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Exercise Training Protects Against Cardiac Pathological Remodeling in Myocardial Infarction rats via Improving Mitochondrial Biogenesis

Dandan Jia, Zhenjun Tian
Institute of Sports and Exercise Biology, Shaanxi Normal University

Objective Growing evidence suggests that exercise training reverses cardiac pathological remodeling and cardiac dysfunction during myocardial infarction (MI), but the underlying mechanisms have not been fully understood. In this study, we investigated the impact of exercise training on cardiac function, myocardial fibrosis, apoptosis, oxidative stress and mitochondrial biogenesis.

Methods Sprague Dawley rats were subjected to MI by permanent ligation of the left anterior descending (LAD) coronary artery or Sham operation. Rats with MI were randomly assigned to sedentary MI group (MI) and MI with exercise training group (MI+EX), and compared to sham-operated group (Sham). Haemodynamics and Masson staining were conducted to evaluate the effect of exercise training on cardiac function and myocardial fibrosis. Myocardial apoptosis, oxidative stress, mitochondrial biogenesis and molecular signaling mechanism were analyzed.

Results Exercise training significantly improves cardiac function and mitigates the MI-induced cardiac pathological remodeling. Meanwhile, Exercise training significantly attenuates MI-induced apoptosis, oxidative stress and mitochondrial biogenesis. In addition, activation of PI3K pathway following MI is further induced by exercise training.

Conclusions Exercise training protects against MI-induced cardiac dysfunction and pathological remodeling through preventing myocardial apoptosis and oxidative stress, and enhancing mitochondrial biogenesis.