Western European Journal of Medicine and Medical Science
Volume 3, Issue 10, October 2025
https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

SARCOPENIA: A LITERATURE REVIEW

Jumayev N.Sh., Sirojiddinov S.Sh., Mirzalieva A.A.
Tashkent State Medical University

Introduction

Sarcopenia is a progressive and generalized skeletal muscle disorder characterized by a loss of muscle mass, strength, and function. Initially considered an inevitable aspect of aging, it is now recognized as an independent clinical condition with significant impacts on morbidity, disability, healthcare expenditures, and mortality [1,4,5]. Given the global demographic shift toward an aging population, sarcopenia has become a critical focus in geriatric medicine, rehabilitation, and preventive healthcare.

Definition and Diagnostic Criteria

The term "sarcopenia" was first introduced by Irwin Rosenberg in 1989 [2]. Since its inception, various expert groups have proposed diagnostic criteria to facilitate the standardized identification and management of this condition. Among them, the European Working Group on Sarcopenia in Older People (EWGSOP) and the Asian Working Group for Sarcopenia (AWGS) have developed the most widely accepted frameworks [1,3].The EWGSOP places particular emphasis on muscle strength as the principal determinant for diagnosing sarcopenia, considering measures of muscle mass and physical performance as confirmatory parameters [1]. Meanwhile, the AWGS offers region-specific diagnostic criteria, taking into account variations in body composition among different populations [3]. Diagnosis of sarcopenia generally involves a comprehensive three-step evaluation. First, muscle strength is measured, most commonly using handgrip dynamometry, which provides a reliable and reproducible assessment of functional decline. Second, muscle mass is evaluated through imaging techniques such as dual-energy X-ray absorptiometry (DXA), magnetic resonance imaging (MRI), or computed tomography (CT), allowing for precise quantification of lean tissue. Third, physical performance is assessed, typically through gait speed tests or standardized batteries such as the Short Physical Performance Battery (SPPB), which help determine the severity and functional consequences of sarcopenia [1].

Illustration: Diagnostic Algorithm for Sarcopenia According to EWGSOP2

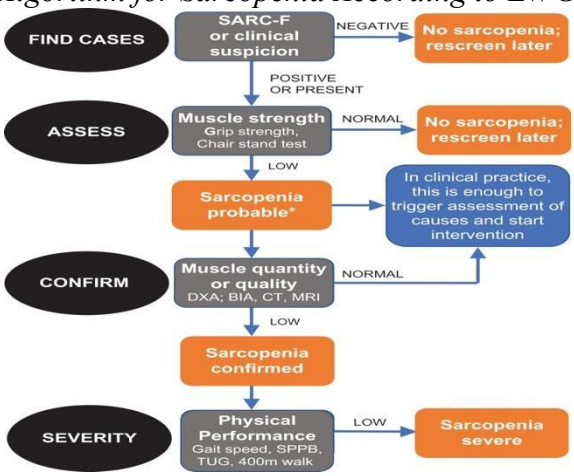

Figure 1. Diagnostic algorithm for sarcopenia proposed by the European Working Group on Sarcopenia in Older People (EWGSOP2) [1].

Western European Journal of Medicine and Medical Science
Volume 3, Issue 10, October 2025
https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

Epidemiology

Sarcopenia is a prevalent and increasingly recognized condition, particularly among older adults, with its prevalence varying depending on the diagnostic criteria, population studied, and assessment methods used. Globally, the estimated prevalence of sarcopenia in individuals aged 60 years and older ranges from **5% to 13%** in community-dwelling populations, and up to **50% or more** in hospitalized or institutionalized elderly individuals [1,4,5].

According to the revised European Working Group on Sarcopenia in Older People (EWGSOP2) criteria, population-based studies in Europe report prevalence rates of **11–20%** in older adults aged 65 and above. In contrast, studies using the Asian Working Group for Sarcopenia (AWGS) criteria in East Asian populations reveal prevalence rates of **7% to 12%** in community-dwelling older men and **5% to 10%** in older women [3].

Age is the strongest risk factor for sarcopenia. Prevalence increases progressively with advancing age:

• **10–20%** among individuals in their 60s
• **20–40%** among individuals in their 70s
• Over **50%** in adults aged 80 and above

Gender differences have also been observed. Men tend to have a higher absolute loss of muscle mass, while women often present with lower muscle strength relative to body size, leading to comparable or even higher functional impairment in women in some cohorts.

Geographic and ethnic variability affects prevalence due to differences in body composition, lifestyle, nutritional status, and healthcare systems. For instance, older adults in low- and middle-income countries may have higher prevalence rates due to increased burden of undernutrition and physical inactivity.

In **clinical settings**, sarcopenia is significantly more prevalent among patients with chronic diseases such as:

• **Cancer:** 30–70%, especially in gastrointestinal and hematologic malignancies
• **Chronic heart failure and COPD:** 20–40%
• **End-stage renal disease (ESRD):** 30–60% in dialysis patients

Sarcopenia is also increasingly recognized in **younger adults with chronic illness, obesity (sarcopenic obesity)**, and those recovering from acute illness or surgery. Its presence in these populations is associated with poor clinical outcomes, including delayed recovery, increased treatment toxicity, and higher mortality.

Given the aging global population, the absolute number of people affected by sarcopenia is expected to rise sharply, with significant implications for healthcare systems and public health planning.

Etiology and Pathophysiology

The pathogenesis of sarcopenia is multifactorial, encompassing a complex interplay of intrinsic biological aging, environmental and lifestyle influences, and a wide range of comorbid medical conditions. These factors converge on several key cellular and molecular pathways that ultimately impair muscle maintenance and regeneration.

a) Hormonal Dysregulation:

One of the central mechanisms involves age-related endocrine alterations, particularly a decline in anabolic hormones such as testosterone, growth hormone (GH), and insulin-like growth factor-1 (IGF-1). These hormones play critical roles in promoting muscle protein synthesis, satellite cell activation, and muscle hypertrophy. The reduction in their levels leads to a diminished anabolic response and contributes to sarcopenic progression. Additionally, insulin

Western European Journal of Medicine and Medical Science
Volume 3, Issue 10, October 2025
https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

resistance—common in aging and in conditions like type 2 diabetes—impairs nutrient uptake and signaling for protein synthesis within muscle cells.

b) Mitochondrial Dysfunction and Oxidative Stress:

Aging is associated with progressive mitochondrial dysfunction, leading to reduced ATP production and accumulation of reactive oxygen species (ROS). Elevated ROS levels promote oxidative damage to DNA, proteins, and lipids in muscle tissue, accelerating cellular senescence and apoptosis. Mitochondrial biogenesis is also impaired, further weakening the muscle's energy-generating capacity and regenerative potential.

c) Chronic Inflammation ("Inflammaging"):

Low-grade, chronic systemic inflammation—termed "inflammaging"—is a hallmark of biological aging and a significant contributor to sarcopenia. Pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) are elevated in older adults and exert catabolic effects on skeletal muscle. These cytokines activate ubiquitin–proteasome and autophagy–lysosome pathways, which degrade myofibrillar proteins and suppress muscle synthesis. Persistent inflammation also impairs satellite cell differentiation and muscle repair.

d) Muscle Regeneration Deficits:

Satellite cells—muscle-resident stem cells—are essential for muscle repair and hypertrophy. In sarcopenia, the number and function of these cells decline due to age-related epigenetic changes and inflammation-induced senescence. Moreover, neuromuscular junction (NMJ) instability, characterized by denervation and impaired reinnervation, leads to motor unit loss, muscle fiber atrophy, and decreased functional capacity.

e) Lifestyle and Nutritional Deficits:

Sedentary behavior and reduced physical activity are major lifestyle contributors. Resistance training normally stimulates muscle protein synthesis and counters muscle atrophy, but physical inactivity leads to disuse atrophy and reduced mechanical stimuli for hypertrophy. Nutritional deficits, especially inadequate protein intake, diminish the amino acid pool required for muscle anabolism. Inadequate intake of specific nutrients such as vitamin D, leucine, and omega-3 fatty acids further impairs muscle metabolism and repair mechanisms.

f) Comorbid Conditions:

Secondary sarcopenia arises from chronic diseases such as diabetes mellitus, chronic obstructive pulmonary disease (COPD), chronic kidney disease, and congestive heart failure. These conditions exacerbate systemic inflammation, promote catabolism, and reduce physical capacity, synergistically worsening muscle wasting. In cancer-associated sarcopenia (also overlapping with cachexia), tumor-derived cytokines and metabolic competition further accelerate muscle loss.

g) Genetic and Epigenetic Factors:

Emerging evidence suggests that genetic predispositions (e.g., polymorphisms in MSTN, ACTN3, and IGF1 genes) and epigenetic modifications (e.g., DNA methylation of muscle-specific genes) influence susceptibility to sarcopenia. These factors may determine baseline muscle mass, responsiveness to interventions, and rate of decline with age.

Clinical Consequences

Sarcopenia is associated with multiple adverse outcomes. Affected individuals exhibit a markedly increased risk of falls, fractures, and functional impairment in activities of daily living (ADLs), leading to a loss of independence and reduced quality of life [4,5]. Moreover, sarcopenia is linked to prolonged hospitalizations, increased rates of institutionalization, and

Western European Journal of Medicine and Medical Science
Volume 3, Issue 10, October 2025
https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

elevated mortality. The syndrome often coexists with frailty and cachexia, complicating diagnosis and management [5]. Additionally, sarcopenia contributes to a cycle of physical inactivity and further muscle degradation, commonly referred to as the "frailty cycle." This can lead to sarcopenic obesity—a condition characterized by the coexistence of reduced muscle mass and increased fat mass—which is associated with worsened metabolic profiles and cardiovascular risk. Cognitive decline and depressive symptoms have also been observed in individuals with sarcopenia, likely due to reduced mobility, social isolation, and systemic inflammation. The economic burden is substantial, as sarcopenia increases direct medical costs related to falls, surgeries, rehabilitation, and long-term care. A 2014 study estimated the annual healthcare cost attributable to sarcopenia in the United States alone to exceed $18 billion.Sarcopenia also negatively influences recovery from acute illnesses and surgical procedures. Older adults with sarcopenia have significantly higher rates of postoperative complications, delayed wound healing, and impaired rehabilitation outcomes. In oncology, sarcopenia is recognized as a predictor of chemotherapy toxicity, poor treatment tolerance, and shorter overall survival across several cancer types, including gastrointestinal and hematologic malignancies.

Assessment Tools

Assessment of sarcopenia incorporates both clinical and instrumental measures. Muscle strength is routinely evaluated using handgrip dynamometry, which serves as a quick and reliable screening tool [1]. Muscle mass is assessed through DXA, MRI, or CT, providing quantitative and qualitative analysis. Physical performance is measured using gait speed tests, the SPPB, or timed-up-and-go (TUG) tests [1]. Emerging research also suggests potential roles for biological markers, such as myostatin and creatinine/cystatin C ratios, in the early detection of sarcopenia [4].

Management Strategies

Management requires a comprehensive, multidisciplinary approach focused on exercise interventions, nutritional optimization, and, potentially, pharmacological therapies. Progressive resistance training remains the cornerstone of non-pharmacological management, demonstrating efficacy in increasing muscle mass, strength, and functional capacity [4,5]. Concurrent aerobic exercise may confer additional benefits. Nutritional strategies emphasize adequate protein intake (1.2–1.5 g/kg/day) and supplementation with leucine, vitamin D, and omega-3 fatty acids to support anabolic processes and attenuate inflammation [5].Pharmacological options are currently under investigation, including selective androgen receptor modulators (SARMs), myostatin inhibitors, and anti-inflammatory agents [5]. Until robust evidence emerges, pharmacotherapy remains adjunctive to lifestyle modification. Implementation of multidisciplinary care, involving physicians, dietitians, physiotherapists, and rehabilitation specialists, is essential to optimize outcomes [5].

Recent Advances and Future Directions

Recent advancements have highlighted the potential role of the gut-muscle axis in the pathogenesis of sarcopenia, suggesting that modulation of the gut microbiome could offer novel therapeutic strategies [4]. The concept of sarcopenia as a marker of biological aging rather than merely chronological aging is gaining attention, with implications for personalized interventions [5]. The use of wearable technologies and digital health tools for continuous monitoring of physical activity and muscle parameters is anticipated to facilitate earlier diagnosis and individualized treatment approaches. Genomic and epigenomic studies are

Western European Journal of Medicine and Medical Science
Volume 3, Issue 10, October 2025
https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

beginning to elucidate individual susceptibilities, paving the way for precision medicine approaches in the prevention and management of sarcopenia [4,5].

Conclusion

Sarcopenia constitutes a significant clinical and public health challenge with profound implications for older adults' functionality and survival. Early identification, targeted intervention, and a multidisciplinary approach are crucial for mitigating its effects. Continued research into molecular mechanisms, diagnostic innovations, and personalized therapeutic strategies holds promise for improving patient outcomes. Integrating sarcopenia awareness and management into routine clinical practice and medical education is imperative to promote healthy aging at the population level [1,5].

References

1. Cruz-Jentoft, A. J., Bahat, G., Bauer, J., et al. (2019). Sarcopenia: Revised European Consensus on Definition and Diagnosis. *Age and Ageing*, 48(1), 16–31. https://doi.org/10.1093/ageing/afy169
2. Rosenberg, I. H. (1989). Summary comments. *American Journal of Clinical Nutrition*, 50(5), 1231–1233.
3. Chen, L. K., Woo, J., Assantachai, P., et al. (2020). Asian Working Group for Sarcopenia: 2019 Consensus Update. *Journal of the American Medical Directors Association*, 21(3), 300–307.e2. https://doi.org/10.1016/j.jamda.2019.12.012
4. Rolland, Y., Czerwinski, S., Abellan Van Kan, G., et al. (2008). Sarcopenia: Its Assessment, Etiology, Pathogenesis, Consequences and Future Perspectives. *The Journal of Nutrition, Health & Aging*, 12(7), 433–450.
5. Landi, F., Calvani, R., Tosato, M., et al. (2016). Sarcopenia: An Overview on Current Definitions, Diagnosis and Treatment. *Current Protein & Peptide Science*, 17(7), 598–608.
6. Alfonso J Cruz-Jentoft 1, Gülistan Bahat 2, Jürgen Bauer 3, Yves Boirie 4, Olivier Bruyère 5, Tommy Cederholm 6, Cyrus Cooper 7, Francesco Landi 8, Yves Rolland 9, Avan Aihie Sayer 10, Stéphane M Schneider11, Cornel C Sieber 12, Eva Topinkova 13, Maurits Vandewoude 14, Marjolein Visser 15, Mauro Zamboni 16; Writing Group for the European Working Group on Sarcopenia in Older People 2 (EWGSOP2), and the Extended Group for EWGSOP2. Sarcopenia: revised European consensus on definition and diagnosis. PMCID: PMC6322506 EMSID: EMS80409 PMID: 30312372