



Review Article

Age-related functional changes and susceptibility to eccentric contraction-induced damage in skeletal muscle cell

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ABSTRACT

Depending upon external loading conditions, skeletal muscles can either shorten, lengthen, or remain at a fixed length as they produce force. Fixed-end or isometric contractions stabilize joints and allow muscles to act as active struts during locomotion. Active muscles dissipate energy when they are lengthened by an external force that exceeds their current force producing capacity. These unaccustomed eccentric activities often lead to muscle weakness, soreness, and inflammation. During aging, the ability to produce force under these conditions is reduced and appears to be due to not only reductions in muscle mass but also to alterations in the basic mechanisms of contraction. These alterations include impairments in the excitation–contraction process, and the action of the cross-bridges. Also, it is well known that age-related skeletal muscle atrophy is characterized by a preferential atrophy of fast fibers, and increased susceptibility to fast muscle fiber when aged muscles are exposed to eccentric contraction followed by the impaired recovery process has been reported. Taken together, the selective loss of fast muscle fiber in aged muscle could be affected by eccentric-induced muscle damage, which has significant implication to identify the etiology of the age-related functional changes. Therefore, in this review the alteration of age-related muscle function and its impact to/of eccentric induced muscle damage and recovery will be addressed in detail.

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1. Introduction

Age-related sarcopenia is characterized by a loss of skeletal muscle mass and a gradual decline in the functional properties of the tissue.¹⁻⁴ Because of the clinical importance of these functional changes, and the demographic changes

projected for the next 20 years, considerable attention has been focused on understanding how aging affects skeletal muscle contractility.

The ability to generate force under isometric or shortening conditions is reduced in old age and appears to be due to not only a selective atrophy or loss of fast contracting fiber types,⁵⁻⁷ impairments in the excitation–contraction

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(EC) coupling process,^{8,9} and perturbations to cross-bridge function.^{10–12} In addition to isometric or shortening contractions, muscle must also actively resist stretch by an external force. This ability to resist, but not prevent, lengthening allows muscles to function as brakes or shock absorbers. These lengthening, eccentric, or pliometric contractions occur frequently during everyday activities. Force production during eccentric contractions appears to be better preserved with age than strength during isometric or shortening contractions.^{13,14} However, in young individuals, unaccustomed or excessive eccentric muscular activity can lead to long-lasting weakness, soreness, and inflammation^{15–19} and these effects may be exacerbated with age.

Aged muscles are more sensitive to eccentric contraction, at least in animal models.^{20–23} However, human studies examining the effect of eccentric-induced muscle damage on old adults have not reached a consensus. Several human studies have reported that older adults show an increased susceptibility to damage by a single bout of eccentric exercise performed by knee extensors compared to younger adults.^{24,25} By contrast, others have reported that the loss of maximal isometric contraction was the same²⁶ or less for old adults vs. young adults following voluntary eccentric exercise by the elbow flexors.^{27,28} These contradictory results of eccentric-induced muscle damage on aging may be due to the inability to adequately control important factors contributing to muscle damage, such as strain magnitude,²⁹ intramuscular fiber pennation, and motor unit recruitment patterns in human muscle. Furthermore, age group differences in muscle mass, fiber type composition, and mechanical stress may complicate experimental design and interpretation.

Few studies have examined the susceptibility of human muscle fibers after eccentric-contraction, especially in muscles experiencing sarcopenia. Furthermore, any preferential damage to fast twitch fibers could exacerbate the symptoms of sarcopenia. It is therefore critical to understand how different fiber types respond mechanically to eccentric-induced muscle injury, especially for aged populations. Therefore, the aim of this review is to explore details about the age-related functional changes particularly in skeletal muscle cell, and its relationship with eccentric-induced muscle damage and recovery.

2. Age-related functional changes in skeletal muscle

Sarcopenia is a general term to describe the gradual decline of muscle mass with aging. It is characterized by not only the loss of skeletal muscle mass, but also the gradual decline in muscle functional properties, such as a decline in force generating capacity, maximum shortening velocity, and a general slowing of contraction.^{1–4} This age-related muscle atrophy is thought to be derived from a decrease in both muscle quantity (mass), and a decrease in muscle quality (force per cross-sectional area of muscle, proportion of fiber types and metabolic characteristics).³

The loss of muscle quantity appears to be mainly due to a degradation of contractile protein, resulting from both the reduction of number of single muscle fibers, and a decrease

in the cross-sectional area (CSA) of residual muscle fibers. For example, the CSA of muscle decrease by up to 30%, and muscle strength by about 30–40% between the ages of 65 years and 75 years.³⁰ This age-related decline of contractile protein content could be explained by different rates of protein degradation and protein synthesis.³¹ The main determinant of the loss of contractile protein content is protein degradation rather than protein synthesis.³² Also type II fibers are primarily atrophied compared to the type I fibers.^{6,7} The age-related decrease in muscle quality indicates that both the time taken to reach peak twitch tension and the time taken for the muscle twitch response to relax are increased in old muscle.³³ This could be due to several different mechanisms, including an increase in the level of intramuscular collagen and fat, a decline in specific force, an alteration of EC coupling,^{34–36} neurogenic factors, or motor unit remodeling (denervation and reinnervation).

Even though it is generally accepted that the contractile properties of muscle are decreased with aging, the effects of aging on the intrinsic ability of single fibers are still equivocal. That is, the results of skinned single fiber studies on maximal isometric force (P_0), and unloaded shortening velocity between young and old humans are not consistent. Several studies have reported that there were significant reduction in the intrinsic contractile properties with aging, such as reductions in P_0 , normalized force by CSA (P_0/CSA), and unloaded shortening velocity.^{11,37–40} This suggests that both size and quality of each individual muscle fiber decrease with age. The proposed mechanisms to explain the declined intrinsic force generating ability of aged fibers are either a lower number of strongly bound cross-bridges during maximal activation or a reduced force-generating ability of each cross-bridge.⁴⁰ According to Lowe and colleagues,¹² when a muscle fiber contracts maximally, about 32% of myosin heads are in the strong-binding state. However, only 22% of myosin heads are in that state in fibers from older rats. Thus, the decreased P_0 in the elderly may due to a decreased numbers of cross-bridges per CSA.¹¹ Also, there is a loss of myofibrillar protein in old rats, which may lead to a reduction in the motor proteins actin and myosin.¹⁰ Slowing of shortening velocity with aging is thought to involve a slowing of the steps within the cross-bridge cycle, such as the actin–myosin cross-bridge detachment rate.³⁹ It is proposed that this occurs without a change in isoform type,^{41,42} and may be related to glycation of myosin.⁴³

By contrast, more recent studies have found that contractile quality of single muscle fiber is maintained during aging^{44–47} or increased in maximal force in both fiber types with aging.^{48,49} In other words, there was no change of intrinsic ability of single fiber with aging among young and old men and women. This suggests that the intrinsic properties of cross-bridge mechanics are preserved with age.⁴⁷

3. Age-related susceptibility to eccentric contraction induced muscle damage

Several studies have considered the different susceptibility of muscles from young and old to damage induced by eccentric contractions using both animal and human models. The general consensus of the animal studies is that an

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