# DOES PRIOR HEAT ACCLIMATION REDUCE MYOCARDIAL INJURY AFTER ACUTE MYOCARDIAL INFARCTION?

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Previous studies have shown that acute heat stress may potentially improve myocardial tolerance to ischemia. This phenomenon was attributed to the expression of heat shock proteins that protect the cells against ischemic damage. In contrast to ischemia, there is no existing information about the effect of heat acclimation on myocardial tolerance to acute myocardial infarction (AMI). This study aimed to evaluate whether prolonged heat acclimation reduces myocardial injury after AMI. The study was approved by the Institutional Animal Ethics Committee. 30 male Sprague Dawly rats weighing 360g were divided into 2 groups: passively heat acclimated for 4 weeks in a heat chamber (34°C, 30%RH) (H, n=15), and 4 weeks of comfort conditions (25°C, 40%RH) (C, n=15). Thereafter AMI was surgically induced in both groups under general anaesthesia (xylazine 10mg/kg and ketamine 90mg/kg). They recovered in comfort conditions for 4 more weeks and were then sacrificed. Body weight and rectal temperature were measured at baseline and after the first 4 weeks. Following the sacrifice, transverse serial sections of the hearts were stained with Mason Trichrome and Hematoxilin Eosin for morphometric measurements that included: necrosis area, relative necrosis area, LV cavitary area, LV muscle area and average LV wall thickness. No differences were found in increase in body weight between H and C groups after 4 weeks of heat acclimation and comfort conditions (20±15g and 30±10g, respectively). Rectal temperature measured one day after the 4 weeks of acclimation/comfort period decreased by 0.6±0.5°C in group H and by only 0.17±0.5°C in group C ($P<0.05$). Myocardial necrosis area and myocardial relative necrosis area were lower in the H group compared with the C group (9±4mm$^2$ compared to 12.5±5mm$^2$ and 24.9±11.8% compared with 30.4±8.6%, respectively) although not significantly. LV cavitary area was lower in the H group compared with the C group (56.9±6.4mm$^2$ and 70.7±16.6mm$^2$, respectively) but not significantly. Average wall thickness was slightly higher in group C compared with group H although not significantly (0.5±0.2mm and 0.4±0.1mm, respectively). All the animals in the C group were successfully heat acclimated. Although not significant, better LV morphometry after AMI in this group suggests that prior heat acclimation may activate mechanisms that reduce myocardial injury after AMI. Nevertheless, further studies should be done (with increased sample population) in order to establish or refute these results.

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