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# SARCOPENIA – CURRENT KNOWLEDGE AND CLINICAL CHALLENGES

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**ABSTRACT**

**Introduction:** Sarcopenia, defined as the progressive loss of muscle mass and strength, is one of the primary threats to the functional independence of older adults. With global population aging and an increasing burden of chronic diseases, the clinical relevance of this condition continues to grow. Despite rising awareness, sarcopenia remains underdiagnosed and undertreated, leading to a decline in quality of life, a higher risk of falls, hospitalizations, complications, and increased mortality.
**Aim of the Study:** The aim of this study is to provide a comprehensive review of current knowledge on sarcopenia, including its definition, pathophysiology, epidemiology, risk factors, diagnostic criteria, and therapeutic and preventive strategies.
**Methodology:** A literature search was conducted using PubMed, Google Scholar, and ScienceDirect for publications from 2005 to 2025. The review included peer-reviewed studies, clinical trials, systematic reviews, and international guidelines (EWGSOP2, ICFSR, GLIS) related to the definition, diagnosis, and management of sarcopenia. Only peer-reviewed articles published in English were included, while non-peer-reviewed materials, conference abstracts, and case reports without broader applicability were excluded.
**Conclusions:** Sarcopenia is a complex and increasingly prevalent condition that requires early diagnosis and a comprehensive, multidisciplinary approach. Effective management relies primarily on resistance training, adequate protein intake, and correction of nutritional deficiencies, while pharmacological options remain limited. Particular attention should be directed to secondary sarcopenia, and routine screening in at-risk populations is essential to reduce functional decline and adverse health outcomes.

**KEYWORDS**

Sarcopenia, Aging, Muscle Loss, Geriatrics, Prevention, Diagnosis, Physical Activity

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## 1. Introduction

The global aging of societies constitutes one of the most significant challenges for contemporary medicine and healthcare systems. According to projections by the World Health Organization (WHO), by 2050 the number of people aged ≥ 60 years will increase to 2 billion, accounting for nearly one-fifth of the world's population [1]. With the extension of average life expectancy, the burden of chronic diseases is also growing, significantly affecting not only survival but also patients 'quality of life. Among the key yet still under-recognized problems is sarcopenia - a progressive loss of muscle mass and strength, which is a crucial contributor to the decline in functional capacity among older adults [2].

Sarcopenia was officially recognized as a disease entity in 2016 and assigned the ICD-10-CM code M62.84 [3]. Current clinical definitions - especially the harmonized framework recommended by the European Working Group on Sarcopenia in Older People 2 (EWGSOP2) – emphasize that diagnosing this condition requires identifying reduced muscle strength, confirmed by low muscle mass, and, in advanced stages, also decreased physical performance [2,3]. The etiology of sarcopenia is multifactorial, involving the interplay of biological aging mechanisms (such as immunosenescence or chronic low-grade inflammation referred to as "inflammaging") and external factors, including inactivity, nutritional deficiencies, polypharmacy, and chronic diseases such as diabetes, COPD, or cancer [4].

Epidemiological data indicate that in the general population over 60 years of age, sarcopenia affects 10% to 16% of individuals, whereas in groups with chronic diseases the prevalence may reach 50–60% [5]. Modifiable risk factors - such as sedentary lifestyle, smoking, and poor diet – are identified as critical determinants of increased disease risk. The presence of sarcopenia significantly elevates the risk of falls, hospitalizations, postoperative complications, loss of independence, and premature death [2,5]. In response to this growing problem, the International Conference on Frailty and Sarcopenia Research (ICFSR) published

recommendations in 2023, emphasizing the necessity of early detection and treatment of sarcopenia as a vital element of geriatric prevention strategies [5].

The aim of this article is to provide a comprehensive and up-to-date review of the current knowledge on sarcopenia -including its definition and classification, pathophysiological mechanisms, epidemiology, risk factors, diagnostic approaches, and therapeutic and preventive strategies. The article emphasizes the practical implementation of international guidelines and highlights the potential for integrated care approaches aimed at improving function and quality of life in older adults.

## 2. Methodology

This review was conducted through an extensive search of the scientific literature to synthesize current knowledge and clinical evidence regarding sarcopenia. Relevant publications were identified using PubMed, Google Scholar, and ScienceDirect databases, covering the period from 2005 to 2025. The search strategy aimed to include the most recent and methodologically robust evidence, encompassing systematic reviews, meta-analyses, randomized clinical trials, and international consensus guidelines issued by leading expert groups such as EWGSOP2, ICFSR, and GLIS.

The search terms combined the keywords “sarcopenia,” “muscle mass,” “aging,” “diagnosis,” and “treatment.” Articles were screened for relevance to the definition, epidemiology, pathophysiology, diagnostic criteria, and management strategies of sarcopenia, including both preventive and therapeutic approaches. Only peer-reviewed studies published in English were included to ensure scientific rigor and accessibility.

Sources such as non-peer-reviewed materials, conference abstracts, narrative commentaries, and case reports lacking broader clinical applicability were excluded. The final selection of studies was based on methodological quality, clarity of results, and clinical relevance. Findings from the selected literature were analyzed comparatively to identify consistent trends, emerging concepts, and gaps in current knowledge.

## 3. Results

### 3.1. Definition and Classification of Sarcopenia

Sarcopenia is currently recognized as a skeletal muscle disease characterized by progressive loss of muscle mass, strength, and quality, regardless of the patient's age, although it is most commonly diagnosed in the elderly population [2]. In 2016, the World Health Organization officially included sarcopenia in the International Classification of Diseases - ICD-10-CM under the code M62.84 [6], which laid the foundation for recognizing it as a distinct disease entity rather than a general geriatric phenomenon.

A turning point in clarifying the definition of sarcopenia was the publication by the European Working Group on Sarcopenia in Older People (EWGSOP2) in 2018-2019, which introduced a standardized, four-stage diagnostic framework [2,7]: (a) screening using the SARC-F questionnaire, (b) assessment of muscle strength (primarily grip strength), (c) measurement of muscle mass, and (d) determination of disease severity based on physical performance. The EWGSOP2 definition specifies that reduced muscle strength (dynapenia) is the first and principal diagnostic criterion (defining probable sarcopenia), followed by confirmation through low muscle mass (confirmed sarcopenia), and impaired physical function – such as slow gait speed or poor chair rise test results – qualifies the condition as severe sarcopenia [2,8].

The EWGSOP2 criteria include specific cutoff points: handgrip strength <27 kg for men and <16 kg for women, while low muscle mass is defined as ASM/height² <7.0 kg/m² in men and <5.5 kg/m² in women [6]. For physical performance, a gait speed <0.8 m/s or poor results in the Short Physical Performance Battery (SPPB) are considered indicators of severe sarcopenia [2,9].

Despite adoption and implementation across many countries, the definition of sarcopenia continues to evolve. In 2023, the Global Leadership Initiative in Sarcopenia (GLIS) launched an effort to establish a global consensus aimed at unifying diagnostic criteria and facilitating therapeutic recommendations worldwide [8]. In the same year, the International Clinical Advisory Forum on Sarcopenia Research (ICAFSR) emphasized the need to distinguish between individual components: primary loss of muscle mass (myopenia), strength (dynapenia), and power (kratopenia) in the definitional framework - a move that may enhance diagnostic accuracy in special populations such as patients with chronic kidney disease [4,10].

Currently, the three-tier model proposed by EWGSOP2 remains clinically and epidemiologically validated [2], yet the development of new GLIS definitions and efforts to tailor criteria for specific populations underscore how dynamic and complex this field continues to be.

### 3.2. Epidemiology and Clinical Significance

Sarcopenia represents a significant health concern in aging societies, as its prevalence increases with age. In the general population aged 60–70, sarcopenia is observed in 5% to 13% of individuals, while among people over the age of 80, this prevalence may reach 11-50% [1]. Epidemiological projections suggest that over 50 million people worldwide currently suffer from sarcopenia, and this number may rise to more than 200 million in the next four decades [1].

Among patients with chronic diseases, the problem becomes even more severe. In studies involving over 68,000 patients with heart failure, the prevalence of sarcopenia averaged 31%, with rates as high as 28–35% in those with reduced ejection fraction (HFrEF) [11]. Furthermore, sarcopenia in these patients increases the risk of poor clinical outcomes (HR 1.64; 1.20-5.25), with the risk rising to 2.77 among those with HFrEF [11].

Sarcopenia also contributes to more severe clinical complications. For instance, osteosarcopenia (coexistence of sarcopenia and osteoporosis) increases mortality risk by approximately 50% (RR 1.53; 1.28-1.78) in elderly populations [12]. Moreover, the number of falls and related injuries among sarcopenic individuals is 1.5 to 2 times higher compared to individuals without this condition [13]. Clinical consequences also include increased risk of fractures, especially femoral neck fractures, reduced mobility, earlier institutionalization, and higher healthcare costs [1,13].

There is growing evidence linking sarcopenia to overall mortality. Meta-analyses of population-level mortality data indicate that sarcopenia nearly doubles the risk of death (HR ≈ 1.87) [14]. In outpatient studies from Korea, functional sarcopenia was associated with an elevated mortality risk (HR 1.78), while confirmed sarcopenia had a hazard ratio of 1.51 [1].

Furthermore, hospitalizations significantly accelerate the deterioration of muscle condition. According to a systematic review from 2025, hospital-associated sarcopenia affects 14% to 75% of patients, with low muscle mass and reduced physical activity being key prognostic factors [15]. Acute muscle mass loss during hospitalization further increases the risk of disability.

Given these epidemiological and clinical associations, sarcopenia must not be underestimated. Its prevalence, impact on motor function, increased risk of falls, fractures, hospitalizations, and poorer outcomes in chronic diseases—as well as its association with higher mortality-justify the need for systematic diagnostic and therapeutic measures in routine geriatric care.

### 3.3. Pathophysiology and Molecular Mechanisms of Sarcopenia

Sarcopenia is a condition with a complex and multifactorial pathophysiology involving interactions among molecular, hormonal, inflammatory, and metabolic processes. A key role in its development is played by age-related degeneration of motor units, chronic low-grade inflammation (so-called "inflammaging"), altered secretion of anabolic hormones, insulin resistance, oxidative stress, and mitochondrial dysfunction [16,17].

One of the earliest observable phenomena is the progressive loss of type II (fast-twitch) muscle fibers, which directly contributes to decreased muscle strength. The degeneration and denervation of motor units, combined with an insufficient regenerative response by satellite cells, lead to their gradual atrophy. Furthermore, aging of skeletal muscle is associated with a decline in the proliferative capacity of satellite cells, primarily due to altered expression of cell cycle–related genes and transcription factors such as Pax7 and MyoD [18].

Another critical mechanism is chronic low-grade inflammation that intensifies with age. Proinflammatory cytokines, including TNF-α, IL-6, and CRP, not only directly suppress muscle protein synthesis but also activate degradation pathways, such as the ubiquitin-proteasome system and autophagy. TNF-α, in particular, activates IKKβ kinase and the NF-κB pathway, increasing transcription of genes encoding key muscle-specific E3 ligases like MuRF1 and atrogin-1, which are responsible for targeted muscle protein breakdown [19,20].

Hormonal axis alterations are also essential. With age, there is a physiological decline in anabolic hormones such as testosterone, growth hormone (GH), and insulin-like growth factor 1 (IGF-1). IGF-1 is particularly important for muscle development and regeneration, as it activates the PI3K–AKT–mTOR pathway, which promotes muscle protein synthesis and inhibits degradation. Impaired activity of this pathway leads to reduced muscle mass and strength [21]. Additionally, insulin resistance impairs glucose uptake in muscle tissue and stimulates catabolic processes, further exacerbating muscle degradation [22].

Mitochondrial dysfunction and oxidative stress represent another vital axis in the pathogenesis of sarcopenia. Aging is associated with a decrease in both mitochondrial number and function, leading to lower ATP production and increased generation of reactive oxygen species (ROS). These ROS damage proteins,

lipids, and DNA and activate pathways leading to muscle cell apoptosis [23]. Moreover, aging mitochondria exhibit defective autophagy and biogenesis, which further compromises their function.

Nutritional deficiencies also play an important role, particularly insufficient intake of protein and vitamin D. Vitamin D deficiency impairs skeletal muscle function by affecting the vitamin D receptor (VDR) present in muscle tissue, reducing protein synthesis and accelerating degenerative processes. Studies confirm that low serum vitamin D levels correlate with decreased muscle strength and poorer physical performance in older adults [24].

At the molecular level, sarcopenia results from a disrupted balance between muscle protein anabolism and catabolism, deterioration of the cellular and hormonal microenvironment, and persistent low-grade inflammation. Understanding these mechanisms is essential not only for prevention but also for developing effective pharmacological and non-pharmacological treatments for sarcopenia.

### 3.4. Risk Factors and Comorbidities

Sarcopenia, as a condition of multifactorial etiology, is closely associated with both non-modifiable factors, such as age and sex, and modifiable ones - including lifestyle, chronic diseases, malnutrition, and systemic inflammation. Understanding the broad spectrum of risk factors is essential for developing effective prevention and treatment strategies.

Age remains the primary independent risk factor. As early as the age of 30, a slow physiological decline in muscle mass is observed, accelerating after the age of 60, and reaching a loss of 3-5% per year among older adults [25]. This phenomenon is exacerbated by immunosenescence, defined as age-related weakening of the immune response, and “inflammaging” – a chronic, low-grade inflammatory state that disrupts muscle homeostasis [26].

Female sex is associated with a higher risk of sarcopenia, particularly after menopause. Estrogens play an essential role in muscle metabolism, and their deficiency contributes to reduced muscle mass and strength [27]. In men, the decline in testosterone is particularly significant, leading to reduced muscle protein synthesis and a loss of type II muscle fibers [28].

Nutritional deficiencies are also significant contributors. Both malnutrition and insufficient dietary protein intake result in negative nitrogen balance and enhanced muscle protein catabolism. Additionally, low levels of vitamin D and antioxidants such as vitamins C and E have been shown to negatively affect muscle function [29].

Physical activity plays a fundamental role in sarcopenia prevention. A sedentary lifestyle and lack of regular resistance exercise significantly accelerate the loss of muscle mass. Studies have demonstrated that even short-term immobilization (e.g., hospitalization) can lead to a marked decline in muscle strength and function, especially in older adults [30].

Sarcopenia is frequently associated with chronic diseases, which may act as both cause and consequence. Among the most commonly described conditions are: type 2 diabetes, chronic obstructive pulmonary disease (COPD), chronic kidney disease, heart failure, cancer, and endocrine disorders (including hypothyroidism and hypogonadism) [31].

In type 2 diabetes, insulin resistance disrupts anabolic signaling pathways in muscle, leading to degradation. Furthermore, hyperglycemia increases oxidative stress and amplifies inflammation. Patients with COPD often experience reduced physical activity, hypoxemia, and frequent use of corticosteroids – all of which contribute to muscle catabolism [32]. In heart and kidney failure, chronic inflammation and hormonal imbalances also promote the progression of sarcopenia.

Polypharmacy should also be highlighted as a relevant risk factor. The use of glucocorticosteroids, statins, chemotherapeutic agents, or diuretics may contribute to the development or progression of sarcopenia through catabolic effects or impaired muscle metabolism [33].

In summary, the risk factors for sarcopenia encompass a wide array of biological and environmental mechanisms. Their multi-layered interactions necessitate a holistic approach to both prevention and treatment.

### 3.5. Diagnosis of Sarcopenia

The diagnosis of sarcopenia is a crucial component of both prevention and treatment, enabling early identification of changes and the implementation of appropriate therapeutic interventions. Due to the multidimensional nature of sarcopenia-including both structural components (muscle mass loss) and functional deficits (decline in muscle strength and performance)—the diagnostic process should be based on a comprehensive approach aligned with current recommendations from scientific societies such as the European Working Group on Sarcopenia in Older People 2 (EWGSOP2) and the International Clinical Practice Guidelines for Sarcopenia (ICFSR).

According to the 2019 EWGSOP2 consensus, sarcopenia should be diagnosed in three stages: (1) screening for risk, (2) confirmation of sarcopenia, and (3) assessment of its severity [2]. The primary diagnostic criterion is muscle strength, currently considered the most sensitive and relevant marker of muscle function. A decrease in strength prompts further evaluation to confirm sarcopenia.

The most commonly used method to assess muscle strength is handgrip strength measurement with a dynamometer. The thresholds suggestive of sarcopenia are <27 kg for men and <16 kg for women [2]. Alternatively, lower limb strength can be assessed using the five-time chair stand test. A time >15 seconds indicates decreased leg muscle strength.

If muscle weakness is confirmed, the next step involves evaluating muscle quantity and quality to confirm the diagnosis. Preferred methods include:

-DXA (Dual-energy X-ray Absorptiometry), considered the gold standard, allowing precise measurement of appendicular skeletal muscle mass (ASM). Sarcopenia is confirmed when ASM is <7.0 kg/m² in men and <5.5 kg/m² in women [34].

-BIA (Bioelectrical Impedance Analysis), which is easier to use but less accurate, can also be applied to estimate ASM.

- In some clinical or oncological cases, CT or MRI may also be used.

Once low muscle mass is confirmed, physical performance must be assessed to determine disease severity. The most common method is measuring gait speed over a 4-meter distance—a result <0.8 m/s suggests severe sarcopenia [2]. Alternative tests include the Short Physical Performance Battery (SPPB), the Timed Up and Go (TUG), or the 6-Minute Walk Test (6MWT).

Screening tools have gained attention in recent years for identifying patients at high risk of sarcopenia before significant loss of function occurs. The SARC-F questionnaire, consisting of five questions concerning strength, rising from a chair, walking, stair climbing, and falls, is most commonly used. A score ≥4 suggests the need for further diagnostics [36]. Other tools include the Ishii Score, the CalF test (calf circumference), and the SARC-CalF tool, which combines the SARC-F questionnaire with calf circumference measurement and is considered more sensitive than SARC-F alone [37].

Importantly, sarcopenia diagnostics should not be limited to biomechanical measurements alone. Increasingly, clinical studies assess molecular biomarkers such as myokines (e.g., myostatin, irisin), inflammatory proteins (CRP, IL-6), or muscle-related metabolites (e.g., serum and urinary creatinine) [38]. While these markers are not yet used in routine clinical practice, they hold promise as future tools for monitoring sarcopenia and therapeutic responses.

In summary, sarcopenia diagnosis requires a multi-step approach-from simple screening tools, through precise measurements of muscle strength and mass, to evaluations of physical performance. Early detection and regular monitoring of muscle status in at-risk populations are key to implementing timely therapeutic strategies and improving patient quality of life.

### 3.6. Non-Pharmacological Treatment of Sarcopenia

Therapeutic management of sarcopenia, in accordance with current recommendations from scientific societies, should be multidimensional and individualized, taking into account the etiology, stage of disease progression, and comorbid conditions. Non-pharmacological treatment methods represent the primary pillar of intervention and should be implemented in all diagnosed cases of sarcopenia-regardless of its underlying cause. The main strategies include lifestyle modifications through appropriately selected physical activity, optimization of protein and energy intake, supplementation of nutrients, and education of the patient and their caregivers.

The strongest scientific evidence supports the effectiveness of resistance training as a key component in improving muscle strength and mass. Regular strength exercises lead to the activation of anabolic pathways in skeletal muscle, increased expression of growth factors such as IGF-1, and inhibition of the myostatin

pathway—a natural inhibitor of muscle development [39]. Training programs should be individualized, progressive, and supervised by physiotherapy or rehabilitation specialists. The optimal frequency is 2–3 sessions per week, each lasting 30–60 minutes, including exercises targeting large muscle groups with a load of approximately 60–80% of one-repetition maximum (1RM) [40].

In addition to resistance training, aerobic exercises (e.g., brisk walking, stationary cycling) should also be implemented, as they improve aerobic capacity and cardiovascular function. Balance and coordination exercises are an important complement, as they reduce the risk of falls-particularly in older patients and those with neurological deficits [41].

The second key component of non-pharmacological treatment is nutritional intervention, aimed at increasing the intake of high-quality protein and energy. Observational and randomized controlled studies have demonstrated that protein intake of ≥1.2 g/kg body weight/day is associated with a reduced risk of sarcopenia and improvements in muscle strength and mass, especially when combined with physical activity [42]. Recommended sources include proteins of high biological value, such as eggs, lean meats, fish, milk, and fermented dairy products. A beneficial effect has also been shown for whey protein, which is rich in leucine-an amino acid particularly important for muscle protein synthesis.

In cases of malnutrition or increased energy requirements, oral nutritional supplements (ONS) containing proteins, carbohydrates, fats, and micronutrients may be indicated [43]. In clinical practice, it is also important to assess and, if necessary, correct vitamin D deficiencies, as low levels are associated with poorer muscle function and a higher risk of falls. Randomized studies have shown that vitamin D3 supplementation (800–2000 IU/day) improves muscle strength and reduces the risk of sarcopenia, especially in individuals with baseline vitamin D deficiency [44].

Complementary to therapy are educational strategies-both regarding physical activity and nutrition-as well as environmental interventions, such as modification of home conditions (e.g., removal of architectural barriers, installation of handrails), which improve patient safety and independence. In some cases, intervention by a multidisciplinary team may be necessary, including a clinical dietitian, physiotherapist, geriatrician, clinical pharmacist, and psychologist.

In summary, non-pharmacological treatment of sarcopenia should serve as the starting point for all therapeutic interventions. Early implementation of individually tailored physical exercise, optimization of protein intake, and supplementation of nutritional deficiencies are strategies with well-documented effectiveness, recommended by international scientific societies as first-line management.

### 3.7. Pharmacological Treatment of Sarcopenia

Although non-pharmacological treatment remains the primary therapeutic strategy in sarcopenia, recent years have seen growing interest in complementing it with pharmacotherapy, particularly in cases of severe or rapidly progressing loss of muscle mass and strength. Currently, there is no single approved drug specifically indicated for the treatment of sarcopenia; however, a number of agents are under clinical investigation or are used off-label. Pharmacological therapies can be classified into several main groups: anabolic hormones, androgen receptor modulators, anti-catabolic agents, as well as pharmacological supplements and metabolic drugs.

One of the most thoroughly studied therapeutic approaches involves the use of testosterone and its analogs. Numerous studies have demonstrated that testosterone positively affects lean body mass and muscle strength in men with androgen deficiency, especially in older age [45]. However, testosterone therapy is associated with adverse effects such as prostate hypertrophy, fluid retention, and increased cardiovascular risk, which limits its widespread application [46].

To minimize the adverse effects of classical androgens, selective androgen receptor modulators (SARMs) have been developed, such as enobosarm (ostarine). In Phase III clinical trials (including the POWER trials), enobosarm demonstrated a beneficial impact on increasing muscle mass and improving physical function, without the typical side effects associated with testosterone therapy [47]. Despite these promising results, these agents have not yet been approved by the FDA for the treatment of sarcopenia, and their long-term safety remains incompletely understood.

Another area of interest includes drugs that target the myostatin and activin pathways, which play a key role in inhibiting muscle growth. Neutralization of myostatin activity through monoclonal antibodies (e.g., bimagrumab – anti-activin type II receptor) in Phase II trials has shown an increase in muscle mass, although without significant improvement in physical function [48]. Further studies are ongoing to optimize this class of drugs, particularly in the context of sarcopenia secondary to chronic diseases.

As part of supportive therapy - especially in malnourished patients-vitamin D is also used, as its deficiency may impair muscle function and contribute to sarcopenia. Supplementation with vitamin D, in doses of 800-2000 IU/day, has shown moderate efficacy in improving muscle strength in individuals with baseline deficiency [49]. Creatine, a popular dietary supplement, supports ATP resynthesis in muscle cells, and its supplementation may enhance strength gains when combined with resistance training-particularly in older adults [50].

Other compounds currently under investigation include metformin, which may improve muscle metabolism by modulating the AMPK pathway, as well as ACE inhibitors, which may positively influence muscle function and reduce chronic inflammation associated with sarcopenia in older individuals [51].

All of the aforementioned pharmacological interventions are considered adjunctive and should be evaluated only in the context of well-documented sarcopenia, following the optimization of non-pharmacological measures. Moreover, further large-scale randomized trials are necessary to definitively confirm the efficacy and safety of these therapies in the geriatric population.

### 3.8. Prevention of Sarcopenia

The prevention of sarcopenia is gaining particular importance in the context of an aging population and the absence of clearly effective pharmacological treatments for this condition. The primary goal of preventive measures is to maintain muscle mass and function at the highest possible level throughout life, particularly in old age. Preventive interventions should be initiated as early as possible-ideally in middle age-and include both non-pharmacological strategies (physical activity, diet) and supportive measures, such as supplementation and the management of chronic diseases.

The cornerstone of sarcopenia prevention remains regular physical activity, especially resistance exercises, which have the strongest impact on stimulating muscle protein synthesis (MPS) and counteracting protein degradation [52]. Consistent strength training, performed at least 2-3 times per week, not only increases muscle strength but also improves functional parameters such as gait speed, balance, and the ability to perform daily activities [53]. Aerobic exercises are equally important, as they have a beneficial effect on muscle metabolism, mitochondrial function, and insulin sensitivity-disturbances of which are recognized as key risk factors for sarcopenia [54].

A second fundamental component of prevention is proper nutrition, including adequate protein intake. According to current ESPEN and PROT-AGE guidelines, older adults should consume at least 1.0-1.2 g of protein per kilogram of body weight per day, and up to 1.5 g/kg/day in individuals at risk of sarcopenia [42]. Equally important is the even distribution of protein intake throughout the day, as well as the presence of branched-chain amino acids in meals-particularly leucine, which exerts a strong anabolic effect on muscle cells through activation of the mTOR pathway [43].

In the prevention of sarcopenia, increasing attention is being paid to the role of vitamins and trace elements—especially vitamin D, deficiency of which is found in as many as 70-80% of older adults in Poland and other Northern European countries. Vitamin D supplementation may not only improve muscle strength, but also reduce the risk of falls and fractures, which are common complications of sarcopenia [55].

Maintaining a healthy body weight is another key preventive factor. Both malnutrition and overweight (particularly abdominal obesity) can contribute to the development of sarcopenia. The phenomenon of "sarcopenic obesity”-the coexistence of excess adipose tissue with loss of muscle mass-is particularly hazardous due to the high pro-inflammatory potential of visceral fat, which accelerates muscle degradation through cytokines (e.g., TNF-α, IL-6) [22].

In addition to these classical interventions, other preventive strategies also deserve attention. A well-documented positive impact on preserving muscle function is associated with the reduction of polypharmacy and optimization of the treatment of chronic diseases, such as diabetes, COPD, heart failure, or depression-all of which can lead to secondary sarcopenia [56]. Increasingly, the importance of regular monitoring of functional status and the use of screening tools, such as SARC-F, is being recognized. These tools allow for the identification of at-risk individuals before the development of full-blown sarcopenia [57].

Systematic reviews and meta-analyses indicate that effective sarcopenia prevention can delay the loss of functional independence, reduce hospitalization rates, and significantly improve quality of life and overall survival [58]. However, a multidisciplinary approach is essential-one that includes physicians, dietitians, physiotherapists, and nurses-to allow for comprehensive assessment and preventive interventions tailored to the individual needs of the patient.

### 3.9. Secondary Sarcopenia – Special Clinical Cases

Secondary sarcopenia develops as a result of chronic diseases, metabolic disorders, cancer, immobilization, or the use of certain medications. In contrast to primary sarcopenia, which is mainly age-related, secondary sarcopenia can also affect younger individuals. Its course is often more rapid and associated with a higher risk of complications. Due to its diverse etiology, secondary sarcopenia poses a particular diagnostic and therapeutic challenge in clinical practice.

One of the best-understood mechanisms leading to secondary sarcopenia is that associated with malignant tumors, particularly in the context of cancer cachexia. In such cases, muscle mass loss occurs even with preserved or increased caloric intake and results from intensified catabolism induced by pro-inflammatory cytokines (IL-6, TNF-α, IFN-γ) and altered hormonal responses [59]. Sarcopenia in oncology patients correlates with poorer treatment response, increased chemotherapy toxicity, and higher mortality rates, regardless of disease stage [60].

Another example is sarcopenia occurring in chronic heart failure (CHF). In this context, muscle hypoperfusion, chronic inflammation, and hormonal axis dysfunction (reduced levels of testosterone, DHEA, GH/IGF-1) combine to cause progressive loss of muscle mass and strength [61]. Studies indicate that clinically significant sarcopenia is present in 20–30% of CHF patients, significantly impacting prognosis and quality of life [62].

Chronic obstructive pulmonary disease (COPD) is another condition frequently coexisting with sarcopenia. In COPD patients, mechanisms contributing to muscle loss include chronic hypoxemia, excessive oxidative stress, inflammation, and limited physical activity due to dyspnea [63]. In this patient group, sarcopenia is associated with more frequent disease exacerbations, increased hospitalization rates, and poorer performance on functional tests (e.g., the 6-minute walk test, 6MWT).

Renal diseases, particularly chronic kidney disease (CKD) in stages 3-5, also play an important role in the development of secondary sarcopenia. In these patients, the accumulation of uremic toxins, chronic inflammation, metabolic acidosis, and hormonal disturbances (decreased GH, IGF-1, testosterone) contribute to skeletal muscle catabolism [64]. Sarcopenia occurs in up to 40–60% of dialysis patients and is associated with increased mortality and hospitalization risk.

It is also important to emphasize the role of drug-induced sarcopenia-especially in the context of glucocorticoids, statins, mTOR inhibitors (e.g., everolimus), and certain anticancer agents. Prolonged exposure to these substances can cause muscle wasting through direct effects on muscle protein degradation pathways (e.g., activation of the ubiquitin–proteasome system and enhanced autophagy) [36].

Sarcopenia may also accompany neurodegenerative diseases such as Parkinson's disease and Alzheimer's disease. In these cases, decreased physical activity, nutritional disturbances, and impaired motor function and balance further contribute to a vicious cycle of declining physical capacity and progressive sarcopenia [65].

All of the above clinical scenarios require heightened diagnostic vigilance. A diagnosis of secondary sarcopenia should always prompt an assessment of nutritional status, muscle strength, and physical function, followed by the implementation of a comprehensive treatment strategy-including modification of the underlying disease treatment, nutritional optimization, and rehabilitation.

## 4. Conclusions

Sarcopenia represents one of the key health challenges in today's aging societies. Although it was long regarded merely as a physiological consequence of aging, it is now recognized as a distinct disease entity with significant clinical, epidemiological, and social implications. An increasing body of scientific evidence confirms that sarcopenia adversely affects not only quality of life but also patient survival, particularly in populations burdened by chronic diseases.

Based on a review of current literature and scientific society guidelines, it can be clearly stated that the proper diagnosis of sarcopenia requires a multi-step diagnostic approach. Early detection of reduced muscle strength is of key importance and should serve as the first warning sign-preceding the loss of muscle mass and functional decline. Modern diagnostic techniques such as dual-energy X-ray absorptiometry (DEXA) and bioelectrical impedance analysis (BIA) allow for objective assessment of body composition and are invaluable in determining disease severity.

A detailed analysis of the pathomechanisms of sarcopenia reveals that it is not a linear process-it involves numerous molecular pathways, hormonal disturbances, inflammatory states, and environmental factors. Changes in satellite cells, oxidative stress, mitochondrial dysfunction, and neurodegeneration are just

some of the mechanisms underlying the progressive loss of muscle mass and strength. It is also important to highlight risk factors such as malnutrition, physical inactivity, multimorbidity, and polypharmacy, which exacerbate the course of sarcopenia and hinder its treatment.

Sarcopenia management should be based on an interdisciplinary approach that incorporates both non-pharmacological and pharmacological interventions. Resistance training, adequate protein intake, and vitamin D supplementation constitute the cornerstone of therapy, with their efficacy confirmed in numerous clinical trials. Although current pharmacological options remain limited, ongoing research into novel drugs-including myostatin pathway modulators and selective androgen receptor modulators (SARMs)-offers hope for more effective future treatments.

Special attention should be paid to secondary sarcopenia, which develops in the course of chronic diseases such as cancer, chronic heart failure, COPD, and kidney disease. In these cases, treatment should be individualized and tailored to the patient's overall condition and therapeutic possibilities.

Equally important are preventive measures-promotion of an active lifestyle, nutritional education, and early interventions in at-risk populations. Implementing routine screening for muscle function into clinical practice, especially among older adults, may significantly reduce the long-term consequences of sarcopenia.

In summary, sarcopenia is a multidimensional clinical problem that requires a comprehensive diagnostic and therapeutic strategy. Improved understanding of molecular mechanisms, early diagnosis, and integrated interventions may significantly enhance patient quality of life and reduce the functional and economic burdens of this condition. In the face of an aging population, continued research into more effective methods of prevention, diagnosis, and treatment of sarcopenia, as well as the integration of these solutions into routine clinical practice, is essential.

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## REFERENCES

1. Yuan, S., & Larsson, S. C. (2023). Epidemiology of sarcopenia: Prevalence, risk factors, and consequences. *Metabolism, 144*, 155533. https://doi.org/10.1016/j.metabol.2023.155533
2. 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
3. Dent, E., Morley, J. E., Bauer, J. M., et al. (2018). International clinical practice guidelines for sarcopenia (ICFSR): Screening, diagnosis, and management. *The Journal of Nutrition, Health and Aging, 22*(10), 1148–1161. https://doi.org/10.1007/s12603-018-1139-9
4. Kirk, B., Cawthon, P. M., Arai, H., et al. (2024). The conceptual definition of sarcopenia: Delphi consensus from the Global Leadership Initiative in Sarcopenia (GLIS). *Age and Ageing, 53*(3). https://doi.org/10.1093/ageing/afae052
5. Morawin, B., Tylutka, A., Bielewicz, F., & Zembron-Lacny, A. (2023). Diagnostics of inflammaging in relation to sarcopenia. *Frontiers in Public Health, 11*, 1162385. https://doi.org/10.3389/fpubh.2023.1162385
6. Anker, S. D., Morley, J. E., & von Haehling, S. (2016). Welcome to the ICD-10 code for sarcopenia. *Journal of Cachexia, Sarcopenia and Muscle, 7*(4), 512–514. https://doi.org/10.1002/jcsm.12147
7. Sanchez-Tocino, M. L., Cigarran, S., Urena, P., et al. (2024). Definition and evolution of the concept of sarcopenia. *Nefrologia (English Edition), 44*(3), 323–330. https://doi.org/10.1016/j.nefroe.2023.08.007
8. Coletta, G., & Phillips, S. M. (2023). An elusive consensus definition of sarcopenia impedes research and clinical treatment: A narrative review. *Ageing Research Reviews, 86*, 101883. https://doi.org/10.1016/j.arr.2023.101883
9. Beaudart, C., Alcazar, J., Aprahamian, I., et al. (2025). Health outcomes of sarcopenia: A consensus report by the outcome working group of the Global Leadership Initiative in Sarcopenia (GLIS). *Aging Clinical and Experimental Research, 37*(1), 100. https://doi.org/10.1007/s40520-025-02995-9

10. Sayer, A. A., & Cruz-Jentoft, A. J. (2022). Sarcopenia definition, diagnosis and treatment: Consensus is growing. *Age and Ageing, 51*(10), afac220. https://doi.org/10.1093/ageing/afac220
11. Chen, R., Xu, J., Wang, Y., et al. (2023). Prevalence of sarcopenia and its association with clinical outcomes in heart failure: An updated meta-analysis and systematic review. *Clinical Cardiology, 46*(3), 260–268. https://doi.org/10.1002/clc.23970
12. de Sire, A., Calafiore, D., Curci, C., et al. (2024). Osteosarcopenia and risk of falls: A concise review. *International Journal of Bone Fragility, 4*(2), 63–66. https://doi.org/10.57582/IJBF.240402.063
13. Wen, Q., Chen, X., Luo, S., et al. (2024). Association between sarcopenia grade and fall history among older adults in West China: A retrospective study. *BMJ Open, 14*(2), e080426. https://doi.org/10.1136/bmjopen-2023-080426
14. Liu, P., Hao, Q., Hai, S., et al. (2017). Sarcopenia as a predictor of all-cause mortality among community-dwelling older people: A systematic review and meta-analysis. *Maturitas, 103*, 16–22. https://doi.org/10.1016/j.maturitas.2017.04.007
15. Cho, M. R., Lee, S., & Song, S. K. (2022). A review of sarcopenia: Pathophysiology, diagnosis, treatment and future direction. *Journal of Korean Medical Science, 37*(18), e146. https://doi.org/10.3346/jkms.2022.37.e146
16. Shafiee, G., Keshtkar, A., Soltani, A., et al. (2017). Prevalence of sarcopenia in the world: A systematic review and meta-analysis of general population studies. *Journal of Diabetes & Metabolic Disorders, 16*, 21. https://doi.org/10.1186/s40200-017-0302-x
17. Walston, J. D. (2012). Sarcopenia in older adults. *Current Opinion in Rheumatology, 24*(6), 623–627. https://doi.org/10.1097/BOR.0b013e328358d59b
18. García-Prat, L., Sousa-Victor, P., & Muñoz-Cánoves, P. (2013). Functional dysregulation of stem cells during aging: A focus on skeletal muscle stem cells. *The FEBS Journal, 280*(17), 4051–4062. https://doi.org/10.1111/febs.12221
19. Li, Y. P., Chen, Y., John, J., et al. (2005). TNF-α acts via p38 MAPK to stimulate expression of the ubiquitin ligase atrogin-1/MAFbx in skeletal muscle. *The FASEB Journal, 19*(3), 362–370. https://doi.org/10.1096/fj.04-2364com
20. Sandri, M. (2008). Signaling in muscle atrophy and hypertrophy. *Physiology, 23*, 160–170. https://doi.org/10.1152/physiol.00041.2007
21. Glass, D. J. (2003). Signaling pathways that mediate skeletal muscle hypertrophy and atrophy. *Nature Cell Biology, 5*(2), 87–90. https://doi.org/10.1038/ncb0203-87
22. Zamboni, M., Mazzali, G., Fantin, F., et al. (2008). Sarcopenic obesity: A new category of obesity in the elderly. *Nutrition, Metabolism and Cardiovascular Diseases, 18*(5), 388–395. https://doi.org/10.1016/j.numecd.2007.10.002
23. Joseph, A. M., Adhihetty, P. J., Buford, T. W., et al. (2012). The impact of aging on mitochondrial function and biogenesis pathways in skeletal muscle of sedentary high- and low-functioning elderly individuals. *Aging Cell, 11*(5), 801–809. https://doi.org/10.1111/j.1474-9726.2012.00844.x
24. Ceglia, L. (2008). Vitamin D and skeletal muscle tissue and function. *Molecular Aspects of Medicine, 29*(6), 407–414. https://doi.org/10.1016/j.mam.2008.07.002
25. Mitchell, W. K., Williams, J., Atherton, P., et al. (2012). Sarcopenia, dynapenia, and the impact of advancing age on human skeletal muscle size and strength: A quantitative review. *Frontiers in Physiology, 3*, 260. https://doi.org/10.3389/fphys.2012.00260
26. Ferrucci, L., & Fabbri, E. (2018). Inflammageing: Chronic inflammation in ageing, cardiovascular disease, and frailty. *Nature Reviews Cardiology, 15*(9), 505–522. https://doi.org/10.1038/s41569-018-0064-2
27. Greising, S. M., Baltgalvis, K. A., Lowe, D. A., & Warren, G. L. (2009). Hormone therapy and skeletal muscle strength: A meta-analysis. *The Journals of Gerontology Series A, 64*(10), 1071–1081. https://doi.org/10.1093/gerona/glp082
28. Morley, J. E., Perry, H. M., Kaiser, F. E., et al. (1993). Effects of testosterone replacement therapy in old hypogonadal males: A preliminary study. *Journal of the American Geriatrics Society, 41*(2), 149–152. https://doi.org/10.1111/j.1532-5415.1993.tb02049.x
29. Welch, A. A. (2014). Nutritional influences on age-related skeletal muscle loss. *Proceedings of the Nutrition Society, 73*(1), 16–33. https://doi.org/10.1017/S0029665113003698
30. Kortebein, P., Ferrando, A., Lombeida, J., et al. (2007). Effect of 10 days of bed rest on skeletal muscle in healthy older adults. *JAMA, 297*(16), 1769–1774. https://doi.org/10.1001/jama.297.16.1772-b
31. Wilkinson, D. J., Piasecki, M., & Atherton, P. J. (2018). The age-related loss of skeletal muscle mass and function: Measurement and physiology of muscle fibre atrophy and muscle fibre loss in humans. *Ageing Research Reviews, 47*, 123–132. https://doi.org/10.1016/j.arr.2018.07.005
32. Benz, E., Trajanoska, K., Lahousse, L., et al. (2019). Sarcopenia in COPD: A systematic review and meta-analysis. *European Respiratory Review, 28*(154), 190049. https://doi.org/10.1183/16000617.0049-2019
33. Thoma, A., & Lightfoot, A. P. (2018). NF-κB and inflammatory cytokine signalling: Role in skeletal muscle atrophy. In *Muscle Atrophy* (pp. 267–279). https://doi.org/10.1007/978-981-13-1435-3_12
34. Buckinx, F., Landi, F., Cesari, M., et al. (2018). Pitfalls in the measurement of muscle mass: A need for a reference standard. *Journal of Cachexia, Sarcopenia and Muscle, 9*(2), 269–278. https://doi.org/10.1002/jcsm.12268

35. Kim, T. N., & Choi, K. M. (2014). The implications of sarcopenia and sarcopenic obesity on cardiometabolic disease. *Journal of Cellular Biochemistry, 116*(7), 1171–1178. https://doi.org/10.1002/jcb.25077
36. Malmstrom, T. K., & Morley, J. E. (2013). SARC-F: A simple questionnaire to rapidly diagnose sarcopenia. *Journal of the American Medical Directors Association, 14*(8), 531–532. https://doi.org/10.1016/j.jamda.2013.05.018
37. Barbosa-Silva, T. G., Menezes, A. M. B., Bielemann, R. M., et al. (2016). Enhancing SARC-F: Improving its performance for screening sarcopenia. *Journal of the American Medical Directors Association, 17*(12), 1136–1141. https://doi.org/10.1016/j.jamda.2016.08.004
38. Carmeli, E. (2020). Sarcopenia in older adults. In *Background and management of muscular atrophy* (pp. 273–288). https://doi.org/10.5772/intechopen.93418
39. Liao, C. D., Tsauo, J. Y., Wu, Y. T., et al. (2017). Effects of resistance exercise combined with protein supplementation on body composition and physical function in older adults: A systematic review and meta-analysis. *The American Journal of Clinical Nutrition, 106*(4), 1078–1091. https://doi.org/10.3945/ajcn.116.143594
40. Fragala, M. S., Cadore, E. L., Dorgo, S., et al. (2019). Resistance training for older adults: Position statement from the National Strength and Conditioning Association. *Journal of Strength and Conditioning Research, 33*(8), 2019–2052. https://doi.org/10.1519/JSC.0000000000003230
41. Sherrington, C., Michaleff, Z. A., Fairhall, N., et al. (2017). Exercise to prevent falls in older adults: An updated systematic review and meta-analysis. *British Journal of Sports Medicine, 51*(24), 1750–1758. https://doi.org/10.1136/bjsports-2016-096547
42. Bauer, J., Biolo, G., Cederholm, T., et al. (2013). Evidence-based recommendations for optimal dietary protein intake in older people: A position paper from the PROT-AGE Study Group. *Journal of the American Medical Directors Association, 14*(8), 542–559. https://doi.org/10.1016/j.jamda.2013.05.021
43. Deutz, N. E., Bauer, J. M., Barazzoni, R., et al. (2014). Protein intake and exercise for optimal muscle function with aging: Recommendations from the ESPEN Expert Group. *Clinical Nutrition, 33*(6), 929–936. https://doi.org/10.1016/j.clnu.2014.04.007
44. Gielen, E., O'Neill, T. W., Pye, S. R., et al. (2015). Endocrine determinants of incident sarcopenia in middle-aged and elderly European men. *Journal of Cachexia, Sarcopenia and Muscle, 6*(3), 242–252. https://doi.org/10.1002/jcsm.12030
45. Srinivas-Shankar, U., Roberts, S. A., Connolly, M. J., et al. (2010). Effects of testosterone on muscle strength, physical function, body composition, and quality of life in intermediate-frail and frail elderly men: A randomized, double-blind, placebo-controlled study. *The Journal of Clinical Endocrinology & Metabolism, 95*(2), 639–650. https://doi.org/10.1210/jc.2009-1251
46. Khoo, T. K. (2010). Adverse events associated with testosterone administration. *The New England Journal of Medicine, 363*(19), 1865–1867. https://doi.org/10.1056/NEJMc1009326
47. Dalton, J. T., Barnette, K. G., Bohl, C. E., et al. (2011). The selective androgen receptor modulator GTx-024 (enobosarm) improves lean body mass and physical function in healthy elderly men and postmenopausal women: Results of a double-blind, placebo-controlled phase II trial. *Journal of Cachexia, Sarcopenia and Muscle, 2*(3), 153–161. https://doi.org/10.1007/s13539-011-0034-6
48. Rooks, D., Swan, T., Goswami, B., et al. (2020). Bimagrumab vs optimized standard of care for treatment of sarcopenia in older adults: A randomized clinical trial. *JAMA Network Open, 3*(10), e2020836. https://doi.org/10.1001/jamanetworkopen.2020.20836
49. Stockton, K. A., Mengersen, K., Paratz, J. D., Kandiah, D., & Bennell, K. L. (2011). Effect of vitamin D supplementation on muscle strength: A systematic review and meta-analysis. *Osteoporosis International, 22*(3), 859–871. https://doi.org/10.1007/s00198-010-1407-y
50. Candow, D. G., & Chilibeck, P. D. (2008). Timing of creatine or protein supplementation and resistance training in the elderly. *Applied Physiology, Nutrition, and Metabolism, 33*(1), 184–190. https://doi.org/10.1139/H07-139
51. Onder, G., Penninx, B. W. J. H., Balkrishnan, R., Fried, L. P., Chaves, P. H. M., Williamson, J. D., et al. (2002). Relation between use of angiotensin-converting enzyme inhibitors and muscle strength and physical function in older women: An observational study. *The Lancet, 359*(9310), 926–930. https://doi.org/10.1016/S0140-6736(02)08024-8
52. Peterson, M. D., Rhea, M. R., Sen, A., & Gordon, P. M. (2010). Resistance exercise for muscular strength in older adults: A meta-analysis. *Ageing Research Reviews, 9*(3), 226–237. https://doi.org/10.1016/j.arr.2010.03.004
53. Liu, C. J., & Latham, N. K. (2009). Progressive resistance strength training for improving physical function in older adults. *Cochrane Database of Systematic Reviews*, (3), CD002759. https://doi.org/10.1002/14651858.CD002759.pub2
54. Steffl, M., Bohannon, R. W., Sontakova, L., et al. (2017). Relationship between sarcopenia and physical activity in older people: A systematic review and meta-analysis. *Clinical Interventions in Aging, 12*, 835–845. https://doi.org/10.2147/CIA.S132940
55. Bischoff-Ferrari, H. A., Dawson-Hughes, B., Staehelin, H. B., et al. (2009). Fall prevention with supplemental and active forms of vitamin D: A meta-analysis of randomized controlled trials. *BMJ, 339*, b3692. https://doi.org/10.1136/bmj.b3692

56. Landi, F., Calvani, R., Tosato, M., et al. (2015). Sarcopenia as the biological substrate of physical frailty. *Clinics in Geriatric Medicine, 31*(3), 367–374. https://doi.org/10.1016/j.cger.2015.04.005
57. Woo, J., Leung, J., & Morley, J. E. (2014). Validating the SARC-F: A suitable community screening tool for sarcopenia? *Journal of the American Medical Directors Association, 15*(9), 630–634. https://doi.org/10.1016/j.jamda.2014.04.021
58. Tieland, M., Trouwborst, I., & Clark, B. C. (2018). Skeletal muscle performance and ageing. *Journal of Cachexia, Sarcopenia and Muscle, 9*(1), 3–19. https://doi.org/10.1002/jcsm.12238
59. Baracos, V. E., Martin, L., Korc, M., Guttridge, D. C., & Fearon, K. C. H. (2018). Cancer-associated cachexia. *Nature Reviews Disease Primers, 4*, 17105. https://doi.org/10.1038/nrdp.2017.105
60. Shachar, S. S., Williams, G. R., Muss, H. B., & Nishijima, T. F. (2016). Prognostic value of sarcopenia in adults with solid tumours: A meta-analysis and systematic review. *European Journal of Cancer, 57*, 58–67. https://doi.org/10.1016/j.ejca.2015.12.030
61. Collimate, A., Marzetti, E., Calvani, R., et al. (2016). Sarcopenia in heart failure: Mechanisms and therapeutic strategies. *Journal of Geriatric Cardiology, 13*(7), 615–624. https://doi.org/10.11909/j.issn.1671-5411.2016.07.004
62. Fülster, S., Tacke, M., Sandek, A., et al. (2013). Muscle wasting in patients with chronic heart failure: Results from the Studies Investigating Co-morbidities Aggravating Heart Failure (SICA-HF). *European Heart Journal, 34*(7), 512–519. https://doi.org/10.1093/eurheartj/ehs381
63. Bernard, S., LeBlanc, P., Whittom, F., et al. (1998). Peripheral muscle weakness in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine, 158*(2), 629–634. https://doi.org/10.1164/ajrccm.158.2.9711023
64. Isoyama, N., Qureshi, A. R., Avesani, C. M., et al. (2014). Comparative associations of muscle mass and muscle strength with mortality in dialysis patients. *Clinical Journal of the American Society of Nephrology, 9*(10), 1720–1728. https://doi.org/10.2215/CJN.10261013
65. Cai, Y., Feng, F., Wei, Q., et al. (2021). Sarcopenia in patients with Parkinson's disease: A systematic review and meta-analysis. *Frontiers in Neurology, 12*, 598035. https://doi.org/10.3389/fneur.2021.598035