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Treatment with Hydrogen-Rich Saline Delays Disease Progression in a Mouse Model of Amyotrophic Lateral Sclerosis

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Abstract

Amyotrophic lateral sclerosis (ALS) is the most frequent adult-onset motor neuron disease, and accumulating evidence indicates that oxidative mechanisms contribute to ALS pathology, but classical antioxidants have not performed well in clinical trials. The aim of this work was to investigate the effect of treatment with hydrogen molecule on the development of disease in mutant SOD1 G93A transgenic mouse model of ALS. Treatment of mutant SOD1 G93A mice with hydrogen-rich saline (HRS, i.p.) significantly delayed disease onset and prolonged survival, and attenuated loss of motor neurons and suppressed microglial and glial activation. Treatment of mutant SOD1 G93A mice with HRS inhibited the release of mitochondrial apoptogenic factors and the subsequent activation of downstream caspase-3. Furthermore, treatment of mutant SOD1 G93A mice with HRS reduced levels of protein carbonyl and 3-nitrotyrosine, and suppressed formation of reactive oxygen species (ROS), peroxynitrite, and malondialdehyde. Treatment of mutant SOD1 G93A mice with HRS preserved mitochondrial function, marked by restored activities of Complex I and IV, reduced mitochondrial ROS formation and enhanced mitochondrial adenosine triphosphate synthesis. In conclusion, hydrogen molecule may be neuroprotective against ALS, possibly through abating oxidative and nitrosative stress and preserving mitochondrial function.

Keywords: Amyotrophic lateral sclerosis; Hydrogen molecule; Mitochondrial function; Oxidative and nitrosative stress.

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