

# Comparative assessment of the effects of resatorvid and sulfasalazine on disease severity, intestinal histopathology, as well as inflammation and oxidative stress markers in a murine experimental model of ulcerative colitis

Baneen Abbas Altaay and Fatima Adnan Alzubaidi

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Ulcerative colitis (UC) is a chronic relapsing inflammatory disease with an incompletely understood aetiology and no definitive cure, in which toll-like receptor 4 (TLR4)-mediated inflammation plays a key pathogenic role and represents a promising therapeutic target. This study aimed at evaluating the protective effects of resatorvid compared with those of sulfasalazine in a dextran sulfate sodium (DSS)-induced experimental model of UC in mice. To this end, 50 adult male BALB/C mice (aged 6–10 weeks; weighing 22–30 g) were randomly assigned into five experimental groups: (i) a control group, (ii) a DSS-induced UC group (in which DSS was administered orally through a cage feeding bottle at a concentration of 5% for 10 days), (iii) a group that has been exposed to DSS and has also received resatorvid (at 3 mg/kg, once daily, intraperitoneally) for 10 days, (iv) a group that has been exposed to DSS and has also received sulfasalazine (at 100 mg/kg, orally, by gavage once, daily) for 10 days, and (v) a vehicle control group that has been exposed to DSS and has also received 1% DMSO (in normal saline, once daily, intraperitoneally) for 10 days. On day 11 of the study, the mice were sac-

rificed, and colonic tissue was collected for biochemical and histopathological analysis. The levels of the oxidative stress markers myeloperoxidase (MPO) and isoprostane-8, of TLR4, and of the pro-inflammatory cytokines tumour necrosis factor alpha (TNF- $\alpha$ ) and interleukin-1 beta (IL-1 $\beta$ ) were quantified in the colonic tissues using ELISA kits. Histological examination was performed in order to assess the extent of the mucosal damage and inflammatory cell infiltration. DSS administration induced severe experimental colitis, as evidenced by a significant elevation of the disease activity index (DAI), macroscopic and histopathological scores, oxidative stress markers, and pro-inflammatory cytokine levels, as compared to those of the control group ( $p < 0.001$ ). The tissue levels of TNF- $\alpha$ , IL-1 $\beta$ , MPO, isoprostane-8, and TLR4 were found to be increased by approximately 274%, 262%, 136%, 135%, and 118.3%, respectively, in the DSS-induced UC model mice when compared to those of the control group. No significant differences were observed in the herein assessed parameters between the DSS-induced UC model group and the vehicle- and DSS-treated group. The treatment

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with sulfasalazine significantly reduced the DAI and the macroscopic and histopathological scores, as well as the colonic tissue TNF- $\alpha$ , IL-1 $\beta$ , MPO, isoprostane-8, and TLR4 levels by approximately 51%, 49%, 56%, 44%, 44%, 37%, 34%, and 25%, respectively, when compared with those of the UC model mice ( $p < 0.001$ ). Similarly, the administration of resatorvid was found to significantly attenuate the DSS-induced colonic injury by reducing the DAI, the macroscopic and histopathological scores, and the tissue levels of TNF- $\alpha$ , IL-1 $\beta$ , MPO, isoprostane-8, and TLR4 by approximately 47%, 42%, 47%, 40%, 38%, 35%, 32%, and 44%, respectively, compared with those of the DSS-induced UC model mice ( $p < 0.001$ ). No significant differences were observed amongst the herein assessed parameters between the resatorvid- and the sulfasalazine-treated groups, with the exception of resatorvid demonstrating a significantly greater suppression of the colonic TLR4 expression compared to sulfasalazine ( $p < 0.001$ ). Our findings are supportive of selective TLR4 inhibition as a promising therapeutic strategy for the treatment of UC.

#### **Keywords**

oxidative stress; sulfasalazine; TAK-242; TLR4; ulcerative colitis

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#### **Conflicts of interest statement**

None to declare.

#### **Affiliation(s)**

College of Pharmacy, University of Babylon, Hillah, Iraq (BAA) ▪ Department of Pharmacology and Toxicology, College of Pharmacy, University of Babylon, Hillah, Iraq (FAA)

#### **Correspondence**

Baneen Abbas Altaay  
College of Pharmacy  
University of Babylon  
Hillah, Iraq  
pha746.baneen.abbas@student.uobabylon.edu.iq