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Free Radic Res. 2015;49(8):1026-37. doi: 10.3109/10715762.2015.1038257. Epub 2015 May 7.

Maternal molecular hydrogen treatment attenuates lipopolysaccharide-induced rat fetal lung injury

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PMID: 25947958 DOI: 10.3109/10715762.2015.1038257

Abstract

Maternal inflammation is associated with spontaneous preterm birth and respiratory impairment among premature infants. Recently, molecular hydrogen (H2) has been reported to have a suppressive effect on oxidative stress and inflammation. The aim of this study was to evaluate the effects of H2 on fetal lung injury caused by maternal inflammation. Cell viability and the production of interleukin-6 (IL-6) and reactive oxygen species (ROS) were examined by treatment with lipopolysaccharide (LPS) contained in ordinal or H2-rich medium (HM) using a human lung epithelial cell line, A549. Pregnant Sprague Dawley rats were divided into three groups: Control, LPS, and HW + LPS groups. Rats were injected with phosphate-buffered saline (Control) or LPS intraperitoneally (LPS) on gestational day 19 and provided H2 water (HW) ad libitum for 24 h before LPS injection (HW + LPS). Fetal lung samples were collected on day 20, and the levels of apoptosis, oxidative damage, IL-6, and vascular endothelial growth factor (VEGF) were evaluated using immunohistochemistry. The number of apoptotic cells, and levels of ROS and IL-6 were significantly increased by LPS treatment, and repressed following cultured with HM in A549 cells. In the rat models, the population positive for cleaved caspase-3, 8-hydroxy-2'-deoxyguanosine, IL-6, and VEGF was significantly increased in the LPS group compared with that observed in the Control group and significantly decreased in the HW + LPS group. In this study, LPS administration induced apoptosis and oxidative damage in fetal lung cells that was ameliorated by maternal H2 intake. Antenatal H2 administration may decrease the pulmonary mobility associated with inflammation in premature infants.

Keywords: IL-6; lipopolysaccharide; lung; reactive oxygen species.

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