Biochem Biophys Res Commun. 2017 Sep 23;491(3):733-739. doi: 10.1016/j.bbrc.2017.07.119. Epub 2017 Jul 22.

Hydrogen-rich medium protects mouse embryonic fibroblasts from oxidative stress by activating LKB1-AMPK-FoxO1 signal pathway

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PMID: 28743498 DOI: 10.1016/j.bbrc.2017.07.119

Abstract

Persistent oxidative stress is recognized as a major cause of many pathological conditions as well as ageing. However, most clinical trials of dietary antioxidants have failed to produce successful outcomes in treating oxidative stress-induced diseases. Molecular hydrogen (H_2) has recently received considerable attention as a therapeutic agent owing to its novel antioxidant properties, a selective scavenger of hydroxyl and peroxynitrite radicals. Beyond this, numerous reports support that H_2 can modulate the activity of various cellular signal pathways. However, its effect on AMP-activated protein kinase (AMPK) signal pathway, a central regulator of energy hemostasis, has remained almost elusive. Here, we report that hydrogen-rich medium activated LKB1-AMPK signal pathway without ATP depletion, which in turn induced FoxO1-dependent transcription of manganese superoxide dismutase and catalase in mouse embryonic fibroblasts. Moreover, hydrogen-rich media effectively reduced the level of reactive oxygen species in cells treated with hydrogen peroxide and protected these cells from apoptosis in an AMPK-dependent manner. These results suggest that the LKB1-AMPK-FoxO1 signaling pathway is a critical mediator of the antioxidant properties of H_2, further supporting the idea that H_2 acts as a signaling molecule to serve various physiological functions.

Keywords: AMP-Activated protein kinase; Antioxidant enzyme; Molecular hydrogen; Oxidative stress.

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Nucleotide (RefSeq)
Nucleotide (Weighted)
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