



Exercise Biochemistry Review

Proceedings of IBEC 2018, Beijing, China, October 23-25
PO-086

Exercise enhances cardiac antioxidant capacity in diabetic rats through the Keap1/Nrf2 signaling pathway

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Objective To investigate the effects of exercise on the myocardial oxidative stress injury of diabetic rats, and discussed the role of Keap1/Nrf2 signaling pathway in this process

Methods Tyep 2 diabetic rat model was established by streptozotocin injection through abdominal cavity and high fat diet. The all the diabetic rats were divided into three groups: control group (NC), diabetes group(T2DM) and diabetes exercise group, NC and T2DM group were kept quiet for 8 weeks, T2DME group was trained for 8 weeks. After the exercise, weight, heart weight and blood were measured. MDA, T-SOD and GSH-PX enzyme were measured by biochemical method. Ho-1, Keap1, Nrf2 gene and protein expression were detected by RT-PCR and WesternBlotting.

Results Compared with NC group, the weight of rats in the T2DM group significantly decreased [(528+/-71g vs 362+/-33g), $P<0.05$], HWI significantly increased [(2.845+/-0.22 vs 3.841+/-0.21, $P<0.05$], blood glucose was significantly increased [(6.4±3.8 vs 26±7.5mmol/L), $P<0.01$], T-SOD and GSH-PX activity decreased significantly ($P<0.05$), Ho-1 protein expression increased ($P<0.01$), Keap1 and Nrf2 showed no significant changes, and Nrf2 nuclear transposition decreased ($P<0.05$). Compared with the T2DM group, no significant change in body weight and heart weight in the T2DME group, with significant decrease in HWI[(3.841±0.21 vs 3.235±0.23) , $P<0.05$], with significant decrease in blood glucose [(26.0±7.5 vs 21.0±6.8) , $P<0.05$]. Ho-1 gene and protein expression increased significantly ($P<0.05$ and $P<0.01$) , with no significant change of Keap1, while Nrf2 expression increased significantly ($P<0.05$), and Nrf2 nuclear transposition increased significantly ($P<0.01$).

Conclusions Exercise activates the myocardial Keap1/Nrf2 signaling pathway in rats, promotes the expression of downstream antioxidant enzymes, increases cardiac antioxidant capacity, and resists diabetic myocardial oxidative stress injury.