J Thorac Cardiovasc Surg. 2015 Sep;150(3):645-54.e3. doi: 10.1016/j.jtcvs.2015.05.052.
Epub 2015 May 22.

Hydrogen ameliorates pulmonary hypertension in rats by anti-inflammatory and antioxidant effects

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PMID: 26095621 DOI: 10.1016/j.jtcvs.2015.05.052

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Abstract

Objective: The pathogenesis of pulmonary arterial hypertension (PAH) involves reactive oxygen species and inflammation. Beneficial effects of molecular hydrogen, which exerts both anti-inflammatory and antioxidative effects, have been reported for various pathologic conditions. We therefore hypothesized that molecular hydrogen would improve monocrotaline (MCT)-induced PAH in rats.

Methods: Nineteen male Sprague-Dawley rats (body weight: 200-300 g) were divided into groups, receiving: (1) MCT + hydrogen-saturated water (group H); (2) MCT + dehydrogenized water (group M); or (3) saline + dehydrogenized water (group C). Sixteen days after substance administration, we evaluated hemodynamics, harvested the lungs and heart, and performed morphometric analysis of the pulmonary vasculature. Macrophage infiltration, antiproliferating cell nuclear antigen-positive cells, 8-hydroxy-deoxyguanosine (8-OHdG)-positive cells, and expressions of phosphorylated signal transducers and activators of transcription-3 (STAT3) and nuclear factor of activated T-cells (NFAT) were evaluated immunohistochemically. Stromal cell-derived factor-1 and monocyte chemoattractant protein-1 expressions were evaluated by quantitative reverse-transcription polymerase chain reaction.

Results: Pulmonary arterial hypertension was significantly exacerbated in group M compared to group C, but was significantly improved in group H. Vascular density was significantly reduced in group M, but not in group H. Adventitial macrophages, antiproliferating cell nuclear antigen - and 8-OHdG-positive cells, and stromal cell-derived factor-1 and monocyte chemoattractant protein-1 expressions were significantly increased in group M, but improved in group H. Expressions of phosphorylated STAT3 and NFAT were up-regulated in group M, but improved in group H.

Conclusions: Molecular hydrogen ameliorates MCT-induced PAH in rats by suppressing macrophage accumulation, reducing oxidative stress and modulating the STAT3/NFAT axis.

Keywords: NFAT; STAT3; molecular hydrogen; pulmonary arterial hypertension; reactive oxygen species.

Comment in

Molecular hydrogen: A novel therapy for the treatment of pulmonary hypertension.
Lazar HL.

J Thorac Cardiovasc Surg. 2015 Sep;150(3):654-5. doi: 10.1016/j.jtcvs.2015.06.004. Epub 2015 Jun 11.
PMID: 26123397 No abstract available.

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