Background: Exercise is considered a method to induce physiological hypertrophy. However, human studies identified evidence for myocardial damage after intense endurance exercise in athletes. We compared two exercise protocols in rats, differing in their intensity, and assessed cardiac function in vivo and respiratory capacity of isolated mitochondria.

Methods: We subjected rats to an incremental training protocol for 10 weeks with either 10% or 16% incline. At 2, 6, and 10 weeks we performed echocardiography and assessed maximal respiratory capacity in isolated mitochondria.

Results: Within ten weeks animals trained with 16% incline developed hypertrophy (LVPWD: 1.6±0.1 vs. 2.4±0.1mm; p<0.05) with normal function (Ejection fraction: 75.2±2.5 vs. 75.6±2.1%; n.s.). However, after 6 weeks there was a temporary impairment in contractile function (EF: 74.5±1.67 vs. 65.8±2.3%; p<0.05; eFS%: 44.92±1.53 vs. 38.12±2.04%; p<0.05), which was associated with decreased maximal respiratory capacity of isolated mitochondria (state 3 respiration: 326±71 vs. 161±22 natsomsO/min/mg; p<0.05) and a gene expression shift from the adult (α) to the fetal (β) isoform of myosin heavy chain (α MHC: from 1.49±0.27 to 0.62±0.07; p<0.05; β MHC: from 0.38±0.06 to 0.64±0.28; p<0.05). Interestingly, the heart failure markers ANF and BNP were reduced at all three time points. In contrast, at 10% incline, there was no hypertrophy at ten weeks (LVPWD: 1.7±0.1 vs. 1.8±0.1mm; n.s.), normal function (EF: 74.3±1.5 vs. 69.0±1.0%; n.s.; eFS%: 45.3±1.4 vs. 40.0±0.8%; n.s.) and maximal respiratory capacity was normal at all time points.

Conclusions: At high intensity, exercise may cause reversible, but significant impairment in cardiac contractile function associated with reduced maximal respiratory capacity and pathological alterations in gene expression. These findings should be considered when selecting an exercise protocol aimed at the induction of physiological hypertrophy.