

Hyperbaric oxygen therapy reduces COX-2 expression in a dimethylhydrazine-induced rat model of colorectal carcinogenesis.

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Source

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Abstract

BACKGROUND:

A better understanding of oncogenesis mechanisms should result in more effective approaches to colorectal cancer (CRC) treatment and prevention. Hyperbaric oxygen (HBO₂) therapy is indicated as adjuvant treatment for infectious diseases as well as hypoxic and inflammatory lesions. The anti-inflammatory effect of HBO₂ could reduce colorectal carcinogenesis.

METHODS:

48 Wistar rats were randomly divided into the following groups: * G1 - control; * G2 - HBO₂ treatment; * G3 - 1,2-dimethylhydrazine (DMH) injection only; * G4 - DMH injection and HBO₂ treatment. These groups were further randomly divided into two subgroups: a. euthanasia at six weeks; and. b. euthanasia at 12 weeks. Animals belonging to G2 and G4 were subjected to 15 HBO₂ sessions, performed every 24 hours at 2.0 atm absolute pressure, 90 minutes each. Cancer was induced via intraperitoneal injection of DMH in G3 and G4. The aberrant crypt foci index (ACFi), the cell nuclear antigen index (PCNA) and the cyclooxygenase-2 index (iCOX-2) were determined.

RESULTS:

After DMH administration, ACFi increased and was higher in subgroups euthanized at six weeks than in those sacrificed at 12 weeks ($p < 0.001$). HBO₂ alone (G2) did not affect ACFi ($p > \text{or} = 0.05$). Larger increases of PCNA were detected in G2 versus G3 ($p < 0.05$). Comparison between G4 and control group mice revealed no differences in PCNA ($p > 0.05$). COX-2 was overexpressed in G3 ($p < 0.0001$) compared to G4.

CONCLUSION:

COX-2 expression was "induced" by DMH and reverted to a "wild"-type level of expression upon exposure to HBO₂.

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