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Effects of oral intake of hydrogen water on liver fibrogenesis in mice

Yukinori Koyama [1], Kojiro Taura, Etsuro Hatano, Kazutaka Tanabe, Gen Yamamoto, Kojiro Nakamura, Kenya Yamanaka, Koji Kitamura, Masato Narita, Hiromitsu Nagata, Atsuko Yanagida, Taku Iida, Keiko Iwaisako, Hikohito Fujinawa, Shinji Uemoto

Affiliations
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Abstract

Aim: Liver fibrosis is the universal consequence of chronic liver diseases. Sustained hepatocyte injury initiates an inflammatory response, thereby activating hepatic stellate cells, the principal fibrogenic cells in the liver. Reactive oxygen species are involved in liver injury and are a promising target for treating liver fibrosis. Hydrogen water is reported to have potential as a therapeutic tool for reactive oxygen species-associated disorders. This study aimed to investigate the effects of hydrogen water on liver fibrogenesis and the mechanisms underlying these effects.

Methods: C57BL/6 mice were fed with hydrogen water or control water, and subjected to carbon tetrachloride, thioacetamide and bile duct ligation treatments to induce liver fibrosis. Hepatocytes and hepatic stellate cells were isolated from mice and cultured with or without hydrogen to test the effects of hydrogen on reactive oxygen species-induced hepatocyte injuries or hepatic stellate cell activation.

Results: Oral intake of hydrogen water significantly suppressed liver fibrogenesis in the carbon tetrachloride and thioacetamide models, but these effects were not seen in the bile duct ligation model. Treatment of isolated hepatocyte with 1 μg/mL antimycin A generated hydroxyl radicals. Culturing in the hydrogen-rich medium selectively suppressed the generation of hydroxyl radicals in hepatocytes and significantly suppressed hepatocyte death induced by antimycin A; however, it did not suppress hepatic stellate cell activation.

Conclusion: We conclude that hydrogen water protects hepatocytes from injury by scavenging hydroxyl radicals and thereby suppresses liver fibrogenesis in mice.

Keywords: hydrogen; hydrogen water; hydroxyl radical; liver cirrhosis; liver fibrosis; liver injury.

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