI 5.77–19.90) and 5 years (HR 5.72; 95%CI 3.57–9.15) SG independently predicted (5) ↓SMI at 5 years (HR 2.31; 95%CI 1.48–3.61)
Ruthes et al. 2022 [60], Brazil	(1) EWGSOP2, 2019: Pre-sarcopenia: ↓MS (HGS) Sarcopenia: ↓MM (DXA: ALST/ht^2) + ↓MS (HGS) (2) FNIH, 2014: Pre-sarcopenia: ↓MM (DXA: ALST/BMI) Sarcopenia: ↓MM (DXA: ALST/BMI) + ↓MS (HGS)	ALST/ht^2: < 5.5 kg/m^2 ALST/BMI: < 0.512 kg/m^2 HGS: < 16 kg	Baseline: EWGSOP2: 0% FNIH: 100% pre-sarcopenia 1 year: EWGSOP2: 0% FNIH: 100% pre-sarcopenia	Baseline vs. 6 months and 1 year (1) LST: 78.66 ± 11.56 vs. 47.06 ± 5.42 vs. 46.46 ± 5.65, $p < 0.001$ (2) HGS: 29.4 ± 8.2 vs. 26.9 ± 6.4, $p = 0.044$
Vassilev et al. 2022 [61], Germany	Only body composition (MRI at 3rd lumbar) SMI (cm^2/m^2) = SMA/ht^2	SMI: ♀ < 38.5 cm^2/m^2, ♂ < 52.4 cm^2/m^2	Baseline 12% 1.5 months 17% 3 months 45% 6 months 57%	SMI during follow-up ($p < 0.001$): (1) Baseline: 52.65 ± 7.06 vs. (2) 1.5 months: 45.67 ± 6.62 vs. (3) 3 months: 43.84 ± 7.14 vs. (4) 6 months: 42.48 ± 7.86

(Continues)

TABLE 3 | (Continued)

Reference, year, country	Sarcopenic obesity diagnostic criteria	Cutoff points	Prevalence	Outcome/additional analyses
Vieira et al. 2022 [62], Brazil	(1) ESPEN/EASO, 2022: Stage I: ↑%FM + ↓MM (DXA: ALST/wt × 100; BIA: SM/wt × 100) + ↓MS (HGS or 30-CST) Stage II: Stage I + ↓PF (↓TUG or ↓6MWD) (2) EWGSOP2, 2019: Sarcopenia: ↑%FM + ↓MM (DXA/BIA: ALST/ht^2) + ↓MS (HGS or 30-CST) Severe sarcopenia: Sarcopenia + ↓PF (↓TUG or ↓6MWD) 3) SDOC, 2020: No staging, no MM assessment ↑%FM + ↓MS (HGS/BMI or HGS/FM or HGS/wt)	%FM: 20–39 years: ♀ > 39%, ♂ > 26% 40–59 years: ♀ > 41%, ♂ > 29% 60–79 years: ♀ > 43%, ♂ > 31% ALST/wt: ♀ < 23.47%, ♂ < 28.27% SM/wt: ♀ 22.1%–27.6%, ♂ 31.5%–37.0% ALST/ht^2: ♀ < 5.5 kg/m^2, ♂ < 7.5 kg/m^2 HGS: ♀ < 16 kg, ♂ < 30 kg HGS/BMI: ♀ < 0.79 kg/m^2, ♂ < 1.05 kg/m^2 HGS/FM: ♀ < 0.65 kg/kg, ♂ < 1.66 kg/kg HGSwt: ♀ < 0.34 kg/kg, ♂ < 0.45 kg/kg 30-CST: 20–29 years: ♀ < 15 rep, ♂ < 14 rep 30–39 years: ♀ < 13 rep, ♂ < 14 rep 40–49 years: ♀ < 13 rep, ♂ < 13 rep 50–59 years: ♀ < 11 rep, ♂ < 13 rep 60–69 years: ♀ < 11 rep, ♂ < 13 rep TUG: 20–29 years: ♀ > 6.96 s, ♂ > 6.56 s 30–39 years: ♀ > 7.41 s, ♂ > 6.75 s 40–49 years: ♀ > 7.59 s, ♂ > 7.46 s 50–59 years: ♀ > 8.24 s, ♂ > 8.42 s 60–69 years: ♀ > 10.60 s, ♂ > 8.26 s 6MWD: Predicted by Enright and Sherrill equation[b], 1998	ESPEN/EASO, DXA: ♀ 21.7%; ♂ 35.7%; total 23.0% Stage I: ♀ 17.4%; ♂ 21.4%; total 17.8% Stage II: ♀ 4.3%; ♂ 14.3%; total 5.2% ESPEN/EASO, BIA: ♀ 6.5%; ♂ 21.4%; total 7.9% Stage I: ♀ 4.3%; ♂ 7.1%; total 4.6% Stage II: ♀ 2.2%; ♂ 14.3%; total 3.3% EWGSOP2, DXA: ♀ 2.2%; ♂ 14.3%; total 3.3% Sarcopenia: ♀ 1.5%; ♂ 0%; total 1.3% Severe sarcopenia: ♀ 0.7%; ♂ 14.3%; total 2.0% EWGSOP2, BIA: ♀ 0%; ♂ 7.1%; total 0.7% Sarcopenia: ♀ 0%; ♂ 0%; total 0% Severe sarcopenia: ♀ 0%; ♂ 7.1%; total 0.7% SDOC, DXA: ♀ 24.6%; ♂ 85.7%; total 30.3% SDOC, BIA: ♀ 21.7%; ♂ 78.6%; total %27.0	Prevalence across postoperative years (1) ESPEN/EASO (BIA): ≥ 2 to ≤ 5 years: 1.8% > 5 to ≤ 10 years:10.3% > 10 years: 14.3% (2) ESPEN/EASO (DXA): ≥ 2 to ≤ 5 years: 16.1% > 5 to ≤ 10 years: 23.5% > 10 years: 35.7% Agreement between ESPEN/EASO vs. other diagnostic criteria (3) EWGSOP2 (BIA) κ 0.14 (95%CI −0.11, 0.40) (4) EWGSOP2 (DXA) κ 0.19 (95% CI 0.04; 0.34) (5) SDOC (BIA) κ 0.27 (95%CI 0.12, 0.42) (6)SDOC (DXA) κ 0.16 (95%CI −0.01, 0.32) Agreement between BIA vs. DXA (7) ESPEN/EASO: κ 0.43 (95%CI 0.26, 0.60) (8) EWGSOP2: κ 0.30 (95%CI −0.15, 0.75)
Florencio et al. 2023 [63], Brazil	EWGSOP, 2010: ↓MM (DXA: ALST/ht^2) + ↓MS (HGS) or ↓PF (GS)	ALST/ht^2: ♀ < 5.45 kg/m^2, ♂ < 7.26 kg/m^2 HGS: ♀ < 20 kg, ♂ < 30 kg GS: < 0.8 m/s	MBS candidates: 0% Postoperative group: 0%	(1) No correlations with phase angle MBS candidates, correlations: (2) HGS and CRP: $r = -0.454$, $p = 0.020$ (3) HGS and leptin: $r = -0.465$, $p = 0.004$ Postoperative group, correlations: (4) HGS and leptin: $r = -0.402$, $p = 0.028$ (5) ALST/ht^2 and leptin: $r = -0.410$, $p = 0.042$

(Continues)

TABLE 3 | (Continued)

Reference, year, country	Sarcopenic obesity diagnostic criteria	Cutoff points	Prevalence	Outcome/additional analyses
Maïmoun et al. 2023 [64], France	Only body composition (DXA): (1) ↓ALST or (2) ↓ALST/ht^2 or (3) ↓ALST/BMI	(1) EWGSOP2, 2019 proposed cutoffs*: ALST: ♀ < 15 kg, ♂ < 20 kg ALST/ht^2: ♀ < 5.5 kg/m^2, ♂ < 7 kg/m^2 (2) IWGS, 2011 proposed cutoffs*: ALST/ht^2: ♀ < 5.67 kg/m^2, ♂ < 7.23 kg/m^2 (3) FNIH, 2014 proposed cutoffs*: ALST: ♀ < 15.02 kg, ♂ < 19.75 kg ALST/BMI: ♀ < 0.512 kg/m^2, ♂ < 0.789 kg/m^2 *No PF evaluation	ALST/ht^2: 0% at any postoperative time ALST/BMI: Baseline: 12% 1 month: 9.6% 12 months: 5.3% 24 months: 3.3%	Age correlated with LST loss: (1) At 12 months: $r = 0.29$, $p < 0.05$ (2) At 24 months: $r = 0.26$, $p < 0.05$
Rodrigues et al. 2024 [65], Portugal	Only body composition (DXA) ↑%FM + ↓MM (ALST/wt × 100)	ESPEN/EASO, 2022 proposed cutoffs*: %FM: ♀ > 43% ALST/wt: < 23.47% *No PF evaluation ALST/wt: Lowest tertile within the study cohort (most severe SO)	Baseline: 89.3% 1 year: 2.9%	Lowest tertile decreased more at 1 year*: (1) BMI: −15.9 ± 4.6 vs. −14.8 ± 3.5 vs. −13.6 ± 3.4, $p = 0.005$ (2) Fat mass: −32.6 ± 8.6 vs. −30.5 ± 7.3 vs. −27.9 ± 7.6, $p = 0.005$ *Baseline values were statistically different, BUT no adjustments were made
Metabolic and bariatric surgery candidates ($n = 10$)				
Mastino et al. 2016[c] [66] France	Only body composition (BIA): SM from Jansen equation, 1985[a]: SMI (kg/m^2) = SM/ht^2	SMI: Lowest tertile within the study cohort	33.3%[d]	Baseline SO vs. no SO: No differences in 6MWD, wall sit test, early complications, %wt loss, and excess wt loss at 12 months
Gaillard et al. 2018[c] [67], France	Only body composition (CT at 3rd lumbar) SMI (cm^2/m^2) = SMA/ht^2	SMI: Lowest tertile within the study cohort using TPA or TMA TPA: ♀ < 6.08 cm^2/m^2, ♂ < 8.2 cm^2/m^2 TMA: ♀ < 59.16 cm^2/m^2, ♂ < 63.5 cm^2/m^2	TPA: 32.2%[d] TMA: 30.7%[d]	Gastric leak at 3 months ($n = 9$, 4.4%) Baseline SO (as per TPA) vs. no SO: 66.7% leak vs. 30.6% no leak ($p = 0.032$)

(Continues)

TABLE 3 | (Continued)

Reference, year, country	Sarcopenic obesity diagnostic criteria	Cutoff points	Prevalence	Outcome/additional analyses
Xiao et al. 2018 [68], United States	Only body composition (BIA): FMI/FFMI FMI = FM/ht^2 FFMI = FFM/ht^2	95th of FMI/FFMI distribution from NHNES BMI 30–39.9 kg/m^2: Age 18–39.9 years: ♀ > 1.01, ♂ > 0.57 Age 40–59.9 years: ♀ > 0.93, ♂ > 0.59 Age 60–69.9 years: ♀ > 0.97, ♂ > 0.59 Age 70–90 years: ♀ > 0.95, ♂ > 0.61 BMI ≥ 40 kg/m^2: Age 18–39.9 years: ♀ > 1.17, ♂ > 0.86 Age 40–59.9 years: ♀ > 1.10, ♂ > 0.79 Age 60–69.9 years: ♀ > 1.02, ♂ > 0.66 Age 70–90 years: ♀ > 1.12, ♂ > 0.69	50.7%	NA
Carvalho et al. 2019 [69], Brazil	Only body composition (BIA): (1) ALST/BMI (2) ALST/wt × 100	ALST/BMI: Lowest quintile within the study cohort ALST/wt × 100: Lowest quintile within the study cohort	ALST/BMI: 20.3%[d] ALST/wt: 30.5%[d]	Low MM (ALST/wt) vs. normal MM (1) HGS: 24.2 ± 3.3 vs. 30.7 ± 4.6, $p < 0.001$ (2) 6MWD: 359 ± 51 vs. 380 ± 61, $p = 0.225$ Low MM (ALST/BMI) vs. normal MM (3) HGS: 23.2 ± 3.3 vs. 30.2 ± 4.6, $p < 0.001$ (4) 6MWD: 338 ± 51 vs. 383 ± 57, $p = 0.017$ Correlations: (5) ALST/wt vs. HGS $r = 0.59$, $p < 0.001$ (6) ALST/BMI vs. HGS $r = 0.65$, $p < 0.001$ (7) ALST/wt or ALST/BMI vs. 6MWD no differences

(Continues)

TABLE 3 | (Continued)

Reference, year, country	Sarcopenic obesity diagnostic criteria	Cutoff points	Prevalence	Outcome/additional analyses
Molero et al. 2020 [70], Spain	Only body composition (BIA): BIA: SM from Jansen equation, 1985[a]: (1) SMI (kg/m^2) = SM/ht^2 (2) %SM = SM/wt × 100	Class I: SMI or %SM between −1 and −2 SD Class II: SMI or %SM < −2 SD Gender-specific regression line of the BMI versus the SMI or %SM relationship in a reference group	As per SMI: Class I: ♀ 17.3%, ♂ 14.8% Class II: ♀ 5.6%, ♂ 1.4% As per %SM: Class I: ♀ 15.6%, ♂ 13.4% Class II: ♀ 5.7%, ♂ 1.7%	(1) Sarcopenia by age groups, as per SMI: ♀ 12.0%, ♂ 15.4% (age 18–39) ♀ 13.9%, ♂ 15.4% (age 40–49) ♀ 22.6%, ♂ 12.9% (age 50–59) ♀ 29.1%, ♂ 16.7% (age ≥ 60) (2) Sarcopenia by age groups, as per %SM: ♀ 10.5%, ♂ 14.6% (age 18–39) ♀ 12.2%, ♂ 12.0% (age 40–49) ♀ 20.8%, ♂ 12.9% (age 50–59) ♀ 27.4%, ♂ 16.7% (age ≥ 60) (3) Agreement between SMI vs. %SM ϰ 0.886, $p < 0.001$ (4) Age (HR 1.04, 95% CI 1.03–1.05) and female sex (HR 1.44, 95% CI 1.04–1.99) increased the likelihood of SO
Bacelar et al. 2022 [71], Brazil	(1) EWGSOP2, 2019: ↓MM (BIA: ALST/ht^2) + ↓MS (HGS) (2) FNIH, 2014: ↓MM (BIA: ALST/BMI) + ↓MS (HGS) (3) SO: ↓MM (BIA: ALST/wt x100) + ↓MS (HGS)	ALST/ht^2: ♀ < 5.5 kg/m^2; ♂ < 7.0 kg/m^2 ALST/BMI: ♀ < 0.512 kg/m^2; ♂ < 0.789 kg/m^2 ALST/wt: ♀ ≤ 22.1%; ♂ ≤ 31.5% HGS: ♀ < 16 kg, ♂ < 27 kg	(1) EWGSOP2: 0%, ↓ALST/ht^2: 0% (2) FNIH: 4.1%, ↓ALST/BMI: 38.1% (3) SO: 1.1%, ↓ALST/wt: 13.2%	(1) Agreement between FNIH vs. SO: ϰ 0.662, $p < 0.001$ (2) ↓HGS: 2.1% (3) ↓GS: 50.3%
Simo-Servat et al. 2022 [72], Spain	Only body composition (BIA and ultrasound): (1) SM from Jansen equation, 1985[a]: SMI (kg/m^2) = SM/ht^2 (2) Thigh muscle ultrasound thickness: rectus femoris + vastus intermedius	SMI: Lowest tertile within the study cohort	BIA: 32.8%[d]	Correlations: (1) LST (BIA) vs. thigh muscle thickness (ultrasound): $r = 0.46$, $p < 0.001$ (2) SMI (BIA) vs. thigh muscle thickness by ultrasound: $r = 0.47$, $p < 0.001$ (3) Proposed cutoff point for thigh muscle thickness (ultrasound): 1.57 cm

(Continues)

TABLE 3 | (Continued)

Reference, year, country	Sarcopenic obesity diagnostic criteria	Cutoff points	Prevalence	Outcome/additional analyses
Crispim Carvalho et al. 2023[c] [73], Brazil	Body composition and/or PF: (1) ↓MM (BIA: ALST/wt) and/or (2) ↓MS (HGS)	ALST/wt × 100: Lowest quartile within the study cohort HGS: Lowest quartile within the study cohort	41.7%[d]	At 1 year: (1) Highest preoperative ALST/wt quartile positively associated with bone mineral density variables ($p < 0.05$) (2) Decrease in absolute SM and HGS, but increase when relative to BMI ($p < 0.05$)
Arnaiz et al. 2024 [74], Spain	ESPEN/EASO, 2022: ↑%FM (DXA) + ↓MM (DXA: ALST/wt × 100; BIA: SM/wt × 100) + ↓MS (HGS)	%FM: 20–39 years: ♀ > 39%, ♂ > 26% 40–59 years: ♀ > 41%, ♂ > 29% ALST/wt (DXA): ♀ < 19.4%, ♂ < 25.7% SM/wt (BIA 1): ♀ 22.1%, ♂ 31.5% SM/wt (BIA 2): ♀ 24.15%, ♂ 32.05% HGS 1: Age < 45 years: ♀ < 18, ♂ < 32.6 Age 45–60 years: ♀ < 15, ♂ < 34.5 HGS 2: ♀ < 16 kg, ♂ < 27 kg HGS 3: ♀ < 16.8 kg, ♂ < 25.1 kg	DXA + HGS1: ♀15.7%; ♂40.0%; total 22.6% HGS2: ♀11.2%; ♂17.1%; total 12.9% HGS3: ♀12.7%; ♂17.1%; total 13.7% BIA 1 + HGS1: ♀14.6%; ♂40.0%; total 21.8% HGS2: ♀11.2%; ♂17.1%; total 12.9% HGS3: ♀13.5%; ♂17.1%; total 14.5% BIA 2 + HGS1: ♀16.9%; ♂40.0%; total 23.4% HGS2: ♀13.5%; ♂17.1%; total 14.5% HGS3: ♀15.7%; ♂17.1%; total 16.1%	SO agreement using HGS 1 + DXA vs. BIA 1: ϰ 0.977 DXA vs. BIA 2: ϰ 0.977 BIA 1 vs. BIA 2: ϰ 0.954
Shang-Guan et al. 2024[c] [75], Spain	Only body composition (CT at 3rd lumbar): SMA (cm^2) + total fat area (subcutaneous + visceral adipose tissues) FM (kg) = 0.042 × total fat area + 11.2 FFM (kg) = 0.3 × SMA + 6.06 SO: FM/FFM	FM/FFM ≥ 0.8	40.4%	SO vs. no SO (baseline) (1) Operation time: 114.0 ± 22.7 vs. 104.4 ± 21.9, $p < 0.001$ (2) Hospital stay: 5.8 ± 2.2 vs. 5.1 ± 1.4, $p < 0.001$ (3) IMAT: 4.4 ± 3.2 vs. 3.3 ± 2.6, $p = 0.007$ SO vs. no SO (postoperatively) (4) ↓%EWL at 1, 3, 6, 9, and 12 months ($p < 0.05$) (5) Quality of life at 6 months[e]: 0.86 ± 1.01 vs. 1.10 ± 1.14, $p = 0.001$

Abbreviations: 5-CST, five-repetition chair stand test; 6MWD, 6-min walk distance; 30-CST, 30-s chair stand test; ALST, appendicular lean soft tissue; BIA, bioelectrical impedance analysis; BMI, body mass index; CI; confidence interval; CT, computed tomography; DXA, dual-energy x-ray absorptiometry; EASO, European Association for the Study of Obesity; ESPEN, European Society for Clinical Nutrition and Metabolism; EWGSOP, European Working Group on Sarcopenia in Older People; FNIH, Foundation of the National Institutes of Health; FFM, fat-free mass; FFMI, fat-free mass index; FM, fat mass; FMI, fat mass index; GS, gait speed; HGS, handgrip strength; HR, hazard ratio; ht, height; IMAT, intramuscular adipose tissue; LST, lean soft tissue; MBS, metabolic and bariatric surgery; MM, muscle mass; MRI, magnetic resonance imaging; MS, muscle strength; NA, not available/not applicable; NHANES, National Health and Nutrition Examination Survey; OR, odds ratio; PF, physical function; rep, repetitions; RYGB, Roux-en-Y gastric bypass; SD, standard deviation; SDOC, Sarcopenia Definition and Outcomes Consortium; SG, sleeve gastrectomy; SM, skeletal muscle mass; SMA, skeletal muscle cross-sectional area; SMI, skeletal muscle index; SO, sarcopenic obesity; SPPB, short physical performance battery tests; TMA, total muscle area; TPA: total psoas area; TSH: thyroid stimulant hormone; TUG, timed-up and go test; wt, weight.

[a]SM = (ht^2/resistance × 0.401) + ([male = 1, female = 0] × 3.825) + (age × − 0.071) + 5.102.

[b]6MWD = ♀ 6MWD = (2.11 × ht) − (2.29 × wt) − (5.78 × age) + 667 m; ♂(7.57 × ht) − (5.02 × age) − (1.76 × wt) − 309 m;

[c]A study focused on post-MBS but only assessed SO at baseline.

[d]Not a prevalence measure; percentage defined based on lowest tertile, quartile, or quintile within the study cohort.

[e]Using Moorhead–Ardelt QOL questionnaire II (MA II).

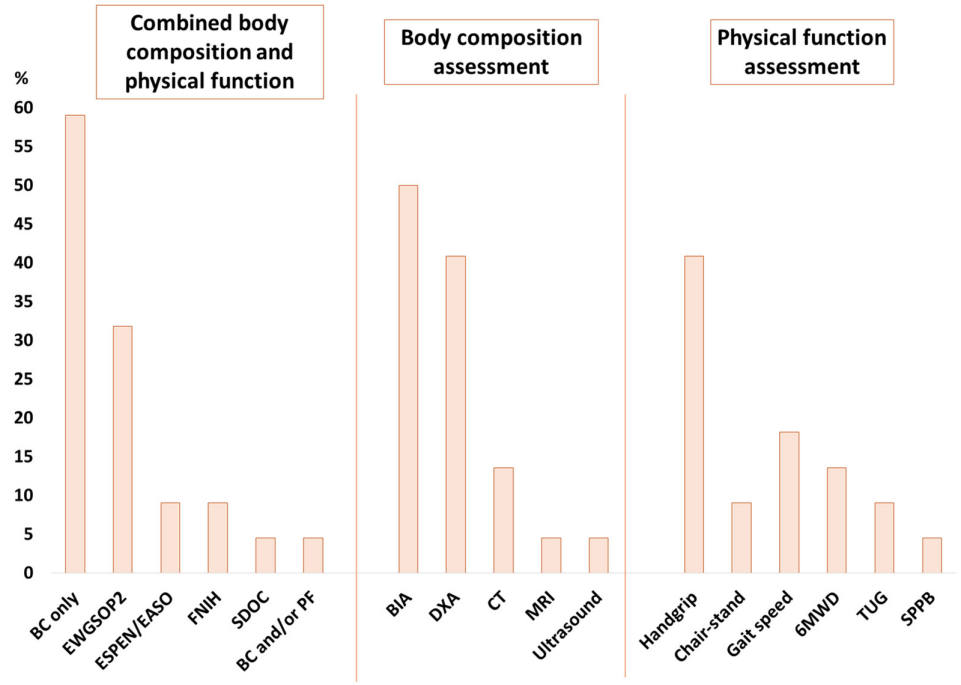

FIGURE 3 | Prevalence of sarcopenic obesity assessment in the context of metabolic and bariatric surgery among the included studies 6MWD, 6-min walk distance; BC, body composition; BIA, bioelectrical impedance analysis; CT, computed tomography; DXA, dual-energy x-ray absorptiometry; EASO, European Association for the Study of Obesity; ESPEN, European Society for Clinical Nutrition and Metabolism; EWGSOP2, The Revised European Working Group on Sarcopenia in Older People; FNIH, Foundation of the National Institutes of Health; MRI, magnet resonance imaging; PF, physical function; SDOC, Sarcopenia Definition and Outcomes Consortium; SPPB, short physical performance battery tests; TUG, timed-up and go test.

4.4 | What Is the Prevalence of SO After MBS?

There were 12 studies reporting the prevalence of SO after MBS, eight of which were prospective cohort studies evaluating patients at specified time points postoperatively [56–58, 61–63, 66, 67], and four cross-sectional studies with variable postoperative times [59, 60, 64, 65]. In prospective studies using body composition alone, the prevalence of SO 3 months after surgery was 45% using MRI [63] and 54% using a definition of ≥ 15% of weight lost from muscle mass as per DXA [56]; 57% at 6 months using MRI [63]; and at 1 year 2.9%–5.3% using DXA adjusted by weight or BMI [66, 67], 6.7% using BIA adjusted for height squared [61], 29.3% using a definition of ≥ 15% of weight lost from muscle mass as per DXA [56], and 32% using CT [57]. Maïmoun et al. [66] and Molero et al. [61] were the only studies with follow-up beyond 1 year, reporting 3.3% at 2 years [66] and 22.7% at 5 years [61], respectively. Of the prospective cohort studies, only two used body composition in combination with physical performance [58, 62]. Using the EWGSOP2 definition with muscle mass derivative adjusted for height squared and unadjusted HGS, Coral et al. reported a 0% prevalence of SO at 6 months [58, 62], whereas Ruthes et al. also reported 0% prevalence at 1 year [62].

There were four cross-sectional studies evaluating body composition in combination with physical performance in patients who were at least 1–2 years post-MBS, with mean postsurgical times of approximately 4–6 years. Vieira et al. compared six definitions of SO, evaluating body composition with both BIA and DXA and using multiple methods to define low muscle strength and performance [64]. They found a prevalence of SO between 0% and 30.3% depending on the definition used (see Table 3) [64]. In this study, HGS was adjusted for BMI and weight, and unlike unadjusted HGS at EWGSOP2 cutoffs, was able to identify individuals with low muscle strength/SO [64]. Significantly more patients were classified as having SO when DXA was used rather than BIA (e.g., 23% vs. 7.9% using ESPEN/EASO and 3.3% vs. 0.7% using EWGSOP2). Likewise, more patients were classified as having SO using the SDOC and ESPEN/EASO definitions compared to EWGSOP2. When prevalence was stratified based on length of postoperative time (2–5 years vs. 5–10 years vs. > 10 years), the prevalence of SO appears to increase with greater postoperative time [64]. The three other cross-sectional studies used the EWGSOP2 to define SO. Although Florencio et al. [65] found a 0% prevalence, the other two reported 22.5%–28.3% [59, 60]. The difference was that instead of using unadjusted HGS for muscle strength evaluation, Buzza et al. used 5-CST [60], whereas Baad et al. used SPPB [59]. Both studies also evaluated unadjusted HGS, which was not sensitive compared to other modalities for identifying low muscle strength.

4.5 | Incident SO After MBS: When Does SO Develop?

As previously noted, most of the cohort data at various time points after MBS come from studies using body composition alone. Within these limitations, SO can be present before surgery and develop as soon as 6 weeks after MBS, as seen by Vassilev et al. using repeated MRI measurements [63]. In the largest study using BIA, Molero et al. found an initial reduction in SO from 20.2% at baseline to 6.6% at 1 year [61], followed by a rebound to 22.7% at 5 years. In contrast, Maïmoun et al. [66]

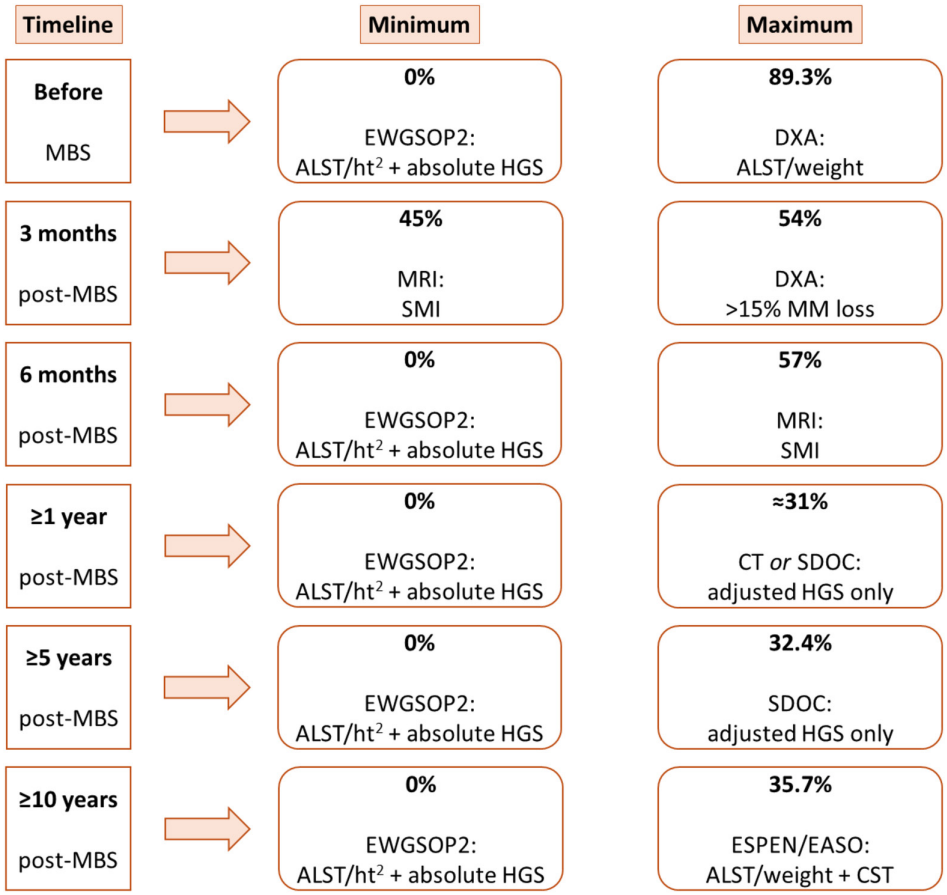

FIGURE 4 | Sarcopenic obesity prevalence according to different diagnostic criteria in the context of metabolic and bariatric surgery (MBS) among the included studies. ALST/ht^2, appendicular lean soft tissue divided by height squared; BIA, bioelectrical impedance analysis; CT, computed tomography; DXA, dual-energy x-ray absorptiometry; EASO, European Association for the Study of Obesity; ESPEN, European Society for Clinical Nutrition and Metabolism; EWGSOP2, The Revised European Working Group on Sarcopenia in Older People; HGS, handgrip strength; MM, muscle mass; MRI, magnetic resonance imaging; SDOC, Sarcopenia Definition and Outcomes Consortium; SMI, skeletal muscle mass index.

found a gradual decrease in SO throughout the postoperative period (0–24 months). Baseline SO independently predicted SO at 1 and 5 years post-MBS, and age was associated with muscle mass loss and SO [61, 66]. Voican et al. developed a predictive score for new sarcopenia (defined as low SMI) at 1 year after SG and found male sex and baseline SMI were independent predictors [57]. However, there was no external validation for this score.

There were two studies using physical parameters in addition to body composition to evaluate SO in the first year after MBS. Coral et al. found no SO at baseline or 6 months after surgery using EWGSOP2 with unadjusted HGS, the limitations of which were discussed above [58]. They reported that despite a significant reduction in muscle mass, there was no change in absolute HGS and significant improvements in TUG and gait speed. Ruthes et al. found a significant decrease in HGS after 1 year [62], although this was absolute and not relative to body size.

4.6 | Is There a Difference in SO Depending on the Type of MBS?

Limited studies compared RYGB with SG [56, 58, 59, 61, 67, 75] in terms of outcomes. In five of them, there were no differences between the surgical modalities on SO overall or using individual parameters of muscle mass or strength [56, 58, 59, 67, 75], even despite a higher total weight loss in RYGB [59]. The only study with a long-term follow-up of 5 years is by Molero et al. [61]; they did not find differences at 1 year, but at 5 years, SG was an independent risk factor for low SM.

4.7 | What Is the Impact of SO on Outcomes After MBS?

Three authors examined the association between body composition-defined SO and perioperative complications. Using CT, Gaillard et al. found an association between baseline SO and increased incidence of gastric leak [69], and Shang-Guan et al. [77] found an increased operation time and hospital stay postoperatively, whereas using BIA, Mastino et al. found no differences [68]. There were six studies that assessed the relationship between SO and postoperative weight loss, with conflicting results. At 1 year after surgery, Voican et al. [57] and Rodrigues et al. [67] found a greater total percent weight loss or BMI, Mastino et al. found no differences in weight loss [68], and Molero et al. [61] and Shang-Guan et al. [77] found a lower total or excess percent weight loss. In two studies with longer postoperative time, both

found significantly lower total percent weight loss in individuals with SO at 5–6 years after surgery [60, 61]. Quality of life evaluated by multiple tools was also shown to be reduced at 6 months post-surgery in individuals with SO [77]. HGS and ALST/ht^2 presented moderate negative correlations with leptin both in MBS candidates and post-MBS [65].

5 | Discussion

In our scoping review of SO in the context of MBS, we found significant heterogeneity in the assessment of SO, leading to a broad range of prevalence estimates from 0% to 89.3%. Despite this variability, when applying specific SO diagnostic criteria, 1 of 4 patients seemed to have SO prior to and after MBS. This is especially interesting considering the mean age of individuals undergoing MBS, emphasizing this is not necessarily an age-related process. SO may develop within the first few months post-MBS; however, low muscle mass and muscle loss during this initial phase do not necessarily result in worse functional outcomes, and improvements in muscle performance can occur. Nonetheless, SO likely presents a significant concern in late stages post-MBS, as the only study with follow-up extending beyond 1 year indicated an increasing prevalence of SO over time, although muscle function was not evaluated. Limited data indicate similar SO prevalence by surgery type, and that SO may lead to an increased risk of early anastomotic leak, prolonged operation time, and hospital stay, and possibly inferior weight loss and quality of life post-MBS.

SO is likely to be a significant issue in cases of late weight regain after MBS, as most of the weight gained back may be FM, and there is limited or no recovery of muscle mass that was lost during the initial rapid weight loss phase [1, 6]. With an increase in FM, poor dietary intake, and a decline in physical activity/exercise, the cascade of factors promoting SO returns, including chronic inflammation and insulin resistance, resulting in a very high risk of SO [78]. However, a proportion of patients who maintain optimal weight loss, adequate protein intake, and physical activity/exercise could be prevented from developing sarcopenia [29, 30, 79]. Given the profound negative health and functional effects of SO and the potential for preventive measures, it is an important condition that deserves further attention and study in this high-risk population.

Our review identified significant heterogeneity in how SO was assessed and defined, which is consistent with historically changing concepts and definitions of this condition. Most studies relied on body composition alone for diagnosing SO, which may impact its prevalence by omitting physical function assessment. As expected, the most used technique for assessing muscle mass surrogates was BIA, given its accessibility in clinical practice. However, BIA has significant limitations as it does not directly measure any body compartment, relying on estimates derived from other body composition methods [80]. In individuals with obesity, especially with increasingly higher BMI, BIA is considered to be even less accurate because it assumes a constant hydration of FFM, when in fact there is increased hydration of FFM within adipose and connective tissue [81]. DXA is considered preferable to BIA in individuals with obesity, but it also has limitations, including indirect assessment of muscle mass and extrapolating ALST to whole body mass. Indeed, DXA may also overestimate FFM in the setting of obesity [81, 82]. SMI derived from CT or MRI is likely a preferred method of assessing body composition in the setting of obesity [80]. Although this imaging is not routinely performed in the clinical setting and its use cannot be advocated for general use in all patients, these scans are routinely performed in cases of surgical complications, which often result in reduced nutritional intake, inflammation, and unintentional weight loss. Since these are the patients who would also be at the highest risk of SO, low SM should be investigated when cross-sectional imaging is available, with further evaluation for functional losses.

Our review found a variety of assessment tools used to assess physical function, but by far, the most popular was HGS, which was done in all studies that employed physical function testing. HGS is a popular method because it is readily accessible, reproducible, and applicable in clinical settings [83]. However, HGS may not reflect overall body strength or physical performance, and it also varies depending on BMI, and there are no validated BMI-specific cutoffs. On the other hand, CST assesses lower body muscle strength and performance, which may be more relevant to overall function and is more sensitive to detect changes over time. Nevertheless, CST could be limited by non-SO-related factors involving physiological and psychological aspects such as lower extremity osteoarthritis and balance [84]. HGS and CST have been recommended by SO/sarcopenia diagnostic criteria as interchangeable methods to assess muscle strength; it is important to consider that these tests evaluate different components of muscle strength and may therefore identify different individuals with the condition and also those at risk for poor clinical outcomes [85].

In studies that evaluated physical function, the most used consensus definition was EWGSOP2 [37]. However, EWGSOP2 was developed for the identification of sarcopenia, without the coexistence of obesity, and for older adults. Thus, it appears to be inappropriate for SO assessment in the context of MBS, as indicated by some studies included in this review that identified 0% SO prevalence. The EWGSOP2 definition recommends adjusting muscle mass by height squared, which may not identify low muscle mass in individuals with obesity, where there can be seemingly normal or high muscle mass, but low relative to total weight or BMI [62, 64]. Another concern is the use of absolute HGS cutoffs (e.g., 27 kg males and 16 kg females), which indicate low muscle strength in older adults [49]. Most participants in MBS studies are in their forties [12], where normative values for HGS are substantially higher (e.g., 38 kg males and 23 kg females represent the bottom 10th percentile at the age of 40) [49]. CST and adjusted HGS were able to identify more individuals with low muscle strength when compared to absolute HGS [60, 64], demonstrating that the absolute HGS cutoffs from EWGSOP2 are insufficiently sensitive for identifying low muscle strength in younger individuals with obesity.

The ESPEN/EASO 2022 consensus definition was the first consensus definition developed specifically for SO [1, 2]. These diagnostic criteria provide specific assessments and multiple cutoff points (age and race-specific) to identify excess adiposity, also indicating that muscle mass should be adjusted for weight (or BMI). This definition should likely be used for future studies

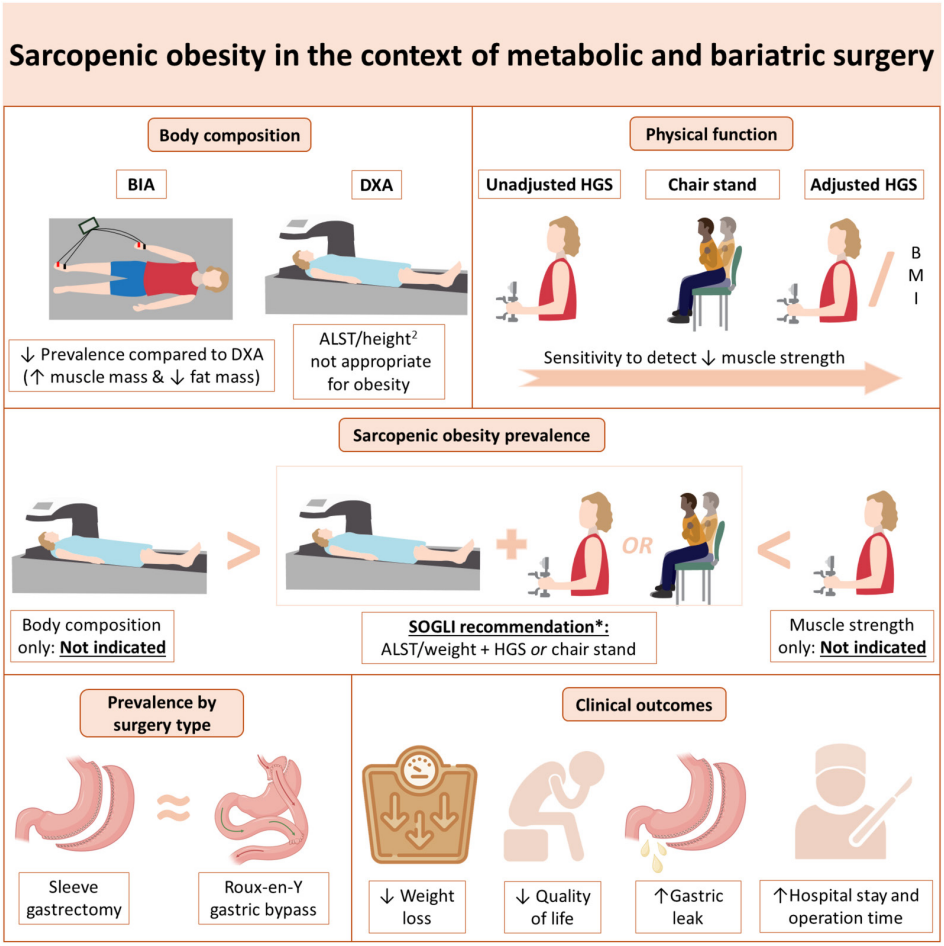

FIGURE 5 | Summary of findings of the investigation of sarcopenic obesity in the context of metabolic and bariatric surgery. ALST, appendicular lean soft tissue; BIA, bioelectrical impedance analysis; DXA, dual-energy x-ray absorptiometry; HGS, handgrip strength; SOGLI, Sarcopenic Obesity Global Leadership Initiative. *SOGLI recommendation may also be adapted to use adjusted HGS (relative to weight or BMI).

related to SO. Although muscle strength adjustment (e.g., HGS/BMI) is discussed by the SOGLI group, it is currently not uniformly recommended due to the lack of validated cutoffs, as discussed elsewhere by Prado et al. [6] This is pointed out as a key area for research in SO to properly identify low muscle strength in individuals with obesity. SOGLI also recommends considering muscle quality (functional [muscle-specific strength] and morphological), since it can be profoundly affected in obesity (e.g., myosteatosis). Poor muscle quality in obesity and assessments are discussed elsewhere [86].

Our scoping review employed systematic methodology: (1) search strategy developed and revised by experts in the field and a trained librarian, following the PRESS guidelines, (2) comprehensive database and gray literature searches, (3) two independent reviewers for screening and selection of studies, and data extraction, and (4) reporting of the data following the PRISMA guidelines. However, there are still significant limitations. There is high variability in how SO/sarcopenia is defined in the literature, which could have led to the exclusion of important studies that evaluated body composition and/or physical function in the context of MBS. Most studies included in this review presented a relatively small sample size, and almost half were from a single country, which limits generalizability to other populations. Many of the studies were also at risk of selection bias, as they comprised convenience samples and/or had high dropout rates. Many of the patients studied were young and would not be at the highest risk of SO. Study heterogeneity prevented meta-analysis or pooling of data.

6 | Conclusion and Future Directions

SO prevalence in the context of MBS is highly heterogeneous, with several variations in applied diagnostic criteria and both body composition and physical function assessments. Following the SOGLI recommendations (ESPEN/EASO 2022), it appears that one out of four individuals, both before and post-MBS, have SO, a hidden condition that requires targeted interventions. SO may be associated with poor surgical outcomes, such as decreased weight loss and quality of life, and increased risk for gastric leak, prolonged operation time, and hospital stay. Increased awareness for SO over time postsurgery is recommended, especially during aging. Standardization of SO diagnosis is urgently needed to improve the identification process and enable comparisons among studies, associations with clinical outcomes, and ultimately improve patient outcomes.

KEY FINDINGS (Figure 5)

Metabolic and bariatric surgery and sarcopenic obesity

- SO involves vicious cycling of muscle mass and strength losses with a concurrent gain of excess fat mass
- There is an increased risk for SO in the context of MBS, both before and after surgery
  - 0–24 months: reduction in appetite and overall energy consumption, including protein, leads to rapid and substantial weight loss
  - After ≈24 months: possibility of weight regain, mostly derived from fat, increasing the risk for excess fat mass
- Risk for SO at any perioperative time: intolerance to protein food sources, low adherence to protein supplementation, restrictive dieting, problematic eating behavior, physical inactivity (especially regarding resistance training), associated comorbidities (inflammation), and aging

Body composition assessment

- BIA overestimates muscle mass and underestimates FM in individuals with obesity. Caution when BIA is used to investigate SO in cross-sectional studies; it is likely better suited for longitudinal assessments during MBS follow-up care.
- DXA should be the first choice for body composition assessment in individuals with obesity, when available.
- CT and MRI are gold-standard techniques for body composition assessment when performed for different purposes (e.g., medical diagnosis); however, they are not suitable for routine use in clinical practice.
- Ultrasound is an emerging technique for body composition and SO assessment but requires further investigation.
- Adjustment of muscle mass surrogates should be performed by weight (or BMI): ALST/weight (DXA) or SM/weight (BIA).
- Adjustment by height squared is not suitable for individuals with obesity.

Physical function assessment

- HGS cutoff points based on older adults may not identify low muscle strength in younger individuals with obesity.
- HGS adjustment for body size (weight and/or BMI) is likely needed to identify low muscle strength in individuals with obesity but is not part of the SOGLI recommendation due to lack of evidence and cutoff points; research in this area is urgently needed.
- CST may represent a fair alternative to assess low muscle strength in individuals with obesity. Special considerations are needed for impairments due to obesity or obesity-related conditions (e.g., osteoarthritis).

Sarcopenic obesity diagnosis and prevalence

- Highly heterogeneous in terms of diagnostic criteria, assessment of body composition, and physical function.
  - Pre-MBS: from 0% to 89.3%
  - Post-MBS: from 0% to 57%
- Neither body composition nor physical function alone should be used to diagnose SO.
- SOGLI recommendation (ESPEN/EASO 2022) appears to be the most appropriate diagnostic criteria; however, there is a lack of studies associating it with clinical outcomes.
- Following SOGLI recommendation (ESPEN/EASO 2022), one out of four individuals may have SO both pre- and post-MBS.
- SO risk and prevalence may increase over time post-MBS, especially with aging.

Metabolic and bariatric surgery type

- RYGB and SG may present similar SO risk and prevalence.

Clinical outcomes

- SO may be associated with decreased total weight loss and quality of life, and increased risk for gastric leak, prolonged operation time, and hospital stay post-surgery.

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Conflicts of Interest

The authors declare no conflicts of interest.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.