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Smoke inhalation-induced alveolar lung injury is inhibited by hyperbaric oxygen.

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Smoke-induced lung injury in rats was assessed in terms of histopathology, gross mortality, neutrophil accumulation and as capillary leak. Administration of hyperbaric oxygen (HBO2), 2.8 atm abs for 45 min, inhibited adhesion of circulating neutrophils subsequent to smoke inhalation. HBO2 reduced pulmonary neutrophil accumulation whether used in a prophylactic manner, 24 h before smoke inhalation, or as treatment immediately after the smoke insult Emphasis was placed on prophylactic administration of HBO2 to avoid the possibility that beneficial effects may be related to hastened removal of carbon monoxide. Based on all parameters tested, smoke inhalation injury was reduced by prophylactic aadministration of HBO2. The beneficial effect appears related to inhibition of neutroophil adhesion to the vasculature.

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