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Association of the expression levels of STAT and SOCS in mucosal tissues with the severity and prognosis in patients with ulcerative colitis

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Abstract: Objective To investigate the expression of signal transducer and activator of transcription (STAT) and suppressor of cytokine signaling (SOCS) in ulcerative colitis (UC) lesion tissue, and to analyze their relationship with the severity of UC and therapeutic outcomes. **Methods** A case-control study design was used, and 70 UC patients admitted to Cangzhou People's Hospital were selected as the UC group. Another 70 cases of normal intestinal mucosal tissue confirmed by pathological examination due to previous trauma, surgery, or other reasons were used as the control group. The expression levels of STAT mRNA and SOCS mRNA in the intestinal mucosal tissues of the two groups were compared. Stratification was conducted based on disease in the active phase (n=55) or in remission phase (n=15). Mesalazine treatment was administered to UC patients in active phase, and the STAT mRNA and SOCS mRNA expression levels were compared among patients with different treatment responses. **Results** The mRNA expression levels of STAT1, STAT3, and STAT5 in the intestinal mucosa of the UC group, were higher than those of the control group, while the mRNA expression levels of SOCS2 and SOCS3 were lower than those of the control group, with the differences being statistically significant (P<0.05). Compared with patients in remission phase, UC patients in active phase had significantly higher mRNA expression levels of STAT5 in intestinal mucosal (P<0.05) , while significantly lower mRNA expression levels of SOCS2 and SOCS3 (P<0.05). Fifty-five UC patients in the active phase were treated with mesalazine, with 38 cases of good curative effect and 17 cases of poor curative effect. Compared with UC patients with good curative effects, UC patients with poor curative effects had higher expression levels of STAT1, STAT3 and STAT5 mRNA (P<0.05), and lower expression levels of SOCS2 and SOCS3 mRNA (P<0.05). **Conclusion** The mRNA expressions of STAT1, STAT3 and STAT5 in UC lesions are higher than those in healthy individuals, while the mRNA expressions of SOCS2 and SOCS3 are lower than those in healthy individuals, which are related to disease activity and treatment efficacy.

Keywords: Ulcerative colitis; Signal transducer and activator of transcription; Suppressor of cytokine signaling; Severity of disease; Treatment outcome
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Ulcerative colitis (UC) is an inflammatory bowel disease of unknown etiology that primarily affects the colon and rectum, leading to symptoms such as abdominal pain, diarrhea, and mucopurulent bloody stools [1-2]. Its core pathological features are abnormal inflammatory responses and immune dysregulation within the intestinal mucosa [3]. In recent years, the roles of the signal transducer and activator of transcription (STAT) family and the suppressor of cytokine signaling (SOCS) family in the pathological mechanisms of UC have received increasing attention [4-5]. As key transcriptional regulators, abnormal expression of STAT proteins is directly linked to the exacerbated inflammatory process in UC. SOCS, as negative regulators of the STAT pathway, are crucial for maintaining immune homeostasis by inhibiting STAT activity [6-7]. The inhibitory effects of SOCS2 and SOCS3 in the JAK-STAT pathway have been demonstrated in animal experiments, and changes in their expression are closely associated with exacerbated intestinal mucosal inflammation in UC models [8]. However, most of these studies have focused on single or a few SOCS family members. Whether other members of the SOCS family (SOCS1, SOCS5, SOCS6, SOCS7) play a role in the regulation of inflammation in UC, their dynamic changes during UC treatment, and their predictive value for therapeutic efficacy-particularly systematic studies based on clinical data-remain insufficient. In view of this, the present study investigates the expression of STAT and SOCS in UC lesions, analyses their association with disease severity and treatment outcomes, aims to provide new perspectives for understanding the pathogenesis of UC, and offers potential molecular targets and biomarkers for optimizing clinical treatment strategy.

1 Data and Methods

1.1 General data

This study used a case-control design. A total of 70 UC patients diagnosed at Cangzhou People's Hospital from March 2022 to June 2024 were enrolled as the UC group. Seventy subjects whose normal intestinal mucosal tissue was confirmed by pathological examination due to previous trauma, surgery, or other reasons were enrolled as the control group. This study was approved by the Ethics Committee of Cangzhou People's Hospital [K2022- Approval-071 (12.16)]. There was a significant difference between the two groups in family history of UC (P<0.05); no significant difference

was found in other baseline data (P>0.05), indicating comparability (see **Table 1**).

Diagnostic criteria: Diagnosis of UC referred to the *Chinese consensus on diagnosis and treatment of inflammatory bowel disease (2018, Beijing)* [9]; diagnosis was confirmed by pathological examination of biopsy samples obtained via electronic colonoscopy.

Inclusion criteria: (1) met the diagnostic criteria for UC; (2) aged 18–65 years; (3) complete medical records and good compliance; (4) interval between endoscopic examination and blood tests ≤ 48 hours.

Exclusion criteria: (1) malignancy or hematological disease; (2) concurrent other infectious diseases; (3) concurrent severe cardiac or pulmonary insufficiency, thrombotic diseases, or autoimmune diseases; (4) use of drugs affecting coagulation function within the past 3 months; (5) use of corticosteroids within 3 months before admission; (6) pregnant or breastfeeding women.

Tab.1 Comparison of baseline data between two groups (n=70, case)

Group	Age (years, $\bar{x} \pm s$)	BMI (kg/m², $\bar{x} \pm s$)	Gender		Smoking	Drinking	Family history of intestinal polyps	Family history of colorectal tumors	Family history of UC
			Male	Female					
UC group	52.6±9.4	23.05±1.95	61	9	45	40	21	4	14
Control group	50.7±8.8	23.36±2.00	53	17	38	32	15	1	3
t/χ^2 **value**	1.235	0.929	3.023		1.450	1.830	1.346	0.831	8.101
P **value**	0.219	0.355	0.082		0.229	0.176	0.246	0.362	0.004

1.2 Colonoscopy and the mRNA detection

Colonoscopy: All subjects underwent colonoscopy performed by physicians with at least 5 years of experience in endoscopic examination. The condition of the intestinal mucosal lesions was recorded in detail.

mRNA detection of SOCS and STAT: Tissue samples of intestinal mucosa from both groups, archived in the pathology department, were collected for examination. Total RNA was extracted from the tissues using Trizol RNA extraction reagent (Invitrogen). Subsequently, cDNA was synthesized from the extracted total RNA samples by reverse transcription using a cDNA synthesis kit (Promega). The cDNA samples were amplified using a PCR reaction kit (Promega). PCR was performed on an ABI PRISM 7500 real-time PCR system using the GoTaq® qPCR Master Mix kit (Promega, containing BRYT Green® dye, catalogue number: A6001) to amplify the target genes SOCS1–7, STAT1, STAT3, and STAT5. The reaction conditions were: pre-denaturation at 95 °C for 10 min, followed by 40 cycles of denaturation at 95 °C for 15 s, annealing and extension at 60 °C for 1 min. Finally, based on the real-time quantitative PCR results, the relative mRNA expression levels of SOCS1 to SOCS 7, STAT1, STAT3, and STAT5 were calculated for each sample group.

1.3 Treatment regimen

All UC patients received active treatment upon admission.

Active phase: Patients were given mesalazine enteric-coated tablets (Losan Pharma GmbH, Germany, 0.5 g/tablet, National Drug Approval Number HJ20171358) combined with prednisone (Jinyao Pharmaceutical Co., Ltd., 5 mg/tablet, National Drug Approval Number H20237053). The specific regimen was: mesalazine enteric-coated tablets 1 g/time, 4 times daily orally; prednisone 30–40 mg/day. After initial control (1–2 weeks), prednisone was tapered by 5 mg per week, and when the dose was reduced to 20 mg/day, it was then tapered by 2.5 mg every 1–2 weeks until discontinuation.

Remission phase: Only mesalazine enteric-coated tablets were used for maintenance therapy, 1 g/time, 4 times daily orally. A course of treatment was 14 days, and 1–2 consecutive courses were given depending on the patient's improvement.

After 2 months of treatment, patients underwent repeat colonoscopy to comprehensively evaluate treatment effectiveness and ensure effective disease control and management.

1.4 Criteria of Disease Severity and Efficacy Evaluation

Disease severity in UC patients was assessed using the Sutherland Disease Activity Index (DAI) criteria [10], which primarily include objective measurement indicators and subjective evaluation indicators: patient clinical symptoms, endoscopic findings, laboratory tests, and histopathological grading. DAI scores of 0–1 as remission phase, and scores of 2–9 as active phase.

The clinical efficacy evaluation for patients with active UC referred to the criteria of the *Chinese Guidelines for Surgical Treatment of Ulcerative Colitis* [11] and was based on post-treatment colonoscopy results. Patients whose diarrhea, mucopurulent bloody stools decreased, abdominal pain was relieved or disappeared, and endoscopy showed ulcer mucosal healing >50% or complete healing were included in the good efficacy group. Patients whose diarrhea, mucopurulent bloody stools, and abdominal pain did not improve significantly, causing severe impact on daily life, and endoscopy showed <50% ulcer healing were included in the poor efficacy group.

1.5 Statistical methods

Data were analysed using SPSS 21.0 software. Continuous data following a normal distribution were described as $\bar{x} \pm s$, and comparisons between groups were performed using independent samples t-tests. Categorical data were described as case (%), and comparisons were performed using the Chi-square test. P<0.05 was considered statistically significant.

2 Results

2.1 Comparison of relative SOCS mRNA expression levels between the two groups

The relative expression levels of SOCS2 and SOCS3 mRNA in the UC group were lower than those in the control group (P<0.05). There was no significant difference in the relative expression levels of SOCS1, SOCS4, SOCS5, SOCS6, and SOCS7 mRNA between the two groups (P>0.05). See **Table 2**.

2.2 Comparison of relative STAT mRNA expression levels between the two groups

The relative expression levels of STAT1, STAT3, and STAT5 mRNA in the intestinal mucosa of the UC group were higher than those in the control group (P<0.05). See **Table 3**.

2.3 Comparison of relative STAT mRNA expression levels at different phase

Among the 70 UC patients, 15 were in remission and 55 had active phase. The relative expression level of STAT5 mRNA in patients with active UC was higher than that in patients in remission (P<0.05). There was no significant difference in the relative expression levels of STAT1 and STAT3 mRNA between patients with active disease and those in remission (P>0.05). See **Table 4**.

2.4 Comparison of relative SOCS mRNA expression levels at different phase

The levels of SOCS2 and SOCS3 mRNA in patients with active UC were lower than those in patients in remission (P<0.05). There were no significant differences in the relative expression levels of SOCS1, SOCS4, SOCS5, SOCS6, and SOCS7 mRNA between patients with active disease and those in remission (P>0.05). See **Table 5**.

2.5 Comparison of relative SOCS mRNA expression levels of active UC patients with different treatment outcomes

The relative expression levels of SOCS2 and SOCS3 mRNA in patients with active UC who had poor outcomes were lower than those in patients with good outcomes (P<0.05). There was no significant difference in the relative expression levels of SOCS1, SOCS4, SOCS5, SOCS6, and SOCS7 mRNA between patients with poor outcomes and those with good outcomes (P>0.05). See **Table 6**.

2.6 Comparison of relative STAT mRNA expression levels of active UC patients with different treatment outcomes

Among the 55 patients with active UC treated with mesalazine, 38 had a good response and 17 had a poor response. The relative expression levels of STAT1, STAT3, and STAT5 mRNA in patients with poor outcomes were higher than those in patients with good outcomes (P<0.05). See **Table 7**.

Tab.2 Comparison of the relative expression of SOCS mRNA in intestinal mucosa between two groups (n=70, $\bar{x} \pm s$)

Group	SOCS1	SOCS2	SOCS3	SOCS4	SOCS5	SOCS6	SOCS7
UC group	1.09±0.21	0.39±0.08	0.33±0.10	0.98±0.10	1.03±0.15	1.06±0.18	0.97±0.17
Control group	1.06±0.18	1.00±0.18	1.00±0.13	1.00±0.14	1.00±0.14	1.02±0.15	1.01±0.14
***t* value**	0.907	25.910	34.178	0.973	1.223	1.428	1.520
***P* value**	0.366	<0.001	<0.001	0.332	0.223	0.155	0.131

Tab.3 Comparison of the relative expression of STAT mRNA in intestinal mucosa between two groups (n=70, $\bar{x} \pm s$)

Group	STAT1	STAT3	STAT5
UC group	1.93±0.45	2.30±0.57	1.76±0.32
Control group	1.22±0.39	1.44±0.39	1.09±0.27
***t* value**	9.976	10.418	13.389
***P* value**	<0.001	<0.001	<0.001

Tab.4 Comparison of the relative expression of STAT mRNA in intestinal mucosa in UC patients at different phase ($\bar{x} \pm s$)

Disease stage	Case	STAT1	STAT3	STAT5
Remission	15	1.77±0.39	2.10±0.50	1.62±0.29
Active Phase	55	1.98±0.42	2.36±0.54	1.80±0.30
***t* value**		1.741	1.678	2.074
***P* value**		0.086	0.098	0.042

Tab.5 Comparison of relative expression of SOCS mRNA in UC patients at different disease stages ($\bar{x} \pm s$)

Disease stage	Case	SOCS1	SOCS2	SOCS3	SOCS4	SOCS5	SOCS6	SOCS7
Remission	15	1.10±0.18	0.51±0.08	0.54±0.09	1.00±0.08	1.01±0.12	1.07±0.16	1.00±0.15
Active Phase	55	1.09±0.20	0.36±0.06	0.27±0.08	0.97±0.09	1.04±0.14	1.06±0.14	0.96±0.12
***t* value**		0.175	7.968	11.282	1.170	0.757	0.238	1.083
***P* value**		0.862	<0.001	<0.001	0.246	0.452	0.813	0.282

Tab.6 Comparison of the relative expression of SOCS mRNA in the intestinal mucosa of active UC patients with different treatment outcomes ($\bar{x} \pm s$)

Different treatment outcomes	Case	SOCS1	SOCS2	SOCS3	SOCS4	SOCS5	SOCS6	SOCS7
Good Efficacy	38	1.08±0.17	0.38±0.08	0.30±0.07	0.98±0.07	1.03±0.13	1.05±0.15	0.97±0.14
Poor Efficacy	17	1.11±0.18	0.32±0.07	0.20±0.06	0.95±0.09	1.06±0.12	1.08±0.12	0.94±0.11
***t* value**		0.594	2.666	5.105	0.342	0.809	0.726	0.781
***P* value**		0.555	0.010	<0.001	0.185	0.422	0.471	0.438

Tab.7 Comparison of the relative expression of STAT mRNA in active UC patients with different treatment outcomes ($\bar{x} \pm s$)

Different treatment outcomes	Case	STAT1	STAT3	STAT5
Good Efficacy	38	1.81±0.35	2.21±0.47	1.68±0.26
Poor Efficacy	17	2.36±0.40	2.70±0.52	2.07±0.28
t value		5.153	3.458	5.021
P value		<0.001	<0.001	<0.001

3 Discussion

Genetic, environmental, and immune factors have been implicated in ulcerative colitis (UC). In recent years, the importance of the STAT family and SOCS in UC pathology has gradually emerged. However, research on how their expression patterns in UC lesions affect disease progression and treatment outcomes remains insufficient [12-13].

Overactivation of STAT family proteins can induce excessive production and amplification of inflammatory cytokines, accelerating intestinal tissue damage and the inflammatory response [14]. The results of this study show that the relative expression levels of STAT1, STAT3, and STAT5 mRNA in the mucosal tissue of UC patients were higher than those in the control group, suggesting that these factors play important roles in the pathogenesis of UC. Possible explanations include that intestinal dysbiosis, impaired mucosal barrier function, and other factors may induce an excessive immune response against intestinal self-antigens in UC patients, promoting the massive release of cytokines, activating STAT signaling pathways, and enhancing the transcription and expression of inflammation-related genes [15-16].

This study also found that the relative expression levels of SOCS2 and SOCS3 mRNA in the UC group were lower than those in the control group, indicating a role for SOCS2 and SOCS3 in the pathophysiology of UC. Some studies have reported that the SOCS family regulates cytokine signaling by inhibiting STAT activity, thereby maintaining immune homeostasis and preventing excessive activation of inflammatory responses [17]. In the pathological context of UC, low expression of SOCS2 and SOCS3 mRNA weakens their inhibitory effect on the STAT pathway, leading to abnormal activation of STAT1, STAT3, STAT5 and other molecules, triggering excessive production of downstream inflammatory cytokines. This is closely linked to the complex immunopathological process of UC, collectively contributing to abnormal immune system responses and exacerbating inflammation. Some studies have suggested that levels of inflammation-related factors are elevated in UC patients [18]; the results of the present study are consistent with those findings, further confirming the persistent inflammatory state in patients with UC.

Stratified analysis of patients with UC showed that the relative expression levels of STAT1, STAT3, and STAT5 mRNA in the intestinal mucosa of the UC group were higher than those in the control group, while the relative expression levels of SOCS2 and SOCS3 mRNA were lower, reflecting a close association between these markers and UC disease activity. A possible explanation is that high expression of STAT1, STAT3, and STAT5 mRNA in patients with active UC leads to sustained activation of inflammatory signaling pathways, promoting excessive production of downstream inflammatory cytokines and exacerbating intestinal tissue inflammation and damage [19]. Low expression of SOCS2 and SOCS3 mRNA in patients with active UC suggests a profound imbalance in immune regulatory mechanisms. The normal function of the SOCS family limits the intensity and duration of inflammatory responses by inhibiting STAT activation. In active UC, downregulation of SOCS2 and SOCS3 mRNA expression significantly impairs their inhibitory effect, leading to abnormal activation of the STAT pathway and uncontrolled inflammation. Building on previous research, this study identifies the potential of STAT and SOCS as biomarkers for assessing disease activity. By closely monitoring changes in these markers, clinicians may better understand disease dynamics in UC patients, providing a scientific basis for developing personalized and effective treatment strategies.

The efficacy of mesalazine is primarily based on suppressing intestinal inflammation, reducing the release of inflammatory mediators, and promoting intestinal mucosal repair. Studies have suggested that different activation states of inflammatory signaling pathways in patients are a key factor contributing to individual differences in treatment response [20]. In this study, patients with poor efficacy had higher relative expression levels of STAT1, STAT3, and STAT5 mRNA and lower relative expression levels of SOCS2 and SOCS3 mRNA compared with those who with good efficacy, reflecting a close relationship between the efficacy of mesalazine in UC and these molecular markers. A possible explanation is that high expression of STAT1, STAT3, and STAT5 mRNA in patients with poor efficacy reflects sustained activation of inflammatory signaling pathways and excessive production of inflammatory cytokines, which may exceed the conventional inhibitory capacity of mesalazine or require higher doses to achieve efficacy. Furthermore, SOCS2 and SOCS3, as negative regulators of the STAT pathway [21], show notably low expression in poor responders, weakening their inhibitory effect on STAT activation and exacerbating inflammation. Therefore, when developing treatment plans for UC, in addition to considering the characteristics of the drug itself, attention should be paid to the patient's inflammatory signaling pathway status and the function of negative feedback regulatory mechanisms to achieve precise and efficient treatment.

This study has unique features in its experimental design compared with previous studies. It performed stratified analysis of UC disease activity (active *vs* remission), examined the dynamic relationship between relative expression levels of STAT and SOCS mRNA and disease severity, and, by investigating the association between mesalazine treatment efficacy and changes in these molecular markers, demonstrated their potential as biomarkers for predicting treatment response, opening

new perspectives for precision medicine strategies in UC.

In summary, the expression of STAT1, STAT3, and STAT5 mRNA in UC lesional tissue is higher than that in healthy individuals, while the expression of SOCS2 and SOCS3 mRNA is lower, and these expression patterns are associated with treatment outcomes. However, this study has certain limitations. Due to financial and geographical constraints, the sample size was limited and sourced from a single region. Future studies will increase the sample size, expand the geographical sources, and conduct large-scale, multicenter investigations.

Conflict of Interest None

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·炎症性肠病专题·论著·

溃疡性结肠炎患者肠黏膜组织中STAT、SOCS表达水平与疾病严重程度及预后的关系

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摘要：目的　探讨溃疡性结肠炎(UC)病灶组织中信号转导与转录激活因子(STAT)、细胞因子信号抑制因子(SOCS)的表达，分析其与UC严重程度及治疗结局的关系。**方法**　采用病例对照研究方法，选取2022年3月至2024年6月沧州市人民医院确诊的70例UC患者作为UC组，既往因外伤、手术等原因获取的70例经病理学检查证实为正常肠黏膜组织作为对照组，对比两组肠黏膜组织中STAT mRNA、SOCS mRNA相对表达量，并根据疾病处于活动期(n=55)和缓解期(n=15)进行分层，对活动期UC患者实施美沙拉秦治疗，对比不同疗效患者的STAT mRNA、SOCS mRNA相对表达量。**结果**　UC组肠黏膜STAT1、STAT3、STAT5 mRNA相对表达量均高于对照组，SOCS2、SOCS3 mRNA相对表达量均低于对照组，差异有统计学意义(P<0.05)。活动期UC患者的肠黏膜STAT5 mRNA相对表达量高于缓解期患者(P<0.05)，SOCS2、SOCS3 mRNA相对表达量低于缓解期患者(P<0.05)。55例活动期UC患者接受美沙拉秦治疗，疗效良好38例，疗效不佳17例。疗效不佳的活动期UC患者STAT1、STAT3、STAT5 mRNA相对表达量高于疗效良好患者，SOCS2、SOCS3 mRNA相对表达量低于疗效良好患者，差异有统计学意义(P<0.05)。**结论**　UC病灶组织中STAT1、STAT3、STAT5 mRNA表达高于健康人群，SOCS2、SOCS3 mRNA表达低于健康人群，并且与疾病活动情况及治疗效果有关。

关键词：溃疡性结肠炎；信号转导子与转录激活因子；细胞因子信号抑制子；疾病严重程度；治疗结局
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Association of the expression levels of STAT and SOCS in mucosal tissues with the severity and prognosis in patients with ulcerative colitis

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Abstract: Objective　To investigate the expression of signal transducer and activator of transcription (STAT) and suppressor of cytokine signaling (SOCS) in ulcerative colitis (UC) lesion tissue, and to analyze their relationship with the severity of UC and therapeutic outcomes. **Methods**　A case-control study design was used, and 70 UC patients admitted to Cangzhou People's Hospital from March 2022 to June 2024 were selected as the UC group. Another 70 cases of normal intestinal mucosal tissue confirmed by pathological examination due to previous trauma, surgery, or other reasons were used as the control group. The expression levels of STAT mRNA and SOCS mRNA in the intestinal mucosal tissues of the two groups were compared. Stratification was conducted based on disease in the active phase (n=55) or in remission phase (n=15). Mesalazine treatment was administered to UC patients in active phase, and the STAT mRNA and SOCS mRNA expression levels were compared among patients with different treatment responses. **Results**　The mRNA expression levels of STAT1, STAT3, and STAT5 in the intestinal mucosa of the UC group, were higher than those of the control group, while the mRNA expression levels of SOCS2 and SOCS3 were lower than those of the control group, with the differences being statistically significant (P<0.05). Compared with patients in remission phase, UC

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patients in active phase had significantly higher mRNA expression levels of STAT5 in intestinal mucosal (P<0.05), while significantly lower mRNA expression levels of SOCS2 and SOCS3 (P<0.05). Fifty-five UC patients in the active phase were treated with mesalazine, with 38 cases of good curative effect and 17 cases of poor curative effect. Compared with UC patients with good curative effects, UC patients with poor curative effects had higher expression levels of STAT1, STAT3 and STAT5 mRNA (P<0.05), and lower expression levels of SOCS2 and SOCS3 mRNA (P<0.05). **Conclusion** The mRNA expressions of STAT1, STAT3 and STAT5 in UC lesions are higher than those in healthy individuals, while the mRNA expressions of SOCS2 and SOCS3 are lower than those in healthy individuals, which are related to disease activity and treatment efficacy.

Keywords: Ulcerative colitis; Signal transducer and activator of transcription; Suppressor of cytokine signaling; Severity of disease; Treatment outcome

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溃疡性结肠炎(ulcerative colitis,UC)作为一种病因尚不明确的炎症性肠病,主要影响结肠与直肠,可导致腹痛、腹泻及黏液脓血便等症状[1-2]。其核心病理特征在于肠黏膜内异常的炎症反应与免疫应答失衡[3]。近年来,信号转导与转录激活因子(signal transducer and activator of transcription,STAT)家族及细胞因子信号抑制因子(suppressor of cytokine signaling,SOCS)在UC病理机制中的角色日益受到关注[4-5]。STAT蛋白作为关键的转录调控因子,其异常表达直接关联UC的炎症加剧过程,SOCS作为STAT通路的负调节器,通过抑制STAT活性,对维持免疫稳态至关重要[6-7]。SOCS2和SOCS3在JAK-STAT通路中的抑制作用已被动物实验所证实,其表达变化与UC模型中的肠黏膜炎症加剧紧密相关[8]。但这些研究多局限于单个或少数SOCS家族成员,SOCS家族其他成员(SOCS1、SOCS5、SOCS6、SOCS7)在UC炎症调控中是否发挥作用,在UC治疗过程中的动态变化及其对疗效的预测价值,尤其是基于临床数据的系统性研究仍显不足。鉴于此,本研究探讨UC病灶中STAT及SOCS的表达,分析其与疾病严重程度及治疗效果的关联,为理解UC的发病机制提供新视角,并为临床治疗策略的优化提供潜在的分子靶点和生物标志物。

1 资料与方法

1.1 一般资料 本研究采用病例对照研究方法,选取2022年3月至2024年6月沧州市人民医院确诊的70例UC患者作为UC组,既往因外伤、手术等原因行病理学检查证实为正常肠黏膜组织的70例受试者作为对照组。本研究通过沧州市人民医院伦理委员会审批[K2022-批件-071(12.16)]。两组患者UC家族史差异有统计学意义(P<0.05),其他基础资料差异无统计学意义(P>0.05),具有可比性。见表1。诊断标准:UC诊断参考《炎症性肠病诊断与治疗的共识意见(2018年·北京)》标准[9],电子肠镜取活组织实施病理学检查确诊。纳入标准:(1)符合UC诊断标准;(2)年龄18~65岁;(3)病历资料齐全,依从性良好;(4)内镜检查与血液检查间隔≤48 h。排除标准:(1)合并恶性肿瘤及血液系统疾病;(2)合并其他感染性疾病;(3)合并严重心、肺功能不全和血栓性、自身免疫性疾病;(4)近3个月使用过影响凝血功能的药物;(5)入院前3个月内曾使用激素类药物;(6)妊娠期或哺乳期妇女。

1.2 肠镜检查及SOCS、STAT mRNA检测 肠镜检查:所有受试者均接受结肠镜检查,由从事内镜检查5年以上的医师进行。详细记录肠道黏膜病变状况。指标检测:取病理科存档的两组受试者肠道黏膜的组织样本进行检查。采用Invitrogen公司的Trizol RNA抽提试剂提取组织中的总RNA。随后,利用Promega公司的cDNA合成试剂盒,将提取到的总RNA样本进行反转录,生成cDNA。使用Promega公司的PCR反应试剂盒对cDNA样本进行扩增,PCR反

表1 两组研究对象基础资料比较 (n=70,例)

Tab.1 Comparison of basic data between two groups of research subjects (n=70,case)

组别	年龄(岁,$\bar{x}\pm s$)	BMI(kg/m^2,$\bar{x}\pm s$)	性别		吸烟	饮酒	肠息肉家族史	结直肠肿瘤家族史	UC家族史
			男	女					
UC组	52.6±9.4	23.0±2.0	61	9	45	40	21	4	14
对照组	50.7±8.8	23.4±2.0	53	17	38	32	15	1	3
t/χ^2值	1.235	0.929	3.023		1.450	1.830	1.346	0.831	8.101
P值	0.219	0.355	0.082		0.229	0.176	0.246	0.362	0.004

应在 ABI PRISM 7500 荧光定量 PCR 仪上进行，采用 Promega 公司的 GoTaq® qPCR Master Mix 试剂盒（含 BRYT Green®染料，货号：A6001）分别针对 *SOCS*1~7、*STAT*1、*STAT*3、*STAT*5 等目标基因进行扩增。反应条件为 95 ℃预变性 10 min，然后 95 ℃变性 15 s，60 ℃退火及延伸 1 min，共进行 40 个循环。最后，根据实时荧光定量 PCR 的结果，计算各组样本中 SOCS1~7、STAT1、STAT3、STAT5 mRNA 相对表达量。

1.3　*治疗方法*　所有 UC 患者入院后均积极给予治疗。活动期：给予美沙拉秦肠溶片（德国 Losan PharmaGmbH，规格：0.5 g/片，国药准字 HJ20171358）联合泼尼松（津药药业股份有限公司，规格：5 mg/片，国药准字 H20237053）。具体用药方案为：美沙拉秦肠溶片 1 g/次，每日 4 次口服；泼尼松 30～40 mg/d，基本控制后 1~2 周开始，每周递减 5 mg，当剂量减至 20 mg/d 后，调整为每 1~2 周递减 2.5 mg，直至停用。缓解期：仅采用美沙拉秦肠溶片进行维持治疗，1 g/次，4 次/d，口服。14 d 为 1 个疗程，根据患者病情的改善情况，连续进行 1~2 个疗程的治疗。治疗满 2 个月后，患者需接受结肠镜复查，以全面评估治疗效果，确保病情得到有效控制与管理。

1.4　*病情及疗效判断标准*　UC 患者的病情程度采用 Sutherland 疾病活动指数（DAI）标准[10]进行判断，主要包括客观测量指标和主观评价指标，即患者的临床症状、内镜检查、实验室检查、病灶组织切片分级，其中 DAI 0~1 分为缓解期，2~9 分为活动期。

活动期 UC 患者临床疗效评估参照《中国溃疡性结肠炎外科治疗指南》[11]标准，依据治疗后内镜检查结果进行评价，治疗后腹泻、黏液血便次数减少，腹痛症状缓解或消失，内镜检查显示溃疡黏膜愈合超过 50%或完全愈合的患者纳入疗效良好组；治疗后腹泻、黏液血便次数和腹痛症状未明显改善，对生活造成严重影响，内镜检查溃疡面愈合不足 50%的患者纳入疗效不佳组。

1.5　*统计学方法*　使用 SPSS 21.0 软件分析数据。计量资料符合正态分布，采用 $\bar{x}\pm s$ 描述，组间比较采用独立样本 t 检验；计数资料用例（%）描述，采用 χ^2 检验。P<0.05 为差异有统计学意义。

2　结　果

2.1　*两组肠黏膜 SOCS mRNA 相对表达量比较*　UC 组 SOCS2、SOCS3 mRNA 相对表达量均低于对照组（P<0.05），两组 SOCS1、SOCS4、SOCS5、SOCS6、SOCS7 mRNA 相对表达量比较无差异（P>0.05）。见表 2。

2.2　*两组肠黏膜 STAT mRNA 相对表达量比较*　UC 组肠黏膜 STAT1、STAT3、STAT5 mRNA 相对表达量均高于对照组（P<0.05）。见表 3。

2.3　*不同病期 UC 组患者肠黏膜 STAT mRNA 相对表达量比较*　70 例 UC 患者中，缓解期患者 15 例，活动期患者 55 例。活动期 UC 患者的 STAT5 mRNA 相对表达量高于缓解期患者（P<0.05），活动期与缓解期患者的 STAT1、STAT3 mRNA 相对表达量比较无差异（P>0.05）。见表 4。

2.4　*不同病期 UC 组患者肠黏膜 SOCS mRNA 相对表达量比较*　活动期 UC 患者 SOCS2、SOCS3 水平均低于缓解期患者（P<0.05），活动期与缓解期患者的 SOCS1、SOCS4、SOCS5、SOCS6、SOCS7 mRNA 相对表达量比较无差异（P>0.05）。见表 5。

表 2　两组肠黏膜 SOCS mRNA 相对表达量比较　（n=70，$\bar{x}\pm s$）

Tab.2　Comparison of the relative expression of SOCS mRNA in intestinal mucosa between two groups　（n=70，$\bar{x}\pm s$）

组别	SOCS1	SOCS2	SOCS3	SOCS4	SOCS5	SOCS6	SOCS7
UC 组	1.09±0.21	0.39±0.08	0.33±0.10	0.98±0.10	1.03±0.15	1.06±0.18	0.97±0.17
对照组	1.06±0.18	1.00±0.18	1.00±0.13	1.00±0.14	1.00±0.14	1.02±0.15	1.01±0.14
t 值	0.907	25.910	34.178	0.973	1.223	1.428	1.520
P 值	0.366	<0.001	<0.001	0.332	0.223	0.155	0.131

表 3　两组肠黏膜 STAT mRNA 相对表达量比较（n=70，$\bar{x}\pm s$）

Tab.3　Comparison of the relative expression of STAT mRNA in intestinal mucosa between two groups　（n=70，$\bar{x}\pm s$）

组别	STAT1	STAT3	STAT5
UC 组	1.93±0.45	2.30±0.57	1.76±0.32
对照组	1.22±0.39	1.44±0.39	1.09±0.27
t 值	9.976	10.418	13.389
P 值	<0.001	<0.001	<0.001

表 4　不同病期 UC 组患者肠黏膜 STAT mRNA 相对表达量比较　（$\bar{x}\pm s$）

Tab.4　Comparison of the relative expression of STAT mRNA in intestinal mucosa in UC patients at different disease stages　（$\bar{x}\pm s$）

期别	例数	STAT1	STAT3	STAT5
缓解期	15	1.77±0.39	2.10±0.50	1.62±0.29
活动期	55	1.98±0.42	2.36±0.54	1.80±0.30
t 值		1.741	1.678	2.074
P 值		0.086	0.098	0.042

表5 不同病期UC组患者肠黏膜SOCS mRNA相对表达量比较 （$\bar{x}\pm s$）

Tab.5 Comparison of relative expression of SOCS mRNA in UC patients at different disease stages （$\bar{x}\pm s$）

期别	例数	SOCS1	SOCS2	SOCS3	SOCS4	SOCS5	SOCS6	SOCS7
缓解期	15	1.10±0.18	0.51±0.08	0.54±0.09	1.00±0.08	1.01±0.12	1.07±0.16	1.00±0.15
活动期	55	1.09±0.20	0.36±0.06	0.27±0.08	0.97±0.09	1.04±0.14	1.06±0.14	0.96±0.12
t值		0.175	7.968	11.282	1.170	0.757	0.238	1.083
P值		0.862	<0.001	<0.001	0.246	0.452	0.813	0.282

2.5 不同治疗结局的活动期UC组患者肠黏膜STAT mRNA相对表达量比较 55例活动期UC患者接受美沙拉秦治疗，疗效良好者38例，效果不佳者17例。效果不佳者的STAT1、STAT3、STAT5 mRNA相对表达量均高于疗效良好（P<0.05）。见表6。

2.6 不同治疗结局的活动期UC组患者肠黏膜SOCS mRNA相对表达量比较 疗效不佳的活动期UC患者的SOCS2、SOCS3 mRNA相对表达量均低于疗效良好者（P<0.05），活动期疗效不佳与疗效良好患者的SOCS1、SOCS4、SOCS5、SOCS6、SOCS7 mRNA相对表达量比较无差异（P>0.05）。见表7。

表6 不同治疗结局的活动期UC组患者肠黏膜STAT mRNA相对表达量比较 （$\bar{x}\pm s$）

Tab.6 Comparison of the relative expression of STAT mRNA in intestinal mucosa in active UC patients with different treatment outcomes （$\bar{x}\pm s$）

不同结局	例数	STAT1	STAT3	STAT5
疗效良好	38	1.81±0.35	2.21±0.47	1.68±0.26
效果不佳	17	2.36±0.40	2.70±0.52	2.07±0.28
t值		5.153	3.458	5.021
P值		<0.001	<0.001	<0.001

表7 不同治疗结局的活动期UC组患者肠黏膜SOCS mRNA相对表达量比较 （$\bar{x}\pm s$）

Tab.7 Comparison of the relative expression of SOCS mRNA in the intestinal mucosa between active UC patients with different treatment outcomes （$\bar{x}\pm s$）

不同结局	例数	SOCS1	SOCS2	SOCS3	SOCS4	SOCS5	SOCS6	SOCS7
疗效良好	38	1.08±0.17	0.38±0.08	0.30±0.07	0.98±0.07	1.03±0.13	1.05±0.15	0.97±0.14
效果不佳	17	1.11±0.18	0.32±0.07	0.20±0.06	0.95±0.09	1.06±0.12	1.08±0.12	0.94±0.11
t值		0.594	2.666	5.105	0.342	0.809	0.726	0.781
P值		0.555	0.010	<0.001	0.185	0.422	0.471	0.438

3 讨 论

UC是一种病因复杂的慢性肠道炎症，发病涉及遗传、环境及免疫因素，近年来，STAT家族与SOCS在UC病理中的重要性逐渐显现，在UC病灶中的表达模式如何影响病情及治疗，研究尚不充分[12-13]。

STAT家族蛋白的过度活化可诱发炎症因子过量生成与放大效应，加速肠道组织损伤与炎症反应进程[14]。本研究结果显示，UC患者黏膜组织中STAT1、STAT3、STAT5 mRNA相对表达量高于对照组，提示上述因子在UC发病中发挥了重要作用。分析原因可能为肠道微生态失衡、黏膜屏障功能受损等因素可能诱导UC患者免疫系统对肠道自身抗原的过度应答，促使细胞因子大量释放，激活STAT信号通路，增强炎症相关基因的转录与表达[15-16]。

本研究还显示，UC组中SOCS2、SOCS3 mRNA相对表达量低于对照组，提示SOCS2与SOCS3在UC病理生理过程中的作用。有研究称，SOCS家族通过抑制STAT的活性，调控细胞因子信号传导，以维护免疫稳态并防止炎症反应的过度激活[17]。在UC病理背景下，SOCS2、SOCS3 mRNA的低表达会削弱其对STAT通路的抑制作用，导致STAT1、STAT3、STAT5等分子的异常激活，引发下游炎症因子的过度生成，与UC复杂的免疫病理过程紧密相连，共同促成免疫系统的异常反应，致炎症反应加剧。有研究认为，在UC患者中，体内炎症相关因子水平呈现升高趋势[18]，本研究结果与其一致，进一步证实UC患者体内持续的炎症反应状态。

对UC患者分层分析显示，UC组肠黏膜的STAT1、STAT3、STAT5 mRNA相对表达量高于对照组，SOCS2、SOCS3 mRNA相对表达量低于对照组，反映上述指标与UC疾病活动状态的紧密关联性。分析原因可能为活动期UC患者体内STAT1、STAT3、STAT5 mRNA高表达，炎症信号通路的持续激活状态，促使下游炎症因子过度生成，加剧肠道组织的炎症反应与损伤[19]。活动期UC患者SOCS2、SOCS3 mRNA的低表达提示免疫调节机制的深度失衡。SOCS家族的正常功能可通过抑制STAT的激活来限

制炎症反应的强度和持续时间。在活动期UC中，SOCS2、SOCS3 mRNA的表达下调显著削弱其抑制作用，导致STAT通路异常激活，使得炎症反应失控。在以往研究的基础上，本研究发现STAT、SOCS作为疾病活动度评估生物标志物的潜力。通过密切监测这些指标的变化，临床医生能够更精准地把握UC患者的病情动态，为制定个性化、高效的治疗策略提供科学依据。

美沙拉秦的疗效主要基于抑制肠道炎症、减少炎症介质释放及促进肠道黏膜修复。研究提出，患者体内炎症信号通路的不同激活状态是导致治疗反应个体差异的关键因素[20]。本研究效果不佳者STAT1、STAT3、STAT5 mRNA相对表达量高于疗效良好者，SOCS2、SOCS3 mRNA相对表达量低于后者，反映美沙拉秦治疗UC效果与上述分子标志物之间的密切关系。分析原因可能为效果不佳者中STAT1、STAT3、STAT5 mRNA的高表达反映其体内炎症信号通路持续激活，炎症因子过度生成，可能超出美沙拉秦的常规抑制能力或需更高剂量方能奏效。另SOCS2和SOCS3作为STAT通路的负性调节器[21]，低表达在效果不佳患者中尤为显著，削弱对STAT激活的抑制作用，加剧炎症反应。因此，在制定UC治疗方案时，除考虑药物本身特性外，还需关注患者体内炎症信号通路的状态及负反馈调节机制的功能，以实现治疗的精准化与高效化。

本研究在实验设计上展现出不同于以往研究的独特之处，对UC的疾病活动状态（活动期与缓解期）进行分层分析，分析STAT与SOCS mRNA相对表达量与疾病严重程度之间的动态联系，通过考察美沙拉秦治疗效果与这些分子表达变化的关联性，展现其作为预测治疗反应生物标志物的潜力，为UC的精准医疗策略开辟了新的视角。

综上所述，UC病灶组织中STAT1、STAT3、STAT5 mRNA表达高于健康人群、SOCS2、SOCS3 mRNA表达低于健康人群，并且与治疗效果有关。但本研究也存在一定不足，受限于资金、地域等限制，导致入组样本量有限，来源地域单一，后续将增加样本量、扩大地域来源，进行大样本、多中心探究。

利益冲突　作者均声明无利益冲突

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