SHORT COMMUNICATION

EFFECT OF THE NITRIC OXIDE DONOR AND THE NITRIC OXIDE SYNTHASE INHIBITOR ON THE LIVER OF RATS WITH CHRONIC HEPATITIS INDUCED BY DIMETHYLNITROSAMINE

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The present study was designed to examine the effects of the donor of nitric oxide (NO), NaNO₂, and the inhibitor of NO synthase, N⁶-nitro-L-arginine (L-NNA), on the development of dimethylnitrosamine (DMNA)-induced chronic hepatitis in rats. L-NNA decreased rat survival and enhanced the severity of hepatic encephalopathy in the DMNA-treated animals. The aggravation of the morphological signs of hepatitis, the activation of serum alanine aminotransferase and cytosolic superoxide dismutase activities and the increase in the liver malondialdehyde content were observed in this group. The treatment with NaNO₂ improved liver morphology, decreased serum marker enzyme activities, lowered the activities of α-D-mannosidase and N-acetyl-β-D-glucosaminidase compared to the DMNA-treated group.

The results of the morphological and biochemical studies suggest that L-NNA increased DMNA-induced liver damage, whereas NaNO₂ partially prevented the development of chronic hepatitis. It is proposed that the opposite effects of L-NNA and NaNO₂ are partially explained by a modulation of the free radical-dependent processes in the liver.

Key words: N-nitro-L-arginine, sodium nitrate, rat liver, hepatitis, dimethylnitrosamine