DISCUSSION PAPERS, LECTURES, NEW TRENDS IN MEDICAL SCIENCE

THE EFFECT OF EXERCISE AND NUTRITIONAL SUPPORT ON ELDERLY AND SENILE PATIENTS WITH SARCOPENIC OBESITY

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ABSTRACT

The aim of the review. *To analyze the prevalence of sarcopenic obesity among elderly and senile people, to assess its causes, and to present modern methods for its prevention and physical rehabilitation.*
This review article discusses the most recent evidence on age-related changes in fat and muscle tissue, and on calorie restriction and exercise that have positive effect on physical performance in older people with sarcopenic obesity. In addition, potential gaps in clinical practice guidelines that merit attention in future research are identified and analyzed.
Search strategy. *We used the following key words to define participation in the review: "sarcopenic obesity", "sarcopenia with obesity", "sarcopenia", "elderly/old age".*
Inclusion and exclusion criteria. *The review included original research results (reviews, meta-analyses). Editorials, proceeding of the conferences, and research protocols were excluded. The study sample included women and men of any race aged ≥ 60 years with a diagnosis of sarcopenic obesity and with preserved locomotion function. Articles involving hospital patients were also excluded. Non-human studies and studies that did not report precise intervention criteria (e. g., nutrition, exercise, duration, etc.) were excluded.*
The literature search was conducted in four electronic databases: PubMed, Cochrane Library, Springer, Scopus, for the period from 2013 to August 1, 2023. There were no restrictions on the language of the publication.

Key words*: sarcopenia, physical activity, aging, obesity, muscle strength, lean body mass, old age, morbidity*

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ВЛИЯНИЕ ФИЗИЧЕСКИХ УПРАЖНЕНИЙ И НУТРИТИВНОЙ ПОДДЕРЖКИ НА ПАЦИЕНТОВ ПОЖИЛОГО И СТАРЧЕСКОГО ВОЗРАСТА С САРКОПЕНИЧЕСКИМ ОЖИРЕНИЕМ

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РЕЗЮМЕ

Цель обзора. *Проанализировать распространённость саркопенического ожирения среди людей пожилого и старческого возраста, оценить причины его возникновения, представить современные методы профилактики и физической реабилитации.*
В данной обзорной статье обсуждаются самые последние данные о возрастных изменениях в состоянии жировой и мышечной ткани, а также об ограничении калорий и комплексах физических упражнений, которые оказывают положительное влияние на физические показатели пожилых людей с саркопеническим ожирением. Кроме того, выявлены и проанализированы потенциальные пробелы в рекомендациях по клинической практике, которые заслуживают внимания в будущих исследованиях.
Стратегия поиска. *Ключевые слова, используемые для определения условий участия в обзоре: «саркопеническое ожирение», «саркопения с ожирением», «саркопения», «пожилой/преклонный возраст».*
Критерии включения и исключения. *В обзор включались оригинальные результаты исследований (обзоры, метаанализы). Редакционные статьи, тезисы конференций, протоколы исследований были исключены. Выборка для исследования включала женщин и мужчин любой расы в возрасте ≥ 60 лет с диагнозом «саркопеническое ожирение» и сохранённой функцией локомоции. Также исключены статьи с участием госпитализированных пациентов. Исследования, не связанные с людьми, и исследования, в которых не сообщалось точных критериев вмешательства (например, питание, упражнения, продолжительность и т. д.), были исключены.*
Поиск литературы был проведён в четырёх электронных базах данных: PubMed, Cochrane Library, Springer, Scopus, – за период с 2013 г. по 1 августа 2023 г. Ограничений на языковой уклон публикации введено не было.

Ключевые слова: *саркопения, физическая активность, старение, ожирение, мышечная сила, мышечная масса тела, пожилой возраст, заболеваемость*

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INTRODUCTION

The number of elderly and senile people is growing worldwide. In 2017, the number of elderly people accounted for 13 % of the world population and is expected to increase to 20 % of the population by 2030, and to reach 2.1 billion by 2050 [1]. Most chronic diseases worsen with age, which is associated with profound changes in body composition, i.e., an increase and redistribution of fat mass and a loss of muscle and bone mass [2].

Among the most common conditions is obesity, which is a complex, multifactorial and relapsing disease that has become a pandemic worldwide [3]. In several countries, the prevalence of obesity reaches 30–40 % of the population, and a further increase in incidence is expected over the next decades [4]. Obesity is characterized by excessive accumulation of white adipose tissue, not only in fat depots, but also ectopically, which significantly reduces physical function [2, 3]. Thus, it is not surprising that obesity is associated with more than 200 medical complications and an increased risk of morbidity and mortality, being the fifth leading cause of death worldwide [3, 4]. Decreased muscle mass and strength, known as sarcopenia, is very common among older adults with obesity (sarcopenic obesity) and is closely associated with frailty, which is a state of impaired homeostatic reserve and stress tolerance that leads to increased vulnerability to adverse health outcomes [5]. Thus, sarcopenic obesity is closely associated not only with cardiometabolic dysfunctions, but also with physical disability [2].

Although we clearly see a significant increase in overall life expectancy, chronic diseases associated with aging, which are exacerbated by obesity, seriously impair quality of life during these "gained years." Therefore, there is increasing effort to identify effective strategies that can curb the obesity pandemic and support the process of healthy aging. Among them, lifestyle interventions, including dietary and training protocols, have been widely studied [6]. In this context, it is important to highlight that diet-induced weight loss involves not only a loss of fat, but also of muscle and bone mass and may further exacerbate age-related sarcopenia and frailty in older adults. Successful approaches that can induce fat loss while preserving muscle and bone mass are critical to reducing the cardiometabolic risks associated with aging and obesity, while preventing or mitigating frailty [2, 7, 8].

Obesity is a chronic metabolic disorder characterized by increased body fat stores, which consequently increases the risk of metabolic diseases, cardiovascular diseases, and mortality. As with sarcopenia, there is currently no consensus on appropriate cutoff values for obesity. The World Health Organization (WHO) uses body mass index (BMI) to define obesity (≥ 30 kg/m2) and overweight (25–29.9 kg/m2) [3]. The American Association of Clinical Endocrinology [9] recommends using cutoff values for body fat percentage to diagnose obesity (> 25% in men and > 35% in women). The amount of abdominal fat is easily estimated using waist circumference (WC), which is highly correlated with intra-abdominal fat content. WHO has also used WC cutoff values (≥ 102 cm for men, ≥ 88 cm for women) as a surrogate for visceral fat. Lower cutoff values for central obesity are required for different ethnic groups, including Asians (2, 8). The Korean Society for the Study of Obesity defines abdominal obesity as WC ≥ 90 cm and ≥ 85 cm in men and women, respectively, based on the results of an epidemiological study [10].

Sarcopenia is a predominantly geriatric disease with a gradual loss of muscle strength, skeletal muscle mass and muscle function. The term was officially adopted at the 2010 meeting of the European Working Group on Sarcopenia in Older People (EWGSOP), and in September 2016, sarcopenia was included in the International Classification of Diseases of the 10th revision (ICD-10) under the code M62.84. In 2018, the updated consensus EWGSOP-2 was aimed at improving the effectiveness of early detection and treatment of sarcopenia and its risk in clinical practice [11]. The group adopted low muscle strength as the main determinant of sarcopenia, since muscle strength is considered to be better than muscle mass in predicting adverse outcomes [11, 12]. EWGSOP-2 focused on low muscle strength (grip strength) as the main parameter of sarcopenia (hand dynamometry); low muscle quantity and quality were also used to confirm the diagnosis of sarcopenia (dual-energy X-ray absorptiometry (DXA), bioelectrical impedance analysis (BIA), ultrasound, etc.), and the assessment of the severity of sarcopenia relied on physical performance indicators based on a series of physical functioning tests (SPPB, Short Physical Performance Battery) [5, 11].

A related disorder is sarcopenic obesity, a term coined by R.N. Baumgartner and used for a specific phenotype of low muscle mass and high body fat [13].

Sarcopenic obesity (SO) is a multifactorial disease characterized by the simultaneous presence of sarcopenia and obesity. For patients, SO poses a greater health risk than either sarcopenia or obesity alone [2, 13]. Therefore, studying sarcopenic obesity and finding effective treatment are important due to the constant increase in the elderly population.

Sarcopenia and obesity lead to decreased physical performance. The hallmark of sarcopenia is slower gait speed. In addition to a higher risk of falls [14], obese older adults have decreased physical performance, as assessed by self-assessment questionnaires or tests such as the SPPB [13]. Sarcopenic obesity is thought to have a synergistic effect on health deterioration compared to sarcopenia or obesity alone. It causes more health problems than sarcopenia or obesity [15] and is a major cause of metabolic disorders, disability, cardiovascular disease, and mortality [16]. Currently, there is no common definition, making it difficult to establish standardized diagnosis and management. Despite progress in defining sarcopenic obesity, according to the recent

Consensus of the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) [17], the discussion on the treatment of this condition is still open. Currently, there is no common definition, making it difficult to establish standardized diagnosis and management. Despite progress in defining sarcopenic obesity, according to the recent Consensus of the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) [17], the discussion on the treatment of this condition is still open.

Sarcopenic obesity is a global health phenomenon driven by both the rapid increase in the elderly population and the obesity epidemic. Aging-associated increase in visceral fat and decrease in muscle mass are associated with numerous adverse cardiometabolic effects and contribute to poor health outcomes [18]. Several biological pathways lead to age-related sarcopenic obesity. Aging reduces resting metabolic rate and metabolic adaptations, including adaptive thermogenesis, maintaining low muscle mass and increasing body fat [13]. Reduced resting metabolic rate, physical activity, mitochondrial volume, and oxidative capacity with age contribute to age-related declines in muscle mass and strength. Furthermore, age-related changes in body fat distribution include loss of subcutaneous fat and accumulation of visceral fat [19]. Also, age-related changes accompanied by a decrease in bone mineral density can lead to the development of osteosarcopenic obesity, the main criterion of which is the deterioration of bone condition and loss of muscle mass, coupled with the presence of sarcopenia and obesity [10, 20].

Sex-specific hormonal changes are an important factor associated with sarcopenic obesity. In women, declining estrogen levels after menopause lead to increases in body weight and fat mass, as well as shifts in fat deposition from subcutaneous to visceral [13, 18]. In older men, total testosterone levels decline by approximately 1% per year, with lower levels associated with sarcopenia, decreased muscle strength, deterioration in physical performance, and increased risk of falls [21].

PREVALENCE AND MORTALITY RISK

As the population ages, the prevalence of sarcopenic obesity increases, as the prevalence of obesity and sarcopenia also increases, especially among adults aged 65 years and older [2]. This is associated with decreased physical activity and energy expenditure, as well as increased body weight [10, 13].

A study of sarcopenic obesity in South Korea, comprising healthy volunteers aged 40–80 years, found the prevalence of sarcopenic obesity ranging from 0.8% to 22.3% in women and from 1.3% to 15.4% in men [22]. Data from individuals aged 18–90 years from the Dutch Lifelines cohort study showed a global prevalence of sarcopenic obesity of 1.4% and 0.9% in women and men, respectively, with a rise in prevalence at 50 years and the prevalence reaching 16.7% in the 80–89 age group [23]. A meta-analysis of 50 studies including 86,285 individuals reported a global prevalence of sarcopenic obesity in adults aged 60 years to be 11% [24]. A meta-analysis of 50 studies including 86,285 participants found a global prevalence of sarcopenic obesity of 11% in adults aged 60 years [24].

In a study of older adults (n = 4652) aged over 60 years, conducted as part of the Third National Health and Nutrition Examination Survey (NHANES III), the prevalence of sarcopenic obesity was 18.1% in women and 42.9% in men. The study defined sarcopenia using the BIA-derived sex-specific cutoffs [25]. Another study conducted among Koreans (n = 2221) aged over 60 years, using the same SD criteria, found that the prevalence of sarcopenic obesity was 6.1% and 7.3% in men and women, respectively [10]. The rapidly increasing prevalence of obesity suggests a likely corresponding increase in sarcopenic obesity in elderly and senile people.

Multiple studies have assessed the association between sarcopenic obesity and mortality risk. NHANES III also conducted a mortality risk analysis in 4652 individuals aged 60 years and older with a 14-year follow-up, and found a significantly higher risk of all-cause mortality among women with sarcopenic obesity compared to women without obesity or sarcopenia. However, there was no statistically significant difference in mortality risk between men with and without sarcopenic obesity [25]. On the other hand, the British Regional Heart Study examined mortality risk in 4107 men aged 60 to 79 years. During an 11-year follow-up period, men with sarcopenic obesity had the highest mortality risk compared to non-obese and non-sarcopenic subjects [26]. A Swedish study of 809 people assessed mortality risk in sarcopenic obesity. Women aged 75 years with SO had a higher 10-year mortality risk compared to women without sarcopenia or obesity. Among men aged 75 years, a similar association with mortality was observed, although it did not reach statistical significance [27]. A cohort study using the UK Biobank (n = 452,931) showed a significantly increased mortality risk in individuals with sarcopenic obesity compared with control subjects with pre-existing cardiovascular disease [28]. Finally, a meta-analysis of 23 studies including 50,866 individuals found that sarcopenic obesity was significantly associated with a higher mortality risk in older adults. Multiple subgroup analyses showed that this higher mortality risk was significant among adults living alone and hospitalized patients. Furthermore, this indicator was consistent across studies that used different criteria to define obesity and sarcopenia [29].

A long-term study of 2,309 elderly Japanese American men in the Honolulu Heart Program in the United States reported a significantly higher mortality risk in the sarcopenic group (measured using DXA) than in the non-sarcopenic, non-obese group [30].

DIAGNOSIS OF SARCOPENIC OBESITY

Since sarcopenic obesity is a subclinical disease and there is no universal consensus on diagnostic criteria and their implementation in clinical practice, the identification of the disease and diagnosis depend on the sum of its components, specifically obesity and sarcopenia.

Recently (2022), ESPEN and EASO published a joint consensus statement on the definition and diagnostic criteria for sarcopenic obesity [31] (Fig. 1). The proposed diagnostic process is as follows: 1) screening of patients by high BMI or increased waist circumference and surrogate parameters for sarcopenia; 2) diagnosis of patients by testing muscle function followed by body composition analysis; 3) making a diagnosis in case of a positive result for sarcopenic obesity [31]. In practice, a person who screens positive for both conditions should consider undergoing diagnostic testing to confirm sarcopenic obesity, which first evaluates the decline in skeletal muscle function and then measures altered body composition, including increased fat mass and decreased muscle mass. In the future, these diagnostic criteria for sarcopenic obesity will need to be validated to establish universal reference values according to the measurement method and ethnic group.

There is currently no ICD code for sarcopenic obesity, and the working definition/differential diagnosis is constantly evolving. Age is a strong risk factor for the onset and severity of sarcopenic obesity, but the disease is not unique to the elderly [32]. Therefore, diagnostic consideration is given to the manifestation of symptoms, with age being a component of risk management [33].

Current definitions of sarcopenic obesity are based on individual definitions of sarcopenia and obesity. However, these definitions vary significantly, making it difficult to accurately diagnose, conduct epidemiological studies, and develop treatment strategies for the disease.

THE EFFECT OF EXERCISE ON PHYSICAL PERFORMANCE

Exercise can affect hormonal balance, reduce oxidative stress, induce mitochondrial synthesis, alter immunological and motor functions, and improve muscle oxidative capacity [34, 35]. Increased muscle protein synthesis with exercise sensitizes muscle sensitivity to insulin and promotes anabolism [6]. Sarcopenia is associated with reduced muscle protein synthesis, partly due to decreased anabolic stimulation (which can result from a lack of regular exercise). Aerobic training, strength training, and a combination of both increase muscle protein synthesis in older adults despite age-related declines in anabolic signaling [7, 10]. Aerobic activity can improve muscle oxidative capacity by counteracting the negative effect of intracellular lipids and accelerating lipolysis, leading to increased capillary density. Mitochondrial synthesis in myocytes is enhanced to meet the demands of increased capillary density, which in turn leads to increased oxygen extraction and metabolism through the induction of calcium and metabolic signaling pathways [34].

Myocyte apoptosis can be abolished by physical activity, while cell quality control mechanisms including autophagy, mitophagy and mitochondriogenesis contribute to the development of sarcopenic obesity and may be potential targets for therapy [36]. Reduction in cytokine production may lead to improvements in glucose metabolism, insulin sensitivity and muscle protein synthesis, which may slow the progression of sarcopenic obesity [2].

Resistance training increases the number and size of fast-twitch muscle fibers (IIA and IIX), which improve muscle glucose metabolism and muscle protein synthesis [37]. Muscle protein synthesis is also improved through nutrient-stimulated vasodilation and nutrient transport to local muscle myofibrils [7]. Muscle fascicle length and muscle tendon stiffness have been reported to increase after resistance training (leg press and extension) for 14 weeks in a group of men and women over the age of 65 [2]. Resistance training has also been shown to decrease levels of cytokines such as resistin, leptin, and IL-6 (REF142) [38].

TREATMENT OF SARCOPENIC OBESITY

Resistance, aerobic, and combined training programs reduce body fat and improve muscle function in older men and women with sarcopenic obesity [39, 40]. Notably, resistance training alone was more effective in improving muscle function. Resistance training improves physical function while reducing body fat in older women with sarcopenic obesity [40, 41]. Similarly, dietary modification to ensure adequate and/or high protein intake prevents muscle loss and in some cases improves muscle function [42]. Dietary restriction is the gold standard first-line treatment for obesity and to some extent counteracts the detrimental effects of aging on skeletal muscle function in mammals [43]. However, evidence for the safety and effectiveness of dietary restriction in patients with sarcopenic obesity is very limited. In a recent study, a very low calorie diet improved muscle performance in patients with sarcopenic obesity, although at the expense of muscle mass [44]. Patients receiving a very low calorie diet combined with physical training showed similar improvements in muscle function while maintaining muscle mass, indicating a synergistic effect of combination therapy in the treatment of the disease. Micronutrients and minerals such as amino acids, vitamin D, selenium, and magnesium can be added to the diet to correct pre-existing deficiencies or to establish supraphysiological concentrations that elicit a biological response

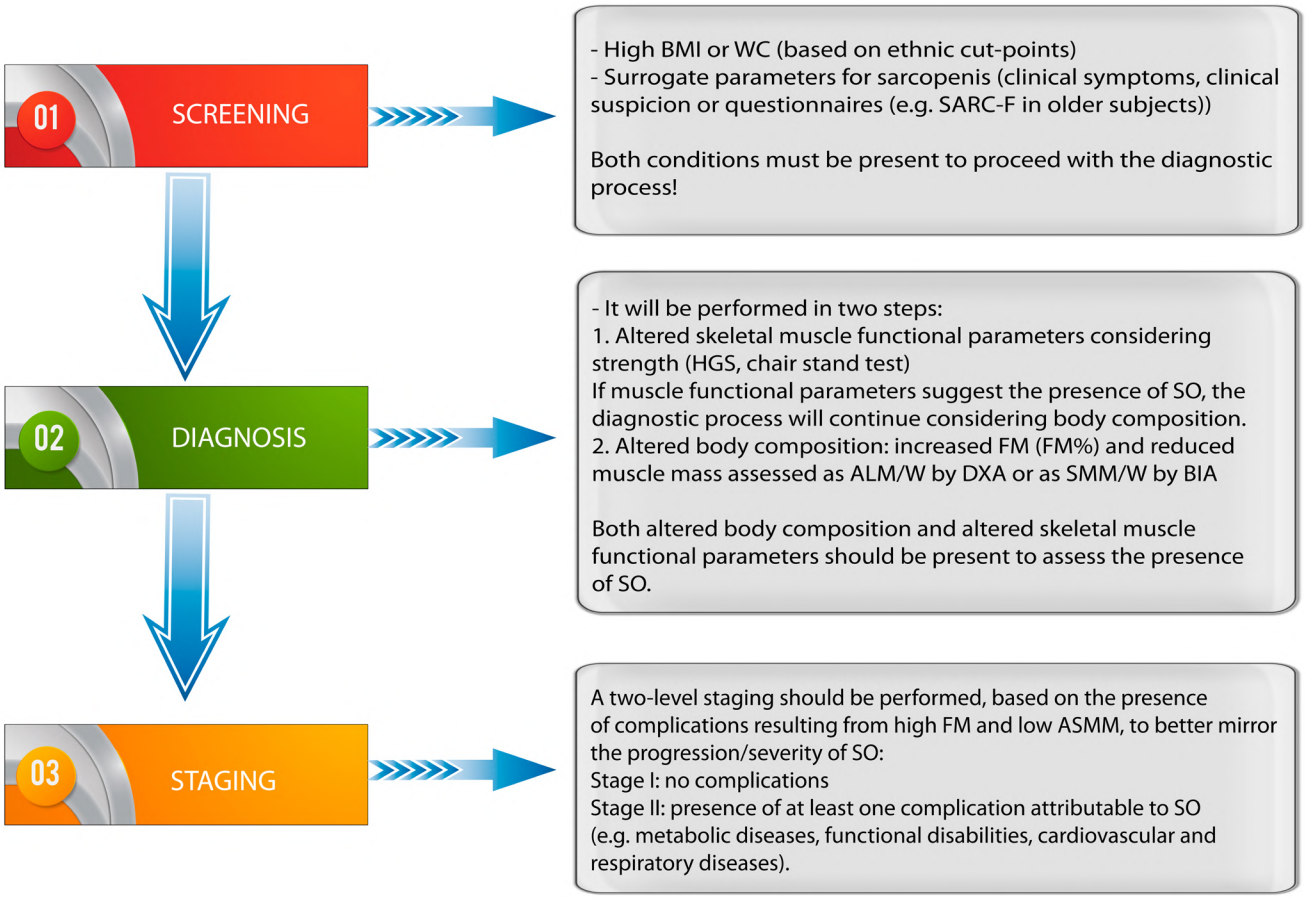

FIG. 1.
Diagnostic procedure for the assessment of sarcopenic obesity SARC-F – Strength, Assistance with walking, Rising from a chair, Climbing stairs and Falls; HGS – handgrip strength; FM – fat mass; ASMM – absolute skeletal muscle mass

[45]. Whey protein supplementation in combination with exercise improves muscle function in adults with sarcopenic obesity [46]. In men, testosterone replacement therapy can be used alone or in combination with diet, exercise, or vitamin supplements to restore androgen balance. Use in elderly and senile patients is somewhat limited due to the high risk of cardiovascular complications [47]. Similarly, in postmenopausal women, estrogen replacement therapy can be used alone or in combination with lifestyle modification with generally positive results in preserving muscle mass [48]. More recently, oxytocin therapy has been used as a trial therapy for sarcopenic obesity to restore the relative decline in oxytocin production with age. Intranasal oxytocin is well tolerated and improves muscle mass in older adults with sarcopenic obesity [49]. However, it remains unclear whether intranasal oxytocin improves muscle function and physical performance. A summary of the prevailing clinical interventions is provided in Table 1. Taken together, treatment options are insufficient and complicated by the competing needs underlying sarcopenia and obesity [33].

The approach to exercise prescription in sarcopenic obesity should be individualized. A program that includes a combination of resistance and aerobic training may be more beneficial than either intervention alone [2, 50]. Aerobic training should be aimed at achieving approximately 65% of peak heart rate, with the goal of increasing to a maximum of 75%. Resistance training should focus on only one or two muscle groups, with the initial 8–12 reps being approximately 65% of the maximum strength a person could develop in one repetition. Progression should be aimed at using 2–3 muscle groups and 75% of maximum intensity [2, 22].

In the sarcopenic obese population, the effects of resistance training on body composition and muscle function are less clear. K.S. Vasconcelos et al. [51] showed that a 10-week resistance training program was not effective in improving physical function in older women with sarcopenic obesity compared to a non-training control group: the SPPB score increased by 0.40 ± 1.3 points ($p > 0.05$), strength increased by 2.36 ± 12.0 N/kg ($p > 0.05$), and power increased by 15.87 ± 13.8 W/kg ($p < 0.05$). The small sample size and short duration of treatment may have

contributed to this result. The study by A.B. Gadelha et al. [52] demonstrated improvements in both strength (12.42 ± 1.5 N/kg; $p < 0.001$) and skeletal muscle mass (0.29 ± 0.4 kg; $p < 0.001$) compared to the control group after 24 weeks of traditional resistance training. Leg press and bench press performance increased by 78% and 70%, respectively. Furthermore, a 12-week study examined the effects of elastic band resistance training in older women with SO and found that skeletal muscle mass (0.73 kg; 95% confidence interval (95% CI): 0.08–1.39; $p < 0.05$) and physical performance (8.58 points; 95% CI: 4.79–12.36; $p < 0.001$) were significantly improved compared to the non-training group [40]. Also, another recent study reported that 8 weeks of resistance training in 60 elderly individuals with sarcopenic obesity resulted in a slight increase in skeletal muscle mass (0.1 kg; $p < 0.05$), a decrease in fat mass (≈ 1.0 kg; $p < 0.05$), and an increase in handgrip strength (3.5 N/kg; $p < 0.05$) compared to a group that did not engage in physical training [38]. W. Kemmler et al. (Germany) conducted a study on men aged ≥ 70 years ($n = 100$) using whole-body electromyostimulation (WBEMS): fat mass loss was 2.1%, handgrip strength increased by 6.3% [53]. A year earlier in Brazil, K. Wittmann et al., using WBEMS, achieved similar results [54]. The advantages of the treatment are its time-effectiveness and accessibility to populations with high levels of frailty. Although electrical myostimulation favorably improves body composition and muscle function compared to noninvasive control [53, 54], the effect sizes are small and may be ineffective in adults with more severe sarcopenic obesity [32, 55].

TABLE 1

CLINICAL TRIALS AIMED AT THE TREATMENT OF SARCOPENIC OBESITY

Sex	Age, number of observations	Study protocol	Duration, frequency	Result	Author, year of publication
Men/women	50-70 years, 24 observations	VLCKD in combination with IT	6 weeks, 30-35 min, 2 days a week	↑ HGS ↓ FM	Camajani E. et al. (2022)
Women	65-80 years, 28 observations	RT	10 weeks, 35 min, 3 days a week	= SPPB = SMM ↑ power	Vasconcelos K.C. et al. (2016)
Women	≥ 60 years, 26 observations	RT with WP	12 weeks, 35-40 min, 3 days a week	↓ FM ↑ LBM	Nabuco H.C.G. et al. (2019)
Women	≥ 70 years, 75 observations	WBEMS and WP + vitamin D	26 weeks, 20 min, 1 session a week	↑ SMM ↓ WT = BP	Wittmann K. et al. (2016)
Men	≥ 70 years, 100 observations	WBEMS and WP	16 weeks, 14-20 min, 2 sessions a week	↑ HGS ↓ FM ↓ BMI	Kemmler W. et al. (2017)
Men/women	65-75 years, 60 observations	RT, AT and CT	8 weeks, 40 min, 3 times a week	↑ SMM ↑ HGS ↓ FM	Chen H.T. et al. (2017)
Women	60-80 years, 56 observations	RT with elastic band	12 weeks	↑ SMM ↑ SPPB	Liao C.D. et al. (2018)
Women	≥ 60 years, 49 observations	RT	16 weeks, 40-45 min, 2 days a week	↑ SPPB ↑ walking speed	de Oliveira Silva A. et al. (2018)
Women	≥ 60 years, 69 observations	RT	24 weeks, 40 min, 3 days a week	↑ LBM ↑ HGS = FM	Gadelha A.B. et al. (2016)

Note. ↑ – increase; ↓ – decrease; = – no changes; VLCKD – very low-calorie ketogenic diet; IT – interval training; HGS – handgrip strength; FM – fat mass; RT – resistance training; SPPB – short physical performance battery; SMM – skeletal muscle mass; WP – whey protein; LBM – lean body mass; WBEMS – whole-body electromyostimulation; BP – blood pressure; WT – waist circumference; BMI – body mass index; AT – aerobic training; CT – combined resistance and aerobic training.

Overall, most of the studies mentioned showed that resistance training is an effective strategy to improve body composition in sarcopenic obesity and that it has the potential to improve physical performance [46, 54, 56].

NUTRITION: CALORIE RESTRICTION AND PROTEIN SUPPLEMENTS

Lifestyle approaches such as calorie restriction and physical training are considered the cornerstone of sarcopenic obesity treatment [15, 24]. With regard to nutrition, the optimal therapeutic approach for sarcopenic obesity remains to be determined due to the limited number of clinical trials conducted in this area [31, 43, 57].

Indeed, weight loss in obese older adults remains controversial as it is a double-edged strategy that has beneficial effects by reducing obesity-related complications and potential negative consequences. Currently, very low calorie intake should be avoided in older adults with sarcopenic obesity as this strategy may compromise overall health and lead to micronutrient and electrolyte deficiencies in the body, which will have a detrimental effect on skeletal muscle mass and reduce bone mineral density [13, 57].

In a pilot study by R. Sammarco et al. (Italy), participants with sarcopenic obesity who underwent a weight loss program supplemented with a high-protein diet showed improvement in muscle strength, and the SF-36 (Short Form 36) questionnaire for assessing the patient's quality of life showed a significant change in general health after 4 months of the study [58].

Although the exact amount of kilocalories (kcal) per day has not yet been determined, it should be less than 750 kcal per day [42]. Generally, high-quality protein intake (1–1.2 g/kg per day), especially those containing sources of leucine, is recommended and can be consumed in conjunction with a calorie-restricted diet [43, 44]. However, caution is needed when consuming high-protein diets due to the risk of renal impairment. Medical and dietary management is important to design a nutritional program that allows for moderate calorie restriction while optimizing protein intake [42, 46].

To minimize the risk of weight loss-induced decrease in bone turnover, generally accepted strategies to minimize the impact of weight loss on bone turnover are needed, including supplemental calcium intake at a dosage of 1200 mg per day and 800–1000 international units (IU) of vitamin D3 per day [59]. Oral calcium should be combined with vitamin D to reduce potential risks associated with over-the-counter supplements [60].

Vitamin D supplementation in patients with sarcopenic obesity has the potential to improve muscle function (25-hydroxyvitamin D3 and 1,25-dihydroxyvitamin D3 have differential effects on human skeletal muscle function and gene expression) and reduce proximal muscle weakness via the action of vitamin D metabolites [61]. Vitamin D deficiency is associated with an increased risk of falls and fractures, as well as decreased muscle mass and strength independent of obesity [62].

CONCLUSION

Physical performance declines with age, and the decline is steeper in sedentary adults with sarcopenic obesity. Physical training, and progressive resistance training in particular, is the most commonly used training method in adults aged 60–80 years [46, 51, 63]. It should be noted that no previous studies have examined differences in exercise prescription by classifying participants in subgroups based on age-level. This is a key aspect to develop in the future.

Sarcopenic obesity is a multifaceted disease with limited treatment options. Systemic energy load due to obesity in the context of aging serves as a pathophysiological cause of the disease, increasing the rate of muscle loss [10, 15, 64]. To this end, reducing energy load and improving muscle function are necessary components of a successful therapeutic intervention.

The lack of a universally used diagnostic method and definition criteria does not allow a clear assessment of the prevalence, which currently remains underestimated [23, 24]. Thus, the diagnosis of sarcopenic obesity is an initial problem.

Overall, the best therapeutic approach to sarcopenic obesity, with the most effective and reliable data to date, is lifestyle modification, including regular combined aerobic and resistance training, with dietary modifications that should include calorie restriction to decrease fat mass and increased protein intake to increase muscle mass and functional capacity to improve quality of life and reduce mortality [22, 52].

Longer term studies assessing the impact of a multimodal approach on sarcopenic obesity and cardiovascular complications in patients will be of great interest.

Early detection of this condition remains important and tailored interventions should be considered to reduce its prevalence and associated adverse outcomes. New therapeutic strategies are required to improve the unfavourable prognosis.

Conflicts of interest

No potential conflict of interest relevant to this article reported.

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