Effect of vitamin C supplementation on lipid peroxidation, muscle damage and inflammation after 30-min exercise at 75% VO\(_2\)max

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Aim. Hypothetically, supplementation with the antioxidant vitamins C could alleviate exercise-induced lipid peroxidation. The purpose of this study was to evaluate the effect of vitamin C supplementation on exercise-induced lipid peroxidation, muscle damage and inflammation.

Methods. Sixteen healthy untrained male volunteers participated in a 30-min exercise at 75% \(\text{Vo2max}\). Subjects were randomly assigned to one of two groups: 1) placebo and 2) vitamin C (VC: 1 000 mg vitamin C). Blood samples were obtained prior to supplementation (baseline), 2 h after supplementation (immediately pre-exercise), post-exercise, 2 and 24 h after exercise. Plasma levels of VC, total antioxidant capacity (TAC), creatine kinase (CK), malondialdehyde (MDA), total leukocytes, neutrophils, lymphocytes, interleukin-6 (IL-6) and cortisol were measured.

Results. Plasma vitamin C concentrations increased significantly in the VC group in response to supplementation and exercise (P<0.05). TAC decreased significantly in Placebo group 24 h after exercise compared to pre-exercise (P<0.05). Although MDA levels were similar between groups at baseline, it increased significantly 2 h after exercise only in the Placebo group (P<0.05). CK increased immediately and 2 h after exercise in both groups and 24 h after exercise only in placebo group compared to pre-exercise (P<0.05). Markers of inflammation (total leukocyte counts, neutrophil counts and IL-6) were increased significantly in response to the exercise (P<0.05). In VC group, there was significant increase in lymphocyte counts immediately after exercise compared with pre-exercise (P<0.05). Serum cortisol concentrations significantly declined after supplementation compared with baseline (P<0.05) as well as declined 2 and 24 h after exercise compared with immediately after exercise in VC group (P<0.05).

Conclusion. VC supplementation prevented endurance exercise-induced lipid peroxidation and muscle damage but had no effect on inflammatory markers.

Key Words: Ascorbic acid - Exercise - Inflammation - Dietary supplementation.

S trenuous exercise causes oxidative stress resulting in lipid peroxidation \(^1\) and DNA damage.\(^4\) Evidence of protein oxidation following exercise-induced oxidative stress is less definitive.\(^5\) In response to endurance exercise, oxygen (\(O_2\)) consumption increases 10- to 20-fold systemically \(^6\) and as much as 100- to 200-fold at the level of the skeletal muscle,\(^7\) leading to substantially increased mitochondrial electron flux. Reactive oxygen species (ROS) "leaking" from the mitochondria during exercise are considered a main source of oxidative stress.\(^7\) Other potential sources of ROS during exercise include enhanced

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