New insights in age-related impairments in muscle function

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When skeletal muscles are activated, muscle fibres attempt to shorten, but whether the fibres shorten, stay at the same length, or are lengthened, depends on the interaction between the force developed by the fibres and the external *load* on the muscle. Consequently, for the incredible complexity of human movement, muscles undergo each of the three types of contractions at different times. Of the three types of contractions, only lengthening contractions have the potential to cause injury to fibres. In contrast, muscles fatigue most readily during shortening contractions when forces are low, but the energy cost is high. Both contraction-induced injury and fatigue cause a loss of function, but 'contraction-induced injury' is due to mechanical factors, whereas 'muscle fatigue' is due to metabolic factors. With aging, the skeletal muscles of old animals become smaller, weaker, slower, more fatigable, and more susceptible to contraction-induced injury. The most significant agerelated change in muscle structure occurs between fifty and eighty years of age with a loss of ~50% of the fibres in essentially all the muscles. The loss appears to be immutable and occurs in the muscles of both females and males and in untrained and trained oganisms. In both humans and rats, the loss results predominantly from the loss of motor units, but whether the initiating factors are neurogenic, myogenic, or both, is unknown. The loss involves motor units composed of fast, powerful, type 2a fibres and the slow, type 1 motor units actually get larger due to the 'capture' of denervated fast, type 2 fibres by axonal sprouting. The loss of the fast, powerful motor units aggravates the age-related loss in power due to the loss of fibres. Compensation for the loss in muscle mass is possible to a limited degree by hypertrophy of the fibres that remain and by the conversion of slow, type 1 fibres to fast, type 2a fibres. Many elderly people eventually become *frail* - a condition typified by muscle atrophy and weakness. The condition of frailty is reversible in large measure by well-designed, carefully administered training programs. The so-called 'pliometric' training programs, that include each of the three types of contractions, have revolutionized training for high performance in many sports. Evidence from welldesigned investigations supports the advantages of such 'pliometric' training programs to be implemented with appropriate modifications for the frail elderly population. Despite the advantages, great care must be exercised in designing such training for the elderly, because of the potential of lengthening contractions to induce permanent injury to muscles, particularly those of the 'frail' elderly. In summary, many age-related changes in skeletal muscle are immutable, but the condition of 'frailty' due to atrophy, weakness, fatigability, and susceptibility to injury, can be slowed and even reversed by the commitment to an active life style that includes a well-designed exercise program.