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Saturated hydrogen saline attenuates endotoxin-induced lung dysfunction

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Abstract

Background: Acute lung injury induced by lipopolysaccharides (LPSs) is caused by pulmonary inflammation and pulmonary vascular permeability. Activation of p38 mitogen-activated protein kinase causes inflammation, and proinflammatory cytokines and oxidative stress induce autophagy, a catabolic mechanism responsible for protein degradation and recycling of damaged proteins and cytoplasmic organelles. If not controlled, excessive autophagy responses can result in cell death.

Materials and methods: In this study, we pretreated rats with saturated hydrogen saline, and examined the molecular mechanism by which saturated hydrogen saline attenuates LPS-induced acute lung dysfunction. Sixty-four male Sprague-Dawley rats were randomly assigned to one of three groups--a control group, an LPS group, or an LPS plus saturated hydrogen saline (LPS + H2) group.

Results: Treatment with saturated hydrogen saline prolonged the median survival time of rats and reduced lung dysfunction induced by LPS. Moreover, saturated hydrogen saline significantly attenuated LPS-mediated induction of serum tumor necrosis factor α, interleukin 6, myeloperoxidase, and malondialdehyde ($P < 0.05$).

Conclusions: Autophagosomes were found in the cytoplasm of type II alveolar epithelial cells of LPS-treated rats, and light chain 3 protein (LC3)I/II was increased by LPS treatment. In contrast, saturated hydrogen saline decreased the number of autophagosomes and LC3I/II expression. Saturated hydrogen saline also attenuated the LPS-mediated increase in apoptosis and p38 expression. Taken together, saturated hydrogen saline may attenuate LPS-induced acute lung dysfunction in rats by reducing inflammation, autophagy, and apoptosis involving the p38 mitogen-activated protein kinase signaling pathway.

Keywords: Acute lung injury; Apoptosis; Autophagy; Lipopolysaccharide; Saturated hydrogen saline; p38MAPK.

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