Post-myocardial infarction exercise training improved calcium sensitivity and cardiac function

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After myocardial infarction (MI), the wounded heart undergoes detrimental remodeling, which induces ventricular dilation, fibrosis, and deteriorated cardiac function. Previous studies reported Post-MI exercise training attenuated remodeling-induced adverse effect. The purpose of this study was to investigate whether post-MI endurance training (ET) improves cardiomyocytes Ca^{2+} sensitivity. MI was surgically induced in 7-wk-old rats. The survivors were assigned to 3 groups: Sham (no MI, no exercise; n=7), MI-Sed (MI, no exercise; n=7), and MI-ET (MI, plus endurance training). Exercise training began 1-week after surgery for MI-ET. MI-ET exercise entailed 10-16m/min running on a rodent treadmill inclined to 5° for 50 minutes per day, 5 days per week for a total of 10 weeks. Animals were treated in accordance with NIH Gide for the Care and Use of laboratory Animals. During the experiment, rats were anesthetized with 2% isoflurane mixed with oxygen. Buprenex (pain killer, 0.05mg/kg body weight) was administered subcutaneously for two days after surgery. Our results showed that both MI-Sed and ET had comparable left ventricular end-diastolic dimension (LVEDd, 11.81±0.10 *vs* 11.84±0.15mm, p>0.05). MI-ET had shorter (P<0.05) LV end systolic-dimension (LVESd) than their sedentary counterparts (9.71±0.26 *vs* 10.46±0.14mm). As such, the fractional shortening (FS%) was higher (P<0.05) in MI-ET (17.99±1.54%) than that of MI-Sed (11.43± 1.12%). The pCa50, a measure of Ca^{2+} sensitivity of tension, was higher in MI-ET than that of MI-Sed (5.9±0.06 *vs* 5.75±0.06). These results suggest that post-MI endurance training improved cardiac function. The increased cardiac contractility may be, in part, due to the enhanced Ca^{2+} sensitivity of the cardiomyocytes.