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[Am J Physiol Heart Circ Physiol](#). 2011 Sep;301(3):H1062-9. doi: 10.1152/ajpheart.00150.2011.

Epub 2011 Jun 3.

Inhalation of hydrogen gas attenuates left ventricular remodeling induced by intermittent hypoxia in mice

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PMID: 21642501 DOI: [10.1152/ajpheart.00150.2011](#)

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Abstract

Sleep apnea syndrome increases the risk of cardiovascular morbidity and mortality. We previously reported that intermittent hypoxia increases superoxide production in a manner dependent on nicotinamide adenine dinucleotide phosphate and accelerates adverse left ventricular (LV) remodeling. Recent studies have suggested that hydrogen (H₂) may have an antioxidant effect by reducing hydroxyl radicals. In this study, we investigated the effects of H₂ gas inhalation on lipid metabolism and LV remodeling induced by intermittent hypoxia in mice. Male C57BL/6J mice (n = 62) were exposed to intermittent hypoxia (repetitive cycle of 1-min periods of 5 and 21% oxygen for 8 h during daytime) for 7 days. H₂ gas (1.3 vol/100 vol) was given either at the time of reoxygenation, during hypoxic conditions, or throughout the experimental period. Mice kept under normoxic conditions served as controls (n = 13). Intermittent hypoxia significantly increased plasma levels of low- and very low-density cholesterol and the amount of 4-hydroxy-2-nonenal-modified protein adducts in the LV myocardium. It also upregulated mRNA expression of tissue necrosis factor- α , interleukin-6, and brain natriuretic peptide, increased production of superoxide, and induced cardiomyocyte hypertrophy, nuclear deformity, mitochondrial degeneration, and interstitial fibrosis. H₂ gas inhalation significantly suppressed these changes induced by intermittent hypoxia. In particular, H₂ gas inhaled at the timing of reoxygenation or throughout the experiment was effective in preventing dyslipidemia and suppressing superoxide production in the LV myocardium. These results suggest that inhalation of H₂ gas was effective for reducing oxidative stress and preventing LV remodeling induced by intermittent hypoxia relevant to sleep apnea.

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