Inflammation. 2015 Oct;38(5):1835-46. doi: 10.1007/s10753-015-0161-x.

H2 Treatment Attenuated Pain Behavior and Cytokine Release Through the HO-1/CO Pathway in a Rat Model of Neuropathic Pain

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PMID: 25820467 DOI: 10.1007/s10753-015-0161-x

Abstract

Neuropathic pain (NP) is characterized by persistent pain, tactile allodynia, or hyperalgesia. Peripheral nerve injury contributes to rapid progress of inflammatory response and simultaneously generates neuropathic pain. Hydrogen (H2) has anti-inflammation, anti-apoptosis, and anti-oxidative stress effects. Therefore, we hypothesized that H2 treatment could alleviate allodynic and hyperalgesic behaviors and the release of inflammatory factors in rats with neuropathic pain. Peripheral neuropathic pain was established by chronic constriction injury of sciatic nerve in rats. H2 was given twice through intraperitoneal injection at a daily dose of 10 mL/kg during days 1-7 after the operation. Hyperalgesia and allodynia were tested, pro-inflammatory factors of dorsal root ganglia (DRG) and the spinal cord were measured by enzyme-linked immunosorbent assay (ELISA) during days 1-14 after the operation, and heme oxygenase (HO)-1 messenger RNA (mRNA) and protein expression and activities were measured at day 14 after sciatic nerve injury in rats. After Sn (IV) protoporphyrin IX dihydrochloride (SnPP)-IX, hemin, and carbon monoxide-releasing molecule (CORM)-2 had been given for chronic constriction injury (CCI) in rats, the above indicators were assessed. We found that H2 clearly inhibited hyperalgesia and allodynia in neuropathic pain and also attenuated the pro-inflammatory cytokines TNF-α, IL-1β, and high-mobility group box (HMGB) 1. H2 improved HO-1 mRNA and protein expression and activities in the process of pain. SnPP-IX reversed the inhibitory effect of H2 on hyperalgesia and allodynia and on pro-inflammatory cytokines in DRG and the spinal cord. The antinociceptive and anti-inflammatory effects of H2 were involved in the activation of HO-1/CO signaling during neuropathic pain in rats.

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