Effects of hyperbaric oxygen on the Nrf2 signaling pathway in secondary injury following traumatic brain injury

X.E. Meng, Y. Zhang, N. Li, D.F. Fan, C. Yang, H. Li, D.Z. Guo and S.Y. Pan

Department of Hyperbaric Oxygen, Navy General Hospital, Beijing, China

Corresponding author: S.Y. Pan
E-mail: shuyipan_oooo@126.com

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ABSTRACT. We investigated the effects of hyperbaric oxygen treatment on the Nrf2 signaling pathway in secondary injury following traumatic brain injury, using a rat model. An improved Feeney freefall method was used to establish the rat traumatic brain injury model. Sixty rats were randomly divided into three groups: a sham surgery group, a traumatic brain injury group, and a group receiving hyperbaric oxygen treatment after traumatic brain injury. Neurological function scores were assessed at 12 and 24 h after injury. The expression levels of Nrf2, heme oxygenase 1 (HO-1), and quinine oxidoreductase 1 (NQO-1) in the cortex surrounding the brain lesion were detected by western blotting 24 h after the injury. Additionally, the TUNEL method was used to detect apoptosis of nerve cells 24 h after traumatic injury and Nissl staining was used to detect the number of whole neurons. Hyperbaric oxygen treatment significantly increased the expression of nuclear Nrf2 protein (P < 0.05), HO-1, and NQO-1 in the brain tissues surrounding the lesion after a traumatic brain injury (P < 0.05) and also significantly reduced the number of apoptotic and injured nerve cells. The neurological function scores also improved with hyperbaric oxygen treatment (P < 0.05). Therefore, hyperbaric oxygen has a neuroprotective
role in traumatic brain injury, which is mediated by up-regulation of the Nrf2 signaling pathway.

**Key words:** Traumatic brain injury; Hyperbaric oxygen; Nrf2