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Maternal molecular hydrogen administration ameliorates rat fetal hippocampal damage caused by in utero ischemia–reperfusion

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Abstract

Molecular hydrogen (H2) scavenges hydroxyl radicals. Recently, H2 has been reported to prevent a variety of diseases associated with oxidative stress in model systems and in humans. Here, we studied the effects of H2 on rat fetal hippocampal damage caused by ischemia and reperfusion (IR) on day 16 of pregnancy with the transient occlusion of the bilateral utero-ovarian arteries. Starting 2 days before the operation, we provided the mothers with hydrogen-saturated water ad libitum until vaginal delivery. We observed a significant increase in the concentration of H2 in the placenta after the oral administration of hydrogen-saturated water to the mothers, with less placental oxidative damage after IR in the presence of H2. Neonatal growth retardation was observed in the IR group, which was alleviated by the H2 administration. We analyzed the neuronal cell damage in the CA1 and CA3 areas of the hippocampus at day 7 after birth by immunohistochemical analysis of the 8-oxo-7,8-dihydro-2′-deoxyguanosine- and 4-hydroxy-2-nonenal-modified proteins. Both oxidative stress markers were significantly increased in the IR group, which was again ameliorated by the H2 intake. Last, 8-week-old rats were subjected to a Morris water maze test. Maternal H2 administration improved the reference memory of the offspring to the sham level after IR injury during pregnancy. Overall, the present results support the idea that maternal H2 intake helps prevent the hippocampal impairment of offspring induced by IR during pregnancy.

Keywords: Free radicals; In utero; Ischemia–reperfusion; Molecular hydrogen; Neuronal damage.

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