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

Shiqiang Wang
Hunan University of Technology

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.