Molecular Mechanisms Involved in the Gastric Ulcer Healing Promoted by Chrysin: Participation of EGF, COX-1 and IL-10

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INTRODUCTION

Gastric ulcer healing is a complex process where prostaglandins, growth factors and cytokines play an important role. EGF is the major angiogenic factor derived from epithelia that promotes cellular proliferation. Some cytokines like IL-10 can be released by the resident macrophages and reduces the local inflammation caused by the ulcer. Prostaglandins are lipid mediators produced by COX iso enzymes involved in the gastric maintenance and inflammation. Besides pharmacological treatment reduced the ulcer size, these drugs are poor angiogenic factors and leads to ulcer recurrence after suspension in use. In this sense, this study aims to test Chrysin in the model of gastric ulcer induced by acetic acid and investigate the molecular mechanism involved in the possible healing activity.

RESULTS

Chrysin reduced the macroscopic lesion area in 46.1% when compared to vehicle treated group with seven days of treatment. Also, elevated the expression levels of COX-1. After 14 days, the flavone elevated the expression of COX-1, EGF and IL-10 and reduced the expression of COX-2, indicating the anti-inflammatory and angiogenic effect in the gastric mucosa.

CONCLUSIONS

Our results suggest that Chrysin has a healing activity in the gastric mucosa, related to inhibition of expression of cyclooxygenase 2, increase in the angiogenesis mediated by EGF and increase the production of prostaglandins trough COX-1. Besides that, Chrysin also elevated the expression of IL-10, an anti-inflammatory cytokine.

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