

OPTIMIZING NUTRITIONAL INTAKE TO MITIGATE FRAILTY : A NARRATIVE REVIEW ON DIET, MUSCLE LOSS, AND PHYSICAL VULNERABILITY IN OLDER ADULTS

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ABSTRAK

Frailty merupakan sindrom klinis geriatri yang ditandai oleh penurunan cadangan fisiologis dan ketahanan terhadap stresor, yang menyebabkan peningkatan kerentanan terhadap disabilitas, meningkatnya lama rawat inap, dan kematian. Faktor nutrisi memainkan peran krusial dalam patogenesis dan progresivitas sindrom ini. Asupan protein yang tidak adekuat, defisiensi mikronutrien, dan perubahan komposisi tubuh, khususnya kehilangan massa otot rangka dan peningkatan lemak viseral, berkontribusi pada gangguan fungsi otot dan sarcopenia. Proses inflamasi kronis dan stres oksidatif memperparah kerusakan otot, mempercepat penurunan fungsi fisik. Tinjauan literatur ini bertujuan untuk mengkaji hubungan antara asupan nutrisi, status gizi, komposisi tubuh, dan kelemahan fisik pada lansia. Pencarian artikel dilakukan secara sistematis pada database PubMed, Scopus, dan Google Scholar untuk publikasi antara Januari 2020 hingga Desember 2024. Temuan menunjukkan bahwa intervensi nutrisi, terutama peningkatan asupan protein dan pola makan antiinflamasi, berpotensi mempertahankan massa otot dan menurunkan risiko frailty. Selain itu, pemantauan dini terhadap status gizi dan komposisi tubuh sangat penting dalam upaya preventif. Heterogenitas metodologis dan variasi definisi menjadi tantangan dalam penerapan klinis. Oleh karena itu, integrasi penilaian gizi, evaluasi fungsional, dan pendekatan interdisipliner diperlukan untuk mengoptimalkan strategi pencegahan dan penatalaksanaan frailty pada populasi lanjut usia.

Kata kunci : asupan nutrisi, frailty, komposisi tubuh, lansia, status gizi

ABSTRACT

Frailty is a clinical syndrome characterized by reduced physiological reserve and resistance to stressors, contributing to increased vulnerability in older adults. Inadequate nutritional intake accelerates systemic inflammation, oxidative stress, and metabolic imbalance, impairing muscle regeneration and worsening physical weakness. Low protein intake, micronutrient deficiencies, and body composition changes such as skeletal muscle loss and visceral fat accumulation serve as central contributors to frailty pathogenesis. This review analyzes the relationship between nutritional intake, nutritional status, body composition, and the risk of physical weakness in the elderly. Articles were retrieved through a structured search of PubMed, Scopus, and Google Scholar for publications between January 2020 and December 2024. Selected studies addressed indicators of nutritional status, dietary protein patterns, fat distribution, and physical manifestations including gait speed, grip strength, and functional capacity. Findings indicate that protein-based and anti-inflammatory dietary interventions may help preserve muscle mass and physical function. Early monitoring of body composition and nutritional status provides a strategic approach to delay frailty progression. Variability in terminology and assessment methods remains a major obstacle in the standardization of clinical interventions for aging populations.

Keywords : body composition, frailty, nutritional intake, nutritional status, older adults

INTRODUCTION

Frailty syndrome is a multidimensional clinical condition marked by reduced physiological reserve and diminished resistance to stressors, leading to increased vulnerability in older adults. It is characterized by unintentional weight loss, weakness, exhaustion,

slowness, and low physical activity, which collectively predict adverse outcomes such as falls, prolonged hospitalization, disability, and mortality (Wylie et al., 2022; Chen et al., 2022). Its prevalence increases significantly with age and is strongly associated with sarcopenia and chronic diseases (Kalache et al., 2019; Kalideen et al., 2022). The underlying mechanisms involve chronic low-grade inflammation, mitochondrial dysfunction, hormonal imbalance, and metabolic dysregulation, all contributing to impaired homeostasis and functional decline (Di Giosia et al., 2022; Cruz-Jentoft et al., 2017).

Malnutrition plays a central role in the development of frailty. Inadequate energy and protein intake impair muscle protein synthesis and accelerate sarcopenia (Phillips et al., 2016; Ganapathy & Nieves, 2020). Age-related factors such as loss of appetite, impaired gastrointestinal function, diminished taste and smell, dental problems, cognitive decline, and polypharmacy further contribute to poor dietary intake (Cox et al., 2020; Engelheart et al., 2021). Micronutrient deficiencies, including vitamins D and B12 and antioxidants, exacerbate oxidative stress and muscle dysfunction, increasing frailty risk (Troesch et al., 2020; Sanchez-Garcia et al., 2024). Changes in body composition, particularly the loss of lean muscle mass and increase in visceral fat, are closely linked to the severity of frailty. Sarcopenic obesity represents a high-risk phenotype in which reduced muscle mass coexists with increased fat mass, leading to impaired physical performance (Polito et al., 2022; Santoso et al., 2025).

Evidence from bioelectrical impedance and DXA studies supports the association between altered body composition and physical impairments such as slow gait, poor balance, and weak grip strength—parameters central to frailty diagnostic tools like the Fried phenotype and the frailty index (Ruslim et al., 2024; Santoso et al., 2024). Understanding the connection between nutritional intake, body composition, and frailty is crucial for developing effective interventions. Nutritional strategies emphasizing adequate protein, essential amino acids, and anti-inflammatory dietary patterns—such as the Mediterranean diet—may reduce frailty risk and preserve physical function in older adults (Shanahan et al., 2017; Yaghi et al., 2023; Mazza et al., 2024).

This review analyzes the relationship between nutritional intake, nutritional status, body composition, and the risk of physical weakness in the elderly.

METHODS

This narrative literature review aimed to synthesize current evidence on the role of nutritional intake in frailty syndrome among older adults, emphasizing nutritional status, body composition, and physical weakness. A structured literature search was conducted using PubMed, Scopus, and Google Scholar for articles published between January 2020 and December 2024. Search terms included combinations of “frailty,” “older adults,” “nutrition,” “nutritional status,” “dietary intake,” “body composition,” and “physical weakness,” using Boolean operators (AND/OR) to refine results. Manual screening of reference lists was also performed to identify additional relevant studies. The review included original research articles in English involving human participants aged ≥ 60 years that addressed nutritional intake or nutritional status in relation to frailty or its physical components. Both observational and interventional studies were considered. Excluded were reviews, editorials, conference abstracts, animal studies, and articles without full-text access. Study selection was based on relevance to the review objective, particularly those assessing dietary intake, anthropometry, or biochemical nutritional markers linked to frailty components such as sarcopenia and reduced muscle strength. Studies using validated methods for body composition were prioritized. Findings were synthesized thematically into three domains: nutritional status, body composition, and physical frailty.

RESULT AND DISCUSSION

Interconnection Between Malnutrition and Frailty Pathogenesis

Malnutrition plays a central role in accelerating the onset and progression of frailty syndrome in older adults. The convergence of chronic low-grade inflammation, oxidative stress, and multiple nutritional deficiencies contributes to a progressive decline in physiological resilience, resulting in vulnerability to external stressors and functional deterioration. Frailty pathogenesis is intricately linked to systemic inflammatory responses, often characterized by elevated levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and C-reactive protein (CRP). These pro-inflammatory mediators promote catabolic processes in skeletal muscle, suppress protein synthesis, and impair regenerative capacity, leading to muscle atrophy and reduced physical strength (Di Giosia, Stamerra, Giorgini, Jamialahamdi, Butler, & Sahebkar, 2022; Polito, Barnaba, Ciarapica, & Azzini, 2022).

Inadequate intake of energy, protein, and essential micronutrients exacerbates the inflammatory state and disrupts metabolic homeostasis. Deficiencies in vitamins D, B12, and E, as well as trace elements such as zinc and selenium, impair cellular antioxidant defense mechanisms, intensify mitochondrial dysfunction, and elevate oxidative stress. Reactive oxygen species (ROS) generated under these conditions cause structural and functional damage to proteins, lipids, and DNA, further compromising muscle integrity and neuromuscular function. These biochemical alterations contribute to the loss of muscle mass, reduced gait speed, and diminished endurance, which are key phenotypic components of frailty (Di Giosia et al., 2022; Cruz-Jentoft, Kiesswetter, Drey, & Sieber, 2017).

Additionally, malnutrition reduces the availability of substrates required for tissue repair, immune competence, and hormonal regulation. Hypoalbuminemia and low serum prealbumin levels, commonly observed in frail individuals, reflect both poor nutritional intake and ongoing catabolic activity. The resulting imbalance between anabolic and catabolic signaling pathways favors sarcopenia and impairs recovery capacity following acute illness or hospitalization. This creates a self-perpetuating cycle in which nutritional deficits and frailty reinforce each other, accelerating functional decline. Collectively, the interplay between chronic inflammation, oxidative stress, and nutritional inadequacy constitutes a biologically plausible mechanism underlying frailty progression in older adults (Polito et al., 2022; Cox, Morrison, Ibrahim, Robinson, Sayer, & Roberts, 2020).

Protein Intake and Muscle Preservation in Aging

Adequate protein intake is essential for maintaining skeletal muscle mass and function in older adults, particularly in the prevention of sarcopenia and frailty-related physical decline. Muscle protein synthesis (MPS) becomes progressively less responsive to dietary protein in aging individuals, a phenomenon known as anabolic resistance. Overcoming this resistance requires both sufficient total protein intake and optimal distribution across meals, with particular emphasis on high-quality protein sources rich in essential amino acids, especially leucine. Leucine activates the mammalian target of rapamycin complex 1 (mTORC1) signaling pathway, which serves as a central regulator of protein translation and muscle hypertrophy (Bunchorntavakul & Reddy, 2020; Phillips, Chevalier, & Leidy, 2016).

Aging is associated with decreased expression and sensitivity of anabolic signaling mediators, including insulin-like growth factor 1 (IGF-1), which further impairs MPS. In this context, protein ingestion stimulates the mTORC1 pathway through both IGF-1-dependent and independent mechanisms, enhancing the phosphorylation of downstream effectors such as p70S6 kinase and 4E-BP1. These molecular processes promote ribosomal biogenesis and the initiation of mRNA translation, ultimately increasing the synthesis of contractile proteins necessary for muscle maintenance. Repeated stimulation of these pathways through dietary

protein contributes to muscle preservation and may offset the catabolic effects of aging and inflammation (Phillips et al., 2016; O'Connell, Coppinger, & McCarthy, 2020).

Moreover, adequate protein intake supports mitochondrial biogenesis and function, which are critical for muscle endurance and resistance to fatigue. Branched-chain amino acids (BCAAs) serve as substrates for energy production during physical activity and facilitate recovery from muscular stress. In addition to their anabolic effects, amino acids also exert anti-inflammatory actions by modulating nuclear factor kappa-light-chain-enhancer of activated B cells (NF- κ B) activity and reducing the expression of pro-inflammatory cytokines. These effects are particularly relevant in frail individuals who often exhibit elevated systemic inflammation. Integrating adequate protein intake into dietary strategies for older adults may therefore serve a dual function: stimulating muscle anabolism through mTORC1-dependent signaling and reducing inflammation-induced catabolism. When combined with resistance-based physical activity, protein supplementation has been shown to further enhance muscle mass and strength, thereby attenuating the progression of frailty (Phillips et al., 2016; Engelheart, Forslund, Brummer, & Ljungqvist, 2021).

Body Composition Alteration and Frailty Risk

Alterations in body composition, particularly the concurrent decline in lean body mass and accumulation of adipose tissue, represent a core component of frailty development in older adults. Sarcopenia, defined as the progressive and generalized loss of skeletal muscle mass and strength, directly impairs mobility, postural control, and metabolic function. Clinically, reduced lean mass has been associated with decreased gait speed, impaired balance, and increased risk of falls, all of which are key physical manifestations of frailty. The decline in muscle quality, reflected in reduced contractility and infiltration of intramuscular fat, further diminishes functional performance and accelerates dependence in activities of daily living (Kosaka, Nakao, Goto, Umezaki, & Ohnishi, 2022; Jyväkorpi, Urtamo, Kivimäki, & Strandberg, 2020).

In parallel, increased fat mass, especially visceral adiposity, contributes to a pro-inflammatory and insulin-resistant metabolic state that exacerbates frailty pathogenesis. Adipose tissue, particularly in central depots, acts as an active endocrine organ that secretes adipokines such as leptin, resistin, and adiponectin. Excess adiposity alters the balance of these adipokines, promoting chronic low-grade inflammation and oxidative stress, which impair muscle regeneration and metabolic flexibility. This adipose-driven inflammatory response amplifies catabolic pathways within skeletal muscle and compromises anabolic signaling, accelerating the decline in muscle mass and strength (Santoso, Destra, & Firmansyah, 2025; Ruslim, Destra, Gunaidi, & Yulishaputra, 2024).

The coexistence of reduced muscle mass and elevated fat mass, a condition referred to as sarcopenic obesity, represents a high-risk phenotype for adverse outcomes. Individuals with sarcopenic obesity experience greater limitations in physical function, higher rates of hospitalization, and increased mortality compared to those with either sarcopenia or obesity alone. From a clinical perspective, conventional anthropometric measures such as body mass index (BMI) may fail to detect these compositional abnormalities, necessitating the use of more precise tools such as dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA) for accurate assessment of muscle and fat compartments. Recognition of body composition changes as integral to frailty underscores the need for targeted interventions. Strategies that preserve lean mass while minimizing fat accumulation through nutritional optimization, resistance training, and modulation of inflammatory processes are essential for maintaining functional independence in aging populations (Santoso, Setiawan, & Wijaya, 2024; Ruslim, Santoso, Soeltanong, & Soebrata, 2024).

Clinical Implications for Screening and Intervention

Findings from current evidence underscore the critical need for early screening and comprehensive intervention strategies to address frailty in older adults. Nutritional factors, including inadequate protein intake, micronutrient deficiencies, and altered body composition, contribute significantly to the development and progression of frailty and must be prioritized in clinical assessments. Integrating nutritional screening tools into routine geriatric evaluation can identify individuals at risk before overt physical decline occurs (Ruiz-Margáin, Macías-Rodríguez, Flores-García, Román Calleja, Fierro-Angulo, & González-Regueiro, 2024; Durlach, Tripoz-Dit-Masson, Massé-Deragon, Subtil, Niasse-Sy, & Herledan, 2024).

Early detection of body composition abnormalities, particularly loss of skeletal muscle mass and the presence of visceral adiposity, provides a valuable window for preventive intervention. Techniques such as bioelectrical impedance analysis or DXA enable the objective quantification of muscle and fat compartments and can guide tailored management approaches. Monitoring changes in grip strength, gait speed, and lower limb function offers additional insight into musculoskeletal resilience and should be incorporated into frailty assessments (Durlach et al., 2024; Mazza, Ferro, Maurotti, Micale, Boragina, & Russo, 2024). From a nutritional intervention standpoint, optimizing protein intake remains central to preserving muscle mass and delaying frailty-related deterioration. Older adults may benefit from dietary plans emphasizing high-quality protein sources, evenly distributed across meals, combined with adequate intake of vitamin D, B12, and antioxidants. Nutritional support is most effective when integrated with resistance-based exercise programs, which synergistically stimulate

CONCLUSION

Malnutrition, reduced protein intake, and altered body composition significantly contribute to frailty in older adults. Chronic inflammation, oxidative stress, and anabolic resistance impair muscle synthesis and accelerate sarcopenia. Adequate protein intake, micronutrient sufficiency, and preservation of lean mass are essential to maintain physical function and delay frailty progression. Early detection through nutritional and functional screening, combined with individualized interventions, is critical for optimizing outcomes. Comprehensive management involving dietary support and physical activity can reduce frailty burden and promote independence. A multidisciplinary approach is needed to integrate nutritional strategies into geriatric care and preserve physiological reserve during aging.

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