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Loss of Physical Performance, Strength, and Muscle in Sarcopenia

Ivan Cavero*

Castilla-La Mancha University, Health and Social Research Center, Cuenca, Spain

Abstract

A characteristic of ageing, sarcopenia is described as the loss of muscular mass, strength, and physical performance. It is usually linked to altered amino acid metabolism, increased muscle protein catabolism in comparison to anabolism, and muscle fiber loss. Sarcopenia can coexist with obesity [sarcopenic obesity (SO)], or it might be linked to a general decrease of body mass. Sarcopenia has been demonstrated to contribute to poor surgical results, increased chemotherapy toxicity associated with both cytotoxic and targeted drugs, as well as negatively affecting survival in patients with malignant illness; however its effects may even be more severe in these individuals. While sarcopenia development is a common age-related phenomenon, the catabolic processes that go along with it seem to be encouraged by physical inactivity, poor nutrition, and systemic low-grade inflammation, in addition to intrinsic muscle and molecular changes, such as mitochondrial dysfunction and impaired muscle stem cell regenerative capacity. Although many older cancer patients do not meet the recommendations for physical activity and nutrition, and cancer treatment can make it more challenging to make positive lifestyle changes, increased physical activity and an adequate protein intake can reduce the incidence and severity of sarcopenia in cancer patients. The detrimental clinical effects of sarcopenia in older people, particularly older cancer patients, are explored. Along with recommendations for therapies, reviews of lifestyle, molecular, and cellular variables that affect sarcopenia and its concomitant consequences are also included.

Keywords: Sarcopenia; Muscle mass; Aging; Obesity; Sarcopenic obesity; Cancer

Introduction

A gradual, all-encompassing muscular condition called sarcopenia is defined by a loss of muscle mass, a reduction in muscle strength, and a change in the composition of the muscles [1-5]. Most frequently, it is linked to age, although it can also be caused by inflammatory and degenerative conditions such rheumatoid arthritis, renal failure, and congestive heart failure [1-5]. Sarcopenia is frequently linked to negative outcomes such deteriorating fragility, fractures, falls, physical impairment, and death. Sarcopenia often manifests as weight loss, however it can also coexist with obesity, in which case the condition is known as sarcopenic obesity (SO). Sarcopenia is more common in elderly cancer patients, who also have a poorer prognosis overall and worse surgical results as well as greater cytotoxicity from chemotherapy. In order to enhance outcomes in older persons, particularly those receiving treatment for malignant illnesses, targeted therapies to lessen the frequency and severity of sarcopenia and related comorbidities are unquestionably required.

Several systemic etiologic factors, such as altered energy balance, mitochondrial dysfunction, oxidative stress, immune cell alterations leading to Senescence Associated Secretory Phenotypes (SASP), extracellular matrix alterations [6], and increased fat mass, are linked to age-dependent muscle catabolic processes that cause sarcopenia. These factors can all contribute to chronic low grade inflammation, which can affect all body systems and organs, but particularly skeletal muscle. Muscle atrophy f-box protein (MAF-box) and muscle ubiquitin ring finger-1 ligase (MURF-1) are upregulated as a result of these pro-inflammatory processes, which also include SASP, increased fat mass, and altered extracellular matrix. This results in the ubiquitination and degradation of muscle protein. CRP, IL-1, IL-6, and TNF are examples of chronic low-grade inflammatory indicators that are elevated throughout the natural ageing process (inflammaging).

Additionally, oxidative stress brought on by mitochondrial malfunction can cause DNA breakage, myofibrillar degradation, and caspase 3 release. Reactive oxygen species (ROS) that are formed by the peroxidation of polyunsaturated fatty acids and circulate in the body have been found to be a reliable indicator of linked oxidative stress, enlarged fat mass, and related meta inflammatory conditions. T cell P16INK4A has been discovered as a senescence marker and suggestive of the SASP, which is characterized by several pro-inflammatory cytokines secreted and mitotic arrest (Muss et al., companion paper, in detail). It has been demonstrated that each of the aforementioned factors influences muscle catabolism, decreased muscle protein synthesis, and enhanced muscle protein degradation patterns both alone and jointly [7].

Further muscle mass loss is caused by the cancers and its treatment's enhanced inflammatory response and metabolic changes [8]. In addition, being inactive is associated with a loss of lean body mass, and receiving chemotherapy is associated with an increase in fat mass, all of which affect the ratio of lean to fat mass. While it is true that many of these markers are upregulated during the normal ageing process [9], their increased association with disease may speed up the ageing process, causing functional decline, age-related comorbidities like sarcopenia, and contribute to inter-individual variation in tolerance and response to cancer therapy [10].

Along with the alterations mentioned above, age-related reductions in testosterone and dehydroepiandrosterone sulphate (DHEAS) in both men and women have also been linked to the development of sarcopenia [11]. Numerous clinical studies on older people using DHEAS or testosterone supplements, frequently in conjunction with

***Corresponding author:** Ivan Cavero, Castilla-La Mancha University, Health and Social Research Center, Cuenca, Spain, E-mail: ivancav89@hotmail.com

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exercise, have demonstrated positive benefits on muscle mass but variable effects on muscular strength and function. There was a negative cardiovascular impact linked to testosterone administration research in several of these investigations. Studies are still being conducted to better methods of enhancing hormone supplementation to stop and slow the onset of sarcopenia while preventing cardiovascular events [11].

Numerous investigations have been started to discover the genes that predispose to sarcopenia on the basis of observational studies, particularly twin studies, that show heritability of muscle mass and muscular strength phenotypes [12]. The vitamin D receptor gene, ciliary neurotrophic factor (CNTF) and its receptors (CNTFR), Myostatin, IGF genes, angiotensin converting enzyme 1, and alpha actinin 3 genes may be associated as a result of these studies, which include genome-wide linkage studies and candidate gene association studies. Although genes like myostatin are particularly interesting since they can prevent muscle development, these investigations have often been inconsistent, and no obvious candidates have been found. Furthermore, none of these genes seem to explain more than 5% of variations in muscle mass.

Conclusion

Sarcopenia is prevalent in elderly cancer patients and negatively affects their ability to tolerate therapy and long-term prognosis. Sarcopenia can be prevented by increasing physical activity, particularly strength training, and getting enough protein, however studies show that many older cancer survivors fall short of their dietary and exercise targets. In order to increase muscle mass, prevent, reverse, and lessen the impact of sarcopenia, as well as to improve outcomes in older patients with and without malignancies, a transdisciplinary, mechanistic, and clinical approach involving regenerative cell therapy with targeted metabolic strategies, pharmacologic, and lifestyle modifications of nutrition and physical activity, including both aerobic and resistance exercise, is clearly required.

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