Increase and decrease of skeletal muscle mass in response to several stimuli are considered obvious examples of cell plasticity. The accepted mechanism underlying such changes is quantitative modifications of gene expression. In particular, the quantitative mechanism (change in cross sectional area) of muscle plasticity seems to be the major accepted factor by which skeletal muscle can adapt to variable functional requirements (increase and decrease in load and pathological states) through changes in mass and fiber size (hypertrophy and atrophy). Among conditions able to induce negative adaptational adjustments through quantitative mechanisms, aging plays a major role. In particular, in aging the overall loss of muscle quality is well described by the term sarcopenia which indicates the loss of muscle mass and strength. In presence of sarcopenia, the loss of mass is mainly related to a quantitative mechanism responsible for an imbalance between protein synthesis and breakdown, primarily described by changes in the two major structural proteins of striated muscles: myosin and actin. Indeed, it has been suggested that in presence of sarcopenia the disproportionate reduction of fibres size and myofibrillar protein content might contribute to reduction of muscle specific force observed in vivo. This evidence strengthens the hypothesis that skeletal muscle fibres containing the same myosin isoforms may show different contractile properties and that sarcopenia may relate to changes in the intrinsic properties of muscle fibres independently from myosin isoforms expression. A possible mechanism which may contribute to the disproportionate loss of muscle mass and size is represented by age-related modifications of myofibrillar proteins at post-transcriptional level. Studies reported that ageing is responsible for alterations of post-transcriptional events as the reduced availability of specific mRNAs encoding myosin and actin and a dysregulation of Insulin/IGF1 activated mTOR/PI3/Akt metabolic pathway known to regulate translation of specific mRNAs encoding for essential components of the protein synthetic machinery. Contrarily to sarcopenia, resistance training is well known to activate quantitative mechanism leading to muscle hypertrophy, selective increase of fibre CSA and gain in force. The mechanism through which heavy work increases muscle mass may be the activation of protein synthesis pathways. The expression of IGF-1, induced by muscle overload, and insulin have been demonstrated to be responsible for regulation of protein synthesis by stimulating the PI3K/Akt pathway, which, in turn, results in the downstream activation of targets required for protein synthesis. Interestingly, recent evidences suggest that heavy work may be associated with increase of specific force of single muscle fibres thus opening the possibility that unknown mechanisms can modulate force developed by a given amount of contractile proteins independently from a quantitative mechanism.

Keywords: Hypertrophy, Atrophy, Muscle Force