J Hepatol. 2011 Mar;54(3):471-80. doi: 10.1016/j.jhep.2010.08.011. Epub 2010 Sep 25.

The protective role of hydrogen-rich saline in experimental liver injury in mice

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PMID: 21145612 DOI: 10.1016/j.jhep.2010.08.011

Abstract

Background & aims: Reactive oxygen species (ROS) are considered to play a prominent causative role in the development of various hepatic disorders. Antioxidants have been effectively demonstrated to protect against hepatic damage. Hydrogen (H(2)), a new antioxidant, was reported to selectively reduce the strongest oxidants, such as hydroxyl radicals (·OH) and peroxynitrite (ONOO(-)), without disturbing metabolic oxidation-reduction reactions or disrupting ROS involved in cell signaling. In place of H(2) gas, hydrogen-rich saline (HS) may be more suitable for clinical application. We herein aim to verify its protective effects in experimental models of liver injury.

Methods: H(2) concentration in vivo was detected by hydrogen microelectrode for the first time. Liver damage, ROS accumulation, cytokine levels, and apoptotic protein expression were, respectively, evaluated after GalN/LPS, CCl(4), and DEN challenge. Simultaneously, CCl(4)-induced hepatic cirrhosis and DEN-induced hepatocyte proliferation were measured.

Results: HS significantly increased hydrogen concentration in liver and kidney tissues. As a result, acute liver injury, hepatic cirrhosis, and hepatocyte proliferation were reduced through the quenching of detrimental ROS. Activity of pro-apoptotic players, such as JNK and caspase-3, were also inhibited.

Conclusions: HS could protect against liver injury and also inhibit the processes leading to liver cirrhosis and hepatocyte compensatory proliferation.

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