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Acute heat stress protects rats against endotoxin shock.

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The purpose of this study was to determine 1) whether prior (24-h) heat stress could render rats cross-resistant to the lethal activity of bacterial lipopolysaccharide (LPS) and 2) whether this acquired state of resistance is associated with endotoxemia during the heat stress event. Four groups (n = 7/group) of rats were examined: 1) saline treated, 2) LPS treated, 3) heat stressed and saline treated, and 4) heat stressed and LPS treated. Saline or LPS (Escherichia coli, serotype 0111:B4, 20 mg/kg body wt) was given intravenously 24 h after exposure to heat (ambient temperature 47-50 degrees C, relative humidity 30%) for heat-stressed rats and at the same time of day for nonheated rats; survival was monitored for 48 h. Thermal responses were similar (P > 0.05); values for maximum core temperature (Tc) and time above Tc of 40 degrees C were 42.7 +/- 0.1 and 42.6 +/- 0.1 degrees C (SE) and 44.0 +/- 2.1 and 47.9 +/- 3.7 (SE) min for the heat-stressed saline-treated and heat-stressed LPS-treated rats, respectively. Administration of LPS to nonheated rats resulted in 71.4% (5 of 7 rats) lethality. In contrast, all (7 of 7) rats subjected to a single nonlethal heat stress event 24 h before LPS treatment survived (P < 0.05). Endotoxin was not detected in arterial plasma immediately after heat stress in rats (n = 6) exposed to a Tc of 42.9 +/- 0.1 degrees C. These findings demonstrate that acute heat stress can protect rats from the lethal activity of LPS.

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