










# The coumaric acid and syringic acid ameliorate acetic acid-induced ulcerative colitis in rats via modulator of Nrf2/HO-1 and pro-inflammatory cytokines

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## Abstract

### Background

Ulcerative colitis (UC) is an inflammatory bowel disease (IBD) that causes uncontrolled inflammation and ulcers in your digestive tract. The coumaric acid and syringic acid are phenolic derivative found in many fruits and vegetables and is widely recognized for the ability of anti-parasitic, anti-microbial, anti-viral, anti-inflammatory, and antioxidant. The purpose of this study was to investigate the anti-inflammatory and antioxidant properties of coumaric acid and syringic acid on acetic acid-induced colitis in rats.

### Methods

A total of 64 male Wistar rats were divided into eight equal groups (n=8). Colitis was induced by intrarectal administration of acetic acid, and rats orally received coumaric acid (100 and 150mg/kg), syringic acid (10, 25, and 50mg/kg), and dexamethasone (2mg/kg) once per day for four days after colitis induction. Then, HO-1, Nrf2, and NQO1 mRNA expression were quantified by real time-PCR. Finally, the tissue levels of TNF- $\alpha$  and IL-1 $\beta$  protein were measured by ELISA.

### Results

Colitis led to a decrease in HO-1, Nrf2, and NQO1 mRNA expression and an increase in the tissue levels of TNF- $\alpha$  and IL-1 $\beta$  protein in the colon tissue. Treatment with dexamethasone significantly increased HO-1, Nrf2, and NQO1 mRNA expression and decreased the tissue levels of TNF- $\alpha$  and IL-1 $\beta$  protein compared to the UC group. Treatment with 150mg/kg of coumaric acid and 50mg/kg of syringic acid significantly increased HO-1, Nrf2, and NQO1 mRNA expression compared to the UC group. Also, treatment with 100 and 150mg/kg of coumaric acid and 10, 25, and 50mg/kg of syringic acid significantly decreased the tissue levels of TNF- $\alpha$  and IL-1 $\beta$  protein compared to the UC group.

### Conclusion

The coumaric acid and syringic acid, especially at high doses, may be an alternative strategy for the treatment of UC by the reduction of TNF- $\alpha$  and IL-1 $\beta$  levels and upregulation of the Nrf2/HO-1 pathway.

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