Muscle wasting and weakness in old age

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Senile sarcopenia, the loss of muscle mass associated with ageing, is a main cause of muscle weakness in old age. This process has an onset at around the 6th decade, and by the 8th muscle mass is about 60% that in the 2nd decade (Lexell et al., 1988). The etiology of sarcopenia is rather complex since involves multiple factors, but there is general consensus that it is mainly driven by neuropathic changes leading to motoneuron death. The loss of motoneurons leads to a decrease in the number of muscle fibres (hypoplasia) (Lexell et al., 1988). Fibre size also decreases with age (atrophy) and this is mainly due to a reduction in physical activity since even ‘physically active’ septuagenarians (individuals aged 70-79 years) are about 20% less active than their vicenarian (individuals aged 20-29 years) counterparts (Morse et al. 2004). In addition to these neuropathic and physical activity-related changes, nutritional, hormonal and immunological factors are also known to contribute to sarcopenia. Malnutrition in ageing is quite common, and this is due to a progressive loss of appetite, a reduction in food intake and also to vitamin D deficiency. Low Vit D, in association with high parathyroid hormone levels, increases the risk of muscle wasting in old age (Visser et al 2003). The hormonal and immunological alterations contributing to sarcopenia are represented by the withdrawal, or resistance, to those factors responsible for anabolism (GH, IGF-1, testosterone) and by an increased catabolic activity (by IL-1, IL-6, TNF-α, myostatin), also due to Vit D deficiency since Vit D is known to protect muscle and bone from inflammatory cytokines (Schach, 1999). Despite the fact that sarcopenia is a major determinant of muscle weakness in old age, the loss of muscle strength exceeds that of muscle size and, as a consequence, there is a decline in force per unit of muscle cross-sectional area (Macaluso et al. 2002; Morse et al 2004). Several factors contribute to this phenomenon, frequently referred to as a deterioration in ‘muscle quality’, these can be grouped under three main categories: muscular, tendinous and neural changes.

Recent evidence obtained both by ours and others’ laboratories shows that ageing is associated with: 1) a decrease in single fibre specific tension (D’Antona et al 2003, Larsson et al 1997), 2) alterations in muscle architecture (Narici et al. 2003, Morse et al 2005), 3) decreased tendon stiffness (Maganaris 2001, Narici et al. 2005), 4) reduced activation capacity of agonist muscles (Morse et al. 2004), 5) increased antagonist co-activation (Klein. et al. 2001; Macaluso et al. 2002) and 6) reduced motor unit firing frequency (Kamen et al. 1995), though this tends to be compensated by a slower contraction time (Narici et al. 1991).

Physical activity, particularly resistive training, has been found to reverse most of the above changes (Reeves et al. 2003, 2004, Morse et al. 2005, Narici et al. 2005, D’Antona et al. 2003, Macaluso and De Vito 2004), with the exception of motor unit firing frequency that cannot be increased by training in old age (Kamen and Knight, 2004). These findings demonstrate that in old age the musculoskeletal and neuromuscular systems maintain the ability to adapt to regimes of increased loading. These training-induced adaptations are most important for maintaining mobility, speed of motion, balance and independence in old age.

References

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This work was supported by the European Commission Funding (Better-ageing; QLRT-2001-00323)