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Protective effects of hydrogen-rich saline against N-methyl-N-nitrosourea-induced photoreceptor degeneration

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Abstract

The N-methyl-N-nitrosourea (MNU)-treated rat is typically used as an animal model of chemically-induced retinitis pigmentosa (RP). Reactive oxygen species (ROS) have been recognized as the crucial contributor to the retinal photoreceptor apoptosis seen in MNU-treated rats. In the present study, we explored the therapeutic effects of hydrogen-rich saline (HRS), a selective ROS scavenger, on MNU-induced photoreceptor degeneration. Intraperitoneal (IP) administration of HRS ameliorated MNU-induced photoreceptor degeneration in terms of morphology and function: Sharply decreased thickness of the retinal outer nuclear layer (ONL) and flattened photopic and scotopic electroretinogram (ERG) waveforms, typically seen in response to MNU treatment, were substantially rescued in rats cotreated with MNU and HRS (MNU + HRS). Moreover, the terminal deoxyuridine triphosphate nick-end labeling (TUNEL) assay revealed a smaller number of apoptotic photoreceptors in the MNU + HRS group compared that in the MNU group. Compared to MNU-treated rats, retinal malondialdehyde (MDA) content in MNU + HRS rats significantly decreased while superoxide dismutase (SOD) activity significantly increased. Morphological and multi-electrode array (MEA) analyses revealed more efficient preservation of the architecture and field potential waveforms in particularly the peripheral regions of the retinas within the MNU + HRS group, compared to that in the MNU group. However, this enhanced protection of structure and function in the peripheral retina is unlikely the result of site-dependent variation in the efficacy of HRS; rather, it is most likely due to reduced susceptibility of peripheral photoreceptors to MNU-induced degeneration. Inner retinal neuron function in the MNU + HRS rats was better preserved, with fewer apoptotic photoreceptors in the ONL. Collectively, these results support the rationale for future clinical evaluation of HRS as a therapeutic agent for human RP.

Keywords: Apoptosis; Hydrogen; N-methyl-N-nitrosourea; Oxidative stress; Photoreceptor degeneration.

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