Volume 273, 1 August 2017, Pages 266-272

Modulatory effect of silymarin on nuclear factor-erythroid-2-related factor 2 regulated redox status, nuclear factor-κB mediated inflammation and apoptosis in experimental gastric ulcer

Arafa Keshk, W.[a], Zahran, S.M.[b], Katary, M.A.[c], Abd-Elaziz Ali, D.[d]

[a] Medical Biochemistry Department, Faculty of Medicine, Tanta University, Egypt

[b] Biochemistry Department, Faculty of Pharmacy and Drug Manufacturing, Pharos University, Alexandria, Egypt

[c] Pharmacology and Toxicology Department, Faculty of Pharmacy, Damanhur University, Egypt

[d] Department of Histopathology, Faculty of Medicine, Tanta University, Tanta, Egypt

Abstract:

Non-steroidal anti-inflammatory drugs (NSAIDs) consumption has been commonly associated with gastric mucosal lesions including gastric ulcer. Silymarin (SM) is a flavonoid mixture with anti-oxidant and anti-inflammatory activities which explain its protective role against hepatic and renal injuries. However, its impact on gastric ulcer has not yet been elucidated. Thus we went further to investigate the potential protective effects of SM against indomethacin-induced gastric injury in rats. Pretreatment with SM (50 mg/kg orally) attenuated the severity of gastric mucosal damage as evidenced by decreasing ulcer index (UI) and ulcer score, improvement of disturbed histopathologicl features to be insignificant with those induced by the reference anti-ulcer drug. Pretreatment with SM also suppressed gastric inflammation by decreasing myeloperoxidase activity, tumer necrosis factor-α (TNF- α) and interleukin 6 (IL6) levels along with nuclear factor kappa B p65 (NF-κB) expression. Meanwhile, SM prevent gastric oxidative stress via inhibition of lipid peroxides formation, enhancement of glutathione peroxidase, superoxide dismutase activities and up-regulation of nuclear factor-erythroid-2-related factor 2 (Nrf2), the redox-sensitive master regulator of oxidative stress signaling. In conclusion, the results herein revealed that SM has a gastro-protective effect which is mediated via suppression of gastric inflammation, oxidative stress, increased the anti-oxidant and the cyto-protective defense mechanisms.

Reference:

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