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Editorial Open Access

Muscle Damage and Human Skeletal Muscle Hypertrophy

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Editorial

Skeletal muscle hypertrophy is a morphological adaptation to strengthen training associated with the activation of satellite cells, different signaling pathways that regulate protein synthesis and the inflammatory, hormonal and growth factors responses [1].

Recently, the role of exercise-induced muscle damage in promoting the adaptation has received attention in literature [2]. Despite its detrimental short-term effects, it has been hypothesized that the damage is necessary for long-term hypertrophic adaptations [3]. However, other researchers have questioned this hypothesis, noting that hypertrophy can occur in the relative absence of damages [4-6].

Evidences against damage-induce hypertrophy are based on the fact that a repeated bout effect after a single bout of exercise has been consistently report, whereas muscle damage is attenuate [2,7]. This observation would suggest that damage does not contribute to muscle hypertrophy since muscle become less susceptible to damages, but hypertrophy remains occurring [2]. A problem with this theory is that muscle damage persists even in well-trained subjects, although in a lesser degree than in novice trainees [8]. In addition, the marker employed to evaluate the damages is the quantification of plasmatic activity of myofibrilar proteins like the creatine kinase. Evidences in literature illustrate that creatine kinase plasma activity is considered as a poor predictor of muscle damage because it does not present a significant linear correlation with muscle functions and ultra-structural changes in muscle following exercise [8-10].

Evidences for damage-induced hypertrophy are based on the fact that the eccentric component of exercise (well consolidated by inducing greater magnitudes of hypertrophy than concentric and isometric muscle actions) is characterized by inducing greater magnitudes of muscle damage [11] and acute-phase inflammatory responses [7,12-15].

Another evidence is that the increase in mTOR signaling can also occur independently of IGF-1 pathway [16-18], due to the PKB-independent and mechanically induced mTOR signaling via phospholipase D-generated phosphatidic acid production. As proposed by Hornberger et al. [16], phospholipase D dissociates from α-actinin during repetitive mechanical work induced by muscle contraction, relieves the inhibition of phospholipase D and subsequently promotes phosphatidic acid production and mTOR activation. Phospholipase D may thus represent a possible link between muscle structure and intracellular signaling by rising the activation of the mTOR-p70S6k pathway in an PKB-independent way [16], consolidating the fact that Z-band streaming may actually be part of the remodeling of muscle fiber, which occurs in response to resistance-training [19,20].

Considering the polemic nature of the relevance of the topic for the sports performance and rehabilitation field, we recommend that future research aim to clarify whether a causal relationship exists between muscle damage and skeletal muscle hypertrophy. We also recommend special care with previous trained subjects. Considering that the ceiling effect slows the rate of muscle growth as one gains training experience, it is possible that myodamage is an increasingly important mediator of hypertrophy in highly trained individuals, but not in untrained ones.

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