Gallic acid attenuates dextran sulfate sodium- induced experimental colitis in BALB/c mice

ABSTRACT

Gallic acid (GA) is a polyhydroxy phenolic compound that has been detected in various natural products, such as green tea, strawberries, grapes, bananas, and many other fruits. In inflammatory bowel disease, inflammation is promoted by oxidative stress. GA is a strong antioxidant; thus, we evaluated the cytoprotective and anti-inflammatory role of GA in a dextran sulfate sodium (DSS)-induced mouse colitis model. Experimental acute colitis was induced in male BALB/c mice by administering 2.5% DSS in the drinking water for 7 days. The disease activity index; colon weight/length ratio; histopathological analysis; mRNA expressions of IL-21 and IL-23; and protein expression of nuclear erythroid 2-related factor 2 (Nrf2) were compared between the control and experimental mice. The colonic content of malondialdehyde and the activities of superoxide dismutase, catalase, glutathione peroxidase, and glutathione reductase activity were examined as parameters of the redox state. We determined that GA significantly attenuated the disease activity index and colon shortening, and reduced the histopathological evidence of injury. GA also significantly ($P<0.05$) reduced the expressions of IL-21 and IL-23. Furthermore, GA activates/upregulates the expression of Nrf2 and its downstream targets, including UDP-GT and NQO1, in DSS-induced mice. The findings of this study demonstrate the protective effect of GA on experimental colitis, which is probably due to an antioxidant nature of GA.

Keyword: IL-21; NQO1; MDA; Enzymic antioxidants; Nrf2