Sarcopenic obesity in the elderly and strategies for weight management

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Sarcopenia is a multifactorial age-related condition associated with a sedentary lifestyle and protein intakes during weight loss that are inadequate to maintain muscle mass. Sarcopenic obesity in the elderly is associated with a loss of independence and metabolic complications and represents a major public health challenge in individuals over the age of 65 years. It is likely that age-related losses of muscle mass and coincident increases in fat mass could be reduced through regular resistance exercise combined with adequate protein intake to maintain muscle mass. It has been established that increased protein intake will maintain muscle mass during calorie-restricted diets to a greater extent than usual protein intake. Other strategies, including the use of high-protein meal replacements or supplementation with specific ergogenic or branched-chain amino acids, may be beneficial.

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INTRODUCTION

Obesity is currently epidemic in the United States, with almost 70% of Americans overweight and one of three obese.1 Obesity is associated with increased morbidity and mortality, and there is unchallenged evidence that obesity increases the risk for the development of hypertension, dyslipidemia, type 2 diabetes mellitus, atherosclerotic heart disease, stroke, osteoarthritis, sleep apnea, cancers of the breast, prostate, and colon, and all-cause mortality.2–4 This review examines the impact of using body composition as an alternative to the body mass index (BMI) to define sarcopenic obesity. The implications for weight-management strategies, especially in elderly persons with reduced lean body mass and increased fat mass, are discussed. Once sarcopenic obesity has been identified, the use of resistance exercise to increase both muscle mass and the resting metabolic rate becomes a goal of weight-management strategies designed to help protect against a variety of age-related chronic diseases associated with obesity.

UTILIZATION OF THE BODY MASS INDEX

The National Heart, Lung and Blood Institute of the National Institutes of Health has issued clinical guidelines that define overweight as a BMI of 25 to 29.9 and obesity as a BMI of 30 or greater.5 The BMI, also known as Quetelet’s Index, is simply the height in meters divided by weight in kilograms squared. A best-fit linear regression of weight to height yields a best fit at height divided by weight to 1.82 power, but this has been rounded to the power of 2. In epidemiological studies, increasing body weights and BMIs have been associated with an increase in all-cause mortality as well as increased mortality for certain cardiac and noncardiac conditions.4,6 Nevertheless, other investigators have described an “obesity paradox” in which geriatric patients have lower mortality rates at higher BMIs.7–9 While an increased BMI is usually correlated with excess fat in populations, individuals with reduced lean body mass and increased fat mass can be subject to increased risks associated with obesity in the absence of an increased BMI. This is particularly evident...
in the elderly, in whom a sedentary lifestyle, a typical age-related decrease in muscle mass, and inadequate protein intake lead to sarcopenic obesity, as discussed in this review.

**Obesity Assessment by Body Composition Versus BMI**

Obesity is strictly defined as the accumulation of excess body fat and not simply excess weight, which can represent either muscle or fat. Since weight and height are easily surveyed in epidemiological studies, the BMI has been practically adopted as a surrogate for obesity in disease risk assessments. However, elderly individuals at normal or even somewhat reduced BMIs can have excess visceral fat as well as visible fat stores.

In sedentary persons, epidemiological studies have shown that from the second to the eighth decade of life, the total lean body mass declines by about 18% in men and by 27% in women. Loss in muscle mass accounts for the age-associated decreases in basal metabolic rate, muscle strength, and activity levels, which in turn are the cause of the decreased energy requirements of the elderly.

The first clues that lean body mass might be important came from correlation studies of BMI and anthropometry versus mortality risk. Zamboni et al. reviewed 20 studies from 1997 to 2004 that had at least 4.5 years of follow-up and in which the relationship between BMI and mortality was examined. Their review of the literature supports the notion that central fat and relative loss of fat-free mass is more important than BMI in determining the mortality risk associated with obesity in the elderly. Wannamethee et al. examined the relationship between different anthropometric indexes of body composition (both muscle mass and body fat) and all-cause mortality in men aged 60–79 years in a prospective study of 4,107 men followed-up for a mean period of 6 years but excluding those with heart failure, which could affect body composition measurements. Increased adiposity measured by BMI, waist circumference (WC), or waist-to-hip ratio (WHR) showed little correlation with mortality after adjustment for lifestyle characteristics. Muscle mass, on the other hand, as estimated by mid-arm muscle circumference, was significantly and inversely associated with mortality. After adjustment for mid-arm muscle circumference, obesity markers, particularly high WC (>102 cm) and WHR (top quartile), were then associated with increased mortality.

Recently, Srikanthan et al. assessed the relationship between three measures of obesity and all-cause mortality in a group of healthy older adults from the MacArthur Successful Aging Study, a longitudinal study of high-functioning men and women aged 70–79 years at baseline. After 12 years, all-cause mortality increased with WHR. There was an association with gender, i.e., there was a graded relationship between WHR and mortality in women (relative hazard 1.28 per 0.1 increase in WHR; 95% confidence interval, 1.05–1.55) and a threshold relationship in men (relative hazard 1.75 for WHR > 1.0 compared with WHR ≤1.0; 95% confidence interval, 1.06–2.91). It was concluded that WHR rather than BMI appears to be the more appropriate yardstick for risk stratification of high-functioning older adults.

Janssen et al. examined the influence of BMI and WC on mortality risk in the Cardiovascular Health Study, a longitudinal study of cardiovascular disease and its risk factors in older people. The risks of all-cause mortality associated with BMI and WC were examined over 9 years of follow-up. In their model, when examined individually, BMI and WC were both negative predictors of mortality, but when BMI and WC were examined simultaneously, after controlling for WC, mortality risk decreased by 21% for every standard deviation increase in BMI. After controlling for BMI, mortality risk increased by 13% for every standard deviation increase in WC. Therefore, BMI was the negative predictor of mortality, whereas WC was a positive predictor of mortality. These data could be interpreted to indicate BMI reflects lean body mass in individuals with similar WC values, whereas WC reflects fat mass in individuals with similar BMIs.

**Measurement and Prevalence of Sarcopenic Obesity in the Elderly**

As suggested by the above studies, advancing age can be associated with remarkable reductions in lean body mass and increases in body fat mass. Decreased lean body mass occurs primarily as a result of losses in skeletal muscle mass. This involuntary loss of skeletal muscles that occurs with advanced age is defined as sarcopenia. Sarcopenic obesity was first defined in 1996 by Heber et al. as reduced lean mass, as determined by bioelectrical impedance analysis (BIA), with excess fat as a percentage of body weight.

Estimation of skeletal muscle using a BIA equation was validated using magnetic resonance imaging in volunteers. The skeletal muscle mass can be estimated from BIA measurements and expressed as skeletal muscle mass index (SMI = skeletal muscle mass/body mass × 100). Subjects with an SMI greater than 1 standard deviation above the sex-specific mean for young adults (18–39 years) are considered normal. Class I sarcopenia occurs at SMIs that are 1 to 2 standard deviations below normal for young adult values, and class II sarcopenia is present when the SMI is 2 standard deviations below young adult values.

Baumgartner et al. used dual x-ray absorptiometry (DEXA) to estimate skeletal muscle mass and developed
their own sex-specific cutoff values for sarcopenia based on the statistical distribution of "relative skeletal muscle mass," which was defined as appendicular skeletal muscle mass (ASM) (sum of the masses of arm and leg lean soft tissues from DEXA) divided by height squared (ASM/H2). The cutoff values for the ASM/H2 index were defined as 2 standard deviations below the sex-specific means of the distributions in a reference sample of young and middle-aged adults from the Rosetta Study. The estimated prevalence of sarcopenia in the New Mexico Elder Health Survey increased from 13% to 24% in people younger than 70 years to >50% in people older than 80 years of age and was slightly greater in Hispanics than in non-Hispanic whites. In another study among healthy elderly women (>70 years of age), prevalence rates of sarcopenia, expressed as ASM and ASM/H2, were found to be 40.2% and 12.3%, respectively.

In the Korean Longitudinal Study on Health and Aging, ASM was measured by DEXA. Sarcopenia was assessed by ASM (kg/m2) divided by height squared (ASM/H2), as proposed by Baumgartner et al., and ASM as a percentage of body weight (ASM/WT), as suggested by Janssen et al. Sarcopenia was defined as < 1 standard deviation below the sex-specific mean for a young reference group. The cutoff point for sarcopenia was 7.09 kg/m2 in men and 5.27 kg/m2 in women, as measured using ASM/H2. For ASM/WT, the cutoff was 29.9% in men and 25.1% in women. In this population, obesity was defined as a visceral fat area exceeding 100 cm2 on abdominal computed tomography. The subjects were classified into sarcopenic obese, obese, sarcopenic, and normal groups, according to the definitions described above. The prevalence of sarcopenic obesity was 16.7% in men and 5.7% in women when sarcopenia was defined by ASM/H2. However, it was 35.1% in men and 48.1% in women when defined by ASM/WT.

Janssen et al. estimated optimal cutoff values for predicting disability in a representative American sample (National Health and Nutrition Examination Survey III) using total skeletal muscle mass (TSM, from BIA) adjusted for stature (TSM/H2). Cutoff values in the Janssen study for women ranged from 5.76 kg/m2 to 6.75 kg/m2 and were associated with moderate levels of disability. Less than 5.75 kg/m2 was associated with a high risk of physical disability. In men, the cutoff values ranged from 8.51 kg/m2 to 10.75 kg/m2. Interestingly, if these cutoff values are adjusted to approximate ones based on ASM rather than total muscle mass, they are similar to those originally derived by Baumgartner et al. Participants with ASM/H2 values of 2 standard deviations or more below the young adult mean were also found to have lower grip strength and greater limitation in climbing stairs and general activities of daily living after adjusting for confounding factors in a 4-year prospective study.

When sarcopenia was defined as DEXA-verified muscle mass more than 2 standard deviations below the sex-specific young-normal mean, the age- and sex-adjusted prevalence of sarcopenia varied between 6% and 15% among subjects 65 years of age or over, depending on the muscle mass parameter that was evaluated. Therefore, both BIA and DEXA estimate the prevalence of sarcopenia and are superior to BMI in identifying abnormal muscle function in the elderly. It has been suggested that the "best" measure should be based on muscle strength rather than on muscle mass, particularly in the context of cardiovascular disease risk. To date, no consensus has been reached as to the "best" definition of sarcopenic obesity.

**Sarcopenic obesity, metabolic syndrome, and chronic disease**

Increased body fat and increased abdominal obesity are factors in the increasing incidence of non-insulin-dependent diabetes mellitus among the elderly. In the Korean Sarcopenic Obesity Study, the prevalence of sarcopenic obesity in patients with diabetes was 15.7% versus 6.9% in the control group. In another study in this population, triglyceride levels in men in the sarcopenic obesity group defined by ASM/WT were significantly higher than those in other groups. The mean fasting glucose concentration of the sarcopenic obesity group seems to be higher than that of other groups, although the difference was not statistically significant. When the odds ratios were calculated from logistic regression models predicting metabolic syndrome controlled for age, sex, smoking status, alcohol consumption, and exercise habits, the sarcopenic obesity group had an 8.2 times (95% CI 4.45–15.40) and the obese group a 5.5 times (95% CI 2.81–10.80) higher risk of metabolic syndrome than the normal group. In contrast, using ASM/H2, the odds ratio for metabolic syndrome was 2.90 (95% CI 1.28–6.57) in the obese group and 4.80 (95% CI 2.63–8.75) in the sarcopenic obesity group.

Baumgartner observed that the odds ratio for three or more physical disabilities in sarcopenic obese subjects was 8.72 in men and 11.98 in women. Similarly, the sex-adjusted odds ratio for three or more physical disabilities was 4.12. When ASM/H2 was 2 or more standard deviations below the young-adult mean, Chinese women had lower grip strength and greater limitation in climbing stairs and in general activities of daily living after adjusting for confounding factors.

In a longitudinal study with 9 years of follow-up, cardiovascular disease risk was not significantly increased in the sarcopenic or the obese groups when determined by WC and muscle strength but was increased by 23% within the sarcopenic obese group.
In a study of Japanese men and women aged 18–40 years, classes 1 and 2 sarcopenia were defined using ASM/H2. In that study, the brachial-ankle pulse wave velocity was significantly higher in women with class 1 or class 2 sarcopenia than in normal subjects.31 In a group of men aged 60–79 years, muscle mass was significantly and inversely associated with mortality. After adjustment for mid-arm muscle circumference, high WC and WHR values were associated with increased mortality, indicating that sarcopenic obesity is related to mortality in older men.12

Weight management in sarcopenic obesity

There has been ongoing debate as to whether recommending weight loss in the elderly with obesity is beneficial or harmful.32,33 Several prospective studies of weight loss in older subjects demonstrated increased mortality compared with elderly subjects at stable weight.34 The cause of observed weight losses in the elderly is complex. When reviewing such data, it is important to differentiate intentional weight loss from unintentional weight loss because the latter may reflect weight loss as the result of an illness, representing an increased risk of mortality.32–35 Wannamethee et al.36 further demonstrated that differentiating between intentional and unintentional weight loss without taking into account the underlying reason for intentional weight loss may lead to biased results as well. Weight reduction in the elderly can have clinically important benefits with regard to osteoarthritis, physical function, type 2 diabetes mellitus, and coronary heart disease.37 Therefore, the observation that fat loss was associated with reduced mortality is not controversial and can be the goal of a weight management strategy that also seeks to retain lean body mass as much as possible through increased protein intake and resistance exercise.38

Protein intake. The current mean dietary protein requirement for healthy adult men and women of all ages is estimated to be 0.6 g protein/kg/day, with a suggested safe level of intake set at 0.8 g protein/kg/day.39 These protein requirements are based on research performed in 1973, and allowance estimates were established using a standard method of nitrogen balance in young men. The 0.6 g protein/kg/day recommendation fulfills the criteria as the minimum daily average dietary intake level that meets the nutritional requirements of nearly all healthy individuals, but it does not promote optimal health or protect the elderly from muscle loss leading to sarcopenia.39 It is not appropriate to extrapolate protein requirements determined in young healthy volunteers to the appropriate protein intake recommendations for weight management in the elderly, especially those with sarcopenia.

Evans40 has shown that healthy, independently living elderly men and women demonstrated an accommodation response to the Recommended Dietary Allowance for protein of 0.8 g/kg/day with decreased urinary nitrogen losses and reduced skeletal muscle mass. Increased dietary protein intake (up to 1.6 g protein/kg/day) may also enhance the hypertrophic response of muscles to resistance exercise, providing a valuable strategy for weight loss and subsequent weight maintenance. In a recent review, Paddon-Jones and Rasmussen41 have recommended 25–30 g of high-quality protein per meal to maximize muscle protein synthesis.

There is significant evidence that high-protein diets have significant advantages over standard-protein diets in weight management through mechanisms that include increased satiety, increased thermogenesis, and better maintenance of lean body mass, which maintains resting metabolic rate during weight reduction. A number of studies have suggested that protein is the most satiating macronutrient and promotes the retention of lean body mass.42 A diet with increased protein-to-carbohydrate ratios where protein intake is increased from 12% to 45% of total energy has been demonstrated to increase satiety and decrease food intake43 in comparison with meals providing standard amounts of protein intake.

Protein has specific effects on satiety hormones, including PYY 3-36.44 In one study, the high-protein diet caused the greatest reduction in hunger and resulted in the greatest increment in both total plasma PYY and integrated PYY levels in normal and obese subjects.44 When protein replaces 15% of carbohydrate within a low-fat diet (45% protein, 35% carbohydrate, and 20% fat), reduced insulinemic and glycemic responses have been observed, resulting in increased fat oxidation.44 A shift in muscle substrate utilization from carbohydrate to fat oxidation may have a positive effect on weight control and may be useful as a strategy for improving insulin sensitivity.

Increased protein intake results in both improved weight loss and improved maintenance of weight loss.42,44–46 This is due to the fact that a higher-protein diet induces more fat loss42,45,46 and results in retention of more lean tissue.41,45–47

Strategies for protein supplementation. A postprandial rise in amino acids and insulin after meals independently stimulates protein synthesis in skeletal muscle.48,49 In the elderly, the consumption of a beverage containing 20 g of protein after aerobic exercise increases whole body protein turnover to a greater extent than a carbohydrate beverage.50 An enhanced muscle protein anabolic response was detected when essential amino acids and carbohydrate were ingested after resistance exercise, primarily due to an increase in muscle protein synthesis, with minor changes in muscle protein breakdown.
observed regardless of carbohydrate dose or circulating insulin level.51,52

Leucine is an important mediator of the response to amino acids. It increases muscle protein synthesis by modulating the activation of mammalian target of rapamycin complex 1 (mTORC1) and signaling components of translation initiation.53 Leucine increases the phosphorylation of mTOR, 70-kDa ribosomal protein S6 kinase-1 (S6K1), eukaryotic initiation factor (eIF) 4E-binding protein-1 (4EBP1), and eIF4G; decreases eIF2α phosphorylation; and increases the association of eIF4E with eIF4G.54 The acute leucine-induced stimulation of muscle protein synthesis is not maintained for prolonged periods, despite continued activation of mTOR signaling, because circulating amino acids fall as they are utilized for protein synthesis. However, when circulating amino acid levels are maintained, the leucine-induced stimulation of muscle protein synthesis is maintained for prolonged periods. Thus, adequate protein intake, especially leucine-enriched balanced amino acids, may enhance muscle protein synthesis and preserve muscle mass and strength (Figure 1).

While both protein-enriched meal replacements and low-fat meats such as poultry and fish can provide a practical means of reaching dietary protein goals in the elderly, meal-replacement shakes as a supplement have the advantage of simplicity and convenience.55 It also makes it practical to supplement adequate amounts of essential amino acids50,56 to preserve lean body mass. Protein-enriched meal replacements also simplify weight-loss regimens by organizing the diet. This strategy consists of replacing one or two meals a day with a product of defined nutrient and calorie content. Meal replacement leads to increased weight losses over 12 weeks compared with simply restricting calories from favorite foods,57,58 and weight losses have been maintained for up to 5 years using meal replacement.59

Resistance training. Resistance training has been shown to be the most effective intervention for reversing sarcopenia in the elderly. Muscle strength and mass progressively decrease as people get older. The maintenance of muscle strength and the prevention of sarcopenia are extremely important for older adults to be able to successfully perform physical tasks, including activities of daily living such as walking, showering, and caring for one’s personal needs.

The effects of strength training on maximal aerobic power and some of its determinants were studied in older men (60–72 years) by Frontera et al. 60,61 The exercise program consisted of 12 weeks of strength conditioning of extensors and flexors of each knee with eight repetitions per set, three sets per session, and three sessions per week at 80% of the one repetition maximum (1 RM). At the completion of the program, extensor strength had more than doubled, and flexor strength had more than tripled. The increase in strength averaged approximately 5% per training session, similar to strength gains observed in younger men. Mid-thigh composition from computerized tomographic scans showed an increase in total thigh area (4.8%), total muscle area (11.4%), and quadriceps area (9.3%). Biopsies of the vastus lateralis muscle revealed similar increases in type I fiber area (33.5%) and type II fiber area (27.6%). The same training program was applied to a group of frail, institutionalized elderly men and women. After 8 weeks of training, muscle strength was increased by almost 180% and muscle size by 11%. The program led to significant gains in muscle strength, size, and functional mobility among frail nursing homes residents up to 96 years of age.62,63

It is vital, however, to establish whether performing regular physical exercise will build or maintain muscle strength and mass in the elderly and to determine which types of exercises are the most effective at preserving muscle function. Klitgaard et al. 64 have examined cross-sectional differences in muscle function and morphology in elderly men (average age, 69 years) who have trained as swimmers, runners, or strength-trainers continuously for 12–17 years. These men were also compared with sedentary young (average age, 28 years) and age-matched controls. The strength-trained elderly men were the only group with muscle strength and size similar to that of the young men. In contrast, the muscle strength and size measurements of the elderly runners and swimmers were generally lower than those in the young group and similar to those in the untrained elderly controls.

The associations between age, training status, muscle function, and morphology can be understood by examining muscle function and individual contractile proteins in young and elderly individuals. Strength training increases the content of specific myosin heavy chain (MHC) proteins in the vastus lateralis muscle in strength-trained elderly in comparison with sedentary elderly.
Specifically, MHC types Ha and Ib are increased, while MHC type I is decreased in comparison with that in sedentary controls or in elderly subjects who participated in solely aerobic activities such as swimming or running.

There are also differences observed in slow versus fast myosin light chains and the ratio of beta- to alpha-tropomyosin isoforms. These changes in muscle protein content reflect the selective atrophy of type II muscle fibers in the absence of strength training and highlight the fact that habitual strength training but not swimming or running may prevent these isoform changes and preserve muscle mass in the elderly. Further research is needed to determine whether strength training in early adulthood will result in maintenance of skeletal muscle mass with aging.

Muscle can change its composition by atrophy of muscle tissue and an increase in the formation of fat cells. Satellite cells in muscle have the ability to form fat cells. Stem cells in muscle form adipocytes rather than myocytes when there is muscle damage due to inflammation, oxidative stress, proteolytic degradation, and nuclear apoptosis. In animal models, exercise-induced muscle injury is used to initiate the events leading to muscle fiber necrosis and sarcopenia. With inactivity, some of these changes may occur in humans as well, leading to sarcopenia, but more research is needed to define the steps in muscle degradation and adipogenesis that cause these changes in aging or inactive human muscle. Human muscle hypertrophy by the fusion of satellite cells rather than, as in other tissues, through cell division. The role of satellite cell function in the pathogenesis of sarcopenia and sarcopenic obesity remains to be established. Nevertheless, the effects of resistance exercise and adequate protein nutrition are likely mediated through satellite cell function and changes in muscle protein metabolism and may play a role in countering age-related and inactivity-related sarcopenic obesity.

**CONCLUSION**

Sarcopenia is a multifactorial age-related condition associated with a sedentary lifestyle and protein intakes during weight loss that are inadequate to maintain muscle mass. Sarcopenic obesity in the elderly is associated with a loss of independence and metabolic complications and represents a major public health challenge in individuals over the age of 65 years. It is likely that age-related loss of muscle mass and the coincident increases in fat mass could be reduced through regular resistance exercise combined with adequate protein intake to maintain muscle mass. It has been established that increased protein intake will maintain muscle mass during calorie-restricted diets to a greater extent than usual protein intakes. However, more research is needed to demonstrate the long-term retention of muscle mass with aging through a strategy of increasing protein intakes above minimum levels but within the upper part of the broad range of 10–35% of caloric intake recommended by the Food and Nutrition Board of the Institute of Medicine (Table 1). Other strategies, including the use of high-protein meal replacements or specific ergogenic or branched-chain amino acid supplements to reach specific goals such as the intake of 6 g of essential amino acids per meal or a total daily protein intake of 25–30% of total calorie intake, may be beneficial and deserve further study to determine whether increased protein intake will improve muscle anabolic responses to resistance exercise in the elderly.

**Acknowledgments**

Declaration of interest. The authors have no relevant interests to declare.

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