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Hydrogen inhalation reduced epithelial apoptosis in ventilator-induced lung injury via a mechanism involving nuclear factor-kappa B activation

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Abstract

We recently demonstrated the inhalation of hydrogen gas, a novel medical therapeutic gas, ameliorates ventilator-induced lung injury (VILI); however, the molecular mechanisms by which hydrogen ameliorates VILI remain unclear. Therefore, we investigated whether inhaled hydrogen gas modulates the nuclear factor-kappa B (NFκB) signaling pathway. VILI was generated in male C57BL6 mice by performing a tracheostomy and placing the mice on a mechanical ventilator (tidal volume of 30 ml/kg or 10 ml/kg without positive end-expiratory pressure). The ventilator delivered either 2% nitrogen or 2% hydrogen in balanced air. NFκB activation, as indicated by NFκB DNA binding, was detected by electrophoretic mobility shift assays and enzyme-linked immunosorbent assay. Hydrogen gas inhalation increased NFκB DNA binding after 1h of ventilation and decreased NFκB DNA binding after 2h of ventilation, as compared with controls. The early activation of NFκB during hydrogen treatment was correlated with elevated levels of the antiapoptotic protein Bcl-2 and decreased levels of Bax. Hydrogen inhalation increased oxygen tension, decreased lung edema, and decreased the expression of proinflammatory mediators. Chemical inhibition of early NFκB activation using SN50 reversed these protective effects. NFκB activation and an associated increase in the expression of Bcl-2 may contribute, in part, to the cytoprotective effects of hydrogen against apoptotic and inflammatory signaling pathway activation during VILI.

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