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Hydrogen alleviates hyperoxic acute lung injury related endoplasmic reticulum stress in rats through upregulation of SIRT1

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Abstract

Hyperoxic acute lung injury (HALI) is a major clinical problem for patients undergoing supplemental oxygen therapy. Currently in clinical settings there exist no effective means of prevention or treatment methods. Our previous study found that: hydrogen could reduce HALI, as well as oxidative stress. This research will further explore the mechanism underlying the protective effect of hydrogen on oxygen toxicity. Rats were randomly assigned into three experimental groups and were exposed in a oxygen chamber for 60 continuous hours: 100% balanced air (control); 100% oxygen (HALI); 100% oxygen with hydrogen treatment (HALI + HRS). We examined lung function by wet to dry ratio of lung, lung pleural effusion and cell apoptosis. We also detected endoplasmic reticulum stress (ERS) by examining the expression of CHOP, GRP78 and XBP1. We further investigated the role of Sirtuin 1 (SIRT1) in HALI, which contributes to cellular regulation including ERS, by examining its expression after hydrogen treatment with SIRT1 inhibitor. Hydrogen could significantly reduce HALI by reducing lung edema and apoptosis, inhibiting the elevating of ERS and increased SIRT1 expression. By inhibition of SIRT1 expression, the effect of hydrogen on prevention of HALI is significantly weakened, the inhibition of the ERS was also reversed. Our findings indicate that hydrogen could reduce HALI related ERS and the mechanism of hydrogen may be associated with upregulation of SIRT1, this study reveals the molecular mechanisms underlying the protective effect of hydrogen, which provides a new theoretical basis for clinical application of hydrogen.

Keywords: Hydrogen; hyperoxic acute lung injury; Sirtuin 1; endoplasmic reticulum stress.

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