



# Protective effects of hydrogen-rich medium on lipopolysaccharide-induced monocytic adhesion and vascular endothelial permeability through regulation of vascular endothelial cadherin

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**ABSTRACT.** We observed the effect of hydrogen-rich medium on lipopolysaccharide (LPS)-induced human umbilical vein endothelial cells (HUVECs), hyaline leukocyte conglutination, and permeability of the endothelium. Endotheliocytes were inoculated on 6-well plates and randomly divided into 4 groups: control, H<sub>2</sub>, LPS, LPS+H<sub>2</sub>, H<sub>2</sub>, and LPS+H<sub>2</sub> in saturated hydrogen-rich medium. We applied Wright's staining to observe conglutination of hyaline leukocytes and HUVECs, flow cytometry to determine the content of vascular cell adhesion protein 1 (VCAM-1) and intercellular adhesion molecule 1 (ICAM-1), enzyme-linked immunosorbent assay to measure the E-selectin concentration in the cell liquor, the transendothelial electrical resistance (TEER) to test the permeability of endothelial cells, and Western blot and im-

munofluorescence to test the expression and distribution of vascular endothelial (VE)-cadherin. Compared with control cells, there was an increase in endothelium-hyaline leukocyte conglutination, a reduction in VCAM-1, ICAM-1, and E-selectin, and the TEER value increased obviously. Compared with LPS, there was an obvious reduction in the conglutination of LPS+H<sub>2</sub> cells, a reduction in VCAM-1, ICAM-1, and E-selectin levels, and a reduction in the TEER-resistance value, while the expression of VE-cadherin increased. Fluorescence results showed that, compared with control cells, the VE-cadherin in LPS cells was incomplete at the cell joints. Compared with LPS cells, the VE-cadherin in LPS+H<sub>2</sub> cells was even and complete at the cell joints. Liquid rich in hydrogen could reduce LPS-induced production of adhesion molecules and endothelium-hyaline leukocyte conglutination, and influence the expression and distribution of VE-cadherin to regulate the permeability of the endothelium.

**Key words:** Hydrogen; Lipopolysaccharide; Sepsis; Endothelial cells; Vascular endothelial-cadherin