Hydrogen-rich pure water prevents cigarette smoke-induced pulmonary emphysema in SMP30 knockout mice.

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Abstract

Chronic obstructive pulmonary disease (COPD) is predominantly a cigarette smoke (CS)-triggered disease with features of chronic systemic inflammation. Oxidants derived from CS can induce DNA damage and stress-induced premature cellular senescence in the respiratory system, which play significant roles in COPD. Therefore, antioxidants should provide benefits for the treatment of COPD; however, their therapeutic potential remains limited owing to the complexity of this disease. Recently, molecular hydrogen (H2) has been reported as a preventive and therapeutic antioxidant. Molecular H2 can selectively reduce hydroxyl radical accumulation with no known side effects, showing potential applications in managing oxidative stress, inflammation, apoptosis, and lipid metabolism. However, there have been no reports on the efficacy of molecular H2 in COPD patients. In the present study, we used a mouse model of COPD to investigate whether CS-induced histological damage in the lungs could be attenuated by administration of molecular H2. We administered H2-rich pure water to senescence marker protein 30 knockout (SMP30-KO) mice exposed to CS for 8 weeks. Administration of H2-rich water attenuated the CS-induced lung damage in the SMP30-KO mice and reduced the mean linear intercept and destructive index of the lungs. Moreover, H2-rich water significantly restored the static lung compliance in the CS-exposed mice compared with that in the CS-exposed H2-untreated mice. Moreover, treatment with H2-rich water decreased the levels of oxidative DNA damage markers such as phosphorylated histone H2AX and 8-hydroxy-2'-deoxyguanosine, and senescence markers such as cyclin-dependent kinase inhibitor 2A, cyclin-dependent kinase inhibitor 1, and β-galactosidase in the CS-exposed mice. These results demonstrated that H2-rich pure water attenuated CS-induced emphysema in SMP30-KO mice by reducing CS-induced oxidative DNA damage and premature cell senescence in the lungs. Our study suggests that administration of molecular H2 may be a novel preventive and therapeutic strategy for COPD.

KEYWORDS: Chronic obstructive pulmonary disease; DNA damage; Emphysema; Molecular hydrogen; SMP30; Senescence

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