Sarcopenia (from the Greek sarx, flesh, and penia, poverty) is a syndrome characterized by progressive and generalized loss of skeletal muscle mass and strength with a risk of adverse outcomes such as physical disability, poor quality of life and death. Recently, the European Working Group on Sarcopenia in the Elderly defined the diagnostic criteria (table 1) [1].

Sarcopenia is a major contributing factor of disability and loss of independence in the elderly. Malnutrition and sarcopenia are interrelated in the cycle of frailty. The loss of muscle mass is often a consequence of senescent musculoskeletal changes that occur with ageing, and is worsened by diseases and enhanced by weight loss. Sarcopenia involves a reduction in muscular strength, rest and total energy expenditure. Because of the anorexia that accompanies ageing, chronic malnutrition develops, thereby worsening sarcopenia.

One of the most important difficulties involved in detecting and monitoring sarcopenia is that there is no gold standard examination for its measurement. Different diagnostic methods are used in both clinical and investigational settings: computed tomography, magnetic resonance imaging, dual energy X-ray absorptiometry and bioimpedance analysis to measure muscle mass; handgrip strength to measure muscle strength, or functional tests to measure physical performance. However, the diagnostic value of sarcopenia is not clearly established. The Working Group on Sarcopenia in Older People has suggested using normal values obtained in healthy young adults and establishing the cutoff point for the diagnosis of sarcopenia at 2 standard deviations below the mean reference value.

The etiopathogenesis of sarcopenia is multifactorial and complex. Several factors affecting the muscle changes associated with ageing have been identified. On the one hand, genetic factors, albeit not well identified, are involved. On the other, the sexual steroid deficit that occurs with ageing has a major impact on both muscle and bone trophism. The decrease in sex hormones is accompanied by activation of inflammatory
mediators that can act as catabolic cytokines for muscle. Growth hormone deficit is also directly involved in the etiopathogenesis of sarcopenia, in synergy with the increase in inflammatory mediators and gonad hormone deficit. IGF-I concentrations in the elderly inversely predict the presence of sarcopenia, acting as a protective factor in men. Weight loss exacerbates sarcopenia, causing a greater loss of lean mass in comparison to fatty mass. Moreover, in patients who recover lost weight, recovery usually involves a greater proportion of fat [2]. However, even with no weight changes, longitudinal studies show progressive loss of muscle mass with ageing. Exercise is inversely and independently related to fat-free mass, especially in women. However, the relationship between spontaneous exercise and muscle mass is difficult because of the relation between exercise and bodyweight.

Different strategies have been tested in the therapeutic approach to sarcopenia [3, 4]; they include:

1 Replacement therapy with testosterone/other anabolic agents
2 Estrogen replacement therapy
3 Human growth hormone replacement therapy
4 Resistance training
5 Nutritional treatment
6 Interventions on cytokines and immune function

Testosterone and human growth hormone treatment increase muscle body mass in hypogonadal men or in human growth hormone-deficient patients, but these results are not accompanied by functional improvement, while side effects are worrisome. Estrogen therapy has showed inconsistent results in muscle mass in short- and long-term therapy in menopausal women.

Of all the therapeutic options available, only resistance training with or without nutritional supplementation has shown its efficacy in increasing skeletal muscle mass. Resistance strength training in the elderly increases muscle mass, muscle strength, balance and resistance. Resistance training is better for increasing muscle strength and mass than endurance training. Exercise must be accompanied by sufficient protein intake. Some studies

<table>
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<tr>
<th>Diagnosis of sarcopenia</th>
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<tbody>
<tr>
<td>1 Low muscle mass</td>
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<tr>
<td>2 Low muscle strength</td>
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<td>3 Low physical performance</td>
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Diagnosis is based on documentation of criterion 1 plus criterion 2 or criterion 3
have shown a synergic effect between protein supplementation and physical exercise, and insufficient protein intake has probably prevented better exercise outcomes.

Nutritional treatment has been focused on increasing or modifying the protein content of the diet [5]. With the current evidence, we know that protein intake greater than the RDA can improve muscle mass, strength and function in the elderly. Therefore, in the absence of contraindications, protein intake should be about 1.5 g/kg per day. The studies that have combined protein supplements with exercise have obtained the best outcomes when supplementation is administered immediately after exercise. The use of protein supplements without exercise, however, has not had any effect on muscle mass. Currently, the role of vitamin D in the pathogenesis of sarcopenia is being considered.

Reference