



Applied nutritional investigation

Association between spicy food consumption and inflammatory bowel disease: A case-control study from Saudi Arabia



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ABSTRACT

Background: Inflammatory bowel disease (IBD), including ulcerative colitis (UC) and Crohn's disease (CD), is a chronic gastrointestinal disorder with increasing global prevalence. Although dietary factors are increasingly implicated in IBD pathogenesis, the role of spicy food remains unclear, especially in Arab populations where such foods are commonly consumed. This study aimed to examine the association between spicy food consumption and the risk of UC and CD in an Arab population.

Methods: We conducted a case-control study at a private clinic in Saudi Arabia, involving 157 patients with UC, 226 with CD, and 390 controls. IBD was diagnosed using laboratory tests, endoscopy with biopsies, and imaging when indicated. Spicy food was defined as dishes made with chili peppers or hot sauces and was assessed using a self-administered questionnaire. Multivariable logistic regression was used to evaluate the association between spicy food intake and IBD risk, and adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were calculated.

Results: Daily consumption of spicy food was significantly associated with higher odds of CD (OR = 1.61; 95% CI: 1.11, 2.33), but not UC (OR = 1.03; 95% CI: 0.67, 1.60). No significant associations were observed between spicy food and IBD extent or severity.

Conclusions: In this Arab population, daily spicy food intake was positively associated with CD risk, while no association was identified for UC. Future prospective cohort studies with detailed information about spicy food types and IBD severity scores are needed to confirm our findings.

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Introduction

Inflammatory bowel disease (IBD), comprising ulcerative colitis (UC) and Crohn's disease (CD), is a chronic gastrointestinal condition characterized by abdominal pain, diarrhea, weight loss, fatigue, and extra-intestinal manifestations, including arthropathies, erythema nodosum, uveitis, and hepatobiliary involvement [1]. Beyond its physical manifestations, IBD significantly affects

mental health and quality of life, often leading to anxiety and depression [2,3]. Globally, the burden of IBD has grown steadily, particularly in newly industrialized countries undergoing rapid lifestyle and dietary transitions. Between 1990 and 2019, age-standardized prevalence rates increased in the majority of Global Burden of Disease (GBD) regions, with an estimated 4.9 million cases and 41,000 related deaths by 2019 [4]. The economic impact is also substantial. Direct annual healthcare costs in the United States range from \$7,824 to \$41,829 per patient, reflecting outpatient care, hospitalization, and medication expenses [5]. Accordingly, IBD is now recognized as a major public health concern [6].

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In Saudi Arabia, IBD has shifted from being considered rare to an increasingly recognized public health concern [7,8]. CD is reported to be more common than UC, with early estimates indicating an incidence of approximately 0.94 cases per 100,000 individuals [9]. More recent evidence suggests that CD incidence continues to rise, mirroring trends observed in other rapidly industrializing regions undergoing lifestyle and environmental transitions. The clinical presentation, disease behavior, and morbidity profile among Saudi patients have been reported to resemble those documented in Western populations [10–12].

The rising prevalence of IBD, especially in developing countries, has been linked to the “westernization” of dietary patterns, including increased consumption of processed and fast foods, food additives, and pro-inflammatory nutrients [13,14]. Among various dietary exposures, spicy food consumption has been proposed as a potential environmental trigger of gastrointestinal symptoms and inflammation [15–17]. Capsaicin, the primary bioactive compound in hot chili peppers, can activate the transient receptor potential vanilloid 1 (TRPV1) receptor in the gastrointestinal tract, leading to neurogenic inflammation, increased intestinal permeability, and release of pro-inflammatory neuropeptides such as substance P and calcitonin gene-related peptide (CGRP). Excessive or frequent capsaicin exposure may also alter gut motility, modify gut microbiota composition, and heighten visceral sensitivity, potentially worsening inflammation in susceptible individuals [18–24].

Despite these plausible mechanisms, the role of spicy food in IBD etiology remains insufficiently explored, as most dietary research has focused on macronutrients or specific food groups (e.g., red meat, dairy, processed foods) rather than spice-related exposures [23–25]. Another important limitation of the existing literature is the lack of cultural and geographic diversity. Most studies have been conducted in Western populations, where the nature, frequency, and culinary use of spicy foods differ from regions where such foods are consumed regularly. A systematic review reported marked differences in dietary risk factors for IBD between Eastern and Western populations, suggesting that region-specific dietary exposures may influence IBD development differently [26].

Spicy food consumption is often reported to be higher in populations living in warmer climates due to cultural preferences and possibly biological adaptation [27]. In Saudi Arabia, a predominantly hot climate country, spicy dishes are commonly incorporated into traditional and modern cuisine [28,29]. However, the term “spicy food” lacks a universal definition, encompassing aromatic spices such as cumin, black pepper, ginger, or garlic, as well as heat-inducing ingredients like chili peppers [30]. In the Arab context, it typically refers specifically to hot chili-based foods. Spicy food is typically incorporated into cooked dishes rather than raw condiments [28,29].

Given the rising incidence of IBD in Saudi Arabia, the cultural integration of spicy foods in local cuisine, and the limited evidence regarding their potential role in disease etiology, this study aimed to investigate the association between spicy food consumption and the risk of UC and CD in a Saudi population.

Methods

Study design and setting

This case-control study was performed at a private clinic in Riyadh, Saudi Arabia, with data collection spanning from January 2009 to December 2017. Details about sample size calculation and participant selection were described elsewhere [31–35]. Briefly, using convenience sampling, 171 patients with UC, 251 with CD,

and 400 controls were enrolled. Participants with IBD were eligible if they were at least 18 years old, newly diagnosed, their diagnosis was confirmed through colonoscopy and histological biopsy, they reported their spicy food consumption, and provided written informed consent. Newly diagnosed IBD refers to cases in which patients received their first diagnosis of CD or UC within the defined study period. Those with prior documented IBD were not involved. Controls, who were also patients visiting the same polyclinic for other gastrointestinal complaints, needed to be at least 18 years old, free from any confirmed or suspected diagnosis of IBD, malignancy, polyposis, or diverticulosis, report on their spicy food consumption, and sign informed consent. After applying the eligibility criteria, the number of participants decreased to 157 with UC (91.8%), 226 with CD (90.0%), and 390 controls (97.5%).

Inflammatory bowel disease ascertainment

All symptomatic patients underwent basic blood tests and stool and urine analyses. Those with suggestive results proceeded to colonoscopy or ileocolonoscopy. Biopsies were collected from affected segments and at least two macroscopically normal areas. These biopsies were examined for features such as crypt architectural distortion, basal plasmacytosis, granulomas, and transmural inflammation. When strictures or fistulas were suspected, CT or MR enterography was performed. UC diagnosis required continuous mucosal inflammation starting in the rectum, whereas CD diagnosis could involve segmental inflammation, cobblestoning, or granulomas. Two gastroenterologists and one pathologist reviewed each case, with disagreements resolved through consensus.

Assessment of spicy food consumption

Spicy food consumption was assessed using a self-administered questionnaire distributed before diagnoses were made. Spicy food consumption in a typical week was assessed using the following question: “How often do you eat sweet food?” Spicy food was defined as dishes containing chili peppers or hot sauces commonly used in Saudi cuisine, such as shatta, harissa, or foods prepared with red chili powder, green chili, or hot pepper paste. The response options were “once per month or less,” “once per week,” “twice per week,” “every day,” “do not remember,” and “not applicable.” To enhance statistical power, the responses “once per month or less,” “once per week,” and “twice per week” were combined into a single category labeled “infrequent consumption,” while the response “every day” was labeled “frequent consumption.” Responses of “do not remember” or “not applicable” resulted in exclusion. Trained data collectors assisted illiterate participants.

Covariates

Additional variables included age, sex, smoking status (current, former, or never), alcohol consumption (current, former, or never), body mass index (BMI), and levels of hemoglobin, creatinine, and liver enzymes. BMI was measured at the clinic and classified as underweight (< 18.5 kg/m²), normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), or obese (≥ 30.0 kg/m²) [36]. Anemia was defined as hemoglobin < 13.0 g/dL in men or < 12.0 g/dL in women [37]. Elevated liver enzymes were identified if levels of alanine transaminase (ALT) were > 36 IU/L or aspartate transaminase (AST) > 30 IU/L [38]. Elevated creatinine was defined as serum creatinine ≥ 1.2 mg/dL in men and ≥ 1.1 mg/dL in women [39].

Statistical analysis

Logistic regression models were used to calculate odds ratios (ORs) and 95% confidence intervals (CIs) for the relationship between daily consumption of spicy foods and the risk of UC and CD. Additional analyses examined the association of spicy food consumption with the extent and severity of both conditions. All models were adjusted for age, sex, BMI, smoking status, anemia, and elevated liver enzymes. All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 773 participants were included in the analysis: 390 controls, 157 with UC, and 226 with CD. Compared with controls, individuals with UC and CD were more likely to be younger than 30 years (20.4% and 34.1% vs. 17.2%, $P < 0.001$). The proportion of men was highest among CD patients (69.0%), followed by controls (64.4%) and UC patients (59.2%), but this difference was not statistically significant ($P = 0.141$). Underweight status (BMI < 18.5 kg/m²) was more prevalent among CD patients (18.1%) compared to UC patients (8.9%) and controls (9.5%), while obesity (BMI ≥ 30 kg/m²) was most common among controls (31.5%), followed by UC (22.3%) and CD (9.3%) ($P > 0.001$). Anemia was substantially more prevalent in UC (43.9%) and CD (23.5%) patients than in controls (15.4%) ($P < 0.001$), while elevated liver enzymes were more frequent among controls (19.2%) than UC (15.3%) or CD (10.2%) patients ($P = 0.012$). Smoking was significantly less common in UC patients compared to controls (11.5% vs. 20.8%, $P = 0.036$) (Table 1).

Spicy food consumption patterns differed notably across groups. Daily consumption was most common among CD patients (41.6%), followed by controls (30.0%) and UC patients (28.7%). In contrast, twice-weekly consumption was highest among UC patients (52.9%), compared to 35.4% in CD and 24.4% in controls.

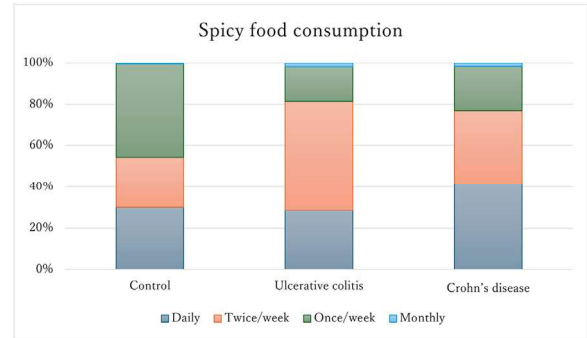


Fig. 1. Distribution of spicy food consumption frequency among controls, patients with ulcerative colitis, and patients with Crohn's disease. Bars represent the percentage of participants in each group reporting daily, twice-weekly, once-weekly, or monthly consumption of spicy foods.

Weekly consumption was more common among controls (45.1%) than UC (16.6%) or CD (21.2%) patients (Figure 1).

In both regression models, the model adjusted for age and sex (Model I) and the model adjusted for age, sex, BMI, smoking, anemia, and liver enzymes (Model II), frequent consumption of spicy food was not associated with UC: ORs (95% CIs) = 0.93 (0.62, 1.41) and 1.03 (0.67, 1.60), respectively (Table 2). Although pancolitis patients reported more frequent consumption of spicy food (34.2%) than those with other extents (27.4%), this association was not statistically significant in either model: ORs (95% CIs) = 1.37 (0.62, 3.04) and 1.33 (0.58, 3.05) (Table 3).

In contrast, frequent consumption of spicy food was associated with higher odds of CD in Model I (OR = 1.59; 95% CI: 1.12, 2.27) and Model II (OR = 1.61; 95% CI: 1.11, 2.33) (Table 4). However, no significant associations were observed between spicy food consumption and ileocolic vs. ileal/colic involvement (Model II OR = 0.72; 95% CI: 0.31, 1.68) (Table 5), nor with complicated disease (defined as strictures and/or penetrating behavior) (Model II OR = 1.12; 95% CI: 0.50, 2.53) (Table 6).

Table 1 Comparison between cases and controls

Characteristics	Control n = 390 (%)	Ulcerative colitis n = 157 (%)	Crohn's disease n = 226 (%)	P-value
Age, years				
< 30	67 (17.2)	32 (20.4)	77 (34.1)	<0.001
30–39	168 (43.1)	54 (34.4)	100 (44.2)	
≥ 40	155 (39.7)	71 (45.2)	49 (21.7)	
Sex				0.141
Men	251 (64.4)	93 (59.2)	156 (69.0)	
Women	139 (35.6)	64 (40.8)	70 (31.0)	
Body mass index, kg/m ²				<0.001
< 18.5	37 (9.5)	14 (8.9)	41 (18.1)	
18.5–24.9	117 (30.0)	72 (45.9)	119 (52.7)	
25.0–29.9	113 (29.0)	36 (22.9)	45 (19.9)	
≥ 30	123 (31.5)	35 (22.3)	21 (9.3)	
Current smoking	81 (20.8)	18 (11.5)	44 (19.5)	0.036
Current alcohol consumption	0 (0.0)	1 (0.6)	1 (0.4)	0.337
Anemia	60 (15.4)	69 (43.9)	53 (23.5)	<0.001
Elevated liver enzymes	75 (19.2)	24 (15.3)	23 (10.2)	0.012
Elevated creatinine	1 (0.3)	3 (1.9)	1 (0.4)	0.083
Extent of ulcerative colitis				–
Proctitis	–	7 (4.5)	–	
Left-sided	–	76 (49.0)	–	
Extensive	–	34 (22.0)	–	
Pancolitis	–	38 (24.5)	–	
Extent of Crohn's disease				–
Ileum	–	–	188 (83.2)	
Colon	–	–	4 (1.8)	
Ileocolonic	–	–	34 (15.0)	
Severity of Crohn's disease				–
No strictures or penetrations	–	–	187 (82.7)	
Strictures only	–	–	20 (8.8)	
Penetrations only	–	–	10 (4.4)	
Strictures and penetrations	–	–	9 (4.0)	

Table 2

Association between spicy food consumption and ulcerative colitis.

Spicy food consumption	Control n = 390 (%)	Ulcerative colitis n = 157 (%)	Model I	Model II
Infrequent	273 (70.0)	112 (71.3)	1 (Reference)	1 (Reference)
Frequent	117 (30.0)	45 (28.7)	0.93 (0.62, 1.41)	1.03 (0.67, 1.60)

Model I: ORs and 95% CIs adjusted for age and sex.

Model II: ORs and 95% CIs adjusted for age, sex, BMI, smoking, anemia, and liver enzymes.

Table 3

Association between spicy food consumption and the extent of ulcerative colitis.

Spicy food consumption	Proctitis, left-sided, or extensive n = 117 (%)	Pancolitis n = 38 (%)	Model I	Model II
Infrequent	85 (72.6)	25 (65.8)	1 (Reference)	1 (Reference)
Frequent	32 (27.4)	13 (34.2)	1.37 (0.62, 3.04)	1.33 (0.58, 3.05)

Model I: ORs and 95% CIs adjusted for age and sex.

Model II: ORs and 95% CIs adjusted for age, sex, BMI, smoking, anemia, and liver enzymes.

Table 4

Association between spicy food consumption and Crohn's disease

Spicy food consumption	Control n = 390 (%)	Crohn's disease n = 226 (%)	Model I	Model II
Infrequent	273 (70.0)	132 (58.4)	1 (Reference)	1 (Reference)
Frequent	117 (30.0)	94 (41.6)	1.59 (1.12, 2.27)	1.61 (1.11, 2.33)

Model I: ORs and 95% CIs adjusted for age and sex.

Model II: ORs and 95% CIs adjusted for age, sex, BMI, smoking, anemia, and liver enzymes.

Table 5

Association between spicy food consumption and the extent of Crohn's disease

Spicy food consumption	Ileum or colic n = 193 (%)	Ileocolic n = 33 (%)	Model I	Model II
Infrequent	112 (58.0)	21 (63.6)	1 (Reference)	1 (Reference)
Frequent	81 (42.0)	12 (36.4)	0.74 (0.34, 1.62)	0.72 (0.31, 1.68)

Model I: ORs and 95% CIs adjusted for age and sex.

Model II: ORs and 95% CIs adjusted for age, sex, BMI, smoking, anemia, and liver enzymes.

Table 6

Association between spicy food consumption and the severity of Crohn's disease

Spicy food consumption	No strictures or penetrations n = 187 (%)	Strictures or penetrations n = 39 (%)	Model I	Model II
Infrequent	111 (59.4)	21 (53.8)	1 (Reference)	1 (Reference)
Frequent	76 (40.6)	18 (46.2)	1.10 (0.54, 2.24)	1.12 (0.50, 2.53)

Model I: ORs and 95% CIs adjusted for age and sex.

Model II: ORs and 95% CIs adjusted for age, sex, BMI, smoking, anemia, and liver enzymes.

Discussion

This study explored the association between spicy food consumption and the risk of IBD in an Arab population. Our results indicate that frequent consumption of spicy food was associated with significantly higher odds of CD (OR = 1.61), but not UC. No association was found between spicy food consumption and CD phenotype (ileocolic vs. ileal/colic involvement) or disease complications such as stricturing or penetrating behavior.

Our findings are consistent with earlier studies showing symptom aggravation linked to spicy food in IBD patients. A study of 6,768 IBD patients from the US Crohn's and Colitis Foundation of America (CCFA) showed that consuming spicy foods was associated with worsening symptoms [40]. In a study of 233 IBD patients from New Zealand, consuming chillis was reported as a symptom exacerbation in 45% of participants [41]. On the other hand, the China Kadoorie Biobank cohort study (n = 512,726) showed an inverse association between spicy food consumption and the risk

of UC [42]. However, spicy food consumers in this cohort were more likely to be heavy smokers [43]. A meta-analysis of nine case-control studies showed a strong inverse association between smoking and UC (pooled OR = 0.26) [44].

Several biological mechanisms may explain the link between frequent consumption of spicy foods and an increased risk of CD. Capsaicin, the active component in spicy foods, activates the TRPV1 receptor, which is abundant in gastrointestinal epithelium. This activation can alter gut motility, increase mucus secretion, and impair intestinal barrier integrity by disrupting tight junction proteins, such as occludin and claudin-1, leading to increased intestinal permeability, also known as a "leaky gut." This may facilitate the translocation of luminal antigens, triggering chronic mucosal inflammation [18–20]. Capsaicin also affects gut microbiota, with high doses shown to reduce beneficial bacteria (e.g., *Lactobacillus*, *Bifidobacterium*) and promote pro-inflammatory species, fostering a Th1/Th17-polarized immune response characteristic of CD [20,21]. The stronger association with CD compared

to UC may reflect their distinct pathophysiology: CD affects the full bowel wall and commonly involves the ileum, where TRPV1 expression is high and microbiota-host interactions are complex, while UC is limited to superficial colonic mucosa [22].

Despite several strengths, such as the inclusion of an understudied population, the use of newly diagnosed IBD cases, and the application of standardized diagnostic criteria, this study has some limitations. First, convenience sampling was employed due to the study's setting in a single private clinic and the need to recruit newly diagnosed participants over an extended period. While this approach facilitated timely recruitment and access to detailed clinical data, it might have introduced selection bias and limited the generalizability of findings since the patients likely represent a higher socioeconomic group. Second, the control group consisted of patients presenting with gastrointestinal complaints but without IBD, cancer, or other exclusionary conditions. While this design helped ensure that controls underwent diagnostic evaluation and shared similar healthcare-seeking behavior, it may also have introduced differential baseline characteristics and potential clinical heterogeneity. Third, dietary exposure was assessed using a non-validated food frequency questionnaire. However, previous findings based on the same tool, particularly associations between dietary factors and IBD, were later supported by meta-analyses [33,35], indicating that the questionnaire likely provided accurate estimates of dietary intake. Fourth, the analysis did not adjust for key dietary factors, including total caloric intake and nutrients that may correlate with spicy food consumption. However, adjustment for BMI, a strong correlate of total caloric and fat intake [45], had little effect on the association, suggesting that residual confounding by overall diet is unlikely to explain the findings. Fifth, due to the limited sample size, non-daily consumers were grouped, limiting the ability to explore dose-response relationships. Sixth, the scope of analysis was limited to the association between spicy food consumption and disease diagnosis and extent. The study did not examine disease activity, biomarker profiles, endoscopic severity indices, or treatment requirements. Seventh, although recall bias cannot be excluded due to the study design of this study, it was likely non-differential, as information on spicy food consumption was obtained before the final confirmation of IBD diagnosis. This temporal sequence reduces the likelihood that participants' reporting was influenced by knowledge of their disease status.

Future research should aim to validate these findings using prospective cohort designs with larger and more diverse populations. The association with IBD endoscopy and clinical severity indices should be measured. Additionally, mechanistic studies are needed to explore the role of capsaicin in intestinal barrier function and gut microbiota composition in humans. Investigating gene-diet and microbiota-diet interactions may clarify individual susceptibility to spicy food. Besides, exploring the impact of reducing spicy food intake on disease onset, symptom severity, or flare-up frequency in at-risk individuals could have practical implications for dietary prevention strategies in IBD.

Conclusion

In this case-control study, daily consumption of spicy food was significantly associated with increased odds of CD, even after adjusting for potential confounders. No such association was observed for UC, nor were significant differences found based on disease extent or phenotype. These findings underscore the potential dietary contribution to CD risk, suggesting that limiting spicy food consumption should be considered in dietary counseling for individuals at high risk of CD. Future studies should employ validated food frequency questionnaires, involve broader populations,

and investigate gene-diet or microbiota-diet interactions to understand the mechanisms linking spicy food consumption to CD.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Anas Almofarreh: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Methodology, Investigation, Formal analysis, Conceptualization. **Haytham A. Sheerah:** Writing – review & editing, Visualization, Validation, Supervision, Conceptualization. **Ahmed Arafa:** Writing – review & editing, Visualization, Validation, Methodology. **Tareq Moh'd:** Writing – review & editing. **Rayan A. Tayyib:** Writing – review & editing. **Ahad N. Yamani:** Writing – review & editing, Conceptualization. **Aidrous M. Ali:** Writing – review & editing, Visualization, Validation.

Data availability

We did not make our data publicly available due to ethical or administrative reasons. However, the corresponding author may provide the original dataset upon a reasonable request after consulting the ethical committee.

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Compliance with ethical standards

The King Fahd Medical City's (KFCC) Institutional Review Board (IRB) approved the study protocol (IRB registration number: H-01-R-012). Informed consent was obtained from all participants. The study was conducted per the Declaration of Helsinki.

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