Muscle damage induced by stretch-shortening cycle (SSC) exercise in humans?

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Introduction

All forms of muscle actions, if performed vigorously enough, lead to a delayed onset of muscle soreness (DOMS) and possible muscle damage (Brockett et al. 2001), which cause impairments of muscle function such as reduced maximal force (Friden et al. 1983) and weakened maximal SSC performance (Gollhofer et al. 1987). On muscular level, repeated eccentric bicycle exercise has been found to cause streaming, broadening and even total disruption of Z-bands (Friden et al 1983). Presumably as a consequence of mechanical disruption, increased levels of myocellular enzymes (Nosaka & Clarkson, 1995) and circulating neutrophils (Cannon et al. 1991) have been reported. These changes are primarily mediated by increased levels of cytokines such as interleukin-1β (IL-1β) associated with acute inflammatory response (Dennis et al. 2004). However, recent studies have suggested that DOMS only reflects an adaptive remodeling of the myofibrils (Yu et.al. 2004). The reduced performance may also be related to breakdown of structural proteins such as titin (Komi, 2000). The present study was designed to evaluate if SSC exercise when repeated until exhaustion in humans can induce muscle damage.

Methods

8 healthy male subjects were measured before, immediately after (IA) and 2 days (2D) after the fatiguing exercise, which consisted of one legged SSC exercises on the sledge apparatus: 100 maximal drop jumps followed by submaximal jumping (mean number of jumps was 876) until complete exhaustion. In the measurements, the subjects performed 2-3 times unilateral (the right leg was the experimental leg and the left leg served as a control) maximal knee extensions (MVC). The muscle biopsies were taken 30 min and 2 days (2D) after the fatiguing exercise from the VL muscles. Titin mRNA levels were analyzed with real-time PCR and protein isoforms with SDS-PAGE. In addition, IL-1β mRNA expression, myosin heavy chain (MHC) isoforms and integrity of dystrophin (immunohistochemistry) were analyzed. Blood samples were drawn for analyzing creatine kinase (CK) activity.

Results

Isometric MVC decreased from the mean (±SD) initial value of 829±102N to 569±92N (IA) (p<0.01), and to 731±118N (p<0.05) on 2D. Figure 1 demonstrates that the whole force-velocity curve shifted to the lower level. SSC exercise induced no changes in the titin mRNA levels (normalized to GAPDH) and in titin isoform contents as compared to the control leg (Fig. 2). Neither any change was observed in dystrophin stainings (Fig. 2) nor in overall morphological appearance of the muscle. IL-1β mRNA values of the exercising leg were 2.4-fold (30 min) and 1.8-fold (2D) higher (p<0.05) as compared to the control leg. However, inter-individual differences were large. After the fatiguing exercise, the relative increase of CK-activity was 194±219% (2D), and the subjects reported increase (p<0.05) in DOMS of the exercised leg during the following days after the SSC fatigue.

Discussion/Conclusion

The present exhaustive SSC-exercise induced a clear decrease in force production not only acutely but also after 2D. This could not be explained either by changes in titin mRNA levels and isoforms, by individual differences in MHC isoforms, or by muscle degeneration. It is possible that in addition to difficulties in timing the muscle biopsy to correspond to the greatest “damage” effects, other factors such as connective tissue weakening (Koskinen et al. 2002) and reduced muscle activation (Nicol et al. 1996) should be considered as relevant factors to weaken the force production. However, the present study supports the study of Dennis (et al. 2004) that inflammatory response in skeletal muscle is an adaptive process to exercise.

References