# Sarcopenia and obesity

*Mauro Zamboni*[a,b], *Sofia Rubele*[a,b], *and Andrea P. Rossi*[a,b]

**Purpose of review**
Together with age-related body composition changes, the increased prevalence of obesity observed in the past few decades in older individuals has led to a condition called sarcopenic obesity, characterized by a mismatch between muscle mass and fat mass. The operative definition of sarcopenic obesity is still under discussion and creates difficulties in clinical practice. Muscle weakness, rather than low muscle mass, was previously proposed as an alternative criterion and, more recently, the dynapenic abdominal obese phenotype is of increasing interest because of its unfavorable health consequences and usability in clinical practice.

**Recent findings**
This review focuses on the most recent findings of pathogenic inter-relationships between adipose tissue and muscle. Recent studies on health consequences of sarcopenic obesity and dynapenic abdominal obesity are also examined. Despite the lack of consensus on a definition for sarcopenic obesity, progress has been made in the delineation of the treatment principles for this condition.

**Summary**
Further research is needed to compare different definitions of sarcopenic/dynapenic obesity to clarify the relationship between obesity and the most important adverse outcomes in the elderly. The next step will be the definition of best possible therapeutic approaches for this condition.

**Keywords**
adipose tissue, disability, dynapenia, mortality, muscle strength, myosteatosis, obesity, sarcopenia

## INTRODUCTION

Definition of sarcopenic obesity combines that of sarcopenia, obtained through variable criteria, to the presence of obesity [1].

The first definition of sarcopenic obesity proposed in the early 2000s stressed the quantitative mismatch between muscle and fat mass [i.e. the presence of absolute or relative low muscle mass coupled with BMI greater than 30 or with high total or percentage fat mass as assessed by dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA)] [2]. Alternative definitions were subsequently proposed, using visceral fat area or waist circumference instead of BMI or fat mass, coupled with total or appendicular muscle mass.

Application of these variable criteria led to a wide range of sarcopenic obesity prevalence (ranging from 4 to 12%) [3].

More recently, definitions of sarcopenic obesity have been proposed that consider muscle impairment, as expressed by muscle strength, rather than muscle mass, associated with waist circumference, which has led to the introduction of the concept of dynapenic abdominal obesity (DAO) [4▪].

There are only a few studies that have compared the prevalence of sarcopenic obesity and DAO in the same population: however, a large difference in prevalence of the two conditions was observed (2.8 and 10.6%, respectively) [4▪,5]. In a recent analysis from the Health ABC Study on a sample of 2896 individuals (Rossi AP *et al.*, unpublished data), only a limited number of individuals (1.3% of total sample) belonged to both groups, suggesting that sarcopenic obesity and DAO are two distinct phenotypes, each with a different health risk profile.

Lack of consensus regarding diagnostic tools and criteria as well as thresholds for defining criteria, in particular in the elderly (e.g. BMI cutoff or appendicular fat-free mass), seems to be the most relevant and as of yet unresolved limitations of sarcopenic obesity definitions.

[a]Geriatric Division, Department of Medicine, University of Verona and [b]HealthyAging Center Verona, Azienda Ospedaliera Universitaria Integrata di Verona, Verona, Italy

Correspondence to Mauro Zamboni, MD, Geriatric Division, Department of Medicine, University of Verona, HealthyAging Center, Ospedale Maggiore, Piazzale Stefani 1, 37126 Verona, Italy. Tel: +39 45 8122537; fax: +39 45 8122043; e-mail: mauro.zamboni@univr.it

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## KEY POINTS

- Many definitions of sarcopenic obesity have been proposed, but a clear and decisive definition is still lacking.
- DAO is a more practical and predictive phenotype, and less affected by inconsistencies associated with definitions that take into account total adiposity.
- Links between loss of muscle mass and adipose tissue dysfunction associated with aging suggest common etiologic factors.
- Further research is needed on the best possible treatment for sarcopenic obesity and other related phenotypes.

## PATHOGENESIS OF SARCOPENIC OBESITY

Muscle and fat are strongly connected pathogenically, in that they share common pathways of damage (Fig. 1).

With aging, there is a progressive decline in fat-free mass and thus in total energy expenditure, derived from decreased physical activity, which may lead to weight gain, primarily with an increase in visceral abdominal fat [2].

Both muscle and fat have been recognized as endocrine organs, capable of producing peptides that communicate with cells in autocrine and paracrine ways [6]. Through the secretion of these peptides, called myokines, skeletal muscle may mediate some of the known positive effects of physical exercise: irisin, a peptide produced by myocytes during exercise, may control fat gain by eliciting browning response in white fat, causing its transdifferentiation into brite adipose tissue, thus determining an increase of UCP 1 and then of energy expenditure complementing the independent effect of physical exercise [7▪▪]. Moreover, irisin may also stimulate myocites differentiation and growth, determining the expression of elevated levels of insulin like growth factor 1 (IGF-1) and lowering levels of myostatin, through an extracellular regulated protein kinases-dependent pathway [7▪▪]. Thus, a reduction of physical activity with aging may determine a decline in the production of irisin by the muscle, which leads to an increase in fat mass and thus to sarcopenic obesity.

Adipocytes secrete several peptides, called adipokines: the secretory profile of adipose cells primarily depends both on their size: the greater

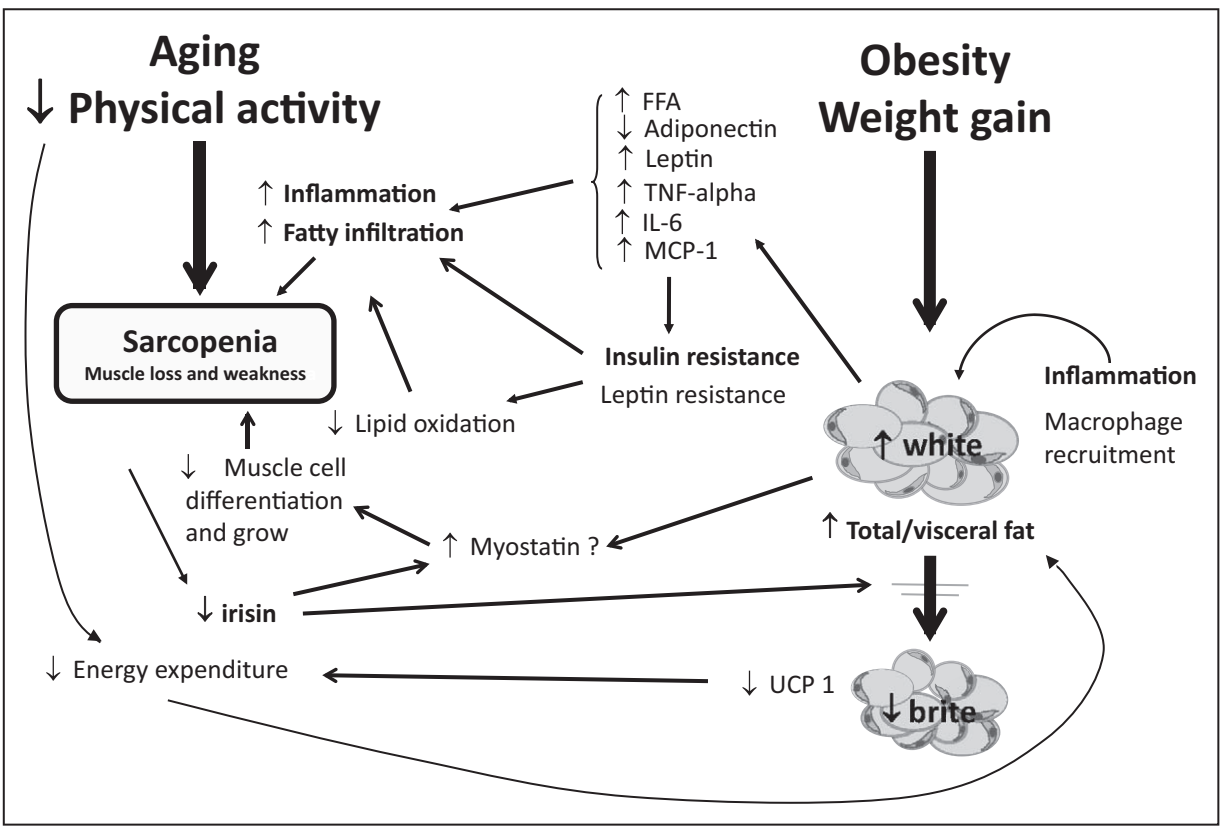


**FIGURE 1.** Cross talk between adipocyte and miocyte in older age: a mechanism leading to sarcopenic obesity. Main steps are given in bold.

the adipose cell size, the higher the profile of proinflammatory adipokines and the lower the anti-inflammatory profile [8▪]. Thus, weight gain may determine an increase in adipose cell size and then an imbalance between proinflammatory and anti-inflammatory adipokines [8▪]. Interestingly, with aging, adipose cells show higher proinflammatory profile as well. Subclinical inflammation derived by both weight gain and aging may be another link between sarcopenia and obesity.

In fact, increased leptin levels may lead to leptin resistance and thus to a reduction of fatty acid (FA) oxidation in muscles, contributing to ectopic fat deposition in organs such as the liver, heart and muscles and, in turn, to a loss of muscle quality in older obese individuals [9]. Decline of adiponectin, a known anti-inflammatory and insulin sensitizer peptide produced by adipocytes, has been shown to be negatively related to muscle mass, because of the lack of its control on NF-kB [10]. As a result of increased inflammatory profile, insulin resistance occurs and promotes muscle catabolism.

Moreover, hypertrophic adipocytes produce excessive free FAs that accumulate into and between muscle fibers ectopically, determining mitochondrial dysfunction, β-oxidation of FA alteration with increased reactive oxygen species production. This phenomenon, coupled with the presence in older individuals of dysfunctional preadipocytes, may determine reduced adipogenesis, compromise the capacity of fat cells to store FA and induce an excess of fat 'overspilled' to nonadipose tissues where they are stored in the form of triacylglycerol [8▪]. However, as the buffer capacity of triacylglycerol soon becomes saturated, excess lipids enter alternative nonoxidative pathways, which results in the production of toxic reactive lipid species, thereby inducing organ-specific toxic responses that lead to apoptosis. Reactive lipids can accumulate in nonadipose tissues of metabolically relevant organs such as skeletal muscle leading to lipotoxicity, a process that contributes substantially to the pathophysiology of insulin resistance and sarcopenia.

Finally, the cross talk between muscle and fat has been further confirmed by the fact that adipocytes and myocytes share the secretion of some chemokines. Increased gene expression and myostatin protein, a well-known inhibitor of myocyte differentiation and proliferation, have been found in obese patients' muscle biopsies and may lead to muscle damage [11].

## HEALTH CONSEQUENCES

Several studies indicate that when obesity and muscle impairment coexist, they act synergistically on the risk of mortality, worsening disability, cardiovascular disease and other unfavorable health conditions.

In the NHANES 1999–2004, the sarcopenic obesity phenotype increased mortality risk in people aged 50–70 years, but not in people aged 70 years and older [12▪]. Similar results were observed in the English Longitudinal Study of Ageing [13▪].

However, studies considering muscle quality measurements, such as handgrip strength, instead of muscle mass, observed a much stronger relationship between sarcopenic obesity and mortality. In fact, Sanada *et al.* [14] showed that all-cause mortality increased in men with sarcopenic obesity defined by waist circumference in a population of 2309 Japanese American men followed up for 24 years.

DAO has been shown to be associated with increased risk of worsening disability [4▪]. The Helsinki Birth Cohort Study has shown that both sarcopenia, defined as lean mass to height ratio, and obesity, evaluated by BMI, waist circumference and percentage body fat, were associated with poorer physical performance [15▪].

Both sarcopenic obesity and DAO have been shown to be related to greater risk of increased falls [16].

Tolea *et al.* [17], in a recent study involving 353 individuals, showed that sarcopenic obesity was associated with higher risk of mental disability and lowest performance on global cognition, compared with individuals with only obesity or sarcopenia, even after adjustment for sociodemographic factors.

Rossi *et al.*, in the InCHIANTI study population, observed that abdominal obesity coupled with low muscle strength strongly increased the risk of hospitalization.

Only a few studies have evaluated the association between sarcopenic obesity, metabolic alterations and cardiovascular diseases and their findings are not conclusive [18,19].

The issue of sarcopenic obesity is today relevant even in clinical oncology: sarcopenic obesity has been shown to be independently associated with higher mortality and a higher rate of complications across multiple cancer types and different treatment plans [20▪▪]. Authors hypothesize that patients with sarcopenia are generally more unfit and vulnerable to stress and large fat mass could inflate drug dose, causing an increased rate of dose-limiting toxicity.

Thus, sarcopenic obesity screening is considered useful for identifying higher risks for complications and worse outcomes in patients undergoing neoadjuvant chemoradiation and resection for rectal cancer [21] and hepatectomy for hepatocellular carcinoma [22▪▪] as well as in acute lymphoblastic leukemia long-term survivors [23▪], and in patients with pancreatic cancer [24▪].

## TREATMENT

As sarcopenic obesity is associated with poor outcomes, its treatment should be taken into consideration. Lifestyle modification, including dietary intervention and physical exercise, is the cornerstone of sarcopenic obesity treatment. The aims of sarcopenic obesity treatment are fat mass loss, together with fat-free mass preservation and physical function improvement.

## DIETARY INTERVENTION

A hypocaloric diet that aims at inducing weight loss should be given carefully to sarcopenic obesity individuals because of its effects on body composition. In fact, weight loss is usually associated, not only with decline in fat mass, but also in fat-free mass, ranging from 20% of total body weight during mild energy restriction to more than 50% during semistarvation [25]. Energy restriction of about 500 kcal/day has proved to be effective in obtaining weight loss, as well as to be safe and able to counteract fat-free mass decline [2,25,26]. A reasonable weight loss goal should not be greater than 5–8% of the initial body weight. Moderate weight loss (nearly 5%) has been found to be effective in reducing muscle fat infiltration and improving muscle function despite a small decrease in appendicular fat-free mass [2].

Quality and quantity of protein intake in sarcopenic obesity patients undergoing to hypocaloric diet should be carefully managed. As older sarcopenic obesity adults have higher protein needs because of the presence of so-called 'anabolic resistance' [27▪▪], the amount of dietary protein included in their hypocaloric treatment should range from 1.0 to 1.2 g proteins/kg of body weight. A recent systematic review and meta-analysis of randomized controlled trials (RCTs) showed that obese or overweight men and women, aged 50 years and older, consuming energy-restricted higher protein rather than normal-protein diets, preserve fat-free mass better, while losing fat mass, which confirms the above-mentioned recommendations [28].

In line with these findings, Sammarco *et al.* [29] showed, in a randomized 4-month clinical trial performed in a sample of sarcopenic obesity women with ages ranging from 41 to 74 years, that a hypocaloric protein enriched diet may improve muscle strength and preserve muscle mass.

The relation between the amount of dietary protein, independent of energy restriction, on muscle mass and quality has been recently evaluated by Wright *et al.* [30], who performed a randomized study in a sample of overweight-obese individuals aged 50–80 years to compare the effects of a 12-week normocaloric high protein diet (1.4 g protein/kg body weight) versus anormo-caloric normal protein diet (0.8 g protein/kg body weight): they observed a small decline in body weight in both groups, with lean mass preservation in the high protein diet group only.

Quality besides amount of dietary protein could be relevant to preserve muscle mass decline during energy restriction. Whey protein has been shown to determine better preservation of postprandial myofibrillar protein synthesis in overweight and obese adults during hypocaloric diet [31▪▪,32▪] and thus should be preferred.

Furthermore, dietary intervention combining proteins, vitamin D and omega-3, may enhance postprandial protein synthesis and muscle mass in both, older healthy and older obese individuals, due to the fact that these supplements have been shown to sensitize skeletal muscle to the anabolic drive of dietary protein [33▪▪].

## EXERCISE

Exercise has multiple positive effects in individuals with sarcopenic obesity. In fact, exercise amplifies anabolic response to endogenous aminoacids, improves insulin sensitivity, provides activation of skeletal muscle satellite cells and increases irisin secretion [7▪▪,34▪▪]. Recently, an 8-week RCT performed on 60 men and women aged 65–75 with sarcopenic obesity compared the effects on body composition and muscle strength of different types of exercise: participants were subdivided in four groups and underwent resistance training, aerobic training, combination training or no training: individuals engaged in the resistance training, aerobic training and combination training interventions had, despite no changes in body weight, a significant increase in muscle mass and muscle strength and greater reduction in total fat mass than those without training, with resistance training showing better results than aerobic training [35▪▪]. Two recent intervention studies, performed in sarcopenic obesity older women, confirmed that skeletal muscle mass and physical performance were significantly improved, in individuals involved in resistance training than in controls without training [36,37▪].

Furthermore, a recent review analyzed eight RCTs that focused on the effects of different types of exercise on sarcopenia and obesity-related parameters in sarcopenic obesity patients: seven out of eight studies reported improvement in at least one sarcopenia-related parameter, whereas only a few studies observed an increase in muscle mass, and an increase in muscle strength was observed

particularly in programmes combining resistance training and aerobic training [38▪▪].

Even though more studies evaluating the effects of exercise in sarcopenic obesity patients are warranted, a combination of different types of exercise, in particular of resistance training and aerobic training, as well as individualized exercise to reduce any risk, seems to be the most practical intervention approach in sarcopenic obesity patients [34▪▪].

A combination of moderate energy restriction diet and exercise is the most effective intervention for both obesity and sarcopenic obesity [25]. Reduction in lean mass observed after weight loss induced by energy restriction may be in fact limited by exercise, with greater muscle strength and physical performance improvement. The combination of energy restriction with both aerobic and resistance exercise seems to be the most effective method to improve physical function and even reduces frailty in older obese individuals [39▪▪].

## OTHER TREATMENTS

Kim *et al.* [40] performed a 3-month RCT in a sample of sarcopenic obese women older than 70 years comparing the effects of four types of treatment (exercise combined with nutritional supplementation, exercise alone, nutritional supplementation alone and just health education) on muscle mass, body fat and muscle strength, observing that combination of exercise and nutritional supplementation improved body fat and strength without additional effects.

Vitamin D supplementation has not been tested in any trials in sarcopenic obesity patients, but it should be taken into account because of its positive effects on muscle function [41]. Thus, vitamin D supplementation could be warranted in all sarcopenic obesity patients undergoing energy restriction.

Whole-body electromyostimulation (WB-EMS) has been recently found to be a technique that is capable of increasing muscle mass and function. It has been recently observed in a study performed in community-dwelling sarcopenic obesity men that WB-EMS is useful, safe and capable of determining a significant increase in skeletal muscle mass, gait speed and handgrip as well as decreasing total fat mass [33▪▪].

## CONCLUSION

With aging, loss of muscle mass and gain in fat seem to be linked with each other and contribute, in the presence of positive energy balance, to the development of sarcopenic obesity.

Identification of elderly individuals with sarcopenic obesity could help in the selection of a group of individuals with a particularly high health risk, and the concept of sarcopenic obesity may help to clarify the relation between obesity, morbidity and mortality in the elderly.

The identification of sarcopenic obesity must be considered not only in the elderly, but also in younger adult obese individuals, without neglecting muscle quality parameters and a body fat distribution evaluation [42▪].

Recently, great effort has been made to improve the definition of sarcopenia in order to use it in the clinical assessment of elderly people. Similar effort seems to be mandatory also for a sarcopenic obesity definition. Clinicians and researchers need to come to a consensus about the definition of sarcopenic obesity or clarify if alternative phenotypes are more predictive of unfavorable health risks.

A definition of DAO may have several advantages compared to sarcopenic obesity definitions. First, it uses two broadly available measurements (handgrip and waist circumference), minimally affected by intercurrent clinical conditions. Second, waist circumference, a good surrogate measure of visceral adipose tissue, is more strictly related to biological changes in fat mass observed in the elderly. In fact, aging determines changes in fat distribution that leads to the increase of inflammatory mediators and cytokine production involved in the physiopathologic interaction between adipose tissue and muscle. Third, visceral adipose tissue is strongly related to intermuscular fat deposition, which is difficult to measure with conventional body composition techniques (i.e. DXA or BIA), but has important consequences on physical performance, insulin resistance and muscle health, determining factors of mysteatosis and myofibrosis.

Future step will be to recognize the best therapeutic approaches for sarcopenic obesity.

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### Conflicts of interest

*There are no conflicts of interest.*

## REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ■ of special interest
- ■■ of outstanding interest


1. Barazzoni R, Bischoff SC, Boirie Y, *et al.* Sarcopenic obesity: time to meet the challenge. Clin Nutr 2018; pii: S0261-5614(18)30169-9. [Epub ahead of print]
2. Zamboni M, Mazzali G, Fantin F, *et al.* Sarcopenic obesity: a new category of obesity in the elderly. Nutr Metab Cardiovasc Dis 2007; 18:388–395.
3. Stenholm S, Harris TB, Rantanen T, *et al.* Sarcopenic obesity: definition, etiology and consequences. Curr Opin Clin Nutr Metab Care 2008; 11:693–700.
4. ■ Rossi AP, Bianchi L, Volpato S, *et al.* Dynapenic abdominal obesity as predictor of worsening disability, hospitalization, and mortality in older adults: results from the InCHIANTI Study. J Gerontol Med Sci 2017; 72:1098–1104.

This study from the InCHIANTI population demonstrates that dynapenia is related to the risk of death regardless of the presence of central obesity, whereas abdominal obesity strongly increases the risk of disability and hospitalization associated with low muscle strength.

5. Rossi AP, Fantin F, Caliari C, *et al.* Dynapenic abdominal obesity as predictor of mortality and disability worsening in older adults: a 10-year prospective study. Clin Nutr 2016; 35:199–204.
6. Kalinkovich A, Livshits G. Sarcopenic obesity or obese sarcopenia: a cross talk between age-associated adipose tissue and skeletal muscle inflammation as a main mechanism of the pathogenesis. Ageing Res Rev 2017; 35:200–221.
7. ■■ Colaianni G, Cinti S, Colucci S, Grano M. Irisin and musculoskeletal health. Ann N Y AcadSci 2017; 1402:5–9.

Review examining the effects of irisin produced during exercise on adipose tissue, muscle and bone mass.

8. ■ Cinti S, Sbraccia P, Finer N editors. The adipose organ. In: Obesity. Cham: Springer; 2018; 1–L 24.

Chapter from the book Obesity, describing the most recent updates on adipose tissue physiology and anatomy.

9. Shimabukuro M. Leptin resistance and lipolysis of white adipose tissue: an implication to ectopic fat disposition and its consequences. J Atheroscler Thromb 2017; 24:1088–1089.
10. Fang H, Judd RL. Adiponectin regulation and function. Compr Physiol 2018; 8:1031–1063.
11. Consitt LA, Clark BC. The vicious cycle of myostatin signaling in sarcopenic obesity: myostatin role in skeletal muscle growth, insulin signaling and implications for clinical trials. J Frailty Aging 2018; 7:21–27.
12. ■ Van Aller C, Lara J, Stephan BCM. Sarcopenic obesity and overall mortality: results from the application of novel models of body composition phenotypes to the National Health and Nutrition Examination Survey 1999–2004. Clin Nutr 2018. pii:S0261-5614(18)30042-6. in press.

Authors aimed to verify, in the wide population of NHANES 1999–2004, if DXA-derived ratio between fat mass and fat free mass predicts survival time better than BMI.

13. ■ Hamer M, O' Donovan G. Sarcopenic obesity, weight loss, and mortality: the English Longitudinal Study of Aging. Am J Clin Nutr 2017; 106:125–129.

Article regarding handgrip strength and weight loss in relation to mortality in an elderly population reporting that weight loss combined with sarcopenia presented the greatest risk of mortality.

14. Sanada K, Chen R, Masaki K. Association of sarcopenic obesity predicted by anthropometric measurements and 24 y-all-cause-mortality in elderly men: the Kuakini Honolulu Heart Program. Nutrition 2018; 46:97–102.
15. ■ Mikkola TM, von Bonsdorff MB, Solonen MK, *et al.* Body composition as a predictor of physical performance in older age: a ten-year follow-up of the Helsinki Birth Cohort Study. Arch Gerontol Geriatr 2018; 77:163–168.

This study assessed how different measures of body composition predict physical performance 10 years later among 1076 men and women aged 57–70 years at baseline.

16. Öztürk ZA, Turkbeyler IH, Soylu G. Health related quality of life and fall risk associated with age related body composition changes: sarcopenia, obesity and sarcopenic obesity. Intern Med J 2018; 48:973–981.
17. Tolea MI, Chrisphonte S, Galvin JE. Sarcopenic obesity and cognitive performance. Clin Interv Aging 2018; 13:1111–1119.
18. Scott D, Cumming R, Naganathan V, *et al.* Association of sarcopenic obesity with the metabolic syndrome and insulin resistance over five years in older men: the Concorde Health and Ageing in men Project. Exp Gerontol 2018; 108:99–105.
19. Fukuda T, Bouchi R, Takeuchi T, *et al.* Sarcopenic obesity assessed using dual energy X-ray absorptiometry (DXA) can predict cardiovascular disease in patients with type 2 diabetes: a retrospective observational study. Cardiovascular Diabetol 2018; 17:55.
20. ■■ Baracos VE, Arribas L. Sarcopenic obesity: hidden muscle wasting and its impact for survival and complications of cancer therapy. Ann Oncol 2018; 29:ii1–ii9.

Review article providing definitions, prevalence and clinical implication of sarcopenic obesity in medical and surgical oncology, discussing possible mechanism, management strategies for sarcopenic obesity patients and potential for reversibility of this condition.

21. Berkel AEM, Klaase JM, de Graaff F, *et al.* Patient's skeletal muscle radiation attenuation and sarcopenic obesity are associated with postoperative morbidity after neoadjuvant chemoradiation and resection for rectal cancer. Dig Surg 2018; 13:1–8.
22. ■■ Kobayashi A, Kaido T, Hamaguchi Y, *et al.* Impact of sarcopenic obesity on outcomes in patients undergoing hepatectomy for hepatocellular carcinoma. Ann Surg 2017. [Epub ahead of print]

Retrospective cohort study evaluating 465 patients who underwent hepatectomy for hepatocellular carcinoma showing that preoperative sarcopenic obesity was an independent factor risk for death and hepatocellular carcinoma recurrence after hepatectomy.

23. ■ Marriot CJC, Beaumont LF, Farncombe TH, *et al.* Body composition in long-term survivors of acute lymphoblastic leukemia diagnosed in childhood and adolescence: a focus on sarcopenic obesity. Cancer 2018; 124: 1225–1231.

This cross-sectional study of body composition involving 75 long-term survivors of acute lymphoblastic leukemia followed for 10 years showed that sarcopenic obesity has a 43% prevalence and is associated with low quality of life in this population.

24. ■ Sandini M, Patino M, Ferrone CR, *et al.* Association between changes in body composition and neoadjuvant treatment for pancreatic cancer. JAMA Surg 2018; 153:809–815.

Retrospective cohort study evaluating body composition changes in 193 individuals with ductal pancreatic cancer undergoing neoadjuvant treatment, showing that increase in muscle tissue was associated with resecability.

25. Zamboni M, Fantin F, Mazzali G. Obesity. In: Michel JP, Beattie L, Martin FB, Walston JD, editors. Oxford textbook of geriatric medicine, 3 ed. Cham: Oxford University Press; 2017. pp. 1–17.
26. Trouwborst I, Verreijen A, Memelink R, *et al.* Exercise and nutrition strategies to counteract sarcopenic obesity. Nutrients 2018; 10; pii:E605.
27. ■■ Morais JA, Jacob KW, Chevalier S. Effects of aging and insulin resistant states on protein anabolic responses in older adults. Exp Gerontol 2018; 108:262–268.

The article describes the development of hyperinsulinemic clamp for the study of glucose and protein metabolism in different nutritional and insulin resistance states in order to studying the contributor to the so-called protein anabolic resistance of aging.

28. Kim JE, O'Connor LE, Sands LP, *et al.* Effects of dietary protein intake on body composition changes after weight loss in older adults: A systematic review and meta-analysis. Nutr Rev 2016; 74:210–224.
29. Sammarco R, Marra M, Di Guglielmo ML, *et al.* Evaluation of hypocaloric diet with protein supplementation in middle-aged sarcopenic obese women: a pilot study. Obes Facts 2017; 10:160–167.
30. Wright CS, Zhou J, Sayer RD, *et al.* Effects of a high-protein diet including whole eggs on muscle composition and indices of cardiometabolic health and systemic inflammation in older adults with overweight or obesity: a randomized controlled trial. Nutrients 2018; 10; pii: E946.
31. ■■ Murphy CH, Shankaran M, Churchward-Venne TA, *et al.* Effects of resistance training and protein intake pattern on myofibrillar protein syntesis and proteome kinetics in older man in energy restriction. J Physiol 2018; 596:2091–2120.

Using deuterated water and muscle biopsies, the researches studied the correlation between resistance exercise and the synthetic rate of muscle tissue-derived proteins in serum (creatine kinase, M-type-type and carbonic anhydrate 3) and muscle sample, observing the stimulatory effects of the exercise on protein synthesis.

32. ■ Devries MC, McGlory C, Bolster DR, *et al.* Protein leucine content is a determinant of shorter- and longer-term muscle protein synthetic responses at rest and following resistance exercise in healthy older women: a randomized, controlled trial. Am J ClinNutr 2018; 107:217–226.

This clinical trial is the first finding that a high-quality mixed protein beverage induces greater increases in acute myofibrillar protein synthesis as compared with isonitrogenous protein beverage with added leucine.

33. ■■ Chanet A, Verlaan S, Salles J, *et al.* Supplementing breakfast with a vitamin D and leucine-enriched whey protein medical nutrition drink enhances postprandial muscle protein synthesis and muscle mass in healthy older men. J Nutr 2017; 147:2262–2271.

Randomized, double-blind, placebo-controlled study, showing that supplementing breakfast with a vitamin D and leucine-enriched whey protein drinks stimulates postprandial muscle protein synthesis and increased muscle mass after 6 weeks of intervention in healthy older adults.

34. ■■ Batsis JA, Villareal DT. Sarcopenic obesity in older adults: aetiology. Epidemiology and treatment strategies. Nat Rev Endocrinol 2018; 14:513–537.

This review outlines current treatment strategies for sarcopenic obesity, with a focus on calorie restriction, protein supplementation and aerobic and resistance exercises.

35. ■■ Chen HT, Chung YC, Chen YJ, *et al.* Effects of different types of exercise on body composition, muscle strength, and IGF-1 in the elderly with sarcopenic obesity. J Am Geriatr Soc 2017; 65:827–832.

RCT involving sarcopenic-obese individuals aged between 65 and 75 showing that resistance training, aerobic training and both increased muscle mass reduced total fat mass and visceral fat mass, increased muscle strength and IGF-1 blood levels compared with the control group without training.

**36.** Chiu SC, Yang ES, Yang EJ, Chang SF. Effects of resistance training on body composition and functional capacity among sarcopenic obese residents in long-term facilities: a preliminary study. BMC Geriatr 2018; 18:21.

**37.** ▪ Liao CD, Tsauo JY, Lin LF, *et al.* Effects of elastic resistance exercise on body composition and physical capacity in older women with sarcopenic obesity: a CONSORT-compliant prospective randomized controlled trial. Medicine (Baltimore) 2017; 96:e7115.

A 12-week intervention study about the effect of elastic band resistance training in sarcopenic obesity older women showing that skeletal muscle mass and physical performance were significantly improved, compared to the no exercise-control group.

**38.** ▪▪ Martínez-Amat A, Aibar-Almazán A, Fábrega-Cuadros R, *et al.* Exercise alone or combined with dietary supplements for sarcopenic obesity in community-dwelling older people: a systematic review of randomized controlled trials. Maturitas 2018; 110:92–103.

The article analyzes 2073 publications from four databases and 72 additional records, for a total of 42 full-text records assessed for eligibility and eight articles finally included, analyzing the effects of resistance training and, in any case, protein supplementation on body composition, muscle strength and physical performance among sarcopenic obesity individuals.

**39.** ▪▪ Villareal DT, Aguirre L, Gurney B, *et al.* Aerobic or resistance exercise, or both, in dieting obese older adults. New Eng J Med 2017; 376:1943–1955.

Clinical trial involving a wide population of 160 obese older adults showing that weight loss combined with both aerobic and resistance exercise is the most effective intervention in improving functional status of obese older adults.

**40.** Kim H, Kim M, Kojima N, *et al.* Exercise and nutritional supplementation on community dwelling elderly Japanese women with sarcopenic obesity: a randomized controlled trial. J Am Med Dir Assoc 2016; 17: 1011–1019.

**41.** Kemmler W, Weissenfels A, Teschler M, *et al.* Whole-body electromyostimulation and protein supplementation favourably affect sarcopenic obesity in community dwelling older men at risk: the randomized controlled FranSO study. Clin Interv Aging 2017; 12:1503–1513.

**42.** ▪ ElGhoch M, Rossi AP, Calugi S, *et al.* Physical performance measures in screening for reduced lean body mass in adult females with obesity. Nutr Metab Cardiovasc Dis 2018; 5. pii: S0939-4753(18)30196-0.

In this study, authors assessed the prevalence of reduced muscle mass in a population of adult women with obesity and evaluated the most accurate functional test for the screening of this condition.