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1: [Toxicol Appl Pharmacol.](#) 2006 Jun 1;213(2):152-9. Epub 2005 Dec 1.

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Hyperbaric oxygen reduces delayed immune-mediated neuropathology in experimental carbon monoxide toxicity.

[Thom SR](#), [Bhopale VM](#), [Fisher D](#).

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The goal of this investigation was to determine whether exposure to hyperbaric oxygen (HBO(2)) would ameliorate biochemical and functional brain abnormalities in an animal model of carbon monoxide (CO) poisoning. In this model, CO-mediated oxidative stress causes chemical alterations in myelin basic protein (MBP), which initiates an adaptive immunological response that leads to a functional deficit. CO-exposed rats do not show improvements in task performance in a radial maze. We found that HBO(2) given after CO poisoning will prevent this deficit, but not eliminate all of the CO-mediated biochemical alterations in MBP. MBP from HBO(2) treated CO-exposed rats is recognized normally by a battery of antibodies, but exhibits an abnormal charge pattern. Lymphocytes from HBO(2)-treated and control rats do not become activated when incubated with MBP, immunohistological evidence of microglial activation is not apparent, and functional deficits did not occur, unlike untreated CO-exposed rats. The results indicate that HBO(2) prevents immune-mediated delayed neurological dysfunction following CO poisoning.

PMID: 16325878 [PubMed - indexed for MEDLINE]

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