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# Physiopathology of muscle in relation to sarcopenia

H. Degens

Skeletal muscle is indispensable for locomotion, respiration and maintenance of body posture. The muscle can perform these functions by generating force and changing its length at the same time, thereby producing power, which is the product of force and velocity. The maximal power a muscle can generate is primarily, but not solely, determined by muscle mass. Loss of muscle mass may thus hamper activities of daily life and in severe cases even cause the transition from an independent to a dependent life-style.

Sarcopenia is the loss of muscle mass during ageing. Figure 1 gives an overview of the main, but not all, factors that contribute to sarcopenia and how regular exercise may minimize the deterioration in muscle mass and function. In humans sarcopenia has primarily been investigated in cross-sectional studies. In those studies it appeared that muscle mass stays fairly stable till about 50 years of age, where after there is a progressive decline [1, 2] of as much as 1 % per year. The rate of loss of muscle mass appeared even faster (~1.5 % per year) in a longitudinal study [3]. The reduction in muscle mass is the consequence of loss and atrophy of muscle fibres [2]. The decline in jumping power and force generating capacity are, however, more than proportional to the loss of muscle mass [4, 5], resulting in lower specific tension (maximal force per muscle physiological cross-sectional area) in both human [6] and rat [7, 8] muscle. These observations suggest that not only the quantity but also the ‘quality’ of the muscle decreases with age. Using isolated single muscle fibres it has indeed been found that the maximal shortening velocity and specific tension are decreased in both rat and human type I and IIa muscle fibres [9–11], though

this is not unequivocal [12]. The loss of muscle fibres is probably related to an incomplete denervation-reinnervation process due to loss of motoneurons [13], while the age-related reduction in the level of physical activity may contribute to muscle fibre atrophy. During ageing the mitochondria tend to leak more resulting in an enhanced production of Reactive Oxygen Species. The oxidative stress may be further increased by chronic low-grade systemic inflammation. In this oxidative environment post-translational modifications of myofibrillar proteins may occur, such as glycation and/or oxidative modifications of the myosin molecule, that have been shown to impair myofibrillar function [14, 15].

So, is the age-related decline in muscle mass and function inevitable? To answer this question we have to understand the underlying causes. One factor that undoubtedly contributes to the decline in muscle mass and function is the age-related reduction in the level of physical activity. The decline in physical activity, however, can not be the sole explanation as even master weight lifters suffer from a progressive loss of muscle strength [16]. It has been suggested that part of the oxidative modifications may be the result of chronic low-grade systemic inflammation at old age [16]. An altered redox-state may also explain the increased expression of inhibitors of differentiation proteins which may have contributed to the apoptosis, sarcopenia and a diminished ability to increase muscle mass as observed in old rats [17]. Regular exercise can, however, attenuate the degree of systemic inflammation, enhance the antioxidant defence system and is usually accompanied with an increase in muscle size and strength [16, 18]. Studies on isolated single fibres indicated that also muscle quality is better in the active than inactive elderly [9], which might be related to a reversal of oxidative modifications of the myosin molecule [19].

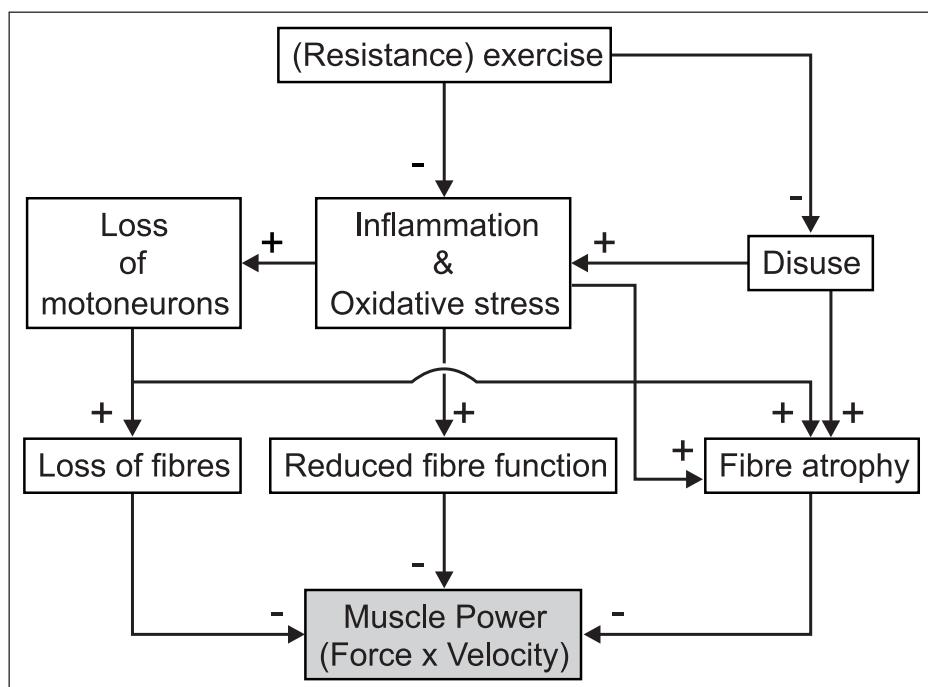


Figure 1: A non-exhaustive depiction of the factors that underlie the age-related muscle wasting (sarcopenia) and the benefits of regular physical exercise.

In rats, life-long exercise increases life span, but when initiated too late in life it does reduce rather than increase life span, suggesting a certain ‘threshold age’ beyond which the prescription of exercise is detrimental [20]. It remains to be seen whether such a phenomenon also occurs in humans, but some studies showing an attenuated response to exercise at old age do hint to this possibility [21, 22]. Overall, regular physical activity appears an effective means to minimize the age-related reduction in muscle mass and function, and helps to prolong a good quality of life.

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