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The protective effects of hydrogen on HO−1 expression in the brainafter focal cerebral ischemia reperfusion in rats

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Abstract

Background/aim: The aim of this study was to investigate whether a hydrogen administration can produce neuroprotective effects after brain ischemia reperfusion in rats.

Materials and methods: A brain ischemia reperfusion injury was induced by a 2-h left middle cerebral artery occlusion (MCAO) using an intraluminal filament, followed by 46 h of reperfusion. A hydrogen-rich saline (1 mL/kg body weight i.p.) was administered at the beginning of reperfusion. Saline (1 mL/kg)-treated animals were used as the control. Sham-operated animals were also used. Subsequently, 48 h after the MCAO, histological alternations, heme oxygenase-1 (HO-1) expression, and levels of malondialdehyde (MDA) and superoxide dismutase (SOD) in the cerebral cortex and the hippocampus were examined.

Results: Hydrogen significantly alleviated brain tissue histological damage, promoted HO-1 expression, upregulated levels of SOD, and decreased the levels of MDA in brain tissue after the ischemia reperfusion injury.

Conclusion: The results suggest that the neuroprotective effects of hydrogen may be mediated by promoting HO-1 expression and attenuated the oxidative injury.

Keywords: Hydrogen; brain; cerebral ischemia reperfusion; heme oxygenase-1; rats.

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