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[Regulative effects of hydrogen-rich medium on monocytic adhesion and vascular endothelial permeability]

[Article in Chinese]

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Abstract

Objective: To explore the regulative effects of hydrogen-rich medium on lipopolysaccharide (LPS)-induced monocytes adhesion to human umbilical vein endothelial cells (HUVEC) and vascular endothelial permeability in vitro.

Methods: Endothelial cells were seeded in 6-well plates and randomly divided into 4 groups (n = 42 each):control (A), hydrogen-rich medium (B), LPS (C) and LPS+hydrogen-rich medium (D). Cells were cultured in plain culture medium in groups A and C or in hydrogen-saturated culture medium in groups B and D.LPS 1 μg/ml was added into groups C and D.When forming a monolayer, monocytes were added into each group after 6, 12 and 24 h respectively. After a 90-minute co-culturing, adhesion status was detected by Wright-Giemsa stain.Supernatants were collected to detect the concentrations of vascular cell adhesion molecule-1 (VCAM-1) and E-selectin by enzyme-linked immunosorbent assay (ELISA). The expression of VE-cadherin was measured by Western blot. Cells were stained with immunofluorescence to show the distribution of VE-cadherin after a 24-hour incubation.

Results: Compared with group A, the adhesion of monocytes to endothelial cells increased ($P < 0.05$) in group C, the levels of E-selectin and VCAM-1 became elevated ($P < 0.05$) while the expression of VE-cadherin decreased significantly ($P < 0.05$). Compared with group C, adhesion decreased in group D ($P < 0.05$), the levels of E-selectin and VCAM-1 decreased ($P < 0.05$) while there was an increased expression of VE-cadherin ($P < 0.05$). Three timepoints showed the same tendency. The results of 24 h fluorescence indicated that, compared with group A, VE-cadherin was incomplete in cell-cell connections in group C.However it was complete and well-distributed in group D versus group C.

Conclusion: Hydrogen-rich medium may reduce the LPS-induced release of adhesion molecules, lessen monocytic adhesion to HUVEC and regulate the expression of VE-cadherin to protect vascular permeability.

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