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Hyperbaric Oxygen Effects on Depression-Like Behavior and Neuroinflammation in Traumatic Brain Injury Rats

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Abstract

Objective: The aim of this study was to determine whether hyperbaric oxygen (HBO) therapy causes attenuation of traumatic brain injury (TBI)-induced depression-like behavior and its associated anti-neuroinflammatory effects after fluid percussion injury.

Methods: Anesthetized male Sprague-Dawley rats were divided into 3 groups: sham operation plus normobaric air (NBA) (21% oxygen at 1 absolute atmosphere [ATA]), TBI plus NBA, and TBI plus HBO (100% oxygen at 2.0 ATA). HBO was applied immediately for 60 min/d after TBI for 3 days. Depression-like behavior was tested by a forced swimming test, motor function was tested by an inclined plane test, and infarction volume was tested by triphenyltetrazolium chloride (TTC) staining on days 4, 8, and 15. Neuronal apoptosis (terminal deoxynucleotidyl transferase dUTP nick-end labeling assay), microglial (marker OX42) activation, and tumor necrosis factor (TNF)-α expression in microglia in the hippocampus CA3 were measured by immunofluorescence methods.

Results: Compared with the TBI controls, without significant changes in TTC staining or in the motor function test, TBI-induced depression-like behavior was significantly attenuated by HBO therapy by day 15 after TBI. Simultaneously, TBI-induced neuronal apoptosis, microglial (marker OX42) activation, and TNF-α expression in the microglia in the hippocampus CA3 were significantly reduced by HBO.

Conclusions: Our results suggest that HBO treatment may ameliorate TBI-induced depression-like behavior in rats by attenuating neuroinflammation, representing one possible mechanism by which depression-like behavior recovery might occur. We also recommend HBO as a potential treatment for TBI-induced depression-like behavior if early intervention is possible.

Keywords: Depression-like behavior; Fluid percussion injury; Forced swim; Hippocampus; Microglia; Tumor necrosis factor–α.