Effects of Protein-based Supplement and Endurance Exercise on Muscle Mass and Oxidative Stress in Rats Treated with Hindlimb Suspension

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Abstract

Introduction: The purpose of the study was to investigate the effect of endurance exercise training with or without protein-based supplementation on muscle mass and oxidative stress in disuse skeletal muscle atrophy caused by hindlimb suspension (HS).

Methods: Thirty-two male Sprague-Dawley (SD) rats (230-280g) were weight-matched and assigned to the following 4 groups: Free control (FC), Hindlimb Suspension Placebo (H), Hindlimb Suspension + Exercise (HE), Hindlimb Suspension + Exercise + Nutrition supplementation (HEN). After 10-days hindlimb suspension (HS) period, all rats were reloaded following one day of rest, thereafter endurance exercise procedure [treadmill running at a speed of 0.6 km/h to 1.2 km/h, 25 min/day, 0% grade] and supplementation (10 ml/kg body wt of solution, containing 300 mg/kg body wt of leucine, 400 mg/kg of $\beta$-Hydroxy $\beta$-methylbutyric acid (HMB), 400 mg/kg of whey protein, 200 mg/kg of casein, 600 mg/kg of glucose) were processed during the two-weeks reloading period. At the end of intervention, the soleus muscle mass and soleus cross-section area (CSA) were measured, and the intracellular level oxidative stress status, 4-Hydroxynonenal protein were measured.

Results: After two weeks of intervention, body weight, muscle mass and muscle fiber CSA in H, HE, and HEN groups could not be restored, and these parameters were still significantly lower than those of control group ($p < 0.01$). 4-Hydroxynonenal (4-HNE) expression was no significantly different among these four groups.

Conclusions: After two weeks of intervention, body weight, muscle mass and muscle fiber CSA in H, HE, and HEN groups could not be restored, and these parameters were still significantly lower than those of control group ($p < 0.01$). 4-Hydroxynonenal (4-HNE) expression was no significantly different among these four groups.

Keywords: Fiber cross-sectional area, Citrate synthase activity, Muscle mass, PGC-1 alpha