

Development and complete morphological and functional reversibility of athletes heart in a rat model

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Purpose: Long-term exercise training is associated with characteristic structural and functional cardiac adaptation termed athlete's heart. However the effect of discontinuation of the training (detraining) on left ventricular (LV) function is still unclear. Our aim was to evaluate the development characteristics of athlete's heart and the reversibility of morphological and functional changes during detraining.

Methods: Rats were divided into trained (n=15) and control (n=17) groups. Trained rats swam 200 min/day for 12 weeks, while control rats were taken into the water for 5 min/day. Detrained rats remained sedentary for 8 weeks after completion of the training protocol. We regularly performed echocardiographic measurements to investigate development and regression of exercise-induced cardiac changes. LV pressure-volume analysis was performed to calculate cardiac functional parameters. LV samples were harvested for histological examination. Myocardial gene expression analysis was performed using qRT-PCR.

Results: Echocardiographic examinations showed rapidly developing LV hypertrophy in the trained group according to wall thickness values (LVmass index after training period: 2.45 ± 0.09 vs. 2.07 ± 0.07 g/ttkg, $p < 0.05$). This adaptation regressed after detraining (LVmass index: 1.94 ± 0.02 vs. 2.05 ± 0.05 g/ttkg, $p = 0.0634$), which was confirmed by post-mortem measured heart weight and histological morphometry. Unchanged myocardial expression of TGF- β and β -MHC and unaltered amount of LV collagen confirmed the physiologic nature of the observed cardiac hypertrophy. Hemodynamic measurements indicated decreased LV end-systolic volume (LVESV: 75 ± 5 vs. 100 ± 7 μ l, $p < 0.05$) along with unchanged end-diastolic volume (EDV), improved systolic function and contractility (slope of the dP/dtmax-EDV relationship: 35.9 ± 2.6 vs. 25.8 ± 2.8 Hgmm/s/ μ l, $p < 0.05$), ameliorated active relaxation and mechanoenergetics (mechanical efficiency: 53 ± 2 vs. $45 \pm 2\%$, $p < 0.05$) after long-term exercise training. After the detraining period regression of exercise-induced cardiac functional changes were observed: LVESV (117 ± 5 vs. 115 ± 6 μ l, $p = 0.7846$), active relaxation, LV contractility (slope of the dP/dtmax-EDV relationship: 30.5 ± 1.7 vs. 28.4 ± 4.3 Hgmm/s/ μ l, $p = 0.5871$) and mechanoenergetic (mechanical efficiency: 50 ± 2 vs. $48 \pm 5\%$, $p = 0.7760$) enhancement reverted completely to control values. Training and detraining did not affect myocardial stiffness.

Conclusions: Our results confirm that the morphological and functional properties of exercise-induced physiologic LV hypertrophy completely regressed after an eight week detraining period.