Published in final edited form as:
J Am Med Dir Assoc. 2011 May ; 12(4): 249–256. doi:10.1016/j.jamda.2011.01.003.

Sarcopenia: An Undiagnosed Condition in Older Adults. Current Consensus Definition: Prevalence, Etiology, and Consequences

International Working Group on Sarcopenia
International Sarcopenia Consensus Conference Working Group Meeting*, Rome, Italy, November 18, 2009

Abstract

Sarcopenia, the age associated loss of skeletal muscle mass and function, has considerable societal consequences for the development of frailty, disability and health care planning. A group of geriatricians and scientists from academia and industry met in Rome, Italy on November 18, 2009 to arrive at a consensus definition of sarcopenia. The current consensus definition was approved unanimously by the meeting participants and is as follows: Sarcopenia is defined as the age-associated loss of skeletal muscle mass and function. The causes of sarcopenia are multi-factorial and can include disuse, altered endocrine function, chronic diseases, inflammation, insulin resistance, and nutritional deficiencies. While cachexia may be a component of sarcopenia, the two conditions are not the same. The diagnosis of sarcopenia should be considered in all older patients who present with observed declines in physical function, strength, or overall health. Sarcopenia should specifically be considered in patients who are bedridden, cannot independently rise from a chair, or who have a measured gait speed less that 1.0 $m{\cdot}s^{-1}$. Patients who meet these criteria should further undergo body composition assessment using dual energy x-ray absorptiometry (DXA) with sarcopenia being defined using currently validated definitions. A diagnosis of sarcopenia is consistent with a gait speed of less than 1 $m{\cdot}s^{-1}$ and an objectively measured low muscle mass (eg: appendicular mass relative to ht^2 that is ≤ 7.23 kg/ m^2 in men ≤ 5.67 kg/ m^2 in men). Sarcopenia is a highly prevalent condition in older persons that leads to disability, hospitalization and death.

Keywords

muscle; aging; body composition; function; disability

"The sixth age shifts
Into the lean and slipper'd pantaloon
With spectacles on nose and pouch on side,
His youthful hose well sav'd, a world to wide
For his shrunk shank"

Shakespeare, As You Like It, Act II, Scene VII, lines 157–161

A reduction in lean body mass and an increase in fat mass is one of the most striking and consistent changes associated with advancing age. Skeletal muscle (1) and bone mass are the

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Financial disclosures: Partial support for this meeting in the form of travel costs were provided by GlaxoSmithKline and Abbott, Chiesi, Danone, Merck, Nestlé, Novartis, Sanofi Aventis

principal (if not exclusive) components of lean body mass to decline with age. These changes in body composition appear to occur throughout life and have important functional and metabolic consequences. The term sarcopenia (From the Greek: sarx for flesh, penia for loss) was first used by Irwin Rosenberg (2). It was originally described by Evans and Campbell (3) and further defined (4) as age related loss of muscle mass. This loss of muscle results in decreased strength, metabolic rate, aerobic capacity and thus, functional capacity. Subsequently, a number of authors have defined sarcopenia more specifically as a subgroup of older persons with muscle mass depletion, usually defined as being two standard deviations below the mean muscle mass of younger persons (usually age 35 years) (5). Since 1994 when 4 articles on sarcopenia were published, there has been an exponential increase in the number of publications reaching 140 in 2006 (6). This has been mirrored by an increase in citations to articles on sarcopenia going from 0 in 1996 to 2221 in 2006. Over this time sarcopenia has become recognized as an important geriatric condition and a key precursor to the development of frailty (7, 8). Much like osteopenia (bone density) predicts risk of a bone fracture, sarcopenia is a powerful predictor of late-life disability. The purpose of this article is to define sarcopenia, provide guidelines for assessment and briefly describe its prevalence, etiology, and consequences. Sarcopenia has “come of age” and should be recognized as a preventable and treatable condition among geriatric patients.

In 1931, Critchley noted that muscle loss occurs with aging and is most noticeable in intrinsic hand and foot muscles (9). Sarcopenia very likely begins in early adulthood (10) with atrophy and loss of type II muscle fibers (11, 12) and continues throughout life as a result of complex interaction of environmental and genetic causes. The direct effect of sarcopenia, on strength is illustrated by the dramatic age-associated decline in the world weight lifting records. These records decline by 30% in men and over 50% in women between the ages of 30 to 60 years. (13). Longitudinal studies have shown a clear decline in muscle mass, strength and power beginning at approximately 35 years of age (14). Strength and power decline to a greater extent than does muscle mass (15). In addition to sarcopenia, intramusculater lipid, termed myosteatosis (16), increases with age and increasing body fatness. Janssen et al. (17) estimated that sarcopenia results in an excess cost to the health care system of the United States of $18.4 billion a year (year 2001), due to associated disability.

Current Consensus Definition

A meeting was convened on November 18, 2009 in Rome, Italy with the express purpose of arriving at a consensus definition of sarcopenia. Because there has been no true consensus of the appropriate criteria for when an individual may be said to be sarcopenic, recognition of this treatable condition has been lacking. The following definition was the current consensus of the group of scientists and geriatricians that were present at the meeting. In addition, this definition was reviewed by a number of researchers in the area of skeletal muscle and aging. All participants are listed in the appendix:

> “Sarcopenia is the age-associated loss of skeletal muscle mass and function. Sarcopenia is a complex syndrome that is associated with muscle mass loss alone or in conjunction with increased fat mass. The causes of sarcopenia are multi-factorial and can include disuse, changing endocrine function, chronic diseases, inflammation, insulin resistance, and nutritional deficiencies. While cachexia may be a component of sarcopenia, the two conditions are not the same.”

There was unanimous agreement that the presence of sarcopenia should be evaluated in older patients who have clinically observed declines in physical functioning, strength, or health status (Table 1). Clinicians should also consider sarcopenia in patients who present with difficulties in performing activitites of daily living, have a history of recurrent falls,

have documented recent weight loss, have recently been hospitalized, or have chronic conditions associated with muscle loss (eg: Type II diabetes, chronic heart failure, chronic obstructive pulmonary disease, chronic kidney disease, rheumatoid arthritis, and malignancies).

There was consensus on the panel that sarcopenia could be effectively targeted by assessing physical functioning in at risk patients (Table 2). Sarcopenia should be considered in patients who are bedridden, non-ambulatory, or who cannot rise from a chair unassisted. In addition, for patients who are ambulatory and can arise from a chair, gait speed should be assessed across a 4 meter course. Patients with a measured gait speed less than 1.0 $m{\cdot}s^{-1}$ should be referred for body composition assessment using whole body dual energy x-ray absorptiometry (DXA).

The diagnosis of sarcopenia should be based on having a low whole body or appendicular fat free mass in combination with poor physical functioning. Current methods index appendicular fat free mass to height squared or whole body fat free mass to height squared. In patients with poor functional capacity, most easily identified using gait speed of than 1 $m{\cdot}s^{-1}$, sarcopenia can be diagnosed when the lean mass is less than 20%tile of values for healthy young adults. Currently objective cutpoints can be made for sarcopenia in men at an appendicular fat lean mass/ ht^2 (aLM/Ht^2) of ≤ 7.23 kg/ m^2 and in women at ≤ 5.67 kg/ m^2 (18).

Muscle Aging

Over the age span from 20 to 80 years of age, there is approximately a 30% reduction in muscle mass and a decline in cross-sectional area of about 20% (14). This is due to a decline in both muscle fiber size and number (10). There is no consensus on whether there is a selective loss of specific muscle fiber types. Early cross-sectional studies demonstrated a shift in muscle fiber composition with a higher type I/type II fiber ratio with advancing age (11). Larsson et al (12) suggested a preferential loss of type II fibers with advancing age, potentially starting in early adulthood. Type II fibers demonstrate selective atrophy (with a preservation of Type I fiber area) with age (19, 20). This is due to a reduction in high intensity activities that recruit these fibers, while type I fibers are used for most activities of daily living and during submaximal exercise (e.g. walking). An increase in hybrid type I and II fibers (21, 22) with advancing age has been described. Within the muscle, there is a decrease in non-contractile area along with a decrease in cross-bridging between the fibers. Single fiber intrinsic force is decreased. There is a decline in the number of T-tubule dihydropyridine receptors and an increase in uncoupled Ryanodine receptors. Twitch contraction time is slower and maximum shortening speed is lower.

Ultrasound studies have demonstrated the importance in tendon changes in altering muscle power with aging (21, 23–25). With aging there is a decrease in tendon stiffness which, coupled with the shortening of muscle fascicles, results in smaller pennation angles (26, 27) and a decrease in specific force (i.e., fascicle force/physiological cross-sectional area). This may be one cause of decreasing strength with advancing age. In general, aging is associated with a greater decline in lower body than upper body and extensor compared to flexor strength (28). Overall, there is a much greater decline in strength than muscle mass with the decline in isometric knee extensor strength being between 55 to 76% (22, 29). These changes may be a cause of the decline in gait velocity that occurs with aging.

Etiology and biochemical basis for Sarcopenia

Sarcopenia is a universal phenomenon with a complex, multi-factorial etiology. Many of the potential causes vary by the age of the individual and are summarized in Table 3. The major

factors considered to be involved include genetic heritability (30–32), nutritional status (protein intake, energy intake, and vitamin D status) (33–38), physical activity (39–42), hormonal changes (declines in serum testosterone and growth hormone) (43, 44), insulin resistance (45–47), atheroscelorosis (48–50) and changes in circulating pro-inflammatory cytokines (51).

At the molecular level, Sarcopenia results from a disproportionate decrease in skeletal muscle protein synthesis and/or an increase skeletal muscle protein breakdown. Anabolic hormones and muscular activity drive the system through activation of the phosphatidyl inositol$_3$ kinase/serine-threonine kinase AKT system (52). This system stimulates muscle protein synthesis through the activation of the mammalian target of rapamycin (mTOR) and SGKI and inhibits atrophy by physophorylating the forkhead protein FOXO. Phosphorylated FOXO is inactivated thus reduces expression of the E3 ligase, Atrogin I and subsequently preventing protein degradation by the ubiquitin-proteasome system (53). A greater expression of MuRF-1 and atrogin-1 expression has been observed in aged rodent muscle compared to young along with a 90% higher level of ubiquitin conjugates. Increased availability of amino acids, particularly, branched chain amino acids stimulate mTOR (54). Elevated levels of angiotensin II inhibit phosphorylation of FOXO and stimulate capsase 3, which cleaves actomysin, allowing the actin and myosin to be degraded by the ubiquitin-proteasome system. This may explain the association of angiotensin converting enzyme inhibitors with increase muscle mass (55). Glucocorticoids inhibit AKT activity (55). Cytokines stimulate MURF 1 (muscle Ring finger), which, like atrogin, activates the ubiquitin-proteasome system (56). Myostatin D inhibits cell cycling through SMAD3 and MyoD, thus inhibiting the production of satellite cells (52) while testosterone increases satellite cell production by stimulating β-catenin. Cytokines cause DNA fragmentation and apoptosis by stimulating NFkB to produce Capsase 8. While little data comparing human skeletal muscle expression of factors affecting the expression of the ubiquitin-proteasome system, a recent study (57) examined baseline characteristics of young (23 ± 2y) and old (85 ± 1y) women as well as their response to a bout of resistance exercise. At baseline, the older women expressed FOXO3A and MuRF-1 genes at higher levels than did the young women. In response to a bout of resistance exercise all of the women demonstrated a substantial increase in the expression of MuRF-1, however the older women also showed a greater expression of atrogin-1, perhaps indicating a greater muscle proteolytic response to exercise.

Epidemiology

The measurement of muscle mass in humans is difficult with most of the available methods requiring assumptions that may not always be valid and with variable degrees of accuracy and difficulty. The most direct measurement currently available is urinary creatinine measured over 24-hour periods (58). Other, more indirect measures, include anthropometry (59), bioelectrical impedance, dual-energy x-ray absorptiometry (60), imaging techniques (e.g., computed tomography and magnetic resonance imaging), ultrasound, total body potassium and neutron activation (61–63). Most indirect measures of fat free mass assume, incorrectly, that skeletal muscle remains a constant component of 60% of fat free mass (64). Thus, some authors use only appendicular skeletal mass and correct this for height. Recent studies have demonstrated that not only is muscle mass reduced with advancing age, but the quality of muscle may also change. Increased skeletal muscle lipid, assessed by computerized tomography, is increased with advancing age and increased total body fatness (65). The Health and Body Composition Study, a longitudinal study of more than 3,000 older (age 70 – 79 y at baseline) has demonstrated the strong association of muscle mass with strength as well as the changing quality of skeletal muscle in late life (66). This and other studies show that sarcopenia is associated with reductions in strength, however the

relationship between muscle mass and force production deteriorates with advancing age (65, 67–69).

Several studies have quantified sarcopenia by indexing fat-free mass or appendicular fat-free mass by divided by height squared, fat mass or total mass. Using an index of aLM/Ht^2 in the New Mexico Study, the prevalence of sarcopenia (i.e. aLM/Ht^2 2 SD below a young reference group) was originally determined to be over 50% in persons older than 80 years (5). Subsequent studies in this population using more direct estimates found a prevalence of 12% for persons 60 to 70 years of age and nearly 30% for persons over 80 years (5, 70). Janssen et al. (71) using an index of lean/total mass and bioelectric impedance data from NH ANES III, found the prevalence of sarcopenia (−2SD) in persons 60 years of age and older was 7% to 10%. Women were more likely to be sarcopenic than were men in this study, but based on different indices, others have reported the opposite (72–76). Table 4 compares a number of different studies on the prevalence of sarcopenia. A common finding of all of these approaches is that sarcopenia, defined as reduced fat free mass, is highly prevalent in older people and that it increases with advancing age.

Sarcopenia and Disability

Sarcopenia is correlated with functional decline and disability (5, 16, 71, 76, 77). Findings are often stronger in men than in women, depending on the indexing method used. Sarcopenia has also been associated with increased mortality (78), although (79) weakness has been demonstrated to be a more powerful predictor of mortality in elderly people than muscle mass. In the longitudinal Rancho Bernardo study, sarcopenia was shown to be predictive of falls (72). Janssen (80) examined 5,036 men and women over 65 enrolled in the Cardiovascular Health Study. He reported that the likelihood of disability was 79% greater for those with “severe” sarcopenia (< SD below that of a 30-yr old person, based on bioelectric impedance and using lean mass/ height squared but not significantly different for those with “moderate” sarcopenia compared with those with normal muscle mass. During 8-year follow-up, only those with severe sarcopenia were more likely to develop physical disability. Sarcopenia has also been found to predict nosocomial infection during hospitalization (81). The term sarcopenic obesity was first used by Heber et al. (82) in 1996 and describes persons with reduced body mass out of proportion to their adipose mass. Sarcopenic obesity is associated with disability, gait problems and falls to a greater extent than persons with “proportionate” sarcopenia (83). In an 8 year longitudinal study, Baumgartner et al. (83), found that “obese sarcopenia” was a better predictor of physical disabilities, abnormalities in gait or balance and falls in the past year than either sarcopenia or obesity alone. This observation has been confirmed in the Framingham and NHANES (National Health and Nutrition Examination Survey) studies demonstrating that elderly people with high body fat and low muscle mass had the highest rate of disabilities (84). These data point to the fact that the development of disability and impaired mobility in older people is a complex etiology. Muscle mass is an important, but not the only, predictor of muscle strength or physical function. Fat has several adverse effects on muscle function. Higher body fatness and older age have been associated with greater intramuscular lipid and reduced muscle quality, defined as reduced strength/cross sectional area (66, 85). It is also possible that higher body fatness decreases the capacity to generate power (force × speed) and muscle power is more closely related to functional capacity that muscle strength (86). Several indices of sarcopenia that account for muscle and fat mass have been examined in relationship to function. These studies illustrate the complexity in defining sarcopenia in relationship to fat mass (18). When fat mass is considered the role of lean mass per se is apparently small. One study of older women compared BMI to lean mass/total mass and aLM/Ht^2 and found only the former two indices were associated with ADL difficulties (87).

As work continues to define the relationship of both lean mass and fat mass together as they relate to disability, refinements in defining sarcopenia are likely to develop. Currently, to classify an individual as sarcopenic, the index of aLM/Ht^2 has had the most support, particularly in men. Better reference values and perhaps sex-specific metrics may prove to provide more precise prediction of future disability in older adults.

Conclusion

Sarcopenia represents a major cause of disability and increased health costs in older persons. It is very common but like most geriatric syndromes, seldom recognized by physicians. Identification of sarcopenic patients at greatest risk can be performed using an easy to perform assessment of mobility, such as gait speed and commonly obtained measures of body composition (88). DXA instruments are used to assess bone density for the identification of those at greatest risk for the development of osteoporosis. Advances in instrumentation and software allow for an accurate and precise measure of fat free mass in elderly people. Similarly, elderly people should be screened for low muscle mass and poor functional capacity. These individuals have a very high risk of loss of independence and premature death. A number of promising treatments for sarcopenia are currently under investigation including physical activity, nutritional therapies, androgen therapy, and other behavioral and pharmacological strategies. However, until professional organizations, Centers for Medicare and Medicaid Services and the Food and Drug Administration recognize sarcopenia as a treatable geriatric condition its identification, treatment and the continued development of potential “anti-sarcopenia” agents will be limited.

We propose here in this manuscript a consensus definition of sarcopenia. This definition defines a population of patients that should be considered for evaluation of sarcopenia, a set of guidelines to target patients who may be sarcopenic for further evaluation, and an objective definition of sarcopenia. The use of the current consensus definition for when an individual can be said to be sarcopenic should provide the criteria for who should be considered for treatment of this condition. Although presently limited, available treatments for sarcopenia include interventions to promote healthy eating and increased physical activity.

Acknowledgments

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Sources of support: Partial support for this meeting in the form of travel costs were provided by GlaxoSmithKline and Abbott, Chiesi, Danone, Merck, Nestlé, Novartis, Sanofi Aventis

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Appendix

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Table 1

Initial Patient Presentation to Evaluate for Sarcopenia

- Noted decline in function, strength, "health" status
- Self-reported mobility-related difficulty
- History of recurrent falls
- Recent unintentional weight loss (> 5%)
- Post-hospitalization
- Other chronic conditions (eg: Type II diabetes, CHF *, COPD **, CKD ***, RA ****, and Cancer)

* CHF = chronic heart failure;

** COPD = chronic obstructive pulmonary disease;

*** CKD = chronic kidney disease;

**** RA = rheumatoid arthritis

Table 2

Targeting Sarcopenia

- Assess patient for reduced physical functioning (or weakness).
- Consider Sarcopenia in patients who are non-ambulatory or who cannot rise from a chair unassisted.
- Assess habitual gait speed over a 4 meter course.
- Patients with a habitual gait speed < 1.0 m/sec. should be considered for quantitative measurement of body composition by DXA.

Table 3

Sarcopenia Etiology by Age

Age	Potential causes	Effects
20–40	Decreased physical activity, decreased type II muscle fiber size and amount, maintenance of type I fibers (10)	Maintenance of VO2max with exercise training, sprinting capacity is reduced
40–60	Loss of motor units accelerates (19). Decreased physical activity, increased body fatness (89), decreased androgens	Decreased aerobic and sprinting capacity even with rigorous exercise, increased body fatness, insulin resistance 204, decreased muscle protein synthesis (90)
60–70	Decreased physical activity, reduced androgen and growth factor levels (44, 91), menopause, increased total body and visceral fat (92), chronic disease, impaired appetite regulation	Inflammation (increased cytokine levels) (93), insulin resistance and type 2 diabetes (94), nutritional deficiencies (protein, vitamin D, and other micronutrients) (38), reduced muscle protein synthesis (90)
70+	Further reduction in physical activity, bouts of enforced inactivity due to illness, hospitalization depression, increased body fatness	Fear of falling, low functional capacity (95), mild cognitive impairment, inflammation and increased muscle protein breakdown (96, 97)

Table 4

Indices and prevalence of Sarcopenia.

Citation	Method	Sarcopenia Index	Reference population	Gender	N	Age (years)	Prevalence
Baumgartner et al. 1998(5)	Anthropometrics	Appendicular lean mass/ht2 m ≤ 7.26 kg/m2 f ≤ 5.45 k/m2	Rosetta study (98) (m/f 18–40 yrs)	m/f	883	61–70 71–80 ≥80	13% 24% 50%
Melton et al. 2000 (76)	DXA	Appendicular lean mass/ht2 m ≤ 7.26 kg/m2 f ≤ 5.45 k/m2	Rosetta study (98) (m/f 18–40 yrs)	m f	100 99	≥70	28% 52%
Morley et al. 2001 (70)	DXA	Appendicular lean mass/ht2 m ≤ 7.26 kg/m2 f ≤ 5.45 k/m2	Rosetta study (98) (ref.) (m/f 18–40 yrs)	m/f	199	<70 ≥80	12% 30%
Janssen et al, 2002 (71)	Bioelectrical impedance	Ratio of muscle mass/total body mass m≤31.5% f ≤22.1%	NHANES III	m f	2,224 2,278	≥60 ≥60	7% 10%
Tanko et al, 2002 (75)	DXA	Appendicular lean mass/ht2 f ≤ 5.4 k/m2	Rosetta study (98) (m/f 18–40 yrs)	f	67	≥70	12%
Ianuzzi-Sacich et al, 2002 (74)	DXA	Appendicular lean mass/ht2 m ≤ 7.26 kg/m2 f ≤ 5.45k/m2	Rosetta study (98) (m/f 18–40 yrs)	m f	142 195	≥65	27% 23%
Gillette-Guyonnet et al, 2003 (73)		Appendicular lean mass/ht2 f ≤ 5.45 k/m2	Rosetta study (98) (m/f 18–40 yrs)	f	1,321	≥75	10%
Newman et al, 2003 (18)	DXA	Appendicular lean mass/ht2 m≤ 7.23 kg/m2 f ≤ 5.67 kg/m2	Health Aging and Body Composition baseline cohort	m f	1,435 1,549	70–79	20% 20%
Castillo et al, 2004 (72)	Bioelectrical Impedance	Fat free mass m ≤ 47.9 kg f ≤ 34.7 kg	(99)(m/f 25–44)	m f	694 1,006	70–75 ≥85	4% 3% 16% 13%
Jansson et al, 2004 (100)	Bioelectrical Impedance	Total muscle mass/ht2 m ≤ 8.50 kg/m2 f ≤ 5.75 kg/m2	NHANES III	m f	2,223 2,276	≥60	11% 9%
Jansson et al, 2004(100)	Bioelectrical Impedance	Total lean mass/ht2 m ≤ 8.50kg/m2 f ≤ 5.75 kg/m2	Cardiovascular Health Study	M f	2,196 2,840	≥65	17% 11%
Schaap et al, 2006(101)	DXA	Longitudinal follow-up LASA study >3% loss of appendicular lean mass	LASA study	m f	328		15%*

longitudinal analysis with sarcopenia defined as a loss of appendicular muscle mass of >3% in three years

DXA = dual x-ray absorptiometry; f = female, m = male