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Sirtuin Type 1 Mediates the Retinal Protective Effect of Hydrogen-Rich Saline Against Light-Induced Damage in Rats

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Abstract

Purpose: Molecular hydrogen has been used as an antioxidant to treat many diseases in clinical and animal studies. However, the therapeutic mechanism of molecular hydrogen remains unclear. We previously reported mitigation of light-induced damage in the rat retina by intraperitoneal injection of hydrogen-rich saline (HRS). In the present study, we investigated whether Sirtuin Type 1 (Sirt1), a class III histone deacetylase, mediates the retinal protective effect of HRS in rats with light-induced retinal damage.

Methods: Rats were treated with HRS for 5 days after intense light exposure, and then ERGs were performed and retinas were collected to evaluate the effect of HRS on Sirt1 expression. The necessity of Sirt1 for the retinal protective effect of HRS was investigated using the Sirt1 activator resveratrol, the Sirt1 inhibitor EX-527, and short interfering RNAs.

Results: In light-damaged retinas, 5 days of HRS treatment increased Sirt1 expression, mitigated a- and b-wave amplitude reduction, and decreased the reduction of outer nuclear cell layers. The Sirt1 activator resveratrol mimicked the effect of HRS in light-damaged retinas. This result supported our hypothesis that Sirt1 mediates the protective effect of HRS. Additionally, the retinal protective effect of HRS was inhibited by both the Sirt1 inhibitor EX-527 and Sirt1 targeted short interfering RNAs. Hydrogen-rich saline also increased B-cell lymphoma 2 (Bcl-2) expression and the activity of the antioxidant enzyme superoxide dismutase (SOD). Conversely, HRS decreased Bcl2-associated X protein expression, cleaved caspase-3, and oxidant-stress product malondialdehyde (MDA) in a Sirt1-dependent manner.

Conclusions: Sirt1 mediates light-induced damage mitigation by HRS through inhibition of apoptosis and oxidant-stress.

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