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Hydrogen gas reduces hyperoxic lung injury via the Nrf2 pathway in vivo

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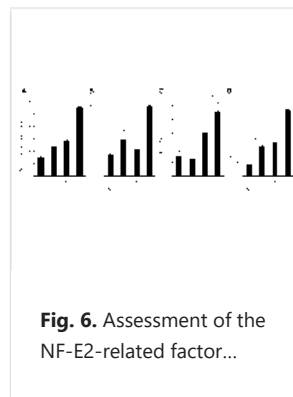
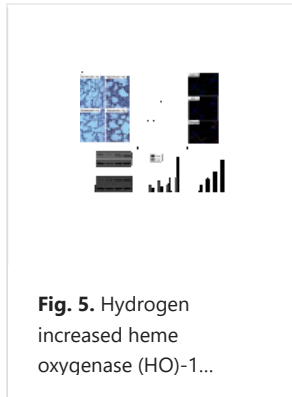
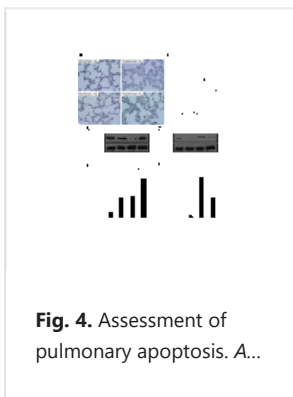
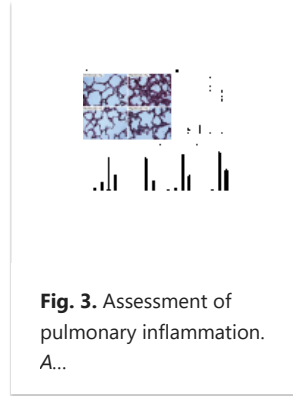
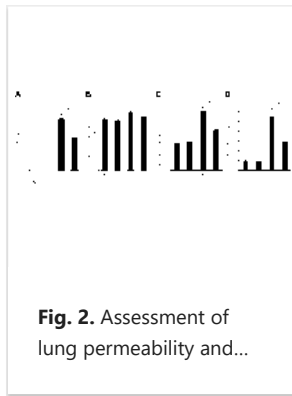
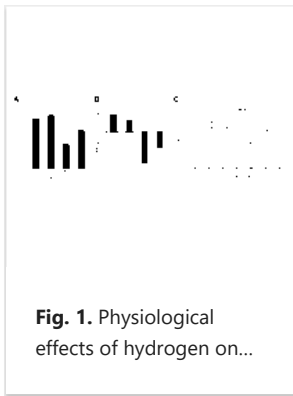
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Abstract

Hyperoxic lung injury is a major concern in critically ill patients who receive high concentrations of oxygen to treat lung diseases. Successful abrogation of hyperoxic lung injury would have a huge impact on respiratory and critical care medicine. Hydrogen can be administered as a therapeutic medical gas. We recently demonstrated that inhaled hydrogen reduced transplant-induced lung injury and induced heme oxygenase (HO)-1. To determine whether hydrogen could reduce hyperoxic lung injury and investigate the underlying mechanisms, we randomly assigned rats to four experimental groups and administered the following gas mixtures for 60 h: 98% oxygen (hyperoxia), 2% nitrogen; 98% oxygen (hyperoxia), 2% hydrogen; 98% balanced air (normoxia), 2% nitrogen; and 98% balanced air (normoxia), 2% hydrogen. We examined lung function by blood gas analysis, extent of lung injury, and expression of HO-1. We also investigated the role of NF-E2-related factor (Nrf) 2, which regulates HO-1 expression, by examining the expression of Nrf2-dependent genes and the ability of hydrogen to reduce hyperoxic lung injury in Nrf2-deficient mice. Hydrogen treatment during exposure to hyperoxia significantly improved blood oxygenation, reduced inflammatory events, and induced HO-1 expression. Hydrogen did not mitigate hyperoxic lung injury or induce HO-1 in Nrf2-deficient mice. These findings indicate that hydrogen gas can ameliorate hyperoxic lung injury through induction of Nrf2-dependent genes, such as HO-1. The findings suggest a potentially novel and applicable solution to hyperoxic lung injury and provide new insight into the molecular mechanisms and actions of hydrogen.

Keywords: NF-E2-related factor 2; heme oxygenase; hydrogen; inflammation.

Figures



All figures (8)

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