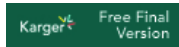


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Hydrogen Suppresses Hypoxia/Reoxygenation-Induced Cell Death in Hippocampal Neurons Through Reducing Oxidative Stress

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Abstract

Background & aims: Deep hypothermic circulatory arrest (DHCA) is a cerebral protection technique that has been used in the operations involving the aortic arch and brain aneurysm for decades. We previously showed that DHCA treated rats developed a significant oxidative stress and apoptosis in neurons. We here intend to investigate the protective effect of hydrogen against oxidative stress-induced cell injury and the involved mechanisms using an in vitro experimental model of hypoxia/reoxygenation (H/R) on HT-22 cells.

Methods: The model of H/R was established using an airtight culture container and the anaeropack. Measurement of mitochondrial membrane potential (MMP) and reactive oxygen species (ROS) production was used H₂DCFDA and JC-1 staining. Western blot was used for the quantification of Akt, p-Akt, Bcl-2, Bax and cleaved caspase-3 proteins. The microRNA (miRNA) profile in hippocampal neurons from rat model of DHCA was determined by miRNA deep sequencing.

Results: The elevation of ROS and reduction of MMP were significantly induced by the treatment with hypoxia for 18 h followed by reoxygenation for 6 h. Hydrogen treatment significantly reduced H/R-caused cell death. The levels of p-Akt (Ser 473) and Bcl-2 were significantly increased while Bax and cleaved caspase-3 were decreased by hydrogen treatment on the model of H/R. The expression of miR-200 family was significantly elevated in model of DHCA and H/R. Hydrogen administration inhibited the H/R-induced expression of miR-200 family in HT-22 cells. In addition, inhibition of miR-200 family suppressed H/R-caused cell death through reducing ROS production.

Conclusions: These results suggest that H/R causes oxidative stress-induced cell death and that the hydrogen protects against H/R-induced cell death in HT22 cells, in part, due to reducing expression of miR-200 family.

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