



Effect of secondary lymphoid tissue chemokine suppression on experimental ulcerative colitis in mice

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ABSTRACT. The secondary lymphoid tissue chemokine (CCL21) is closely associated with lymphoid homing and anti-tumor immune responses. CCL21 also has a chemotactic effect on intestinal lymphocytes. This study mainly focused on CCL21 expression in experimental ulcerative colitis and on the effects of CCL21 suppression on this disease in mice. The mouse colitis model was induced by dextran sulfate sodium (DSS) in 40 female BALB/c mice that were equally distributed into five groups: control, DSS, propylene glycol, triptolide (TL), and dexamethasone treatment groups. The disease activity index, general morphology score of the colon, and histological pathology score of colon tissues were evaluated. CCL21 expression was examined in colons of mice by immunohistochemistry, reverse transcription-polymerase chain reaction, and Western blotting analysis. CCL21 was upregulated in the mouse model of ulcerative colitis (control group *vs* DSS group/propylene glycol group, $P < 0.01$). The TL and dexamethasone treatments improved colitis symptoms and decreased CCL21

expression (TL group/dexamethasone group vs DSS group/propylene glycol group, $P < 0.05$). In conclusion, CCL21 was shown to be involved in the induction of ulcerative colitis. Suppression of CCL21 expression decreased damage induced from ulcerative colitis, indicating that CCL21 targeted therapy might be an effective treatment for this disease.

Key words: Ulcerative colitis; Secondary lymphoid tissue chemokine; Triptolide; Dexamethasone