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VEGF-B inhibits skeletal muscle apoptosis after exercise in Chronic heart failure rats

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Objective To investigate the effects of vascular endothelial growth factor B in aerobic exercise mediated chronic heart failure rats cardiac function improvement and skeletal muscle remodeling.
Methods We employed transverse abdominal aortic constriction (TAC) inducing CHF in Sprague Dawley rats. Controls were sham-operated animals. At 4 weeks after surgery, rats were randomized to 4 weeks of aerobic exercise (CHF+E) or to untrained groups (CHF). After 8 weeks, all rats went echocardiography test. After which, rats were sacrificed and samples were collected. Muscular cytokine VEGFB and its receptor NRP1 expression were analyzed. Expression of apoptosis and muscle atrophy markers were assessed in cardiac muscle、gastrocnemius.
Results TAC rats developed CHF (preserved LV ejection fraction, hypertrophy of myocardial cells, decreased FS, increased LVAW d and LVID s). Exercise ameliorate CHF rat cardiac function. TAC rat skeletal muscle developed irregular muscle fiber distribution.The two atrophy-related ubiquitin ligases atrogin-1 and MuRF1, as well as genes involved in apoptosis and autophagy were upregulated in muscles in CHF rats. Exercise inhibited muscle atrophy and skeletal muscle apoptosis.VEGFB and its receptor NRPI decreased significantly in CHF muscle. Exercise promoted VEGFB and NRP1 expression in cardiac tissue, gastrocnemius.
Conclusions Exercise ameliorates CHF rat cardiac function. VEGFB inhibits cardiac muscle and gastrocnemius apoptosis in CHF rats.