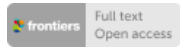


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# Inhalation of Hydrogen Attenuates Progression of Chronic Heart Failure via Suppression of Oxidative Stress and P53 Related to Apoptosis Pathway in Rats

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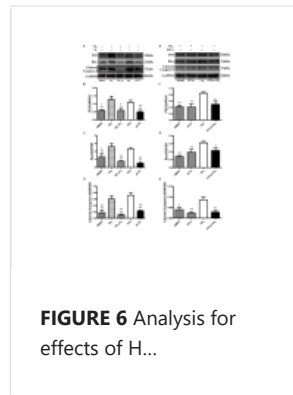
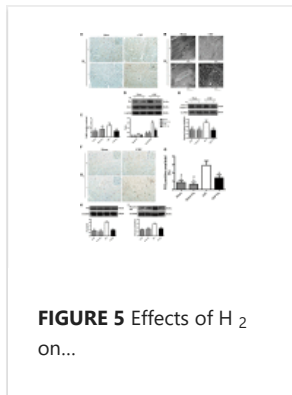
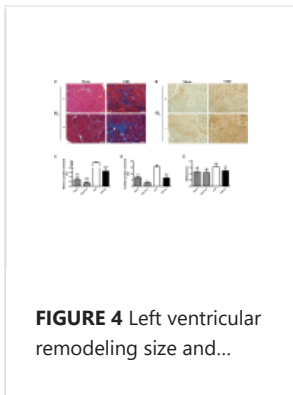
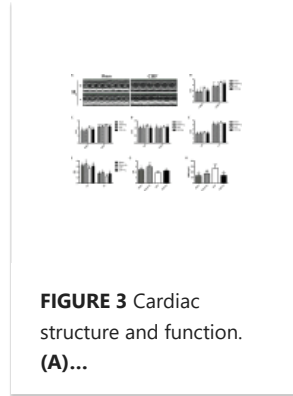
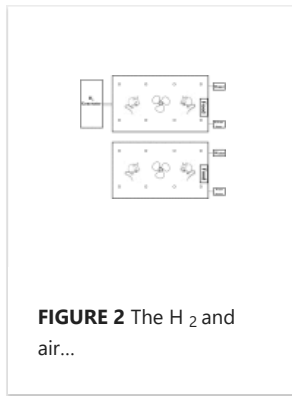
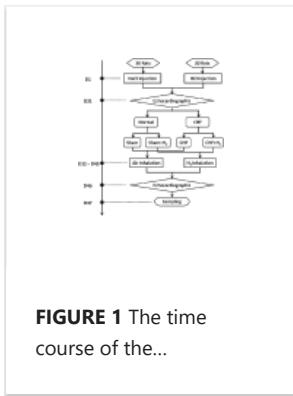
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## Abstract

**Background:** Continuous damage from oxidative stress and apoptosis are the important mechanisms that facilitate chronic heart failure (CHF). Molecular hydrogen (H<sub>2</sub>) has potentiality in the aspects of anti-oxidation. The objectives of this study were to investigate the possible mechanism of H<sub>2</sub> inhalation in delaying the progress of CHF. **Methods and Results:** A total of 60 Sprague-Dawley (SD) rats were randomly divided into four groups: Sham, Sham treated with H<sub>2</sub>, CHF and CHF treated with H<sub>2</sub>. Rats from CHF and CHF treated with H<sub>2</sub> groups were injected isoprenaline subcutaneously to establish the rat CHF model. One month later, the rat with CHF was identified by the echocardiography. After inhalation of H<sub>2</sub>, cardiac function was improved vs. CHF ( $p < 0.05$ ), whereas oxidative stress damage and apoptosis were significantly attenuated ( $p < 0.05$ ). In this study, the mild oxidative stress was induced in primary cardiomyocytes of rats, and H<sub>2</sub> treatments significantly reduced oxidative stress damage and apoptosis in cardiomyocytes ( $p < 0.05$  or  $p < 0.01$ ). Finally, as a pivotal transcription factor in reactive oxygen species (ROS)-apoptosis signaling pathway, the expression and phosphorylation of p53 were significantly reduced by H<sub>2</sub> treatment in this rat model and H9c2 cells ( $p < 0.05$  or  $p < 0.01$ ). **Conclusion:** As a safe antioxidant, molecular hydrogen mitigates the progression of CHF via inhibiting apoptosis modulated by p53. Therefore, from the translational point of view and speculation, H<sub>2</sub> is equipped with potential therapeutic application as a novel antioxidant in protecting CHF in the future.

**Keywords:** apoptosis; chronic heart failure; hydrogen; oxidative stress; p53.

## Figures



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