1: Crit Care Med. 1997 Oct;25(10):1727-32.

Comment in:
Crit Care Med. 1997 Oct;25(10):1636.

Heat stress increases survival rates in lipopolysaccharide-stimulated rats.

Chu EK, **Ribeiro SP**, **Slutsky AS**.

Department of Medicine, Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Toronto, ON, Canada.

OBJECTIVE: To examine the hypothesis that heat stress applied after the administration of bacterial endotoxin is protective. DESIGN: Prospective, randomized, laboratory study. SETTING: University research laboratory. SUBJECTS: One hundred eleven adult male Sprague-Dawley rats (weight range 250 to 400 g). INTERVENTIONS: Production of endotoxemia by the administration of a bacterial endotoxin and exposure to heat stress by heating animals in a neonatal incubator until their rectal temperatures reached 105.8 degrees F (41 degrees C). MEASUREMENTS AND MAIN RESULTS: The rats (n = 111) were anesthetized and were injected with 15 mg/kg of Escherichia coli endotoxin (lipopolysaccharide, LPS) intravenously to produce septic shock. Immediately thereafter, a set of 50 rats were randomly assigned to one of two treatment groups: a) LPS-treated (control); or b) LPS-treated and heated to 105.8 degrees F (41 degrees C). The animals were then observed for the development of fever, and survival rates were monitored for 72 hrs. In another set of 40 animals, the same experimental protocol was used to determine plasma cytokine concentrations in heated and nonheated groups. Blood samples were obtained at 0, 2, 4, or 6 hrs after LPS injection for tumor necrosis factor-alpha and interleukin (IL)-1 beta detection. In a third set of animals, the same experimental protocol was applied to nine animals for the detection of heat-shock proteins of 72-kilodalton molecular weight. LPS injection in the control group did not produce fever. Heat stress increased the abundance of heat-shock proteins of 72-kilodalton molecular weight in the rats' lungs (analysis of variance, p = .016). Twelve hours after the initiation of sepsis, the survival rates of the control group injected with LPS alone and the group heated to 105.8 degrees F (41 degrees C) were 48% and 80%, respectively (p = .039). The peak plasma IL-1 beta concentrations occurring at 2 hrs after LPS injection were significantly reduced in rats heated to 105.8 degrees F (41 degrees C) when compared with nonheated rats (p = .003). CONCLUSION: We conclude that heat stress applied after the initiation of endotoxemia can provide protection against an otherwise lethal stimulus and that the mechanism of protection may be related to the attenuation of plasma IL-1 beta concentrations.

PMID: 9377890 [PubMed - indexed for MEDLINE]