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ELABELA Facilitates Exercise-Induced Cardioprotection in Post-Infarction Cardiac Remodeling

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Objective Exercise has been reported to have positive function after myocardial infarction (MI), but the mechanisms of exercise-induced cardioprotection are poorly understood. ELABELA (ELA) is a hormone and plays a vital role in cardiovascular system. The aim of this research is to investigate the effect and the mechanisms of exercise and exogenous ELA administration on cardioprotection in MI rat.
Methods MI was established by left coronary artery ligation. MI rats underwent 4 weeks of sustainedaerobic exercise and Fc-ELA-21 or Fc-ELA-32 injection. H9C2 cells were treated by ELA-14 peptide. Akt signal was inhibited by LY294002. Heart function was evaluated by hemodynamics; Myocardium fibrosis, cell proliferation, angiogenesis and apoptosis were visualized by Masson's staining, immunohistochemistry and TUNEL, respectively; Protein expression was quantified by Western blotting.
Results Exercise induced angiogenesis and cardiomyocyte proliferation, reduced fibrosis and improved the function of MI heart. Fc-ELA-21 injection further facilitated the exercise-induced cardioprotection effects. APJ expression was up-regulated and Akt-mTOR-P70S6K signal was activated by both exercise and Fc-ELA-21. In H9C2 cells, ELA-14 activated Akt signal by dose dependent manner. Akt signal inhibitioninvalidated ELA-14 function. Fc-ELA-32 directly improved structural and functional recovery of post-MI hearts.
Conclusions ELA is a heart protector and facilitates exercise-induced cardioprotectionthrough Akt-mTOR-P70S6K signal inpost-infarction cardiac remodeling.