Antioxidant supplementation inhibits exercise-induced skeletal muscle signaling but did not alter markers of muscle adaptations

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Acute endurance exercise induces the generation of reactive oxygen species (ROS) in muscle (Davies *et al.*, 1982). It has been shown that exercise-induced production of ROS also regulates skeletal muscle adaptations. First, Gomez-Cabrera *et al.* (2008) have reported that antioxidant vitamin C supplementation prevents the mitochondrial biogenesis induced by exercise training in rat skeletal muscle. Since then, evidence supporting that antioxidant supplementation attenuates exercise training-induced adaptations in human and rodent skeletal muscle has been published (Meier *et al.*, 2013; Ristow *et al.*, 2009). On the other hand, we and another research group have shown that antioxidant supplementation does not alter exercise training-induced adaptations (Higashida *et al.*, 2011; Strobel *et al.*, 2011). Accordingly, the effects of antioxidant supplementation on endurance exercise training-induced skeletal muscle adaptations are still not definitive.

The aim of this study was to investigate the effects of vitamin C and E supplementation on acute exercise-induced changes of markers of skeletal muscle adaptation and its signaling pathways in mice. Male C57BL/6 mice were assigned to one of four groups: a control group; exercise group; vitamin C and E supplemented group; and vitamin C and E supplemented exercise group. Mice in vitamin C and E supplemented group were given vitamin C (750 mg/kg weight/day) and vitamin E (150 mg/kg weight/day) for two weeks. One hour after the last supplementation, exercise group mice ran on a treadmill at 25 m/min, 8% grade for 120 min. Vitamin C and E supplementation attenuates exercise-induced oxidative stress. However, vitamin C and E supplementation did not alter the acute exercise-induced increase in gene expression of peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α), citrate synthase (CS) and vascular endothelial growth factor (VEGF). On the other hand, vitamin C and E supplementation prevented the phosphorylation of AMP activated kinase (AMPK) and p38 mitogen-activated protein kinase (p38 MAPK). These results suggest that reactive oxygen species (ROS) inhibits exercise-induced skeletal muscle signaling but does not alter mitochondrial biogenesis and angiogenesis in skeletal muscle.

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