



Sarcopenia Dietary Treatment and Adapted Exercise

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Abstract

Aging can be defined as the regression of the physiological functions of the human body, which manifests itself with advancing age. One of the consequences that can cause numerous negative implications during this process is called Sarcopenia.

This phenomenon affects about 20% of the population aged between 65 and 70 years up to 50% in the elderly over 80 years.

The depletion of lean mass already begins after the age of 30 in men and, after the age of 50 in women.

After the seventh decade of age, this loss of functional mass tends to increase exponentially in humans (15% every 10 years).

The progressive decrease in lean body mass can lead, if not adequately contrasted, to a series of consequential disorders which generate the establishment of a state of increased vulnerability of the elderly to stressful events, which is defined as "frailty".

The causes of the onset of the sarcopenic process are multifactorial. Basically, we know that this complex pathogenic framework includes the progressive increase in physical inactivity, the increase in intramuscular, subcutaneous and visceral adipose tissue, the alteration of muscle protein metabolism (proteolysis), the reduced capacity for muscle protein synthesis, as well as a state of chronic inflammation. In addition to the loss of strength and decreased muscle function that causes postural instability, with consequent risk of falls and reduction of bone trophism, sarcopenia triggers a much wider spectrum of homeostatic alterations that can modify the profile metabolic rate of the elderly subject; the reduction of lean mass considerably reduces the basal metabolic rate, the increase in visceral fat establishes a persistent insulin sensitivity, which in turn is mainly responsible for the development or worsening of conditions such as diabetes, dyslipidemia, and liver disorders.

Furthermore, the aforementioned pathologies are factors that in turn trigger chronic inflammation, if added to the high levels of circulating insulin, the risk of cognitive disorders such as Alzheimer's and vascular dementia increase in elderly subjects.

Sarcopenia is a problem that must be faced with the right countermeasures as it turns out to be, if not adequately treated, a factor that causes a series of reactions that tend to establish in the elderly the loss of autonomy from a psycho-physical point of view.

Through adequate nutritional support that is able to ensure the right energy intake and a balanced intake of proteins, associated with adequate exercise, it will be possible to guarantee a positive maintenance of the efficiency of cognitive and physical functions, essential prerequisites for it to be ensured for the elderly maximum autonomy for an excellent life expectancy.

Keywords: Sarcopenia, Lean Body Mass, Aging

Introduction

The clinical definition of aging can be summarized as the regression of physiological functions that manifests itself with advancing age. One of the consequences that can cause numerous negative implications during this natural, as delicate, process, occurs through the progressive reduction of lean body mass. Event that takes the name of *Sarcopenia*.

It is obviously associated with a gradual loss of strength and muscle function [1,2], which in turn takes the name of *Dynapenia*.

This phenomenon affects about 20% of the population aged between 65 and 70 years up to 50% in the elderly over 80 years.

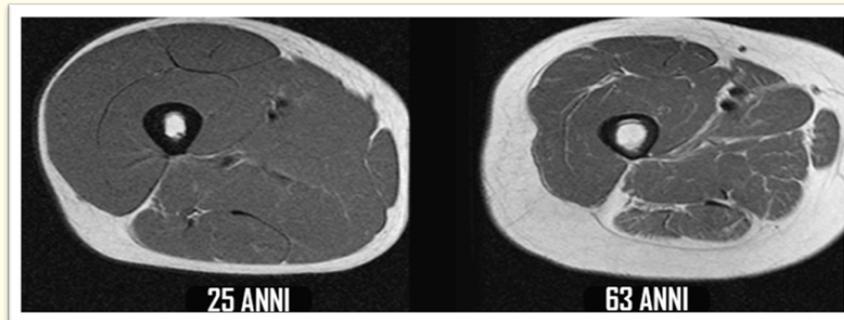


Figure 1: MRI of a muscle in a healthy 25-year-old and a 63-year-old sarcopenic subject.

The slow process of depletion of lean mass begins already after the age of 30 in men and, after the age of 50 in women, with an average loss of 8% for every ten years between 40 and 70 years [3].

After the seventh decade of age this loss of functional mass tends to increase exponentially in humans (15% every 10 years [3]). In women, the reduction is less marked because of the lower amount of lean mass she has. It is good to remember that the loss of lean mass is also associated with an increase in fat mass, an event that then diminishes between 75-80 years.

The progressive decrease in lean body mass can involve, if not adequately counteracted, a series of consequential disorders¹ that generate the establishment of a state of increased vulnerability of the elderly to stressful events, which is called “fragility”.

This condition can occur more markedly becoming clinically definable as pathological, thus increasing the risk of negative outcomes in the “frail elderly”, including falls, physical and cognitive disability, institutionalization, hospitalization and death [4].

Cause

The causes of the occurrence of the sarcopenic process are multifactorial. According to some authors, the loss of muscle mass represents a natural survival mechanism of the organism, which activates more incisively those metabolic processes responsible for catabolism, without an adequate counterbalance of protein synthesis. Basically, we know that this complex pathogen includes the progressive increase in physical inactivity, the increase in intramuscular adipose tissue [1] 5 subcutaneous and visceral 5, the altera-

tion of muscle protein metabolism (proteolysis) [6], the reduced capacity for muscle protein synthesis, as well as a state of chronic inflammation [5].

It has been shown that lifestyle changes, with the progressive increase in sedentary lifestyle, seem to be one of the main factors that cause the gradual increase of sarcopenic subjects [7] with all its consequences associated with it. A review of 2014 [8], underlined how the gradual and progressive physical inactivity is a key factor that accelerates the onset and course of muscle decay, especially in those subjects who lead a purely sedentary life (the loss of lean mass in inactive elderly subjects is parts at 1.5 kg for 10 days; the same value is reached in just 3 days for hospitalized elderly [9]).

People who follow a sedentary lifestyle have a higher production of ROS, compared to moderately active subjects, it has been shown that ROS activate multiple cellular signals involved in the evolution of the sarcopenic process [8].

Reduced physical exercise or complete inactivity can affect the energy expenditure of an adult by halving it. The loss of lean body mass, the reduction of total caloric expenditure if added to the decline of other hormonal factors, (lower GH activity, lower sensitivity to thyroid hormones, reduced levels of testosterone, IGF-1 and leptin-resistance [10]), are all elements that determine an unfavorable body recomposition (loss of lean mass, and increase in fat mass [11]).

The decrease in muscle mass, and the accumulation of visceral adiposity are two closely related parameters in the genesis of in-

sulin resistance, whose cause is to be attributed to the reduction in the amount of muscle fibers and a decrease in GLUT 4 [10]. It can also be related to a state of chronic inflammation. Studies have shown that IL-6, in addition to actively contributing to the establishment of insulin resistance [12], is a marker with catabolic action as well as being an important predictor of the decline in muscle strength [13] by inhibiting, among other things, insulin-like growth factor [12].

Despite the disagreements in the literature, regarding the influence of eating habits in the onset of sarcopenia, some studies [14-17] show a favorable role, in accelerating the evolution of the onset of the sarcopenic event, of unsuitable protein intakes, already prior to the age of onset of sarcopenia itself.

Protein intakes must be reviewed in quantitative and chronological terms, (optimal distribution of protein meals throughout the day), to cope with the now proven reduced anabolic effect of the meal in elderly subjects. This reduced effect has been adversely affected by muscle protein synthesis [18]; It is also not to neglect the need for calcium through those nutrients (dairy products) to protect bone health as much as possible. Milk and dairy products have the dual effect of being foods with a high calcium content and at the same time being sources of excellent quality protein, and well appreciated for their palatability and ease of consumption by elderly subjects, who often automatically become reluctant to introduce other protein sources.

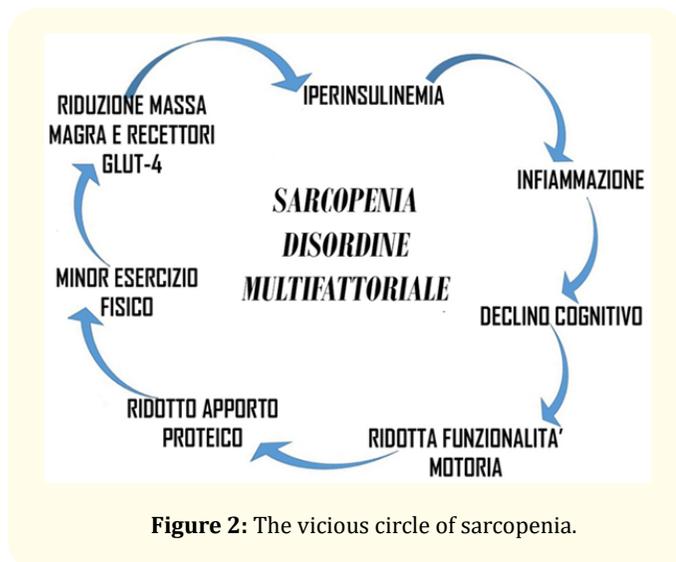


Figure 2: The vicious circle of sarcopenia.

Aftermath

In addition to the loss of strength and lower muscle function that causes postural instability, with consequent risk of falls and reduction of bone trophism, sarcopenia triggers a much wider spectrum of homeostatic alterations that can modify the metabolic profile of the elderly subject; The reduction of lean mass gre-

atly reduces the basal metabolic rate, the increase in visceral fat establishes a persistent insulin sensitivity, which in turn is mainly responsible for the development or aggravation of conditions such as diabetes, dyslipidemia, and liver disorders [19]. In addition, the concomitant increase in fat mass is associated with a higher incidence of cardiovascular problems.

It should be noted, in fact, that there is also a close relationship between abdominal fat and epicardial fat [20]; The latter is closely associated as a cardiovascular risk, especially if it is added to the factors already mentioned above.

The aforementioned pathologies, moreover, are factors that trigger in turn feed chronic inflammation, if we add to it the high levels of circulating insulin, increase in elderly subjects, the risk of cognitive disorders such as Alzheimer's and vascular dementia [21-23].

The onset or aggravation of diabetes further worsens the clinical picture of the sarcopenic subject [19]. Diabetes accelerates the reduction of muscle mass and strength also due to complications of neuropathy and vasculopathy, overweight or associated obesity and overproduction of inflammatory cytokines [19].

Last but not least, the loss of lean mass causes a reduction in proteins and body amino acids which, in addition to being used as a possible reserve energy substrate, are used in the formation of proteins of the acute phase (immune action). This reduction causes at first an accentuation of the sarcopenic process, because, during a metabolic stress, initially, the proteins of the skeletal muscle are able to satisfy the amino acid needs that the immune system needs, but, in a second moment if the amino acids are not adequately introduced with the diet there is an increase in susceptibility to infectious diseases in the elderly [24].

Treatment

The rationale of the preventive and therapeutic approach lies substantially in 3 key points

- Remodulation of protein and amino acid intake: It is correct to re-evaluate these intakes from the canonical 0.8g/kg/day to 1.2 up to 2g/kg/day [25], also taking into account the possible integration with rapidly absorbed proteins 26, essential amino acids 27, arginine [28], leucine 27 and HMB [27] 28, lysine [28] and OKG [29] as valid supports, given their anabolic potential to increase the rate of muscle protein synthesis, in order to safeguard lean mass during aging. As already mentioned, the key role of the fractionation of protein intake during the day should also be considered, inserting a protein source in all 3 main meals of the day. This strategy would optimise nitrogen balance by ensuring optimal protein synthesis [25].

- The use of Vitamin D and calcium supplements should not be neglected. The use of amino acid formulas enriched with Vitamin D and calcium seem to have positive effects on the prevention of the risk of fractures, on the reduction of symptoms such as fatigue, and on the ability of the muscle itself to produce contraction force, which would therefore guarantee a greater ability on the part of the elderly to undertake physical exercise. There is also growing interest in the role of a diet rich in antioxidants as a therapeutic support in sarcopenic subjects. Many studies have shown the preventive and protective role of substances such as beta-carotene, selenium, zinc, vitamin C and E on the functionality of the organism [14].
- Exercise should be promoted and recommended in all sarcopenic patients. It must be modulated on the clinical condition of the patient, on his possible complications and without neglecting his abilities the possible lack of resistance, the premature fatigue that often characterizes these subjects [30]. The greatest benefits are obtained with a combination of resistance training (through the use of overloads such as free weights and isotonic machines), coordination and balance activities, and aerobic activities [31]. The optimal frequency of sessions could be two days a week activities that involve muscle contraction essential to slow down, stop or even promote the development of lean mass; and another 2-3 weekly sessions of aerobic activity [32] for the improvement of cardiorespiratory functions [33], mitochondrial and metabolic [34].
- There is, for the sake of completeness, a fourth point, relating to what is, in the medical field known by the acronym of TRT (Testosterone Replacement Therapy, or Testosterone Replacement Therapy), a practice that arouses conflicting opinions because of the actual effectiveness of this therapy in the elderly and obviously on the potential side effects that such treatment can entail [35,36]. We believe that this topic deserves further investigation, useful to clarify whether the effects of this practice can actually be useful in contrasting, together with diet and exercise, the process of depletion of lean mass in the elderly.

Conclusion

Over the years the definition of sarcopenia has seen a constant evolution, given the continuous new implications that it entails in terms of health. Certainly it is a problem that must be faced with the right countermeasures as it turns out to be, if not adequately treated, a factor that causes a series of reactions that tend to establish in the elderly the loss of autonomy from a psycho-physical point of view.

Through adequate nutritional support that is able to ensure the right energy intake and a balanced intake of proteins, associated

with adequate exercise, it will be possible to guarantee a positive maintenance of the efficiency of cognitive and physical functions, essential prerequisites for ensuring maximum autonomy for an excellent life expectancy to the elderly.

We are well aware that each subject is tied to a thread that will inexorably lead him to a gradual process of functional decay, however to counteract in the best way through the right preventive and therapeutic formulas this process can guarantee as far as possible a more than dignified senescence.

“No one is so old that they don't think they can live another year.” CICERONE.

Bibliography

1. Cruz-Jentoft AJ, *et al.* “Sarcopenia: European consensus on definition and diagnosis.” Report of the European Working Group on Sarcopenia in Older People”. *Age Ageing* 39 (2010): 412-423.
2. Muscaritoli M, *et al.* “Consensus definition of sarcopenia, cachexia and pre-cachexia: Joint document elaborated by Special Interest Groups (SIG) “cachexia-anorexia in chronic wasting diseases” and “nutrition in geriatrics”. *Clinical Nutrition* 29 (2010): 154-159.
3. Grimby G and Saltin B. “The ageing muscle”. *Clinical Physiology* 3 (1989): 209-218.
4. Clegg A, *et al.* “Frailty in elderly people”. *Lancet* 381 (2013): 752-762.
5. Baylis D, *et al.* “Understanding how we age: insights into inflammaging”. *Longevity and Healthspan* 2 (2013): 8.
6. Combaret L, *et al.* “Skeletal muscle proteolysis in aging”. *Current Opinion in Clinical Nutrition and Metabolic Care* 12.1 (2009): 37-41.
7. Berger MJ and Doherty TJ. “Sarcopenia: prevalence, mechanisms, and functional consequences”. *Interdisciplinary Topics in Gerontology* 37 (2010): 94-114.
8. Derbré F, *et al.* “Inactivity-induced oxidative stress: A central role in age-related sarcopenia?” *European Journal of Sport Science* 14 (2014): S98-S108.
9. English KL and Paddon-Jones D. “Protecting muscle mass and function in older adults during bed rest”. *Current Opinion in Clinical Nutrition and Metabolic Care* 13 (2010): 34-39.
10. Vendemiale G. “Metabolic syndrome and frailty”. *Gerontology* 55 (2007): 48-53.

11. Villareal DT, et al. "Obesity in older adults". *The American Journal of Clinical Nutrition* 82 (2005): 923-934.
12. Hamada K, et al. "Senescence of human skeletal muscle impairs the local inflammatory cytokine response to acute eccentric exercise". *FASEB Journal* 19 (2005): 264-266.
13. Stenholm S, et al. "Anabolic and catabolic biomarkers as predictors of muscle strength decline: the in CHIANTI study". *Rejuvenation Research* 13 (2010): 3-11.
14. Robinson S, et al. "Nutrition and sarcopenia: a review of the evidence and implications for preventive strategies". *Journal of Aging Research* (2012): 6.
15. Sayer AA, et al. "The developmental origins of sarcopenia". *The Journal of Nutrition, Health and Aging* 12 (2008): 427-432.
16. Scott D, et al. "Associations between dietary nutrient intake and muscle mass and strength in communitydwelling older adults: the Tasmanian Older Adult Cohort Study". *Journal of the American Geriatrics Society* 58 (2010): 2129-2134.
17. Houston DK, et al. "Dietary protein intake is associated with lean mass change in older, community-dwelling adults: the Health, Aging, and Body Composition (Health ABC) Study". *The American Journal of Clinical Nutrition* 87 (2008): 150-155.
18. Mosoni L, et al. "Age-related changes in protein synthesis measured *in vivo* in rat liver and gastrocnemius muscle". *Mechanisms of Ageing and Development* 68 (1993): 209-220.
19. Angela Shin-Yu Lien, et al. "Diabetes related fatigue sarcopenia, frailty". *Journal of Diabetes Investigation* 9.1 (2018).
20. Rosito GA, et al. "Pericardial Fat, Visceral Abdominal Fat, Cardiovascular Disease Risk Factors, and Vascular Calcification in a Community-Based Sample". *The Framingham Heart Study* 117 (2008): 605-613.
21. Solfrizzi V, et al. "Metabolic syndrome and the risk of vascular dementia: the Italian Longitudinal Study on Ageing". *Journal of Neurology, Neurosurgery, and Psychiatry* 81 (2010): 433-440.
22. Cholerton B, et al. "Insulin, cognition, and dementia". *European Journal of Pharmacology* 05 (2013): 170-179.
23. Kim B and Feldman EL. "Insulin resistance as a key link for the increased risk of cognitive impairment in the metabolic syndrome". *Experimental and Molecular Medicine* 47 (2015): e149.
24. Roth E. "Immune and cell modulation by amino acids". *Clinical Nutrition* 26 (2007): 535-544
25. Baum JL, et al. "Protein Consumption and the Elderly: What Is the Optimal Level of Intake?" *Nutrients* 8 (2016): 359.
26. Penning B, et al. "Whey protein stimulates postprandial muscle protein accretion more accretion more effectively then do casein and casein hydrolysate in older men". *The American Journal of Clinical Nutrition* (2011): 93997.
27. Cruz-Jentoft AJ, et al. "Prevalence of and interventions for sarcopenia in ageing adults: a systematic review. Report of the International Sarcopenia Initiative (EWGSOP and IWGS)". *Age and Ageing* 43 (2014): 748-759.
28. Flakoll P, et al. "Effect of beta-hydroxy-beta-methylbutyrate, arginine, and lysine supplementation on strength, functionality, body composition, and protein metabolism in elderly women". *Nutrition* 20 (2004): 445-451.
29. Walrand S. "Ornithine alpha-ketoglutarate: could it be a new therapeutic option for sarcopenia?" *The Journal of Nutrition, Health and Aging* 14.7 (2010): 570-577.
30. Cobo A, et al. "Impact of frailty in older patients with diabetes mellitus: an overview". *Endocrinología y Nutrición* 63 (2016): 291-303.
31. Zaleski AL, et al. "Coming of Age: Considerations in the Prescription of Exercise for Older Adults". *Methodist DeBakey Cardiovascular Journal* 12.2 (2016): 98-104.
32. Bowen TS, et al. "Skeletal muscle wasting in cachexia and sarcopenia: molecular pathophysiology and impact of exercise training". *Journal of Cachexia, Sarcopenia and Muscle* 6 (2015): 197-207.
33. Yu J. "The etiology and exercise implication of sarcopenia in the elderly". *International Journal of Nursing Sciences* 2 (2015): 199-203.
34. Lanza IR and Nair KS. "Muscle mitochondrial changes with aging and exercise". *The American Journal of Clinical Nutrition* 89.1 (2008): 467S-471S.
35. Skinner JW, et al. "Muscular responses to testosterone replacement vary by administration route: a systematic review and meta-analysis". *Journal of Cachexia, Sarcopenia and Muscle* 9.3 (2018): 465-481.
36. Basualto-Alarcón C, et al. "Sarcopenia and Androgens: A Link between Pathology and Treatment". *Frontiers in Endocrinology (Lausanne)* 5 (2014): 217.