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Hydrogen inhalation improves mouse neurological outcomes after cerebral ischemia/reperfusion independent of anti-necroptosis

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Abstract

This study aimed to investigate the role of necroptosis in the neuroprotection of hydrogen in a mouse model of cerebral ischemia/reperfusion (I/R) injury. C57BL mice were randomly divided into sham group, I/R group, hydrogen/oxygen group (HO), nitrogen/oxygen group (NO). Middle cerebral artery occlusion (MCAO) for 1 hour followed by reperfusion was introduced to animals which were allowed to inhale 66.7% hydrogen/33.3% oxygen for 90 minutes since the beginning of reperfusion. Mice in NO group inhaled 66.7% nitrogen/33.3% oxygen. 24 hours after MCAO, brain infarction, brain water content and neurological function were evaluated. The protein expression of mixed lineage kinase domain like protein (MLKL) was detected at 3, 6, 12, 24 and 72 hours after reperfusion in HO group and the protein and mRNA expression of MLKL at 24 hours after MCAO in four groups. Hydrogen inhalation significantly reduced infarct volume, attenuated brain edema and improved neurobehavioral deficit in MCAO mice. The MLKL expression increased after MCAO and peaked at 6-24 hours after reperfusion. However, hydrogen inhalation had no significant effect on the MLKL expression at transcriptional and translational levels after MCAO. This study indicates high concentration hydrogen improves mouse neurological outcome after cerebral I/R injury independent of anti-necroptosis.

Keywords: high concentration hydrogen; necroptosis; ischemia/reperfusion; mixed lineage kinase domain like protein; neurological function.

Figures

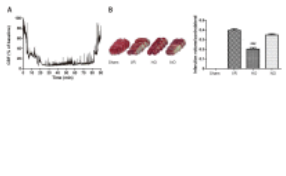

Figure 1 High concentration hydrogen (HCH) reduces...

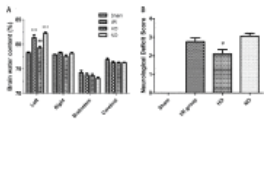

Figure 2 High concentration hydrogen (HCH) improves...

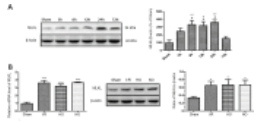

Figure 3 Mixed lineage kinase domain like...

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