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Effect of Exercise-Induced Skeletal Muscle FSTL1 on Cardiac Structure and Function in Myocardial Infarction Rats

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Objective The aim of this study is to investigate the effect of skeletal muscle-derived FSTL1 on cardioprotection in myocardial infarction rats after resistance exercise or tibialis anterior muscle injection of follistatin-like protein 1 (FSTL1) adeno-associated virus vector and its possible signaling mechanisms.
Methods The male Sprague-Dawley rats were randomly divided into five groups (n=10): Sham-operated group(S), sedentary MI group (MI), MI with resistance exercise group (MR), MI with empty adeno-associated virus (AAV) vector group (MV) and MI with FSTL1-AAV group (MF) after the MI model established which was induced by left anterior descending (LAD) coronary artery ligation. S group underwent threading without ligation. 1 week post MI, rats in MR group underwent resistance exercise for 4 weeks, rats in MV and MF group were injected AAV empty vector and FSTL1-AAV in the tibialis anterior muscle of the left limb, respectively. The next day after exercise, rats were anesthetized and heart function was measured. Collagen volume fraction(%) of myocardium were observed and calculated by Masson staining; cardiomyocyte proliferation was measured by immunofluorescence; cardiomyocyte apoptosis was detected by TUNEL staining; The protein expression of skeletal muscle and serum FSTL1 and myocardium FSTL1, DIP2A, pAkt/Akt, p-mTOR/mTOR, CyclinD1, CDK4 and Bcl2/Bax in myocardium were measured by Western blotting.
Results The skeletal muscle FSTL1 protein expression was decreased but the serum and myocardium FSTL1 were upregulated in MI group. The myocardium fibrosis, cardiomyocyte proliferation and cardiomyocyte apoptosis were increased and the heart function was declined after MI. After resistance exercise or tibialis anterior muscle injection of FSTL1-AAV, the skeletal muscle, serum and myocardium FSTL1 protein expression were significantly increased, and there was a significant positive correlation between each data. Myocardium fibrosis and cardiomyocyte apoptosis were also decreased, cardiomyocyte proliferation was increased and the heart function was significantly improved after FSTL1-AAV injection.
Conclusions Resistance exercise increases skeletal muscle FSTL1 expression. Skeletal muscle-derived FSTL1 can reach the heart through blood circulation, promote cardiomyocyte proliferation, inhibit cardiomyocyte apoptosis, reduce myocardium fibrosis and improve heart function in MI rats. Myocardium FSTL1 binds to its receptor, DIP2A, and activates the Akt-mTOR signaling pathway might be the potential mechanism of this protective effect.