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Hydrogen-rich saline reduces oxidative stress and inflammation by inhibit of JNK and NF- κ B activation in a rat model of amyloid-beta-induced Alzheimer's disease

Cai Wang ¹, Jian Li, Qiang Liu, Rui Yang, John H Zhang, Yun-Peng Cao, Xue-Jun Sun

Affiliations

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Abstract

This study is to examine if hydrogen-rich saline reduced amyloid-beta (A β) induced neural inflammation and oxidative stress in a rat model by attenuation of activation of JNK and NF- κ B. Sprague-Dawley male rats (n=18, 280-330 g) were divided into three groups, sham operated, A β 1-42 injected and A β 1-42 plus hydrogen-rich saline treated animals. Hydrogen-rich saline (5 ml/kg, i.p., daily) was injected for 10 days after intraventricular injection of A β 1-42. The levels of IL-1 β were assessed by ELISA analysis, 8-OH-dG by immunohistochemistry in the brain slides, and JNK and NF- κ B by immunohistochemistry and western blotting. After A β 1-42 injection, the level of IL-1 β , 8-OH-dG, JNK and NF- κ B all increased in brain tissues, while hydrogen-rich saline treatment decreased the level of IL-1 β , 8-OH-dG and the activation of JNK and NF- κ B. In conclusion, hydrogen-rich saline prevented A β -induced neuroinflammation and oxidative stress, possibly by attenuation of activation of c-Jun NH₂-terminal kinase (JNK) and nuclear factor- κ B (NF- κ B) in this rat model.

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