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In vivo cellular and molecular gastroprotective mechanisms of chrysin: Emphasis on oxidative stress, inflammation and angiogenesis

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Gastric ulceration is one of the major gastrointestinal disorders affecting people worldwide. Despite medical advances, management of gastric ulcer and its complications remains a challenge facing medicine nowadays. In addition, currently available medicines exhibit limited efficacy and several side effects. Hence, the potential protective effects of chrysin -naturally occurring flavonoid- were tested against indomethacin-induced gastric ulcer model in rats. In a preliminary study, chrysin was administered to spargue-Dawly rats (200-220 g) at three different doses; 25, 50 and 100 mg/kg, single oral dose (S.O.D) compared to omeprazole given at a dose of 30 mg/kg, S.O.D. Indomethacin was administered at a dose of 48 mg/kg, S.O.D. Chrysin in both doses; 50 and 100 mg/kg were effective in promoting mucus secretion and preventing the rise in ulcer and lesion indices, acid production and histologic changes induced by indomethacin. During investigation of the possible underlying mechanisms, chrysin pretreatment significantly attenuated indomethacin-induced oxidative injury proved by its effects on catalase, reduced glutathione and lipid peroxidation levels. In addition, chrysin reduced inflammatory response caused by indomethacin owing to its effects on nuclear factor-kappa B (NF-κB), tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β). Moreover, chrysin activated peroxisome proliferator activated receptor-ɣ (PPAR-γ) leading to a phenotypic switch from pro-inflammatory M1 macrophages to the anti-inflammatory M2 macrophages evidenced by the upregulated mRNA expression levels of PPAR-γ and M2 marker genes (Arg-1 and CD206) and downregulation of M1 marker genes (IL-6 and CCL3). Furthermore, chrysin initiated angiogenesis via increasing expression of vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF) and cluster of differentiation-31 (CD31) resulting in tissue repair. Collectively, these findings indicate that chrysin possesses a potential protective effect against indomethacin-induced gastric ulcer via suppressing oxidative stress, inflammation and initiating angiogenesis.

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