

## Accepted Manuscript

Title: Cirrhosis induced by bile duct ligation alleviates acetic acid intestinal damages in rats: Involvements of nitregeric and opioidergic systems

Authors: Nastaran Rahimi, Mahsa Hassanipour, Narges Sistany Allahabadi, Fatemeh Sabbaghziarani, Maryam Yazdanparast, Ahmadreza Dehpour



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**Cirrhosis induced by bile duct ligation alleviates acetic acid intestinal damages  
in rats: Involvements of nitrenergic and opioidergic systems**

**Manuscript (without Author Details)**

**Abstract**

Background: Colitis, a colonic inflammatory condition, showed a linkage with hepatobiliary disorders such as cirrhosis. It has been reported that both endogenous opioids and nitric oxide (NO) play critical roles in colitis pathogenesis. Moreover, opioid and NO levels showed elevation in patients with cirrhosis. The aim of this study was to evaluate the effect of cirrhosis on the experimental model of colitis and the possible involvement of opioidergic/nitrenergic systems in rats.

Methods: Colitis was induced by acetic acid 28 days after bile duct ligation (BDL). L-NAME, as an inhibitor of nitric oxide synthase and naltrexone, as an antagonist of opioid receptors were administered intraperitoneally to animals during 3 days after induction of colitis. Macroscopic colitis lesion area, inflammatory mediators change, NO metabolite levels, and colon microscopic injuries were assessed 3 days after induction.

Results: Cirrhosis significantly reduced the severity of damages to the colon. Administration of L-NAME (10 mg/kg), naltrexone (10 mg/kg) and co-administration of L-NAME (1 mg/kg) and naltrexone (5 mg/kg) significantly decreased the protective effect of BDL on colitis. Nitrite elevated levels in BDL rats were significantly diminished in L-NAME- and naltrexone-treated animals. Histopathology parameters and cytokines level alterations in the colon of acetic acid-treated animals after BDL was

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