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# Health-related implications and management of sarcopenia

This age-related deterioration in functionality is, to some extent, preventable. Furthermore, encouraging patients to adopt healthy eating and exercise habits can even reverse some of the effects.

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**S**arcopenia is defined as the age-related loss of skeletal muscle mass, strength, and function. The condition is both a process and an outcome. Beginning in the fourth decade of life, adults lose 3% to 5% of muscle mass per decade,<sup>1</sup> a rate of decline that increases to 1% to 2% per year after age 50 years.<sup>2</sup> Muscular strength is independently associated with functionality,<sup>3-5</sup> while loss of skeletal muscle mass and strength is associated with declining health.<sup>3</sup> The loss of muscle mass and function can be both a fundamental cause of and a contributor to disability and disease progression.

Given its ubiquitous nature and deleterious consequences, sarcopenia is believed to have a far-reaching and costly impact on health care in America. Much of

this cost could be averted, however, as sarcopenia is thought to be preventable and, to some extent, reversible. Unfortunately, most clinicians are not familiar with sarcopenia. This article presents a thorough review of the condition, discusses current treatment recommendations, and provides concrete action plans for implementing them.

Sarcopenia should be differentiated from other disorders associated with skeletal muscle loss, including cachexia and wasting. The term *wasting* was originally used to describe the rapid, unintentional loss of both muscle and adipose tissue in persons with HIV disease. Wasting results primarily from anorexia and inadequate dietary intake, with subsequent negative protein and calorie balance. *Cachexia* is defined as the accelerated loss of muscle mass seen with chronic inflammation caused by acute disease. It is a slower process than wasting, and its pathogenesis includes metabolic alterations resulting from a disease-mediated increase in cytokine production.<sup>6</sup> Sarcopenia has an even more insidious course than cachexia. There are numerous underlying mechanisms behind sarcopenia, which collectively result in decreased anabolism leading to net muscle loss over time. Unlike cachexia and wasting, sarcopenia occurs in persons who are otherwise free of disease.

The progressive decline in muscle mass and strength characteristic of sarcopenia impairs functionality and eventually leads to significant morbidity, suggesting a



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**Learning objectives**

- Review the pathophysiologic processes that occur with sarcopenia
- Describe the mortality and morbidity associated with sarcopenia
- Delineate treatment recommendations and how to implement these recommendations in practice

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pathologic state. The disorder is analogous to osteoporosis. In that case, a disease state is defined when bone mass reaches a “zone of fracture risk” (2.5 standard deviations below the young normal mean), with fracture being the primary adverse outcome. Sarcopenia could be considered a disease when the loss of muscle mass and strength reaches the level of functional impairment or disability. Unlike for osteoporosis, however, there is no agreed-upon chief adverse outcome for sarcopenia.

In their work involving the New Mexico Elder Health Study, Baumgartner and colleagues defined sarcopenia as “lean body mass more than two standard deviations below the young normal mean.”<sup>7</sup> This definition has been used in subsequent studies. Some researchers conclude that muscle power is a better predictor of physical function, suggesting that some measure of strength or power should be included with measurements of muscle mass when defining sarcopenia.<sup>8,9</sup> More recent efforts focus on determining a skeletal muscle “cutpoint” that correlates with physical disability risk in the elderly.

Epidemiologic studies estimate the prevalence of sarcopenia to be as high as 50% in people 80 years and older.<sup>7,10,11</sup> In 2000, health care costs attributed to the disorder were estimated to be \$18.5 billion.<sup>12</sup> This figure represents only the direct costs of sarcopenia, including hospital, outpatient, and home care expenditures. As a means of comparison, the annual economic costs of osteoporotic fractures (adjusted to 2000 dollars) were estimated to be \$16.3 billion.<sup>12</sup>

## Etiologies

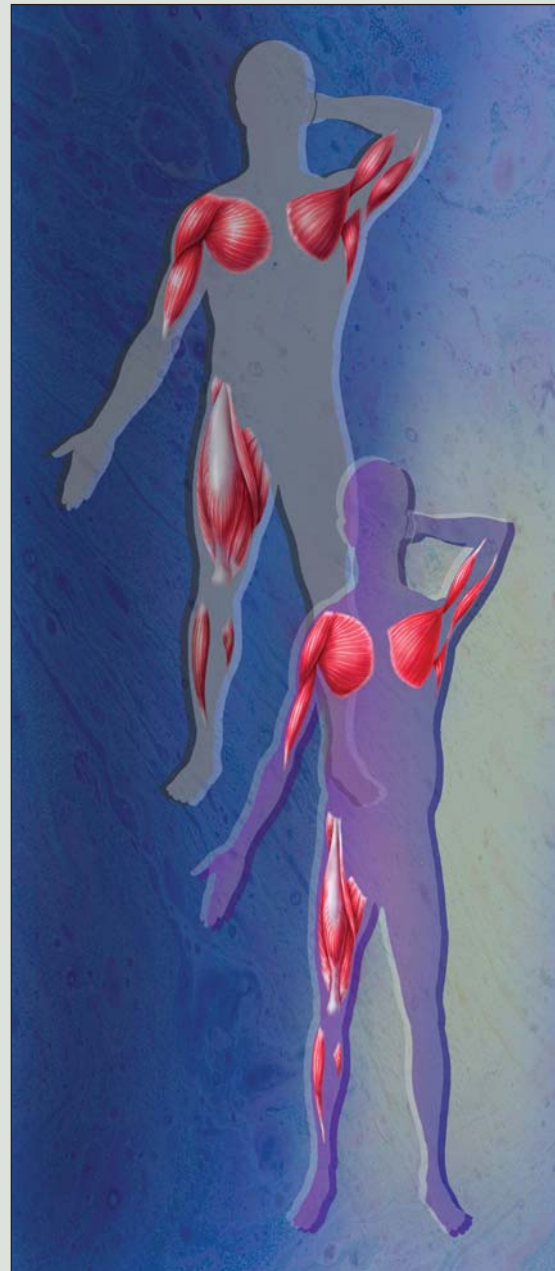
**Protein metabolism** Skeletal muscle homeostasis is achieved through a continuous repair process involving the breakdown and synthesis of protein. Studies consistently find that muscle-protein synthesis rates are approximately 30% lower in older adults than in younger adults.<sup>13</sup> Over time, this results in a net loss of muscle mass consisting of a numeric loss of muscle fibers, as well as a qualitative change in the cross-sectional area of the remaining fibers. Most atrophy is seen in the type II, or “fast twitch,” muscle fibers. Type II fibers have a high glycolytic capacity and are used for short bursts of anaerobic power.

The ability of skeletal muscle to regenerate following injury or overload decreases with age. Satellite cells, the specialized cells located in the basal membrane of the muscle cell, are necessary to develop new muscle tissue. The number of satellite cells in skeletal muscle decreases as a person ages, also contributing to the loss of muscle mass and strength.<sup>14</sup>

**Hormones** Levels of anabolic hormones uniformly decline with age. Testosterone declines at a rate of

FIGURE 1

## Effect of sarcopenia on muscle mass



Age-related changes in protein metabolism, hormone levels, muscle mechanics, and nutrition contribute to the eventual deterioration of muscle fibers in patients with sarcopenia. This process can be halted, and even reversed, through exercises that overload the major muscle groups, including the quadriceps, hamstrings, gluteals, latissimus dorsi, pectorals, and deltoids. The smaller muscle groups—biceps, triceps, trapezius, and calves—can also be strengthened.

Here and on the cover: Kevin Somerville

## IN THIS ARTICLE

### Key Points

- The progressive decline in muscle mass and strength characteristic of sarcopenia impairs functionality and eventually leads to significant morbidity.
- Unlike cachexia and wasting, sarcopenia occurs in persons who are otherwise free of disease.
- Epidemiologic studies estimate the prevalence of sarcopenia to be as high as 50% in people aged 80 years and older.
- The most prudent approach is to encourage adequate nutrition and physical activity, including strength training, for all patients who could benefit.

### Competencies

Medical knowledge	◆◆◆◆◆
Interpersonal & communication skills	◆◆◆
Patient care	◆◆
Professionalism	◆
Practice-based learning and improvement	◆
Systems-based practice	◆

For an explanation of competencies ratings, see the table of contents.

approximately 100 ng/dL per decade and has been shown to parallel the decline in muscle mass and strength occurring in elderly men.<sup>15</sup> Bioavailable testosterone levels in elderly women also are decreased, particularly immediately following menopause. However, the relationship between declining testosterone levels in women and sarcopenia has not been adequately studied.

Growth hormone (GH) is secreted in a pulsatile fashion from the pituitary gland, stimulating peripheral production of insulinlike growth factor-1 (IGF-1). GH secretion declines steadily by approximately 14% per decade, with a proportionate decline in IGF-1 levels.<sup>16</sup> GH deficiency results in a loss of muscle mass and an increase in adipose tissue.

Dehydroepiandrosterone sulfate (DHEAS) is an androgen produced by the adrenal cortex. It is considered to be a metabolic intermediate in the steroid hormone synthesis pathway of testosterone, estrone, and estradiol. Although the biological role of DHEAS is not precisely understood, levels decline with age in a linear fashion by 10% to 20% per decade.<sup>17</sup>

Although not an androgen, estrogen may have a role in the development of sarcopenia in women. Menopause has been linked to a reduction in lean body mass in women, independent of lifestyle factors such as diet and exercise.<sup>18</sup>

**Neuromuscular changes** Movement is initiated by motor neurons sending signals from the brain to the muscles. A motor neuron and all the muscle fibers it innervates make up a motor unit. The number of spinal cord motor neurons and functioning motor units declines with age, by as much as 50% after age 60 years.<sup>14</sup> This age-associated denervation is thought to be continuous and irreversible,<sup>19</sup> causing the muscle fibers to atrophy and motor neurons to eventually die.<sup>14</sup>

When a motor neuron dies, an adjacent—usually a “slow twitch”—motor neuron may reinnervate the muscle fibers, preventing atrophy. This process is called motor unit remodeling. A slow twitch motor unit has a slower firing rate, is slower to contract, produces less muscle force, and is smaller in both size and muscle fiber number. Ultimately, motor unit remodeling leads to a less efficient motor unit producing less precise control of movements, less force production, and an overall slowing of muscle mechanics.<sup>14,19</sup>

**Physical activity** Although sarcopenia cannot be prevented with physical activity, it is clearly accelerated when physical activity is lacking.<sup>1</sup> More precisely, a lack of the kind of activity that *overloads* the skeletal muscles accelerates sarcopenia. The most effective method of overloading the skeletal muscles is resistance exercise, or strength training (ST).

**Nutrition** Obesity receives more public attention, but inadequate intake of nutrients is common in elderly populations. For instance, US Department of Agriculture surveys of household food intake demonstrate that approximately 25% of women older than 65 years do not consume the recommended daily allowance (RDA) for protein. In addition, malnutrition in elderly persons appears to affect muscle mass more severely than it does in younger persons,<sup>20</sup> and correcting malnutrition may be more challenging in the elderly. The RDA for protein is set at 0.8 g/kg, which some studies show to be inadequate in the elderly.<sup>21</sup>

**Other mechanisms** Generating adenosine triphosphate within the mitochondria is necessary to create contractile force within the muscle. It is postulated that reductions in mitochondria may contribute to the increased fatigue, reduced endurance capacity, and loss of strength observed with aging.<sup>16</sup>

The delivery of oxygen and fuel to working muscles also is required for normal energy metabolism and muscular endurance. Cardiovascular disease and reduced cardiac output from heart disease result in decreased perfusion of skeletal muscles, contributing to sarcopenia.

### Effects and implications

The loss of muscle mass and strength is consistent with a loss in functional parameters including gait speed, balance, stair-climbing speed and power, and

## Strength training basics

Strength training (ST) is synonymous with resistance exercise or progressive resistance exercise. It implies muscle movement against resistance such as weights, rubber tubing, or one's own body weight against gravity. ST movements are performed at higher intensity levels and for briefer durations than aerobic-type activities. The intensity is measured as a percentage of the exerciser's one-repetition maximum (1RM), or the maximum amount of resistance that can be overcome when performing one repetition. Intensities of 60% to 90% 1RM are typically utilized in ST programs, though the initial resistance used may be as low as 30%. Patients can also be instructed to achieve a "comfortably hard level" of exertion as an alternative method of quantifying intensity.

As strength increases, a progressively higher level of resistance is needed to permit further improvement. This is accomplished by increasing repetitions and/or resistance. ST places unique stress on the musculoskeletal system, which in turn causes an anabolic adaptation response in both muscle and bone. Aerobic training does not elicit such a response.

**Exercise basics** The typical ST program consists of up to three sets of several exercises that target the major muscle groups—quadriceps, hamstrings, gluteals, latissimus dorsi, pectorals, and deltoids. Additional exercises can be added for the smaller muscle groups—biceps, triceps, trapezius, and calves. One complete cycle of lifting and then lowering a weight is called a repetition. Repetitions are performed in a strict and controlled manner, and full range of motion should be attempted unless limited by pain. Repetition ranges are usually 8 to 15 per set, with a 1- to 2-minute rest interval between each set. Attention is given to proper lifting mechanics, and breathing patterns should remain normal to avoid the Valsalva response. The average ST session lasts 20 to 30 minutes.

Compound, multiple-joint exercises are generally recommended over single-joint, isolation-type movements.

As strength and coordination improve, free-weight or body-weight movements can be incorporated. These exercises challenge patients with dynamic movement and an increased need to balance, improving kinesthetic awareness, which further supports gains in functional strength.

ST may be performed up to three times per week. If the same muscle groups are to be exercised, individual sessions should be separated by a minimum of 48 hours. There are innumerable variations that may be incorporated as experience and familiarity grow or to individualize the program to a patient's specific needs or limitations.

**Patient safety** ST has been demonstrated to be safe and effective in patients with chronic diseases, including diabetes and cardiovascular disease.<sup>1</sup> Because of the significant complications and comorbidities associated with these diseases, however, exercise prescriptions must be individualized. Absolute contraindications include unstable angina, uncontrolled hypertension, uncontrolled arrhythmias, hypertrophic cardiomyopathy, and certain stages of retinopathy. Patients with heart failure, myocardial ischemia, poor left ventricular function, or autonomic neuropathies must be carefully evaluated before initiating a ST program.

**Maximizing success** A safe, friendly environment to exercise in is needed. The ideal is a facility staffed by knowledgeable exercise professionals. Health care providers can help patients find a suitable facility by creating a comprehensive list of resources within the community for referral. Many exercise facilities have staff qualified to work with patients with special needs and are willing to build relationships with health care providers. Your list of resources should also include options for those patients who elect to exercise at home.

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time required to perform five "stand-up" (rise-from-a-chair) tests. A number of studies have attempted to quantify this loss in the elderly.<sup>22-25</sup> Diminished physical performance was proven to be a good predictor of future disability requiring nursing home institutionalization.<sup>26</sup>

Daily energy expenditure in adults declines progressively with age, even in weight-stable persons. A major contributor to energy expenditure at rest is fat-free skeletal muscle and viscera. Sarcopenia contributes to decreased energy expenditure at rest.<sup>19</sup> Therefore, the condition impacts both resting energy expenditure and physical-activity energy expenditure. These significantly contribute to the age-associated accumulation of visceral and total body fat. The implications for obesity, type 2 diabetes, or metabolic syndrome have not been adequately investigated.

Although sarcopenia does not result in the extreme immune dysfunction and subsequent mortality brought on by wasting or cachexia, it is associated with higher all-cause mortality in the elderly, even after adjusting for major clinical variables. Muscle strength, in particular, is a good predictor of mortality. One longitudinal study focused on the impact of grip strength and the rate of change in strength on all-cause mortality. Lower and declining levels of strength resulted in increased mortality, independent of both physical activity and muscle mass.<sup>27</sup>

### Treatment options

**Testosterone** In hypogonadal men, testosterone replacement increases rates of muscle protein synthesis, lean body mass, muscle strength, and task-specific performance.<sup>28</sup> Other beneficial effects include decreased fat

mass and improved bone mineral density, cognition, and sexual function. In eugonadal men, physiologic dosages of testosterone increased lean body mass but did not increase muscle strength. Patient selection and ongoing monitoring is important if replacement therapy is being considered, as testosterone may adversely affect hematocrit levels, prostate health, lipid profiles, and mood.

**Growth hormone** Supplemental GH in elderly men stimulates IGF-1 production to levels on a par with those of men in their thirties, and in most replacement trials, GH administration increased lean body mass. However, GH treatment does not increase muscle protein synthesis rates, muscular strength, or functional ability.<sup>29</sup> The techniques used to measure lean body mass did not always distinguish retained water from skeletal muscle, making the interpretation of increased lean body mass dubious. Furthermore, some studies had high dropout rates due to adverse effects, including carpal tunnel syndrome, gynecomastia, arthralgias, hypertension, and hyperglycemia.<sup>16</sup> In elderly men, ST with GH did not yield additional gains in muscle protein synthesis or strength compared to ST without supplemental GH.<sup>30</sup>

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published exercise guidelines.**

**DHEAS** Supplementation with DHEAS increases IGF-1 levels, decreases body fat, and increases muscle strength in both men and women. Additionally, DHEAS increases bone mineral density and libido in women.<sup>29</sup> Actual end points such as improvement in gait or functional tasks, however, have not been demonstrated. A lack of large-scale, randomized, controlled trials demonstrating efficacy and potential adverse effects are impediments to using DHEAS in managing patients with sarcopenia.

**Estrogen** One study that directly investigated the effects of hormone replacement therapy (HRT) on body composition found that lean body mass increased, while relative fat mass was significantly reduced.<sup>31</sup> The increase in lean body mass with HRT is thought to be a result of increased muscle anabolism and consequent increased skeletal muscle mass. Strength or function, however, was not assessed.

**Nutrition** Adequate nutrition is fundamental. Geriatric-specific nutrition guidelines that emphasize

nutrients such as vitamin B<sub>12</sub>, protein, calcium, and vitamin D are useful in managing sarcopenia. However, what constitutes optimal nutrition for treating or preventing sarcopenia is not clear. Nutritional therapies that have been studied include supplementing the diet with fast-acting whey protein, pulsatile protein feedings, and amino acid infusions.

### **Evidence-based practice guidelines**

Sarcopenia is not well known outside of certain research circles, and there are currently no established practice guidelines for clinicians pertaining to screening, diagnosis, treatment, or management. Most therapies beyond nutrition and exercise, particularly hormone replacement, remain controversial.

The most prudent approach is to encourage adequate nutrition and physical activity, including ST, in all patients who could benefit. ST results in muscle hypertrophy and improved muscular strength and power.<sup>32,33</sup> This translates into improved functionality, including walking mechanics, speed, endurance, and stair-climbing power.<sup>34-36</sup> Those already affected by sarcopenia, such as the frail elderly, receive the greatest benefit.

In one study, nursing home residents (average age, 87 years) assigned to a 10-week ST program coupled with nutrition supplements increased their muscle strength by 125% versus 3% in a control group. The ST participants also had improved gait velocity, stair-climbing power, and levels of "spontaneous physical activity."<sup>22</sup> In a 2-year study of the effects of an at-home ST program on frail women, those in the ST group experienced 31% fewer falls.<sup>32</sup> Research to date shows that ST is an important intervention for preventing falls, frailty, and disability in the elderly.

Increasing levels of lean body mass through ST also improves resting metabolic rate (RMR). Most experts believe that maintaining muscle mass is fundamental in the long-term management of obesity, diabetes, and metabolic syndrome. Limited, short-term (8- and 12-week) studies do not show muscle-mass preservation through ST while dieting to be an effective method of preserving RMR.<sup>37,38</sup> The consequences of years of so-called yo-yo dieting, with its subsequent depletion of muscle mass and perpetually diminishing RMR, have not been thoroughly investigated.

Despite its demonstrated benefits, ST continues to receive only brief mention in published exercise guidelines. Most patients cite their front-line health care providers as their primary source of information regarding healthy lifestyle decisions. There is encouraging evidence that counseling patients on exercise leads to increased exercise participation and levels of physical activity.<sup>39-41</sup>

## There is an almost universal lack of awareness of sarcopenia, its consequences, and its prevention or management.

Providers, however, cite inadequate time, knowledge, and/or experience as the most common reasons for not counseling their patients on exercise.<sup>41</sup> Time constraints will likely remain a ubiquitous problem. Health care providers who participate in aerobic exercise or ST are more likely to recommend it to their patients,<sup>41</sup> although lacking familiarity with ST should not keep a clinician from recommending it (see “Strength training basics,” page 27, for a primer on ST programs).

### Summary

The loss of muscle mass experienced in sarcopenia is a slow, progressive process. The condition is analogous to osteoporosis as a normal, albeit deleterious and equally costly, consequence of aging. There is an almost universal lack of awareness of sarcopenia, its consequences, and its prevention or management, however—which is not the case for osteoporosis.

Sarcopenia has a multifactorial etiology, and interventions targeting the various mechanisms contributing to its pathogenesis have been investigated. Exercise is the only intervention that reliably increases muscle mass, strength, and power. The benefits of exercise, particularly ST, include a reduction in disease, better balance with fewer falls, and fewer fractures. Equally compelling, exercise is associated with increased independence and quality of life. Obtaining adequate nutrition via a healthy diet is a fundamental adjunct to physical activity in managing sarcopenia.

Even though diet and exercise guidelines are an established part of public health recommendations, most people are not meeting target levels for physical activity or nutritional goals. Greater efforts are needed to achieve behavioral and lifestyle change on a national level. Until those changes occur, health care providers must champion the cause and make wellness strategies a priority in their practices. □

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