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Molecular hydrogen protects against oxidative stress-induced SH-SY5Y neuroblastoma cell death through the process of mitohormesis

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Abstract

Inhalation of molecular hydrogen (H₂) gas ameliorates oxidative stress-induced acute injuries in the brain. Consumption of water nearly saturated with H₂ also prevents chronic neurodegenerative diseases including Parkinson's disease in animal and clinical studies. However, the molecular mechanisms underlying the remarkable effect of a small amount of H₂ remain unclear. Here, we investigated the effect of H₂ on mitochondria in cultured human neuroblastoma SH-SY5Y cells. H₂ increased the mitochondrial membrane potential and the cellular ATP level, which were accompanied by a decrease in the reduced glutathione level and an increase in the superoxide level. Pretreatment with H₂ suppressed H₂O₂-induced cell death, whereas post-treatment did not. Increases in the expression of anti-oxidative enzymes underlying the Nrf2 pathway in H₂-treated cells indicated that mild stress caused by H₂ induced increased resistance to exacerbated oxidative stress. We propose that H₂ functions both as a radical scavenger and a mitohormetic effector against oxidative stress in cells.

Figures

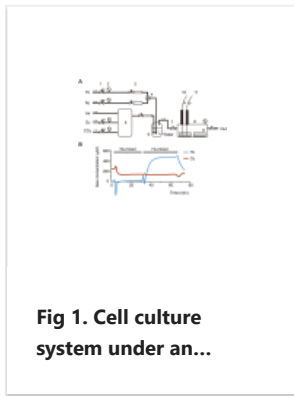


Fig 1. Cell culture system under an...

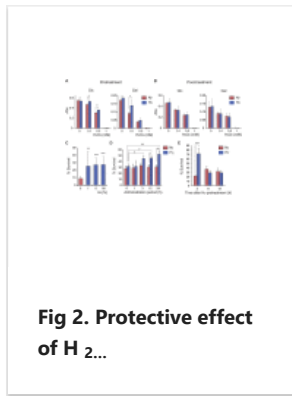


Fig 2. Protective effect of H2...

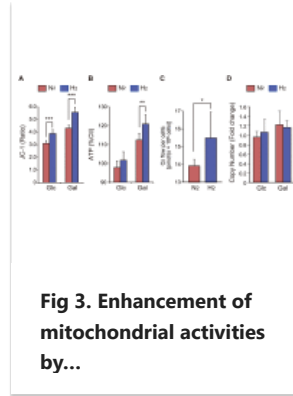


Fig 3. Enhancement of mitochondrial activities by...

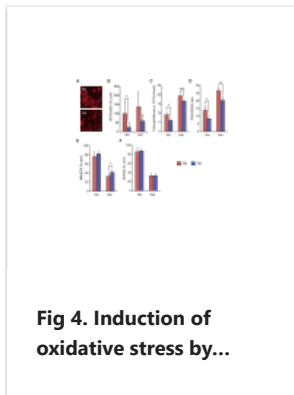


Fig 4. Induction of oxidative stress by...

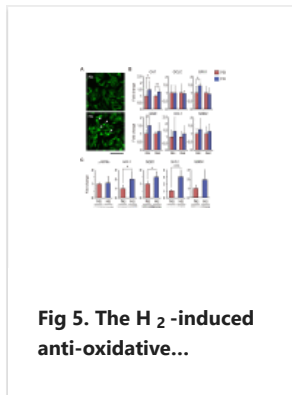


Fig 5. The H₂-induced anti-oxidative...

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