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Hydrogen-rich water protects against ischemic brain injury in rats by regulating calcium buffering proteins

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Abstract

Hydrogen-rich water (HRW) has anti-oxidant activities, and it exerts neuroprotective effects during ischemia-reperfusion brain injury. Parvalbumin and hippocalcin are two calcium buffering proteins, which are involved in neuronal differentiation, maturation and apoptosis. The aim of this study was to investigate whether HRW could moderate parvalbumin and hippocalcin expression during ischemic brain injury and glutamate toxicity-induced neuronal cell death. Focal brain ischemia was induced in male Sprague-Dawley rats by middle cerebral artery occlusion (MCAO). Rats were treated with H2O or HRW (6 ml/kg per rat) before and after MCAO, and cerebral cortical tissues were collected 1, 7 and 14 days after MCAO. Based on our results, HRW treatment was able to reduce brain infarct volume and improve neurological function following ischemic brain injury. In addition, HRW prevented the ischemia-induced reduction of parvalbumin and hippocalcin levels in vivo and also reduced the glutamate toxicity-induced death of neurons, including the dose-dependent reduction of glutamate toxicity-associated proteins in vitro. Moreover, HRW attenuated the glutamate toxicity-induced elevate in intracellular Ca(2+) levels. All these results suggest that HRW could protect against ischemic brain injury and that the maintenance of parvalbumin and hippocalcin levels by HRW during ischemic brain injury might contribute to the neuroprotective effects against neuron damage.

Keywords: Hippocalcin; Hydrogen-rich water; Ischemic brain injury; Neuroprotection; Parvalbumin.

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