Attenuating heatstroke-induced acute lung inflammation, edema, and injury in rats by exercise preconditioning.

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Abstract

BACKGROUND:

This study aimed to ascertain whether heat-induced acute lung edema, inflammation, and ischemic damage can be affected by heat shock protein 70 (HSP-70)-mediated exercise preconditioning (EP) in rats.

METHODS:

Wistar rats were assigned to one of the following four groups: the non-EP + nonheated group, the non-EP + heated group, the EP + heated group, and the EP + HSP-70 antibodies + heated group. EP groups of animals were subjected to a protocol of running on a treadmill for 30 minutes at 20 m/min, 30 minutes at 30 m/min, and 60 minutes at 30 m/min after 1, 2, and 3 weeks of training, respectively. Heated group of animals, under general anesthesia, were put in a folded heating pad of 43°C for 68 minutes. Then, the heated animals were allowed to recover at room temperature. HSP-70 antibodies were injected intravenously 24 hours before heat exposure.

RESULTS:

As compared with the non heated + non-EP rats, the heated + non-EP rats had significantly higher scores of alveolar edema, neutrophil infiltration, and hemorrhage,
acute pleurisy, and increased bronchoalveolar fluid levels of proinflammatory cytokines and ischemic and oxidative damage markers. EP, in addition to inducing overexpression of HSP-70 in lung tissues, significantly attenuated heat-induced acute pulmonary edema, inflammation, and ischemic and oxidative damage in the lungs. HSP-70 antibodies, in addition to reducing HSP-70 expression in the lungs, significantly attenuated the beneficial effects of EP in reducing acute lung inflammation and injury.

CONCLUSION:

EP may attenuate the occurrence of pulmonary edema, inflammation, as well as ischemic and oxidative damage caused by heatstroke by up-regulating HSP-70 in the lungs.