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Complexity of Stomach–Brain Interaction Induced by Molecular Hydrogen in Parkinson's Disease Model Mice

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Abstract

Molecular hydrogen (H_2), as a new medical gas, has protective effects in neurological disorders including Parkinson's disease (PD). In our previous report, the neuroprotective effect of drinking water with saturated H_2 (H_2 water) in PD mice might be due to stomach-brain interaction via release of gastric hormone, ghrelin. In the present study, we assessed the effect of H_2-induced ghrelin more precisely. To confirm the contribution of ghrelin in H_2 water-drinking PD model mice, ghrelin-knock out (KO) mice were used. Despite the speculation, the effect of H_2 water was still observed in ghrelin-KO PD model mice. To further check the involvement of ghrelin, possible contribution of ghrelin-induced vagal afferent effect was tested by performing subdiaphragmatic vagotomy before treating with H_2 water and administration of MPTP (1-methyl- 4-phenyl-1,2,3,6-tetrahydropyridine). The protective effect of H_2 water was still observed in the vagotomized mice in substantia nigra, suggesting that stimulation of vagal afferent nerves is not involved in H_2-induced neuroprotection. Other neuroprotective substitutes in ghrelin-KO mice were speculated because H_2-induced neuroprotection was not cancelled by ghrelin receptor antagonist, D-Lys^3 GHRP-6, in ghrelin-KO PD model mice, unlike in wild-type PD model mice. Our results indicate that ghrelin may not be the only factor for H_2-induced neuroprotection and other factors can substitute the role of ghrelin when ghrelin is absent, raising intriguing options of research for H_2-responsive factors.

Keywords: Ghrelin; Ghrelin-knock out mice; Molecular hydrogen; Parkinson's disease; Vagal afferents.

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