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### The Effects of Interval Exercise on oxidative stress and Smyd1-related myocardial hypertrophy in Rats with Myocardial Infarction

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**Objective** This study was carried out to investigate interval exercise on Smyd1 expression and F-actin sarcomere assembly in non-infarcted myocardium of normal and myocardial infarction (MI) rats and its possible mechanism.

**Methods** Male SD rats were randomly divided into normal control group (C), normal interval exercise group (CE), sham-operated group (S), MI group (MI), MI with interval exercise group (ME) and MI with ROS Tempol group (MT), n=10. MI model was established by left anterior descending coronary artery ligation. Interval exercise was carried out on a small animal treadmill. MT group was given an oral solution of Tempol (2mmol/L). Hemodynamics was performed to evaluate cardiac function. HE and Masson staining were used to analyze the cross-sectional area (CSA) of cardiomyocytes and collagen volume fraction, respectively. T-SOD and MDA kits were used to detect oxidative stress. H9C2 cells were treated with H<sub>2</sub>O<sub>2</sub>. Immunofluorescence staining was used to determine Smyd1 expression and F-actin sarcomere assembly. RT-qPCR and Western blotting were used to detect the gene or protein expression of Smyd1, Trx1, Hsp90, MuRF1, cTnI,  $\alpha$ -actinin and BNP.

**Results** Smyd1, Trx1, Hsp90, MuRF1 and BNP expression in the peri-infarcted area were up-regulated, but cTnI and  $\alpha$ -actinin expression and F-actin assembly were decreased. The cardiac function was reduced. Both interval exercise and Tempol intervention significantly increase the CSA and expression of Smyd1, Trx1, cTnI and  $\alpha$ -actinin, improve the antioxidation capacity and F-actin sarcomere assembly and cardiac function, reduce the expression of Hsp90, MuRF1, BNP and ROS level, and inhibit the fibrosis of myocardium. The oxidative stress level was closely related to the Smyd1 expression. Improvement of cardiac function were correlated with Smyd1 expression. H<sub>2</sub>O<sub>2</sub> can induce oxidative stress injuries of H9C2, and its closely related to cardiomyocytes oxidative stress level and Smyd1 expression.

**Conclusions** Interval exercise could promote antioxidant capability and physiological cardiomyocyte hypertrophy, regulate the expression of Smyd1, Hsp90 and MuRF1 in infarcted heart; so as to improve the cardiac function. Smyd1 may participate in pathologic hypertrophy of cardiomyocytes caused by oxidative stress.