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PHYT 875

Sarcopenia: A Multifaceted and Serious Condition Among the Elderly

Introduction

By 2030, 72.1 million Americans will be 65 or older, representing 20% of the US population.[1] As our "baby boomers" age and life expectancies increase, age related conditions will continue to create a huge public health problem. Along with diseases like arthritis and osteoporosis, sarcopenia is a serious condition associated with advanced age.[2,3] Sarcopenia is the term used to describe the loss of muscle mass and strength caused by aging.[4,5] As a true age-driven disorder, it is distinct from cachexia or muscle wasting caused by inflammatory disease, starvation, and advanced illness.[5] There is evidence of reduced muscle mass and strength universally in all elderly persons compared to young, healthy, and physically active adults.[5] Although evidence shows varied results due to how the condition is defined, the prevalence of clinically significant sarcopenia ranges from 6% to 50%.[6,7] Skeletal muscle mass will peak in an individual's early adult years and will gradually decline beginning around age 45.[8] By the seventh and eight decade of life, maximal voluntary contractile strength is decreased by an average of 20-40% in both men and women.[9] This age-related loss of muscle mass and strength can lead to functional impairments and disability.[4,5,8] In 2001, disability relating to sarcopenia in the elderly population cost the US health care system an estimated $18.4 billion.[10]

Due to the pervasiveness and significance of sarcopenia, the purpose of this paper is to define sarcopenia and identify the multifactorial causes of the condition. The numerous detrimental effects of sarcopenia and current treatment options will also be discussed in order to provide a comprehensive overview of this serious disorder.

Definition and Diagnosis of Sarcopenia

Currently, there is debate as to how sarcopenia should be defined, measured, and diagnosed. A universally implemented definition of sarcopenia is warranted to help healthcare professionals identify those individuals who could be at risk for complications of the condition.[8,11] As of 2000, absolute levels of lean mass, body cell mass, and muscle mass at which sarcopenia was undeniably present were not yet defined.[5,10] Further, the levels at which muscle mass and strength become deficient leading to functional limitations and disability in the elderly population were also not concretely determined.[4] Previous research on starvation, Aquired Immune Deficiency Syndrome (AIDS), and those with critical illness demonstrated that a loss of more than 40% lean mass from baseline was fatal. However, the lethal level of muscle loss has yet to be determined for elderly patients with sarcopenia.[3]

In Janssen et al's 2002 cross-sectional survey study examining the prevalence and consequences of sarcopenia in older Americans, researchers used their validated equation for calculating skeletal muscle mass via bioelectrical impedance analysis (BIA).[Appendix I][8] Using BIA to assess body composition has been validated by underwater weighing and dual energy x-ray absorptiometry (DEXA).[12] In their study, Janssen et al converted absolute skeletal muscle mass to percentage skeletal muscle mass to get the skeletal muscle index (SMI). The SMI appropriately adjusts for height and mass of nonskeletal muscle tissues. Using a method similar to that for defining osteopenia and osteoporosis, Janssen at al developed classification criteria for muscle loss in older persons by comparing their SMI scores to a young reference group aged 18-39.[8] If older adults' SMI scores were within one standard deviation of the sex-specific mean for young adults then their muscle mass was considered normal. Class I sarcopenia was defined as having a SMI within one to two standard deviations of young adult values while those placed in the class II category had SMIs below two standard deviations of

young adult values.[8] This method provides reliable estimates of skeletal muscle mass in adults and is simple and inexpensive to administer.[8] Since Janssen et al published his work, this method of measuring and defining sarcopenia has been advocated for by other experts.

In 2010, Muscaritoli et al reported on the opinions of the special interest group concerned with cachexia in chronic wasting diseases within the European Society for Clinical Nutrition and Metabolism (ESPEN).[11] The definition and assessment of sarcopenia was a key topic in the publication. This group of experts supported the use of the method described by Janssen et al to help diagnosis sarcopenia in the elderly population.[11] In conjunction with the criteria developed by Janssen et al, these experts also advocated for using gait speed to help define sarcopenia.[11] Along with a low SMI, Muscaritoli et al believes a walking speed less than 0.8 m/s on the Four Meter Walking Test is indicative of sarcopenia.[11]

Fielding et al also encouraged the use of gait speed in diagnosing sarcopenia in his paper from 2011.[13] This publication from the Journal of the American Medical Directors Association summarized agreement on the definition of sarcopenia from a group of geriatricians and scientists who met in Rome, Italy to discuss the topic.[13] Slightly different from Muscaritoli et al's group, Fielding et al stated that patients walking less than one m/s should be referenced for body composition assessment using DEXA.[13] While DEXA is a valid and reliable way to assess body composition, it is expensive and not readily available. Further, DEXA may underestimate the prevalence of sarcopenia.[4]

Clearly, the literature shows variations in the exact details of how to clinically define and diagnose sarcopenia. However, clinicians should consider this condition in patients who present with low gait speed, a history of recurrent falls, documented weight loss, recent hospitalization, difficulties in performing activities of daily living (ADLs), and/or chronic conditions associated

with muscle loss.[13] Type II diabetes, chronic heart failure, chronic obstructive pulmonary disease, chronic kidney disease, rheumatoid arthritis, and malignancies are all associated with muscle loss.[13]

Multifactorial Causes of Sarcopenia

While the most optimal way to define and diagnosis sarcopenia is still under debate, numerous internal and external factors that lead to the development of sarcopenia have been recognized.[Appendix II and II][4,5,9,11,13,14] One of the major internal contributors to sarcopenia is the loss of skeletal muscle fibers due to reduced numbers of motonuerons.[5,9,14] Loss of motor neurons or age-related neurodegeneration occurs throughout life.[5,14] With aging, the "adoption" of muscle fibers by surviving neurons partially compensates for the loss of neurons and muscle cells, but this results in larger and less efficient motor units.[5] Thus, the remaining motor units have to work harder.[14] To further compound the problem, there is a preferential loss for fast motor units compared to slow motor units as well as fiber atrophy or loss of cross-sectional area of type II fast glycolytic fibers.[13,14] Various studies have demonstrated reductions in type II fiber area ranging from 20 to 50% while type I area losses range from only 1 to 25%.[9] In addition to the preferential loss of type II fibers, as remaining motor units recruit denervated fibers, the fibers change their type to that of the motor unit's.[14] Therefore, there is a net conversion of type II fibers to type I fibers with aging and evidence of an increase in hybrid type I and II fibers.[13,14] Furthermore, although still controversial, with age there is evidence of loss in the number and function of muscle satellite cells, which are responsible for proliferating and differentiating into skeletal muscle fibers.[14] Overall, age-related neurodegeneration, loss of type II fibers, and decreased ability to regenerate fibers results in significant loss of muscle mass and force.[14]

More possible internal causes of sarcopenia include an increase of noncontractile area and cross-bridging between fibers within the muscle.[13,15] Moreover, the neuromuscular junction undergoes changes with advancing age creating a decreased number, but increased size of terminal areas as well as a reduction in the number of synaptic vesicles.[14] Age-related losses in peripheral nerve fibers and alterations of their myelin sheaths have also been described in the literature.[14] The above factors contribute to a slower twitch contraction time, lower maximum shortening speed, decreased single-fiber intrinsic force, and overall decrease in muscle power.[13,14]

For skeletal muscle mass to be maintained, an active balance must be sustained between protein synthesis from free amino acids and the division of muscle protein into free amino acids.[14] If defects occur within this balance where there is a decrease in skeletal muscle protein synthesis and/or an increase in skeletal muscle protein breakdown, then severe losses in muscle mass can occur.[13,14] Anabolic hormones, such as growth hormone, testosterone, and estrogen that promote protein synthesis, are known to decrease with age and may lead to muscle atrophy and sarcopenia through increasing protein degradation.[5,14] For example, it is well known that a woman's estrogen levels significantly decrease during menopause. There is also evidence of accelerated loss of lean mass and gain in fat during this period, suggesting that estrogen could have a supporting role in maintaining muscle mass.[5,9]

Other humoral factors of muscle loss can be contributed to defects in the aging individual's immune system.[5] Activation of the immune system leads to production of inflammatory cytokines which can cause amino acid export from muscles, further altering the balance between protein synthesis and degradation.[5,14] Some evidence has shown that with aging, the immune system is less able to identify "self" from "non-self" antigens and may

increasingly target self antigens.[5] The higher prevalence of autoimmune diseases as one ages also supports these findings.[5] One theory on aging, states that aging is associated with a subclinical inflammatory state. This inflammatory state leads to an increased production of catabolic cytokines thus creating a greater rate of muscle degradation leading to sarcopenia.[4,5,9,14]

Lastly, in relation to internal processes, oxidative metabolism generates reactive oxygen species (ROS) that accumulate overtime and damage cell components like mitochondria and DNA sequences.[11,14] Evidence has demonstrated that the frequency of abnormal mitochondrial regions is higher in those muscles which are strongly affected by sarcopenia.[14] Currently, the relationship between alterations in mitochondrial DNA and age-related skeletal muscle loss is under investigation. Structural modifications in mitochondria may affect the electron transport chain and compromise respiration.[14] Reductions in maximal oxygen consumption with age has been attributed mainly to loss of muscle mass and reduced cardiac output, but altered mitochondrial mechanisms causing poorer muscle cell respiration may also be a contributing factor.[14]

Obviously, there is an abundance of internal factors in the aging individual that can contribute to the development of sarcopenia. However, several external factors negatively influence muscle mass in the elderly and warrant examination as well.[11] Disuse and physical inactivity are significant contributing factors to developing sarcopenia at an increased rate and to a greater extent.[5,9] Only 10-15% of individuals who are over 65 years of age can be classified as "active".[16] Elderly patients who are less physically active have lower skeletal muscle mass as a sedentary lifestyle promotes loss of muscle and gains in fat content.[5,9] However, since evidence demonstrates that even master athletes develop sarcopenia, the condition remains a result of aging and not solely a consequence of disuse.[5]

The relationship between decline in food intake as one ages, known as anorexia of aging, and the pathogenesis of sarcopenia has also been suggested as another external factor that causes sarcopenia.[4,11] The reasons for anorexia of aging are complex.[Appendix IV][4] With aging, individuals become more satisfied at an earlier stage when eating the same amount of food compared to younger individuals. This mainly occurs due to changes in the fundus of the stomach, which has a decreased ability to respond to adaptive relaxation.[4] Hormone and neurotransmitter changes can also affect anorexia of aging.[4] With reduced food ingestion, protein intake typically falls below the level necessary to maintain muscle mass and decreased consumption of essential dietary nutrients for muscles, such as creatine, may contribute to sarcopenia.[4] Getting adequate amounts of food, especially protein, is often difficult for elderly persons due to higher cost of nutrient dense foods, perceived intolerance to certain food groups, difficultly chewing fibrous foods, and/or fear of consuming too much fat or cholesterol.[17] Further, depression rates are higher in the elderly as compared to the general population and this condition is the most common cause of protein energy malnutrition.[1,4] Compared to younger people, when an elderly person is depressed, they are more likely to experience weight loss.[4]

Along with decreased physical activity and anorexia of aging, Castillo et al found current smokers to have an increased risk of developing sarcopenia.[12] Yet, medications, such as thyroid hormones, corticosteroids, and hormone replacement therapy, were not associated with sarcopenia.[12] While it is important to recognize the vast internal and external causes that lead to sarcopenia, clinicians must also identify how this condition affects elderly patients.

Effects of Sarcopenia

The complex causes of sarcopenia lead to well documented detrimental effects on elderly patients. Overall, sarcopenia creates decreased muscle mass and strength, which is correlated

with functional decline, loss of independence, increased risk of injury, disability, and mortality.[13,14] Age-related changes to the motor units and the concomitant loss of type II fibers result in loss of coordinated muscle contraction and muscle power needed for actions like rising from a chair, climbing steps, or maintaining balance after a perturbation.[4,14] Most importantly, sarcopenia is predictive of falls in elderly patients.[12] A study by Castillo et al found elderly men with sarcopenia to be twice as likely to have fallen in the past year compared to men with preserved muscle mass.[12] Lower extremity weakness is especially predicative of falls. Evidence shows that reduced strength in the hip and other leg muscles along with impaired neuromuscular activation is highly associated with in falls in the elderly.[14]

Fall-related injuries are the main cause of accidental death as well as a major cause of hospitalizations among the elderly.[18,19] The disability from falls causes a significant impact on the public, the caregivers, and the individual. Fall-related injuries in the elderly cause increased usage of publicly funded programs like Medicare and Medicaid in both the acute and chronic phases.[18] When falls occur, caregivers to dependent older adults can sustain monetary cost, increased stress, and loss of time at work.[18,20] In addition, falls or the fear of falling can cause decreased mobility, feelings of hopelessness, loss of confidence, depression, and institutionalization for the individual.[19,21] Decreased mobility may result in elderly persons limiting their activities which can further contribute to muscle loss, osteoporosis, impaired balance, orthostatic hypotension thus further increasing their risk of falls.[19,22] Evidence has shown that knee extension torque and low thigh muscle cross-sectional area are correlated with increased incidence of hip fracture by 50-60%, independent of bone mineral density.[14] Mortality post fracture in the geriatric population is estimated between 10-20% with 50% of fractures resulting in permanent disability.[18,23]

Not only are individuals with sarcopenia at greater risk for falls, subsequent fractures, and the consequences that follow, they are also at an increased risk for being unable to carry out ADLs. Men and women who are in the lowest quartile of thigh muscle cross-sectional area and leg muscle mass had a 30-40% increased risk for the inability to perform normal ADLs.[14] This creates a greater dependence and extra burden on caregivers. The loss of maximal oxygen consumption with age has also been attributed to loss of muscle mass, which further compounds the problem of elderly people performing usual ADLs, participating in optimal levels of physical activity, and consuming adequate nutrition.[14]

Research has shown that those with severe sarcopenia as measured by BIA and defined by Janssen et al, have a 79% greater likelihood of disability.[13] Sarcopenia has even been found to increase a person's risk of nosocomial infection during hospitalization which can lead to further debility or even mortality.[13] However, those with moderate sarcopenia do not have significantly different rates of disability compared to those with normal muscle mass.[13] Therefore, it is imperative to identify the modifiable risk factors for sarcopenia to lessen and combat the detrimental effects this condition can have on the elderly population.

Treatment of Sarcopenia

Currently, evidence has identified physical inactivity as the main modifiable risk factor for sarcopenia. Numerous research studies have shown that resistance training is the most efficacious treatment for this condition.[4,5,9] Resistance training has even demonstrated improvements in strength and increased muscle mass in very frail elderly individuals over 90 years old.[9] Clearly then, aging muscles do not lose the ability to adapt to physical training.[5] Resistance programs lasting 10 to 12 weeks in duration with training two or three times a week have consistently resulted in strength gains in both men and women.[9] However, exercise should

be continued to maintain the gains resulting from the exercise program.[14] Typically, in the first few weeks of a training program, elderly individuals will begin to notice changes in strength. As there is no change in muscle mass at this time, this increase in strength is thought to occur via changes in the central nervous system (CNS) by increased neuronal firing rates, improved recruitment of motor units in response to the signal to contract a muscle, and by increased innervation to the muscle.[5] This phenomenon has been supported by the cross-educational effect seen in contralateral muscles when only one extremity is trained. Some studies have shown strength gains on the untrained side almost as much as half as the increases found on the trained side.[5] In fact, even imagining exercise can significantly increase strength, which is also hypothesized to be the result of changes that occur within the CNS.[5]

With resistance training in the elderly, changes not only occur in the CNS, but evidence has also shown increases in muscle fiber size, capillary density in the muscle, and muscle protein synthesis.[4] Further, resistance training seems to have greater effects on increasing bone mineral density compared to walking and aerobic training.[14] High-intensity resistance training has shown significant effects for improving bone density at hip and vertebral sites, two common places for fractures in the elderly.[14] Since those with sarcopenia are already at a greater risk for falls, resistance training for these individuals is essential to help improve balance and decrease the risk of fractures.

Following a resistance training program for the elderly, both muscle mass and strength are increased.[24] Improvement in muscle quality, strength relative to muscle mass, is imperative in preventing functional decline and disability in elderly adults.[24] Goodpaster et al found that in aging adults, strength loss is actually more rapid compared to loss of muscle mass, which produces a decline in muscle quality.[25] His groups' research also showed that maintaining

muscle mass does not prevent age-associated declines in strength.[25] Further, Newman et al determined lower muscle strength to be a strong and independent predictor of mortality in geriatric patients where simply accounting for muscle size did not.[26] Therefore, resistance training and other treatments as well as outcome measures for this population need to be focused on improving and assessing strength, not solely muscle mass.

In addition to resistance training for preventing and reversing sarcopenia, inadequate nutrition is another modifiable risk factor.[13] Decreases in skeletal muscle mass are one of the most noticeable consequences of the reduction in total body protein as one ages.[17] Recently, experts have recommended that older adults need at least 1.0 gram of protein per kilogram of body weight daily as opposed to the suggested amount of 0.8 grams of protein per kilogram of body weight for younger adults.[17] In a recent study of 2,066 older community-dwelling adults, researchers found that increased dietary protein might be effective in preserving lean muscle, thus preventing sarcopenia and its negative effects.[27] In addition, nutritional interventions that enhance insulin sensitivity and muscle perfusion may be beneficial to elderly adults by improving muscles' ability to use nutrients and respond to anabolic stimulation.[7]

Numerous pharmacological agents and their effect on sarcopenia have also been tested. Sex hormones such as testosterone and estrogen may suppress inflammatory cytokines that exert catabolic effects on muscle.[14] Since these two hormones are known to decrease with age, the effectiveness of hormone replacement therapy has been studied intensely. Mixed results have ensued with this therapy, which proved to be less effective in women than men.[4,14] Using growth hormone to combat sarcopenia in the elderly population has also been studied.[4,14] However, most results have shown this treatment to be unsuccessful in improving strength in the elderly, even if it is efficacious in increasing muscle mass.[4,5,14] Newer treatments with growth-hormone-

releasing hormone maybe more effective, as they have shown increases in growth hormone production and produced moderate gains in muscle strength.[14] Therefore, future research into the value of pharmacological agents in treating sarcopenia is necessary.

Conclusion

Sarcopenia is a serious condition caused by a multitude of factors affecting many individuals as they age. Muscle mass decrease and subsequent strength loss in the elderly lead to many adverse consequences such as loss of independence, reduced mobility, and possible mortality. As our population ages, sarcopenia and its negative effects will create a huge public health problem. Therefore, medical professionals need to establish a clear consensus on how to diagnose the disorder. With proper recognition of sarcopenia, elderly patients can begin treatment to prevent and reverse the disease through strength training and nutritional interventions. Innovative advances in pharmacological agents may also help avert and manage sarcopenia in the future.

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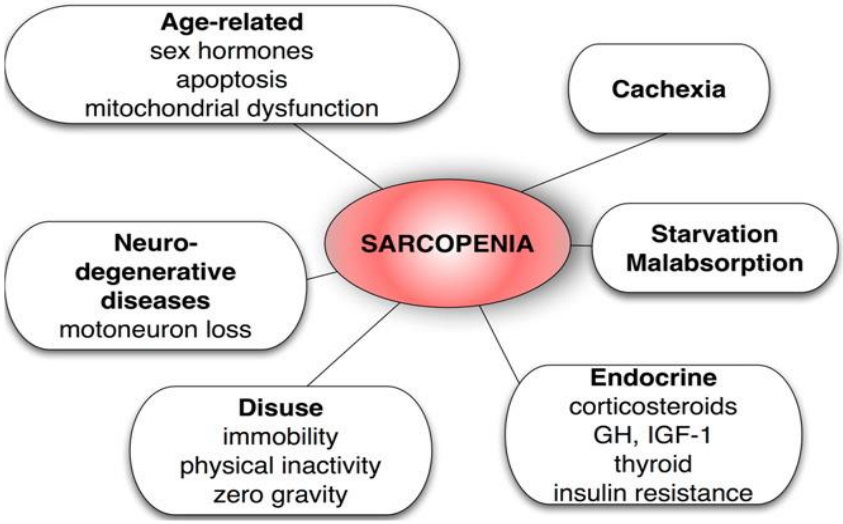
Appendix

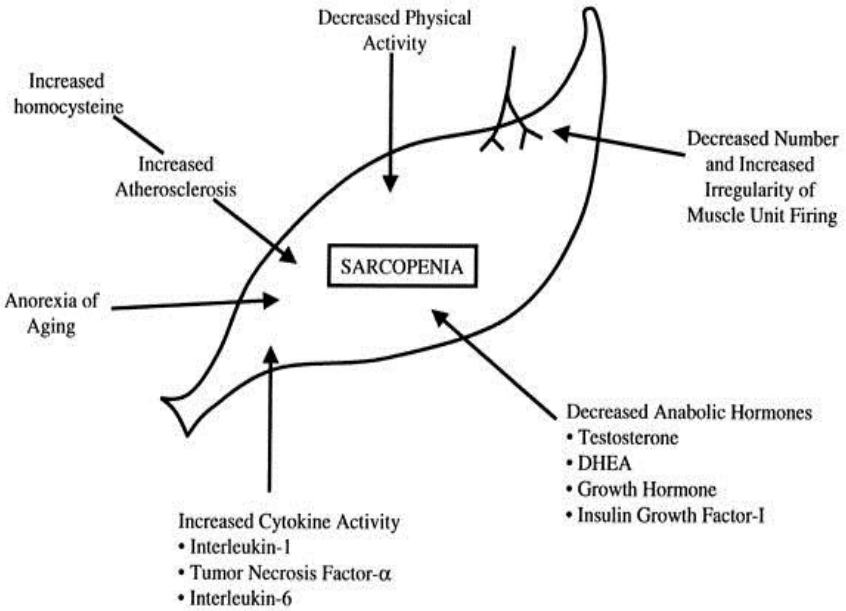
Appendix I: Janssen et al calculated muscle mass using the following BIA equation[8]

$$\text{skeletal muscle mass (kg)} = [(\text{height}^2/\text{BIA-resistance} \times 0.401) + (\text{gender} \times 3.825) + (\text{age} \times -0.071)] + 5.102$$

Appendix II: Conditions potentially leading to sarcopenia[11]



Appendix III: Multifactorial causes leading to sarcopenia[4]



Appendix IV: Reversible causes of weight loss in older adults; MEALS ON WHEELS mnemonic[4]

M edications (eg, degoxin, theophylline)
E motional (eg, depression)
A lcoholism, obesity, anorexia tardive
L ate-life paranoia
S wallowing
O ral problems
N osocomial infections (eg, tuberculosis, clostridium difficile, Helicobacter pylori)
W andering and other dementia-related behaviors
H yperthyroidism, hypoadrenalism, hypercalcemia
E ntene problems (eg, gluten enteropathy)
E ating problems
L ow-salt, low-cholesterol diet
S tones (cholecystitis)