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Association between Dietary Inflammatory Index and Ulcerative Colitis: a case–control study

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Abstract

Introduction Diet plays a crucial role in the activity and onset of ulcerative colitis (UC). The aim of this study was to comprehensively explore the association between the dietary inflammatory index (DII) and UC.

Methods Participants completed the Food Frequency Questionnaire to obtain data on their dietary intake. Individual DII scores were calculated to assess inflammatory potential of each participant's diet. A logistic regression model was used to analyze the correlation between the DII and UC activity, including the active and remission phases.

Results In this study, 100 controls and 106 patients with UC were enrolled, including 50 patients in remission and 56 patients with active UC. Dietary nutrient intake was generally slightly lower in patients with UC than in the controls, including energy, protein, dietary fiber, vitamin D, vitamin E, vitamin B1, vitamin B2, vitamin C, folic acid, fat, monosaturated fatty acids, and n-3 fatty acids ($P < 0.05$). Compared with the low pro-inflammatory potential diet, patients with higher DII had a higher correlation with UC before and after adjustment for relevant confounders. In consecutive DII, the correlation with UC increased with each 1 increase in DII. No significant correlation was observed between DII and UC activity.

Conclusions Diets with a high inflammatory index are correlated with UC. Therefore, consuming a diet with a low inflammatory index may be beneficial for patients with UC.

Keywords Dietary inflammatory index, Ulcerative colitis, Pro-inflammatory diet, Anti-inflammatory diet, Diet

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Introduction

Ulcerative colitis (UC) is one of the conditions that make up the group of diseases known as inflammatory bowel disease (IBD) [1]. UC is a chronic inflammatory disease of the colonic and rectal mucosa characterized by alternating episodes of flare-ups and remissions [2, 3]. Genetic, immune, and environmental factors have been implicated in the pathogenesis of UC [4]. In recent times, there has been a rise in the prevalence of IBD, which, in part, has been attributed to dietary changes. Western diets with higher protein and saturated fats but lacking fruits, vegetables, and fiber have been shown to play a role in the development of UC [5]. Also the modern Western diet containing so much sugar, sweeteners, trans fats and ultra-processed foods has pro-inflammatory properties that increase the risk of chronic, inflammatory diseases [6]. Studies have shown that intake of a diet high in omega- 3 polyunsaturated fatty acids may have a protective effect against UC [7]. However, a high intake of sucrose, animal fat, cholesterol, and linoleic acid has been associated with an increased risk of UC [8]. In two cross-sectional studies on people living with IBD, approximately 70% of individuals thought that diet could affect their condition, 60% thought that diet was a significant factor in relapse, and 16% believed that diet could influence the onset of the disease [9, 10]. The development of UC is significantly influenced by persistent inflammation, and the inflammatory potential of the diet may be able to influence disease progression [11]. Diet is a complex process, and single foods or nutrients do not respond well to the impact of diet on health. Studying dietary patterns provides a more accurate assessment of the impact of diet on health, and evaluating the overall inflammatory index of foods for inflammatory diseases may be more meaningful than analyzing specific foods or nutrients in isolation. The dietary inflammatory index (DII) was first proposed by Cavicchia in 2009 to clarify the effects of pro-inflammatory diets on human health [12]. It was further refined and designed by Shivappa in 2014 [13]. The DII aims to investigate the association between inflammation, diet, and disease by evaluating the potential of food to cause inflammation. Since its development, studies have assessed the relationship between DII and IBD. An Iranian study showed that UC was associated with increased DII [14]. Lamers demonstrated that the relationship between DII and disease activity is not significant in UC; however, it is strongly associated with Crohn's disease [15]. Further studies are required to elucidate the relationship between DII and UC.

In this study, we aimed to retrospectively observe the association between DII and UC in adults in Xinjiang. The understanding of this is beneficial for clinical dietary recommendations for UC prevention and disease modification.

Materials and methods

Study populations

In total, 206 participants were recruited from the First Affiliated Hospital of Xinjiang Medical University in Urumqi between September 2022 and October 2023. Among these, 106 patients were diagnosed with UC either in the remission (50 cases) or active (56 cases) phases based on the Mayo scores [15]. The control group consisted of 100 volunteers who underwent physical examinations at the First Affiliated Hospital of Xinjiang Medical University during the same period. The inclusion criteria for the case group were as follows: ① Patients attending the First Affiliated Hospital of Xinjiang Medical University who had not used antibiotics or probiotics in the past 6 months and had a clinical diagnosis of UC. Inclusion criteria for the control group were as follows: ① undergoing routine physical examination; ② plausible daily energy intake (male: 800–4000 kcal; female: 500–3500 kcal) [16]; ③ no dietary restrictions, not pregnant, lactating, or suffering from certain chronic diseases (diabetes mellitus, hypertension, or hypothyroidism); ④ no acute or chronic intestinal diseases (e.g. enteritis, functional bowel disease, constipation, intestinal polyps, or malignant tumors); ⑤ no long-term use of aspirin, calcium channel blockers, or other drugs.

Participant characteristics

Baseline demographic information, including sex, age (in years), height (in meters), weight (in kilograms), body mass index (BMI; in kg/m^2) and smoking status, was collected via an interviewer-administered questionnaire. Medication use was collected from the case group. Combined with the number of bowel movements, blood in the stool, endoscopy, and the physician's overall assessment,the Mayo score was used to evaluate the disease activity categorize the cases into those in remission (Scores ≤ 2 and no individual score > 1) and active (scores ≥ 3) phases [16].

Food Frequency Questionnaire (FFQ)

We used a regionally sensitive FFQ with acceptable reliability and validity among populations in Northwest China; it was designed by the gastroenterology team at the West China Hospital of Sichuan University. Details of the FFQ can be found in the supplementary document. The FFQ covers 6 months of dietary information. The FFQ was administered to all participants for the evaluation of their dietary intake for energy, macronutrients, and micronutrients. The FFQ consisted of 57 food items categorized into eight groups: staple foods, soybeans, fruits, vegetables, meat, other proteins, beverages, and ultra-processed foods. The three frequency options were "daily," "weekly," and "several times a month." Dietary

mapping and food models were provided to ensure that the food intake measurements were accurate. Two trained investigators collected and analyzed the data according to strict quality control standards, and incomplete or missing information was checked and corrected. Food and nutrient intake was calculated using the 2009 Chinese Food Composition Table [17] and the CDC-authorized Nutrition Calculator (version 2.8.0).

Dietary Inflammatory Index (DII) Calculation

The DII was calculated based on individual dietary inflammation data and average intake worldwide using the following formula:

$$Z-score = \frac{average\ daily\ intake\ of\ a\ nutrient\ in\ the\ study\ population - world\ wide\ per\ capita\ daily\ intake\ of\ this\ nutrient}{standard\ deviation\ of\ global\ per\ capita\ daily\ intake\ of\ this\ nutrient}$$

The value was changed to a percentile score in order to reduce the impact of"right skewness." Each percentile score was doubled, and then subtracted by one to create a symmetrical distribution. The results ranged between +1 (maximal pro-inflammatory effect) and − 1 (maximal anti-inflammatory effect). The individual DII score for each nutrient was calculated by multiplying transformed z-score by the total inflammatory effect score. The results for each nutrient were summed to create an individual DII.A positive score on the DII indicates that dietary components have pro-inflammatory potential, a negative score indicates anti-inflammatory potential, and zero indicates no inflammatory effect. We obtained 26 dietary components, including energy, protein, fat, carbohydrates, dietary fiber, cholesterol, vitamin A, vitamin D, vitamin E, vitamin B1, vitamin B2, vitamin B6, vitamin B12, vitamin C, folate, niacin, magnesium, iron, zinc, selenium, saturated fatty acids, monounsaturated fatty acids, polyunsaturated fatty acids, omega- 3 fatty acids, omega- 6 fatty acids, and black or green tea. Checked various dietary components and nutrient related parameters. The inflammatory effect scores we used ranged from—0.663 for fiber to +0.373 for saturated fat. The contribution of the food parameters to the total DII score depended on the individual intake and the deviation of the inflammatory effect score from 0.The mean DII score was calculated separately for patients with UC and the healthy controls without UC. The populations were categorized into three groups according to the DII tertile: the T1 group (DII ≤1.214), the T2 group (DII =1.214–1.630), and the T3 group (DII ≥1.630). This study was approved by the Medical Ethics Committee of First Affiliated Hospital of Xinjiang Medical University. (approval number: K202303 -20), and all participants provided informed consent.

Statistical analysis

Continuous variables were tested for normal distribution using the Kolmogorov–Smirnov test. Continuous variables that conformed to normal distribution were expressed as mean ± standard deviation (X ± SD). Non-normally distributed continuous variables were expressed as medians and 25 th/75 th percentiles (M [P25, P75]). Independent samples t-test and one-way analysis of variance (or Kruskall–Wallis test, when not normally distributed) were performed for continuous variables. Categorical data as frequencies with proportions were compared between groups using the chi-square test.In the analysis of nutrients,a more conservative threshold of statistical significance was adopted, and the adjusted *p*-values was calculated using the Benjamini Hochberg method. Logistic regression modeling was used to determine the relationship between the inflammatory potential of diet and disease using the DII as a categorical and continuous variable. The lowest tertile (T1) was used as a reference. Unadjusted and adjusted models for relevant covariate factors (total energy intake and BMI) were used to determine the odds ratio (OR) and 95% confidence interval (CI) for the impact of dietary intake. The relationship between DII and disease activity was assessed by comparing the DII in the remission phase with that in the active phase. Relevant covariate factors (e.g. total energy

Table 1 Baseline characteristics and DII in UC patients and healthy control cases

[a]Variable	Cases (*n* = 106)	Controls (*n* = 100)	**P-value
Sex			0.981
Male	50 (47.2%)	47 (47.0%)	
Female	56 (52.8%)	53 (53.0%)	
Age (years)	46.58 ± 11.32	46.99 ± 12.37	0.789
Height (m)	1.67 ± 0.08	1.67 ± 0.09	0.719
Weight (kg)	60 (55,70)	64 (56,75)	0.059
BMI (kg/m2)	22.36 ± 3.52	23.39 ± 3.30	0.031
Smoking			0.376
Yes	10 (10%)	7 (6.6%)	
No	90 (90%)	99 (93.4%)	
Energy (Kcal)	1697 (1451,2052)	1844 (1693,2126)	0.007
DII	2.147 (1.569,2.419)	1.455 (1.016,1.702)	< 0.001

BMI Body Mass Index, *DII* Dietary inflammatory index

[a] Frequency count and (%) OR Mean ± SD and [M (P25, P75)]

[**] Independent samples t-test or chi-square test or Kruskall-Wallis test,significant at $p < 0.05$

Table 2 Baseline characteristics and DII in remission and active group of cases

[c]Variable	Remission (*n* = 50)	Active (*n* = 56)	**P-value
Sex			0.820
Male	23 (46.0%)	27 (48.2%)	
Female	27 (54.0%)	29 (51.8%)	
Age (years)	50 (40,54)	47 (37,56)	0.651
Height(m)	1.67 ± 0.08	1.67 ± 0.08	0.767
Weight(kg)	61 (55,71)	60 (54,70)	0.340
BMI (kg/m2)	22.73 ± 3.64	22.03 ± 3.41	0.310
Smoking			0.251
Yes	5 (10.0%)	2 (3.6%)	
No	45 (90.0%)	54 (96.0%)	
Medication use			0.036
No medication use	0 (0.0%)	3 (5.4%)	
Single drugs[a]	48 (96.0%)	45 (80.0%)	
Multiple drugs[b]	2 (4.0%)	8 (14.0%)	
Energy (Kcal)	1862 (1590,2342)	1569 (1306,1852)	0.001
DII	1.996 (1.447,2.310)	2.274 (1.664,2.508)	0.314

BMI Body Mass Index, *DII* Dietary inflammatory index

** Independent samples t-test or chi-square test or Kruskall-Wallis test, significant at $p < 0.05$

[a] Consumption of aminosalicylic acid compounds (5-ASA) or biological preparation, each per

[b] Combination at least two classes of drugs, including ASA,biological preparation, and hormones

[c] Frequency count and (%) OR Mean ± SD and [M (P25, P75)]

intake and use of medication) were adjusted before assessing the adjusted relationship between DII and disease activity. Statistical analysis was performed using SPSS version 26.0 and R language.

Results

Descriptive data

A total of 206 participants were included in this study, including 100 controls and 106 patients with UC. Of these cases, 50 were in remission and 56 were in the active stage. There were no statistically significant differences between the cases and the control groups in terms of sex, age, or height. Compared to healthy controls, patients with UC seemed to have lower BMI and calorie consumption but higher DII ($P < 0.05$) (Table 1). The remission group consumed slightly more energy but had lower DII than that of those with active UC (Table 2).

Dietary nutrient intake

Compared with the control group, the dietary nutrient intake of the cases was generally slightly lower in energy, protein, dietary fiber, vitamin D, vitamin E, vitamin B1, vitamin B2, vitamin C, folic acid, fat, monosaturated fatty acids, and omega- 3 fatty acids. Additionally, the case group had a higher intake of cholesterol, saturated fatty acids, and omega- 6 fatty acids ($P < 0.05$). There were no statistically significant differences in the intake of polyunsaturated fatty acids, carbohydrates, vitamin A, or tea (Table 3).

The relationship between DII and UC

In the unadjusted model, when the analysis was carried out with DII expressed as tertiles,a high inflammatory index diet (T3) was associated with UC 4.50 times (OR = 5.50, 95% CI = 2.61–11.60)more than low inflammatory index diet (T1). After adjusting for BMI and energy intake, a high-inflammatory index diet was associated with 3.92 times increase in the rate of UC (OR = 4.92,95% CI = 2.27–10.66). Using DII as a continuous variable, each 1-unit increase in DII was associated with a 3.64 times increase in the association with UC (OR = 4.64,95% CI = 2.74–7.84), and this association remained significant in the adjusted model (Table 4).

The relationship between DII and UC Activity

In patients with UC in the remission and active stages, there was no significant association between DII and UC activity in the unadjusted and adjusted models (OR = 1.77, 95% CI = 0.98–3.21; OR = 1.50, 95% CI = 0.78–2.89, respectively) (Table 5).

Discussion

Currently, most patients are treated for ulcerative colitis with medication, while a few may undergo surgery if their condition requires it [18, 19]. Although medication can temporarily relieve disease activity, most patients believe that medication is insufficient to control the disease or maintain the disease in remission for a protracted duration. In addition, chronic intestinal inflammation affects multiple systems, causing a wide range of symptoms and complications. Treatment with drugs over the long term can be problematic, with side effects and progressive effectiveness loss [20]. Moreover, the onset and therapeutic effect of UC treatment are greatly influenced by the environment, of which diet is an important factor. Exploring the influence of diet between the pathogenesis and treatment of UC makes more sense for long-term disease management, including reducing incidence, alleviating symptoms, improving response to conventional treatments, avoiding medication side effects, and decreasing disease burden. Additionally, dietary guidance may be beneficial to improve their overall quality of life.

Table 3 Comparison of nutrient intake between UC patients and healthy controls [*M(P25, P75)*]

*Nutrients	Cases	Controls	**P-value	***Adjustment of P-value
Energy (Kcal)	1697.0 (1451.0,2052.0)	1844.0 (1693.0,2126.0)	0.007	0.010
Protein (g)	63.6 (52.7,76.5)	76.3 (68.6,86.9)	< 0.001	0.002
Total fat (g)	55.4 (46.3,68.3)	60.2 (54.9,66.4)	0.023	0.028
SFA (g)[a]	27.6 (19.6,36.7)	18.3 (16.4,21.3)	< 0.001	0.002
MUFA (g)[a]	11.4 (9.2,13.9)	24.4 (20.3,27.0)	< 0.001	0.002
PUFA (g)[a]	16.2 (12.4,22.1)	17.7 (14.4,20.5)	0.134	0.145
n- 3 Fatty acids (g)	6.7 (4.9,12.1)	9.4 (7.9,12.3)	< 0.001	0.002
n- 6 Fatty acids (g)	8.4 (6.9,10.9)	7.9 (6.3,9.1)	0.030	0.035
Carbohydrates (g)	239.5 (195.3,309.4)	257.9 (217.9,295.2)	0.082	0.093
Fibre (g)	8.1 (6.5,10.3)	10.8 (9.5,12.4)	< 0.001	0.002
Cholesterol(mg)	434.5 (361.0,495.0)	257.9 (217.9,295.2)	< 0.001	0.002
Vitamin A(μg)	288.0 (216.0,472.0)	301.0 (259.0,332.0)	0.311	0.323
Vitamin D(μg)	1.4 (1.1,1.9)	1.7 (1.4,1.9)	0.002	0.003
Vitamin E(mg)	9.9 (7.9,13.4)	11.5 (9.7,14.3)	0.004	0.006
Thiamin(mg)	0.8 (0.7,1.1)	1.0 (0.8,1.2)	0.005	0.007
Riboflavin(mg)	0.9 (0.7,1.0)	1.1 (1.0,1.2)	< 0.001	0.002
Vitamin B6 (mg)	0.1 (0.1,0.1)	0.1 (0.1,0.2)	< 0.001	0.002
Vitamin B12(μg)	0.0 (0.0,0.0)	0.0 (0.0,0.2)	< 0.001	0.002
Vitamin C (mg)	68.7 (52.8,85.7)	88.4 (76.7,98.7)	< 0.001	0.002
Folic acid(μg)	40.9 (26.8,61.2)	59.2 (49.1,76.3)	< 0.001	0.002
Niacin(mg)	13.2 (10.4,16.5)	16.3 (15.0,17.9)	< 0.001	0.002
Magnesium(mg)	238.0 (195.0,291.0)	283.0 (250.0,311.0)	< 0.001	0.002
Iron(mg)	17.6 (14.1,20.8)	18.7 (16.5,21.7)	0.011	0.014
Zinc(mg)	9.2 (7.3,11.1)	10.7 (9.7,11.6)	< 0.001	0.002
Selenium(μg)	41.6 (33.0,49.6)	45.7 (40.3,55.7)	0.001	0.002
Tea(g)	0.0 (0.0,2.0)	0.0 (0.0,2.0)	0.344	0.344

SFA, Saturated fatty acid, *MUFA* Monounsaturated fatty acid, *PUFA* Polyunsaturated fatty acid

[a] [M (P25, P75)]

** Independent samples t-test, significant at $p < 0.05$

*** Adjusted *P*-values using the Benjamini Hochberg method

Table 4 Results of logistic regression analysis of DII and UC ($n = 206$)

DII	Cases/Controls	Unadjusted			Adjusted[a]		
		OR	95%*CI*	P	OR	95%*CI*	P
T1(≤ 1.214)	14/33	1.00	-	-	1.00	-	-
T2(1.214 ~ 1.630)	15/34	1.40	[0.44,2.47]	0.930	0.93	[0.38,2.27]	0.871
T3(≥ 1.630)	77/33	5.50	[2.61,11.60]	< 0.001	4.92	[2.27,10.66]	< 0.001
DII[b]	106/100	4.64	[2.74,7.84]	< 0.001	4.62	[2.68,7.96]	< 0.001

DII Dietary inflammatory index

[a] Adjusted for BMI and total energy intake

[b] DII as a continuous variable

Our research results showed that compared with the low pro-inflammatory potential diet, patients with higher DII had 3.92 times (OR =4.92, 95% CI =2.27–10.66) increased association with UC after adjusting for relevant confounders. In continuous DII, the correlation with UC increased 3.62 times for each 1 point increase in DII (OR =4.62,95% CI =2.68–7.96). Consistent with our findings, Shivappa et.al revealed a correlation between DII and UC [13]. In this study, after multivariate-adjusted, with the DII being used as both a categorical variable(OR

Table 5 Results of logistic regression analysis of DII and UC activity (*n*=106)

	Remission/Active	Unadjusted			Adjusted[a]		
		OR	95%CI	*P*	OR	95%CI	*P*
DII[b]	50/56	1.77	[0.98,3.21]	0.061	1.50	[0.78,2.89]	0.226

[a] Adjusted for total energy intake and medication use

[b] DII as a continuous variable

DII Dietary inflammatory index

=2.58; 95% CI =1.03–6.48) (T3 vs T1) and continuous variable(OR =1.55; 95% CI =1.04—2.23).Xinjiang province has populations from diverse multiethnic backgrounds that are dominated by high-calorie, high-fat, meat (beef and lamb) and dairy products [21], which are more likely to be associated with pro-inflammatory diets. This may explain the higher prevalence of ulcerative colitis in the Xinjiang population. Regarding the correlation of DII and UC activity, there was no significant difference between participants in remission and with mild and moderately active disease [14]. Similar to these studies, our findings suggest a correlation between DII and UC, not with disease activity. There may be overall differences in the confounding variables of the study design and that the sample size of patients in remission and in active disease was not large enough, which may limit comparisons and extrapolation of results. There is also the fact that dietary habits are a long-established process, and it is more difficult to change the dietary patterns of patients who have developed the disease. They need long-term, proper dietary guidance. So whether diet-related inflammation affects disease activity in IBD patients deserves further study.

Diet affects intestinal inflammation through different mechanisms, including altering the composition of the intestinal flora and even interactions with the local immune system [22, 23]. Cytokines include pro-inflammatory and anti-inflammatory factors, which are involved in the inflammatory response throughout the development of chronic inflammation in patients with IBD [24]. A pro-inflammatory diet can contribute to the pathogenesis of UC by raising serum levels of inflammatory cytokines. The gut microbiota has been linked to several inflammatory and immunological disorders, such as asthma, rheumatoid arthritis, eczema, colorectal cancer, and irritable bowel syndrome [25]. The gut microbiota can affect intestinal immunity by defending the body against infections, preserving the integrity of the gastrointestinal barrier, and encouraging the growth and maintenance of the mucosal immune system [26]. The microbiota in inflammatory bowel disease has an abnormal biome structure and reduced diversity. The composition of the microbiota can be influenced by many factors, including age, genetics, host environment, and diet [27]. Microorganisms are dependent on the dietary substrate in the gut, and the gut microbiota is often recognized as a mediator of the pro-inflammatory and anti-inflammatory effects of food. Pro-inflammatory diets may affect UC by influencing the composition of the gut flora [28].

Animal models used in in vivo studies have shown that diets high in saturated fat enhance chronic inflammation. It tends to raise the likelihood of IBD progression and recurrence risk, especially UC [29]. A recent prospective cohort study reported that a carnivorous dietary pattern consisting of poultry, processed meats, and red meats were connected to a high risk of UC developing [30]. Several studies have suggested that adding dietary fiber may have beneficial effects on patients with UC in remission [31]. This study also confirmed that low dietary fiber, vitamin D, unsaturated fatty acids, high cholesterol, and saturated fatty acids may increase the risk of UC. These studies provide evidence for the inflammatory effects of diet and UC.

However, diet-related research is challenging because foods include many ingredients and nutrients that are consumed in varied amounts and combinations. Diet involves complex molecular and physiologic pathways, making clear that their full health effects cannot be extrapolated from any single surrogate outcome [32]. In this way, regarding the overall diet effect for patients with UC, various diet patterns have been referred rather than isolated single nutrients, including the Groningen Anti-Inflammatory Diet (GrAID diet) [33], the Specific Carbohydrate Diet (SCD diet) [34], the Low Fermented Oligosaccharides, Disaccharides, Monosaccharides, and Polyols Diet (Low FODMAP Diet) [35] and the UC Exclusion Diet (UCED Diet) [36]. However, most of these above dietary patterns were based on the combined effects of different food groups, rather than the association of foods with inflammatory markers. As additional and alternative methods to provide stronger evidence for the association between dietary patterns and chronic illnesses, dietary indices and dietary pattern analysis have been suggested [37]. In order to evaluate an individual diet's propensity for inflammation, dietary inflammatory index (DII) was first created which was drawn from the literature and

validated using a variety of inflammatory markers, including C-reactive protein [13, 38], interleukin- 6 [39, 40] and homocysteine [40]. As for dietary indices, DII in contrast to particular meals or nutrients, may offer a more complete picture of food and nutrient consumption and, as such, present a viable method for forecasting the risk of illness [41]. Among the food parameters provided in this study, monounsaturated fatty acids, polyunsaturated fatty acids especially n- 3 fatty acids, dietary fiber, vitamins A, B1, B2, B6, C, D, E, niacin, folate, magnesium, zinc, selenium, and tea have anti-inflammatory effects. Total fat, saturated fatty acids, cholesterol, protein, carbohydrates, vitamin B12, iron have pro-inflammatory effects.

Inflammatory cytokines are crucial in the development of UC since they are persistently present in the bloodstream and tissues categorizing as chronic inflammation. One of the possible DII-related mechanisms of UC was attributed to the interaction of a pro-inflammatory diet with mucosal inflammation, which is characterized by increased cytokine production by dendritic cells in the lamina propria of the colon [42]. Additionally, diet could exert a significant impact on the function of the epithelial barrier, which in turn affects mucosal inflammation [43]. Diet alteration may improve intestinal mucous lesions, modify gut microbiota dysbiosis, and reduce inflammatory response. It is advised that individuals with UC limit their intake of pro-inflammatory foods and increase their intake of foods with potent anti-inflammatory properties, rich dietary fiber, vitamin D, and unsaturated fatty acids. This study provides a new way of thinking about the treatment of UC.

Limitations

The study is a retrospective case–control study. The correlation analysis between DII and UC was performed after adjusting for confounders, which had a strong reliability. However, this study still has some limitations. The FFQ questionnaire does not contain all food items, and incomplete food collection may lead to underestimation of some specific nutrients since the consumption of vitamin D, B6, and B12 was not very similar to the international standard intake. Due to the lack of information on the intake of some specific nutrients, only 26 of the 45 food parameters are suitable for DII calculations, which may exert a difference in the overall study results. In addition, recall bias cannot be avoided in dietary investigation studies [44]. However, we prepared standardized data collection guidelines, trained the data collectors, and supervised the data collection to minimize recall bias. Patients with UC may adjust their diet after realizing diet will affect following gastrointestinal symptoms, and the possibility of diet habit variance should be taken into consideration. Whereas, further validation should be conducted on bigger scales of newly diagnosed UC patients and populations to obtain more reliable conclusion. The small number of people in remission and active disease in the case group in this study may have biased the results, although it has been noted in the literature that no significant difference in mean DII between patients in remission and those with mildly and moderately active disease has been found [15]. However, there is still a need for more cases to be included in subsequent studies to further clarify the relationship between the activity of UC and DII.This is due to the objective reason of the low prevalence of the disease. The sample size we were able to collect was limited.DII was developed using the literature on the association between diet and systemic markers of inflammation rather than gastrointestinal markers.In order to study the relationship between intestinal disease and inflammation, subsequent quantitative measurements of clinical GI markers, such as calprotectin, should be performed.

Conclusion

DII served as a bridge between diet, inflammation, and UC, validating the correlation between a high-inflammatory diet and UC and providing a basis for future dietary guidance. Participants did not develop UC while in the study. Pro-inflammatory diet scores were associated with more with UC patients than controls. Patients with UC were advised to consume foods with strong anti-inflammatory effects, high dietary fiber, vitamin D, and unsaturated fatty acids, and to reduce dietary intake of more pro-inflammatory foods.

Abbreviations

UC	Ulcerative colitis
DII	Dietary inflammatory index
OR	Odds ratio
CI	Confidence interval
IBD	Inflammatory bowel disease
BMI	Body mass index
FFQ	Food frequency questionnaire
X ± SD	Mean ± Standard deviation
M [P25, P75]	Medians and 25th/75th percentiles
SFA	Saturated fatty acid
MUFA	Monounsaturated fatty acid
PUFA	Polyunsaturated fatty acid
GrAIDt	Groningen anti-inflammatory
SCD	Specific carbohydrate
Low FODMAP	Low Fermented Oligosaccharides, Disaccharides, Monosaccharides, and Polyols
UCED	UC Exclusion

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12876-025-03869-0.

Supplementary Material 1.

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Authors' contributions
HP: Conceptualization, methodology, investigation, writing—original draft preparation. LZ: writing—review and editing, data curation. PY: Supervision, visualization, writing, review, and editing. MC: Validation. LL: Project administration; YL: Investigation; LS: Investigation.

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Data availability
The data analyzed in the study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
The study was approved by the Medical Ethics Committee of Xinjiang Medical University's First Affiliated Hospital (approval number: K202303-20). All methods were carried out in accordance with relevant guidelines and regulations. All the participants were provided oral informed consent. Ethics This investigation adhered to the principles outlined in the Helsinki Declaration (World Medical Association Declaration of Helsinki).

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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References

1. Chicco F, Magrì S, Cingolani A, Paduano D, Pesenti M, Zara F, et al. Multidimensional impact of Mediterranean diet on IBD patients. Inflamm Bowel Dis. 2021;27:1–9.
2. Pravda J. Can ulcerative colitis be cured? Discov Med. 2019;27:197–200.
3. Ungaro R, Mehandru S, Allen PB, Peyrin-Biroulet L, Colombel JF. Ulcerative colitis. Lancet. 2017;389:1756–70.
4. Lautenschlager SA, Barry MP, Rogler G, Biedermann L, Schreiner P, Siebenhüner AR. Swiss IBD Cohort Study Group. Lifestyle factors associated with inflammatory bowel disease: data from the Swiss IBD cohort study. BMC Gastroenterol. 2023;23(1):71.
5. Schreiner P, Martinho-Grueber M, Studerus D, Vavricka SR, Tilg H, Biedermann L, et al. Nutrition in inflammatory bowel disease. Digestion. 2020;101(Supplement 1):120–35.
6. Whelan K, Bancil AS, Lindsay JO, Chassaing B. Ultra-processed foods and food additives in gut health and disease. Nat Rev Gastroenterol Hepatol 2024;21(6):406–427. (6)
7. Radziszewska M, Smarkusz-Zarzecka J, Ostrowska L, Pogodziński D. Nutrition and Supplementation in Ulcerative Colitis. Nutrients. 2022;14(12):2469.
8. de Castro MM, Pascoal LB, Steigleder KM, Siqueira BP, Corona LP, Ayrizono MLS, et al. Role of diet and nutrition in inflammatory bowel disease. World J Exp Med. 2021;11:1–16.
9. Casanova MJ, Chaparro M, Molina B, Merino O, Batanero R, Dueñas-Sadornil C, et al. Prevalence of malnutrition and nutritional characteristics of patients with inflammatory bowel disease. J Crohns Colitis. 2017;11:1430–9.
10. Zallot C, Quilliot D, Chevaux JB, Peyrin-Biroulet C, Guéant-Rodriguez RM, Freling E, et al. Dietary beliefs and behavior among inflammatory bowel disease patients. Inflamm Bowel Dis. 2013;19:66–72.
11. Akour A, Kasabri V, Afifi FU, Bulatova N. The use of medicinal herbs in gynecological and pregnancy-related disorders by Jordanian women: a review of folkloric practice vs. evidence-based pharmacology. Pharm Biol 2016;54:1901–18.
12. Cavicchia PP, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS, et al. A new dietary inflammatory index predicts interval changes in serum high-sensitivity C-reactive protein. J Nutr. 2009;139:2365–72.
13. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hébert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr. 2014;17:1689–96.
14. Shivappa N, Hébert JR, Rashvand S, Rashidkhani B, Hekmatdoost A. Inflammatory potential of diet and risk of ulcerative colitis in a case–control study from Iran. Nutr Cancer. 2016;68:404–9.
15. Lamers CR, De Roos NM, Witteman BJM. The association between inflammatory potential of diet and disease activity: results from a cross-sectional study in patients with inflammatory bowel disease. BMC Gastroenterol. 2020;20:316.
16. Pabla BS, Schwartz DA. Assessing severity of disease in patients with ulcerative colitis. Gastroenterol Clin North Am. 2020;49:671–88.
17. Yang Y, Wang G, Pan X. Chinese food composition table Appendix. 2009;2:338–43.
18. Łodyga M, Eder P, Bartnik W, Gonciarz M, Kłopocka M, Linke K, et al. Guidelines for the management of Crohn's disease. Recommendations of the Working Group of the Polish National Consultant in Gastroenterology and the Polish Society of Gastroenterology. Prz Gastroenterol 2012;7:317–38.
19. Magro F, Gionchetti P, Eliakim R, Ardizzone S, Armuzzi A, Barreiro-de Acosta M, et al. Third European evidence-based consensus on diagnosis and management of ulcerative colitis. Part 1: definitions, diagnosis, extra-intestinal manifestations, pregnancy, cancer surveillance, surgery, and ileo-anal pouch disorders. J Crohns Colitis 2017;11:649–70.
20. Beard JA, Franco DL, Click BH. The burden of cost in inflammatory bowel disease: a medical economic perspective and the future of value-based care. Curr Gastroenterol Rep. 2020;22:6.
21. Zhai F, He Y, Wang Z, Hu Y. Status and characteristic of dietary intake of 12 minority nationalities in China. Wei Sheng Yan Jiu. 2007;36:539–41.
22. Khalili H, Chan SSM, Lochhead P, Ananthakrishnan AN, Hart AR, Chan AT. The role of diet in the aetiopathogenesis of inflammatory bowel disease. Nat Rev Gastroenterol Hepatol. 2018;15:525–35.
23. Witkowski M, Witkowski M, Gagliani N, Huber S. Recipe for IBD: can we use food to control inflammatory bowel disease? Semin Immunopathol. 2018;40:145–56.
24. Kunnumakkara AB, Sailo BL, Banik K, Harsha C, Prasad S, Gupta SC, et al. Chronic diseases, inflammation, and spices: how are they linked? J Transl Med. 2018;16:14.
25. Aron-Wisnewsky J, Warmbrunn MV, Nieuwdorp M, et al. Metabolism and metabolic disorders and the microbiome: the intestinal microbiota associated with obesity, lipid metabolism, and metabolic health—pathophysiology and therapeutic strategies. Gastroenterology. 2021;160(2):573–99.
26. Minihane AM, Vinoy S, Russell WR, Baka A, Roche HM, Tuohy KM, et al. Low-grade inflammation, diet composition and health: current research evidence and its translation. Br J Nutr. 2015;114:999–1012.
27. Castro F, de Souza HSP. Dietary composition and effects in inflammatory bowel disease. Nutrients. 2019;11:1398.
28. Khademi Z, Saneei P, Hassanzadeh-Keshteli A, Daghaghzadeh H, Tavakkoli H, Adibi P, et al. Association between inflammatory potential of the diet and ulcerative colitis: a case-control study. Front Nutr. 2020;7:602090.
29. Barnes EL, Nestor M, Onyewadume L, de Silva PS, Korzenik JR, DREAM Investigators. High dietary intake of specific fatty acids increases risk of flares in patients with ulcerative colitis in remission during treatment with aminosalicylates. Clin Gastroenterol Hepatol 2017;15:1390–1396.e1.

30. Peters V, Bolte L, Schuttert EM, Andreu-Sánchez S, Dijkstra G, Weersma RK, et al. Western and carnivorous dietary patterns are associated with greater likelihood of IBD development in a large prospective population-based cohort. J Crohns Colitis. 2022;16:931–9.
31. Forbes A, Escher J, Hébuterne X, Kłęk S, Krznaric Z, Schneider S, et al. ESPEN guideline: Clinical nutrition in inflammatory bowel disease. Clin Nutr. 2017;36:321–47.
32. Mozaffarian D. Foods, nutrients, and health: when will our policies catch up with nutrition science? Lancet Diabetes Endocrinol. 2017;5:85–8.
33. Campmans-Kuijpers MJE, Dijkstra G. Food and food groups in inflammatory bowel disease (IBD): the design of the Groningen anti-inflammatory diet (GrAID). Nutrients. 2021;13:1067.
34. Damas OM, Garces L, Abreu MT. Diet as adjunctive treatment for inflammatory bowel disease: review and update of the latest literature. Curr Treat Options Gastroenterol. 2019;17:313–25.
35. Cox SR, Prince AC, Myers CE, Irving PM, Lindsay JO, Lomer MC, et al. Fermentable carbohydrates [FODMAPs] exacerbate functional gastrointestinal symptoms in patients with inflammatory bowel disease: a randomised, double-blind, placebo-controlled, cross-over, re-challenge trial. J Crohns Colitis. 2017;11:1420–9.
36. Sarbagili-Shabat C, Albenberg L, Van Limbergen J, Pressman N, Otley A, Yaakov M, et al. A novel Uc exclusion diet and antibiotics for treatment of mild to moderate pediatric ulcerative colitis: a prospective open-label pilot study. Nutrients. 2021;13:3736.
37. Moeller SM, Reedy J, Millen AE, Dixon LB, Newby PK, Tucker KL, et al. Dietary patterns: challenges and opportunities in dietary patterns research an Experimental Biology workshop, April 1, 2006. J Am Diet Assoc. 2007;107:1233–9.
38. Wirth MD, Burch J, Shivappa N, Violanti JM, Burchfiel CM, Fekedulegn D, et al. Association of a dietary inflammatory index with inflammatory indices and metabolic syndrome among police officers. J Occup Environ Med. 2014;56:986–9.
39. Wood LG, Shivappa N, Berthon BS, Gibson PG, Hebert JR. Dietary inflammatory index is related to asthma risk, lung function and systemic inflammation in asthma. Clin Exp Allergy. 2015;45:177–83.
40. Shivappa N, Steck SE, Hurley TG, Hussey JR, Ma Y, Ockene IS, et al. A population-based dietary inflammatory index predicts levels of C-reactive protein in the Seasonal Variation of Blood cholesterol Study (SEASONS). Public Health Nutr. 2014;17:1825–33.
41. Miller PE, Lazarus P, Lesko SM, Muscat JE, Harper G, Cross AJ, et al. Diet index-based and empirically derived dietary patterns are associated with colorectal cancer risk. J Nutr. 2010;140:1267–73.
42. Neurath MF. Cytokines in inflammatory bowel disease. Nat Rev Immunol. 2014;14:329–42.
43. Assa A, Vong L, Pinnell LJ, Avitzur N, Johnson-Henry KC, Sherman PM. Vitamin D deficiency promotes epithelial barrier dysfunction and intestinal inflammation. J Infect Dis. 2014;210:1296–305.
44. Freedman LS, Carroll RJ, Wax Y. Estimating the relation between dietary intake obtained from a food frequency questionnaire and true average intake. Am J Epidemiol. 1991;134:310–20.

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