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Effects of a low FODMAP diet on gut microbiota in inflammatory bowel disease: a systematic review

Sebastian Arambulo-Castillo[a], Zhamanda N. Ortiz-Benique[b], Judith Sanchez-Zavaleta[c], Doyler Cubas-Garcia[d], Nicole Collantes-Silva[e], Antony Gonzales-Uribe[f], Carlos Yengle-Garcia[g], Josh Elefteratos[a], Lingyu Bao[a]

Albert Einstein School of Medicine, Montefiore Medical Center, Wakefield Campus, Bronx, NY, USA; Universidad Nacional de San Agustín de Arequipa, Arequipa, Peru; Universidad Nacional Mayor de San Marcos, Lima Perú; Hospital Nacional Edgardo Rebagliati Martins, Lima, Perú; University of New Mexico, Albuquerque, NM, USA; Jacobi Medical Center/Albert Einstein College of Medicine, Bronx, NY, USA; niversidad Privada Antenor Orrego, La Libertad, Perú

Abstract

The low FODMAP diet is commonly used to alleviate gastrointestinal symptoms in patients with quiescent inflammatory bowel disease, particularly those experiencing irritable bowel syndrome–like symptoms. While short-term symptomatic benefits have been reported, its effects on gut microbiota composition remain unclear. We conducted a systematic review of randomized controlled trials following PRISMA guidelines. A comprehensive search of PubMed, Embase, Scopus, and Web of Science through April 2025 identified studies involving adults with inflammatory bowel disease who underwent a low FODMAP diet intervention with pre- and post-diet microbiota assessment. Two reviewers independently screened records, extracted data, and assessed risk of bias using the Cochrane risk of bias tool, version 2. Outcomes included gut microbiota composition, short-chain fatty acid levels, gastrointestinal symptoms, inflammatory markers, and quality of life. Of 3,421 records screened, two randomized controlled trials enrolling 61 patients met inclusion criteria. Both studies demonstrated improvement in gastrointestinal symptoms among patients receiving the low FODMAP diet. However, consistent alterations in gut microbiota were observed, including reductions in several beneficial butyrate-producing and immunoregulatory taxa, as well as lower fecal short-chain fatty acid levels, without significant changes in inflammatory biomarkers. In conclusion, the low FODMAP diet appears effective for short-term symptom relief in patients with quiescent inflammatory bowel disease, but it may adversely affect gut microbiota composition. Further long-term randomized controlled trials are needed to clarify its impact on disease course and relapse risk.

Keywords Inflammatory bowel disease, low FODMAP diet, gut microbiota, gastrointestinal symptoms, systematic review

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Correspondence to: Sebastian Arambulo-Castillo, 600 E 233rd St, Bronx, NY 10466, e-mail: sebastianrambulo@hotmail.com

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Introduction

Inflammatory bowel disease (IBD), which includes Crohn's disease (CD) and ulcerative colitis (UC) is a group of chronic immune-mediated inflammatory disorders characterized by a relapsing-remitting course affecting millions worldwide [1]. The global prevalence of IBD was estimated in 3.83 million cases in 2021, reflecting a rising trend worldwide, particularly in newly industrialized countries [2,3]. However, despite this rising prevalence, age-standardized rates (ASR) for mortality and disability-adjusted life years (DALYs) have shown a steady decline [4]. This trend likely reflects advances in diagnostics and therapeutic strategies [1].

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The pathogenesis of IBD involves dysregulated immune responses to luminal antigens in susceptible individuals, resulting in chronic inflammation. Among the contributing factors, alterations in the gut microbiota, commonly referred to as dysbiosis, have been studied as a key element in both the initiation and perpetuation of the inflammatory response [5]. Dysbiosis in IBD is characterized by reduced functional diversity and imbalances in dominant phyla [6,7]. Notable alterations include a reduction in the Firmicutes phylum and an increased abundance of Proteobacteria [6-8]. These changes can lead to significant metabolic consequences for the host, most notably a reduction in short-chain fatty acids (SCFAs) [8-10], which play an essential role in maintaining intestinal barrier integrity and modulating mucosal immune responses [9,10].

Given the close relationship between gut microbiota composition and intestinal inflammation in IBD, dietary strategies have gained increasing attention not only for symptom control but also for their potential impact on the microbiome. Among nutritional interventions, the low fermentable, oligo-, di-, monosaccharides and polyols (low FODMAP) diet, originally designed for managing symptoms in functional gastrointestinal disorders, has also been studied in patients with IBD with reported improvements in abdominal pain, bloating and diarrhea [11-13].

However, the impact of the low FODMAP diet on gut microbiota composition in IBD remains uncertain. Understanding these effects is crucial to determine whether symptom improvement associated with this dietary approach is accompanied by potentially adverse alterations in the gut microbiome, which could have long-term implications for disease management and mucosal health.

This systematic review aims to evaluate the impact of the low FODMAP diet on gut microbiota composition in patients with IBD, focusing on changes in microbial diversity and the relative abundance of bacterial taxa.

Methods

Search strategy

A systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Suppl. Table 1) and was registered in PROSPERO (CRD420251026701) [14]. A comprehensive literature search was performed in PubMed, Scopus, Embase, and Web of Science including all the studies published through April 7th, 2025. A search formula was constructed using MeSH terms related to 'Inflammatory Bowel Diseases', 'Low FODMAP diet' and 'Gastrointestinal microbiome'; this formula was adapted to each database from above. We also performed a manual reference review of included articles to identify potential studies.

Selection criteria

Eligible studies included cohorts and clinical trials, either randomized or nonrandomized, that involved adult patients (aged 18 years or older) diagnosed with IBD, including CD or UC, confirmed by clinical, endoscopic, histological, or radiological criteria. To be included, studies were required to report the following as a primary outcome: effect of a low FODMAP diet on gut microbiota composition or function. Additionally, eligible studies had to report at least one microbiota-related outcome (e.g., bacterial diversity, relative or absolute abundance, or SCFAs levels).

Data collection

All records identified through the search strategy were uploaded to https://www.rayyan.ai/, an online platform designed to streamline the study selection process. After duplicate entries were removed, two reviewers (JS-Z and NC-S) independently screened the titles and abstracts based on the predefined eligibility criteria. Any disagreements were resolved by a third reviewer (SA). Subsequently, two other independent reviewers (DC and CY) assessed the full texts to determine final study inclusion, with a third author (SA) resolving any discrepancies.

Data extraction

Data from included studies were extracted using a standardized Excel spreadsheet by two independent reviewers (CY and DC). All data extracted from included studies are presented in Tables 1–5 and Supplementary Tables of this manuscript.

The following categories were used:

Study characteristics

First author, year of publication, trial registration number (if available), country, setting, study design, duration of intervention, sample size, main inclusion and exclusion criteria, comparison/control diet, and primary aim of the study.

Participant characteristics

Median age (with interquartile range [IQR]), sex distribution, type of IBD (CD vs UC), and race/ethnicity (if reported).

[a]Division of General Internal Medicine, Department of Medicine, Albert Einstein School of Medicine, Montefiore Medical Center, Wakefield Campus, Bronx, NY, USA (Sebastian Arambulo-Castillo, Josh Elefteratos, Lingyu Bao); [b]Universidad Nacional de San Agustín de Arequipa, Arequipa, Peru (Zhamanda N. Ortiz-Benique); [c]Gastroenterología, Unidad de Postgrado, Universidad Nacional Mayor de San Marcos, Lima Perú (Judith Sanchez-Zavaleta); [d]Servicio de Trasplante de órgano sólido, Hospital Nacional Edgardo Rebagliati Martins, Lima, Perú (Doyler Cubas-Garcia); [e]Department of Internal Medicine, University of New Mexico, Albuquerque, NM, USA (Nicole Collantes-Silva); [f]Jacobi Medical Center/Albert Einstein College of Medicine, Bronx, NY, USA (Antony Gonzales-Uribe); [g]Universidad Privada Antenor Orrego, La Libertad, Perú (Carlos Yengle-Garcia)

Table 1 Study design and patient characteristics of RCTs evaluating low FODMAP diet in IBD

Author (Year)	*Halmos, et al (2016)*	*Cox, et al (2020)*
Period	March 2009 and May 2011 (Recruitment)	Feb 2016 - May 2017 (Recruitment)
Country/ Setting	Australia / Monash University	United Kingdom/ Multicenter (2 gastroenterology clinics in London)
Study design	Randomized, controlled, cross-over feeding trial	Randomized, parallel, single-blind, placebo-controlled trial
Sample size	9 participants	52 participants
Participant age*	35 (29 - 41)	31 (11)
Female (%)	6 (67%)	29 (56%)
CD/CU (n)	8/0	26/26
Main inclusion criteria	Patients with quiescent CD (Harvey–Bradshaw Index ≤5), clinically stable, on stable therapy for >=8 weeks.	Adults (≥18 years) with quiescent CD or UC, persistent gut symptoms, stable medications, and naïve to low FODMAP diet.**
Main exclusion criteria	Celiac disease (excluded via biopsy or serology on gluten diet), prior abdominal surgery, comorbidities (e.g., diabetes), antibiotic/probiotic use within previous 2 months	Recent changes in azathioprine/mercaptopurine/ methotrexate/biologics (<12 weeks), 5-ASA (<4 weeks), antibiotics/probiotics/prebiotics (<8 weeks), perianal CD, stoma, strictures, extensive GI resection, bile acid malabsorption, pregnancy, lactation, or major comorbidities.
Outcomes	Primary Changes in fecal microbiota in CD patients. Secondary: GI symptoms, Fecal frequency, fecal weight, FWC, pH, SCFAs, whole gut transit time differences in fecal pH, total and specific fecal SCFAs concentration, gastrointestinal symptoms, fecal weight, FWC, whole-gut transit time,	Primary: IBS-SSS symptom score Secondary: gut symptoms, disease-specific HRQOL, stool frequency and consistency, clinical disease activity, inflammatory markers, dietary intake, microbiome composition and function, SCFAs, and peripheral T-cell phenotype

*Age is presented as median (IQR) for Halmos *et al*. and mean (standard deviation) for Cox *et al*
**Quiescent IBD was defined by all of the following: physician global assessment, stable medications, no IBD flare in the previous 6 months, fecal calprotectin <250 ug/g, and serum C-reactive protein (CRP) <10 mg/L
Note: Differences in study design (cross-over vs. parallel), inclusion criteria, and microbiota analysis methods may influence outcome comparability
CD, Crohn's disease; UC, ulcerative colitis; IBD, inflammatory bowel disease; RCT, randomized controlled trial; FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides and polyols; SCFAs, short-chain fatty acids; FWC, fecal water content; GI, gastrointestinal; HRQoL, health-related quality of life; IBS-SSS, irritable bowel syndrome severity scoring system

Table 2 Dietary Interventions and microbiota analysis of the included randomized controlled trials investigating Low FODMAP diets in microbiota of patients with IBD

Author	Halmos, *et al*	Cox, *et al*
Follow up period	≥ 9 weeks*	4 weeks (intervention)
Intervention/ Control	Low FODMAP diet vs. habitual Australian diet	Low FODMAP diet vs. sham diet
Diet delivery	Meals provided, frozen and delivered weekly; strict menu and food diaries; high control.	Dietitian-led advice with written materials and weekly follow-up calls; self-administered.
Sampling collection timing	Last 5 days of trial	At baseline and end of trial.
Sample storage conditions	Thawed at 4 °C, aliquoted, stored at −20°C	Homogenized and stored at -80 °C
Sampling analysis technique	SCFAs by GC; pH measured; DNA extracted with bead-beating; qPCR for bacterial abundance.	SCFAs by gas-liquid chromatography protocol, pH probe. DNA quantitated via Qubit; metagenomics pipeline per IHMS

*Halmos *et al* used a 3-period cross-over design: 3 weeks for intervention, 3 wash out and 3 control
SCFAs, Short-chain fatty acids; GC, gas chromatography; DNA, deoxyribonucleic acid; PCR, polymerase chain reaction; qPCR, quantitative polymerase chain reaction; IHMS, international human microbiome standards; FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides, and polyols

Microbiota-related variables

Sampling technique and site (e.g., fecal sample), storage conditions, analytical technique (e.g., 16S ribosomal RNA [16S rRNA] sequencing, quantitative polymerase chain reaction [qPCR], metagenomics), microbial targets (e.g., *Bifidobacterium spp., F. prausnitzii*), diversity measures (alpha and beta diversity), SCFAs levels (e.g., acetate, butyrate, propionate), and key microbiota-related findings.

Table 3 Microbiota-related outcomes and microbial shifts

Author	Halmos, *et al*	Cox, *et al*
Microbiota analysis method	q PCR	Metagenomics (IHMS pipeline)
Targeted bacteria groups	*Clostridium* cluster IV (*Clostridium leptum* group) including *Faecalibacterium prausnitzii* and *Clostridium* cluster XIVa (*Clostridium coccoides* group) and *Roseburia* spp., traditionally prebiotic bacteria (*Lactobacilli* and *Bifidobacteria* spp.), and mucus-degrading bacteria (*Akkermansia muciniphila*, *Ruminococcus gnavus* and *Ruminococcus torques*)	*Bifidobacteria* family and *Faecalibacterium prausnitzii*
Functional classification of bacteria	*Clostridium leptum* group, *Clostridium coccoides* group and *Roseburia* spp., traditionally butyrate-producing bacteria, prebiotic bacteria and mucus-degrading bacteria	NI
Diversity (α / β)	NS/ NS	NS/NS
SCFAs levels measured	Yes (GC) NS	↓ Total SCFAs in UC (not in CD)
Statistical differences in absolute abundance	↓ *Clostridium cluster* XIVa, ↓ *A. muciniphila*	NI
Statistical differences in relative abundance	↓ *Clostridium* cluster XIVa, ↓ *A. muciniphila*, ↑ *R. torques*	↓ *Bifidobacterium adolescentis*, ↓ *Bifidobacterium longum*, ↓ *Bifidobacterium dentium*, ↓ *Faecalibacterium prausnitzii* (total), ↑ *F prausnitzii* SL3/3-M21/2
Statistical test used	Wilcoxon matched-pairs signed rank test. Mann–Whitney test	ANCOVA, Mann–Whitney, FDR

CD, Crohn's disease; UC, ulcerative colitis; SCFAs, short-chain fatty acids; qPCR, quantitative polymerase chain reaction; GC, gas chromatography; IHMS, international human microbiome standards; NS, not significant; NI, not informed; ANCOVA, analysis of covariance; FDR, false discovery rate

Table 4 Microbiota compositional changes associated with a Low-FODMAP diet in patients with IBD

Bacteria	Change in IBD + Low FODMAP diet	Clinical significance
Bifidobacterium spp.	↓	Reduced immune regulation and epithelial barrier maintenance
Faecalibacterium prausnitzii	↓	Loss of anti-inflammatory butyrate producers; impaired mucosal healing
Akkermansia muciniphila	↓	Weakened mucus layer renewal and compromised mucosal barrier
Clostridium cluster XIVa	↓	Decrease in short-chain fatty acid production and mucosal protection
Ruminococcus torques	↑	Excessive mucin degradation leading to mucus thinning and higher intestinal permeability

Clinical outcomes (if applicable)

Symptom scores (eg, Irritable Bowel Syndrome Symptom Severity Scale [IBS-SSS], Visual Analog Scale [VAS], Inflammatory Bowel Disease Questionnaire [IBD-Q]), quality of life measures, changes in stool consistency or frequency, biomarkers of inflammation (e.g., fecal calprotectin, C-reactive protein [CRP]), and adverse effects.

Risk of bias (RoB)

The Cochrane RoB 2.0 tool [15] was used to assess bias in the retrieved randomized controlled trials (RCTs), with judgments falling in the categories of "low RoB", "some concerns", or "high RoB". Two independent researchers (NC-S and CY) assessed RoB and a third researcher (SAC) intervened when an agreement was not reached.

Data analysis

Data from the included studies were summarized descriptively. Microbiota-related, clinical, inflammatory, and metabolic outcomes were compared qualitatively across studies. When outcomes were reported using both intention-to-treat and per-protocol analyses, these were extracted as reported by the original authors and identified accordingly in the narrative synthesis. Given the small number of eligible studies and substantial heterogeneity in study design, intervention duration, microbiota assessment techniques, and outcome reporting, a quantitative synthesis was not considered appropriate. Therefore, findings were synthesized narratively.

Table 5 Clinical, symptomatic and inflammatory outcomes

Author	Halmos, *et al*	Cox, *et al*
Improvement criteria used	VAS symptom score	IBS-SSS ≥50 reduction; Adequate relief of GI symptoms.
Symptom scores used	VAS score to measure overall gastrointestinal symptoms, abdominal pain, bloating, and passage of wind	IBS-SSS score
Quality of Life changes	Not reported	Reported improvement in HRQoL (IBD-Q)
Inflammation markers evaluated	Fecal calprotectin	Fecal calprotectin and high-sensitivity CRP
Changes in calprotectin or CRP	No differences between diets.	No difference between diets in CD or UC
Stool changes	No differences in fecal frequency, weight, FWC, or whole-gut transit time across dietary periods.	↓ severity of flatulence (GSRS: 0.9 vs. 1.2; P=0.035); ↓ stool frequency (1.7 vs. 2.1/day; P=0.012); ↔ stool consistency (P=0.478)
SCFAs changes	No differences in total or specific fecal SCFAs Molar proportions of the major SCFAs unchanged	No differences between diet groups at end of trial in the ITT population. In the PP population, there were significantly lower concentrations following low FODMAP. ↓ fecal acetate. "↓ Total SCFAs (398 vs 505 mg/100g)" cell (P=0.049)
Duration of intervention	21 days each intervention (Cross-over)	4 weeks
Adherence assessment	High adherence; all completed Australian diet; 8/9 adhered to low FODMAP ≥17 days. The only measured nutrient that differed between the two interventional diets was the average daily intake of FODMAPs	Of the 46 patients completing the trial, 3 were noncompliant, leaving 43 participants (21 low FODMAP diet, 22 sham diet) in the PP analysis.
Adverse effects	No reported	6 adverse events. 2 IBD relapse (1 in each group) and 1 commenced antibiotic unrelated to IBD (low FODMAP). One participant reported a worsening of abdominal pain lasting 2 days that resolved (sham diet). Flu-like symptoms and sinusitis were reported (1 in each group), unrelated to the diet.

GI, Gastrointestinal; VAS, visual analogue scale; IBS-SSS, irritable bowel syndrome severity scoring system; HRQoL, health-related quality of life; CRP, C-reactive protein; FWC, fecal water content; GSRS, gastrointestinal symptom rating scale; SCFAs, short-chain fatty acids; ITT, intention-to-treat; PP, per-protocol; SEM, standard error of the mean; NS, not significant

Results

Study selection

Our literature search strategy (Suppl. Table 2) identified 4,418 articles across four databases: PubMed (n=2,436), Web of Science (n=899), Scopus (n=1,058), and Embase (n=25). After removing 997 duplicates, 3,421 unique articles remained for screening. After screening by title and abstract, we excluded 3,399 articles that did not meet the eligibility criteria. We assessed 22 articles in full text. Of these, 20 were excluded: 9 assessed different outcomes (e.g., dietary interventions without microbiota-related endpoints), 7 were excluded due to publication type (e.g., narrative reviews, letters, and conference abstracts), 2 included an ineligible study design (such as observational or uncontrolled trials) and 2 assessed a different intervention. A complete summary of exclusion reasons is presented in Supplementary Table 3. Ultimately, two randomized controlled trials were included in the qualitative synthesis. The study selection process is illustrated in the PRISMA flow chart (Fig. 1).

Characteristics of the included studies

Although our eligibility criteria included cohort and non-randomized designs, none of these met inclusion criteria at the full-text stage. Ultimately, two RCTs were included in this systematic review (n = 61 participants). Study characteristics are summarized in Table 1. One trial employed a crossover design [16], while the other was a parallel-group RCT [17]. Both were conducted in outpatient settings and included adult patients with quiescent IBD, defined by stable medications, no IBD flare in the previous 6 months, fecal calprotectin < 250 ug and serum CRP <10 mg/L in both, CD and UC. The parallel-group was conducted in the United Kingdom and enrolled 52 participants of whom 46 completed follow-up and 43 were included in the per-protocol analysis. The crossover trial was conducted in Australia and included 9 participants with similar disease characteristics [16,17].

Both trials evaluated the effects of a low FODMAP diet compared to a higher one or habitual diet. In the parallel-group, participants were randomized to either continue their habitual diet or transition to a structured low FODMAP diet over a

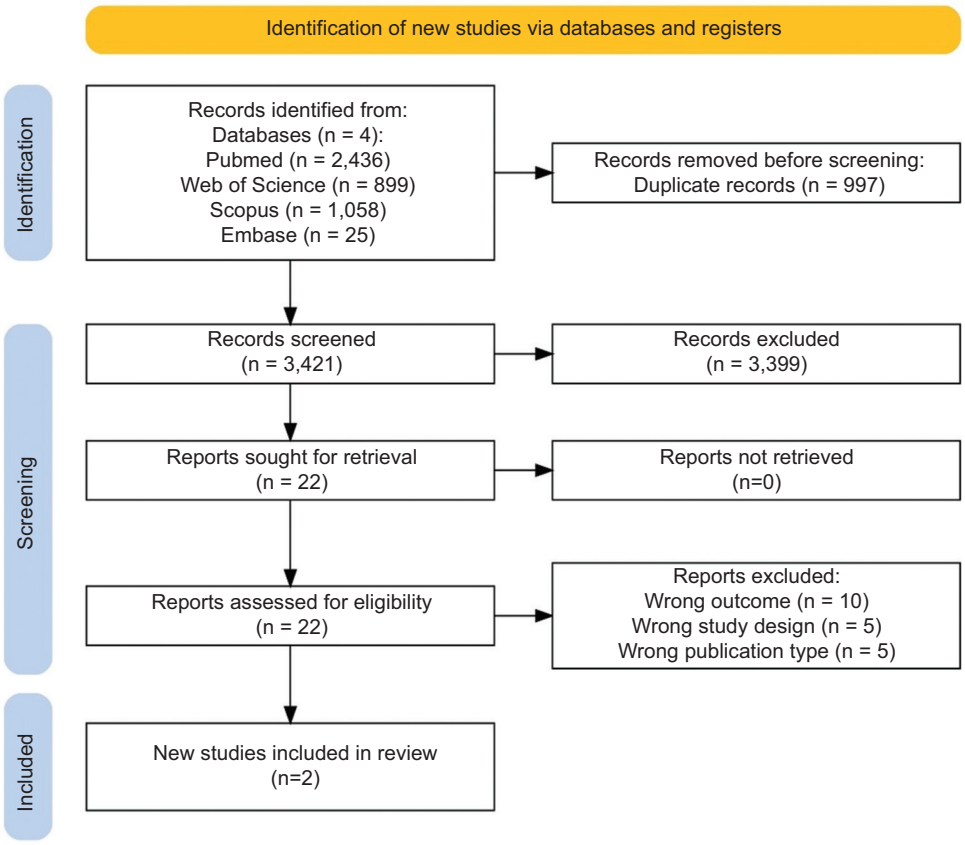


Figure 1 PRISMA flow diagram of study selection

6-week period [17]. In the crossover trial [16] participants underwent a 21-day intervention comparing a low FODMAP diet to a typical Australian diet matched for macronutrient content.

The primary objectives of both studies were to assess changes in gastrointestinal symptoms, gut microbial composition (both absolute and relative abundance), inflammatory biomarkers, and stool characteristics. Both trials included patients in clinical remission as defined by standard disease activity scores. Exclusion criteria included recent antibiotic use, corticosteroid therapy, or presence of concurrent infections.

Additional details, including recruitment period, location, patients' characteristics, target outcomes, inclusion and exclusion criteria, are presented in Table 1.

Intervention characteristics

The crossover trial [16] and the parallel-group study [17] evaluated a low FODMAP diet against different comparators (Table 2). The crossover trial employed a typical Australian diet matched for energy, macronutrients, sugars, starch, and fiber, whereas the parallel-group study [17] used a sham dietary intervention designed to mirror the intensity and attention of low FODMAP diet counseling [16,17]. The duration of the interventions varied between 21 days [16] and 4 weeks [17]. In both studies, the low FODMAP diet was defined and quantified using the Monash University FODMAP composition database; additionally, the crossover trial used high-performance liquid chromatography and enzymatic assays to validate FODMAP content [16]. Adherence in the crossover trial was assessed through detailed food diaries and considered acceptable if participants completed at least 17 of 21 days of the prescribed meals. In the parallel-group study [17], adherence was evaluated using a self-reported question at the end of the trial and based on a 5-point Likert scale ranging from "never/rarely" to "always" following the diet [17]. Only the crossover trial [16] adjusted the fiber content of the low FODMAP diet by adding supplemental psyllium and resistant starch to match the control diet.

Microbiota-related outcomes

Both studies assessed microbiota-related outcomes following the low FODMAP intervention (Table 3), including bacterial abundance, SCFAs levels, and diversity measures. Sample storage conditions and analysis techniques were discussed on Table 2. The crossover trial [16] quantified targeted bacterial groups using qPCR, while the parallel-group performed shotgun metagenomic sequencing [17]. Neither study reported changes in total bacterial load or alpha/beta diversity.

In the crossover trial [16], the low FODMAP diet resulted in a significant reduction in both absolute and relative abundance of *Clostridium cluster XIVa* (absolute: 7.94 vs. 8.73 $\log_{10}$ copies/g; relative: 2.81% vs. 19.2%; both p = 0.008) and *Akkermansia muciniphila* (absolute: 3.75 vs. 5.08 $\log_{10}$ copies/g; relative: 0.01% vs. 0.15%; both P=0.016), compared to the typical Australian diet. *Ruminococcus torques* showed increased relative abundance during the low FODMAP diet (0.16% vs. 0.10%; P=0.039), while other taxa—including *F. prausnitzii*, *Clostridium cluster IV*, *Roseburia spp.*, *Bifidobacteria spp.*, and *Lactobacilli*—did not show significant changes.

The parallel-group study [17] reported a reduction in several *Bifidobacterium* species following the low FODMAP diet, including *B. adolescentis*, *B. longum*, and *B. dentium* (P<0.05). Shifts in *F. prausnitzii* were strain-specific: some variants (e.g., SL3/3, M21/2) increased, while others (e.g., L2/6) decreased. No changes were observed in global diversity metrics or total bacterial load.

Overall, the main gut microbiota compositional changes reported following the low FODMAP intervention are summarized in Table 4.

Clinical, symptomatic, and inflammatory outcomes

Gastrointestinal symptoms were evaluated using validated scoring tools: a VAS for overall discomfort, abdominal pain, bloating, and flatulence; and the IBS-SSS, with clinical response defined as a ≥50-point reduction. Symptom scores were consistently lower during the low FODMAP phase, with reductions in bloating, abdominal pain, and flatulence. Flatulence severity decreased (GSRS score: 0.9 vs. 1.2; P=0.035), and stool frequency was lower in the low FODMAP group (1.7 vs. 2.1/day; P=0.012), while stool consistency remained unchanged (P=0.478).

Health-related quality of life, assessed using the IBD-Q, improved following the intervention. No changes were observed in inflammatory markers; fecal calprotectin and high-sensitivity CRP remained stable across dietary arms. Similarly, no significant differences were found in fecal SCFAs levels in the intention-to-treat analysis. However, per-protocol analysis revealed lower total SCFAs concentrations and reduced fecal acetate in the low FODMAP group (398 vs. 505 mg/100g, P=0.049).

A summary of symptomatic, inflammatory, and metabolic outcomes is presented in Table 5.

Risk of bias

The RoB assessment of the included RCTs showed that the parallel-group study [17] was judged to have a low risk across all evaluated domains. In contrast, the crossover trial [16] presented some concerns, particularly related to the randomization process and overall bias. Additional concerns were noted in the crossover trial [16] regarding missing outcome data. A detailed breakdown of the RoB for each study is presented in Supplementary Table 4.

Discussion

This systematic review examined the impact of low FODMAP dietary interventions on gut microbiota composition in patients with IBD. The two included randomized controlled trials, both performed in patients with inactive or mildly active IBD, reported symptom improvement following dietary restriction, especially in bloating, abdominal pain, and stool form [16,17]. These findings were similar to the ones described by Ville et all where they also found no significant difference between low FODMAP diet and controls at the end of intervention for faecal calprotectin [18,19]. Its clinical effect is thought to result from reduced colonic fermentation and luminal osmotic load through restriction of poorly absorbed short-chain carbohydrates [20,21].

However, changes in microbial composition varied between studies, likely reflecting important methodological differences, including study design (cross-over versus parallel), duration of intervention, dietary control, microbiota assessment techniques (qPCR versus metagenomic sequencing), and reporting of SCFAs outcomes. Shifts were primarily observed in bacterial taxa with immunoregulatory and mucosal protective properties, such as *Faecalibacterium prausnitzii*, *Akkermansia muciniphila*, *Bifidobacterium* spp., and *Clostridium* cluster XIVa [16,17]. Similar findings were reported by Grammatikopoulou *et al* [19] suggesting that the low FODMAP diet, while effective in symptom control, could potentially reduce beneficial commensals, with uncertain long-term consequences for mucosal immunity and inflammation control in IBD [21-24].

Faecalibacterium prausnitzii, a major butyrate-producing bacterium with recognized anti-inflammatory properties, was significantly reduced after a low FODMAP diet in the parallel-group study [17]. This aligns with previous studies reporting decreased abundance of *F. prausnitzii* in patients with CD, particularly those with active inflammation or post-resection recurrence [25-27]. The clinical relevance is though to lie in its ability to secrete metabolites such as butyrate and microbial anti-inflammatory molecules (MAM) that modulate intestinal inflammation, improve barrier integrity, and promote T reg cell expansion elements that play a significant role in patients with IBD [28,29].

On the other hand *Akkermansia muciniphila*, a mucin-degrading bacterium involved in maintaining the gut barrier and metabolic regulation, was reported to be reduced in the crossover trial [16]. While this bacterium is often associated with metabolic health, its role in IBD remains complex and context-dependent. Several studies have reported reduced abundance of *A. muciniphila* in CD patients, particularly those with mucosal disruption, supporting its function in mucus layer integrity [30,31]. Previous studies suggested than can reduce the level of colon infiltrating macrophages and cytotoxic T lymphocytes and downregulate pro-inflammatory cytokines

such as tumor necrosis factor alpha, interferon gamma, interleukin 1 beta, and interleukin 6 to alleviate colitis [31,32]. In contrast, *Ruminococcus torques* exhibited a significant increase following the low FODMAP intervention [16]. This species has been implicated in the erosion of the intestinal mucus barrier due to its high mucinase activity, a phenomenon associated with increased epithelial permeability and translocation of luminal antigens in patients with CD [30]. Several studies have consistently reported enrichment of *R. torques* in the ileal and colonic mucosa of individuals with active IBD, linking it to disruption of barrier integrity and promotion of proinflammatory responses [33,34]. Therefore, although symptom improvement is frequently observed with low FODMAP diet, the consequent changes observed on the mucin-related bacteria could represent a trade-off with potential implications for long-term intestinal health in IBD patients, although the long-term implications remain uncertain [35,36]. Importantly, these microbial shifts were not accompanied by worsening of inflammatory biomarkers or clinical disease activity in the included trials, and therefore no direct adverse clinical consequences can be inferred from the available evidence.

Bifidobacterium spp., a genus of saccharolytic, SCFAs-producing bacteria widely regarded as beneficial, showed a statistically significant decrease in both included trials following a low FODMAP intervention [16,17]. These microbes are integral in the fermentation of oligosaccharides such as galacto-oligosaccharides and fructo-oligosaccharides —substrates notably restricted in low FODMAP diet — and contribute to immune regulation, epithelial integrity, and competitive exclusion of pathogens [37,38]. Clinical studies have reported reduced abundance of *Bifidobacterium* in IBD patients, particularly in active disease, where its decline correlates with increased mucosal permeability and inflammation [39]. Although the reduction observed with low FODMAP diet may be transient, and symptom relief is evident in IBS and IBD populations, the suppression of *Bifidobacterium* could potentially have unintended consequences on mucosal immune homeostasis. To address these concerns, strategies such as the reintroduction phase, dietary personalization, and the incorporation of *Bifidobacterium*-enriched probiotics have been proposed to help restore microbial diversity and promote gut health while maintaining symptom relief [40,41].

Finally, *Clostridium* cluster XIVa, a group encompassing several butyrate-producing and regulatory T-cell-inducing species, was significantly reduced following the low FODMAP intervention in the crossover trial [16]. This cluster plays a key role in gut immune homeostasis by promoting the differentiation of regulatory T cells (Tregs) via SCFAs-mediated epigenetic modulation [42]. In IBD, decreased representation of *Clostridium* XIVa has been consistently associated with active mucosal inflammation and reduced T reg density, suggesting a protective function [43,44]. Its depletion under dietary restriction may have theoretical implications for mucosal tolerance mechanisms. Interestingly, reductions in *Clostridium* spp. were also seen in patients with IBS following low FODMAP diet, though without accompanying inflammatory burden, suggests differential implications across diseases [45]. While the symptom relief observed in IBD patients may be partially independent of these microbial shifts, the suppression of butyrogenic and immunoregulatory taxa underscores the need to consider microbial preservation in long-term dietary management strategies.

Among the strengths of this review is its exclusive focus on randomized controlled trials with microbiota endpoints, which allowed for a clinically meaningful synthesis grounded in mechanistic context. However, important limitations must be acknowledged. Only two small randomized controlled trials were included, both of short duration. Therefore, the overall confidence in the available evidence was considered limited due to sample size and methodological heterogeneity, and findings should be considered hypothesis-generating rather than confirmatory. In addition, differences in study design, intervention duration, dietary control, microbiota assessment techniques, and outcome reporting limit direct comparability and synthesis. Furthermore, one of the included trials was judged to have some concerns regarding RoB, particularly related to the randomization process and missing outcome data, further reducing confidence in the overall evidence base. No firm conclusions can be drawn regarding the sustained impact of a low FODMAP diet on microbiota composition or disease course in IBD.

Overall, the low FODMAP diet may be recommended as a short-term strategy for symptom control in patients with quiescent IBD who experience persistent functional gastrointestinal symptoms. However, current evidence does not support its use as a disease-modifying therapy, as no improvement in inflammatory biomarkers, endoscopic findings, histological parameters, or relapse rates has been demonstrated in the available trials. While alterations in specific microbial taxa were observed, these changes should not be interpreted as evidence of adverse clinical consequences in the absence of objective markers of disease worsening. Therefore, it should be implemented in a structured and individualized manner, ideally under dietitian supervision, and followed by a reintroduction phase to minimize unnecessary long-term dietary restriction. These findings are limited to adults with inactive or mildly active IBD and should not be extrapolated to patients with active disease, pediatric populations, or long-term dietary implementation without appropriate supervision. The low FODMAP diet should be considered an adjunct to standard medical therapy rather than a substitute for it [46,47].

Conclusion

In this systematic review of randomized controlled trials, low FODMAP dietary interventions were associated with symptomatic improvement in gastrointestinal manifestations in patients with inactive IBD. These interventions were also accompanied by changes in specific microbial taxa, including reductions in *Bifidobacterium* spp., *Faecalibacterium prausnitzii, Akkermansia muciniphila,* and *Clostridium cluster XIVa, as well as an increase in Ruminococcus torques*. Although these taxa are linked to pathways related to mucosal integrity, immune

modulation, and barrier function, the clinical significance of these microbial shifts remains uncertain. Given the limited number of short-term trials and the overall low certainty of evidence, these findings should be considered hypothesis-generating. Future studies should prioritize longitudinal designs, incorporate mucosal and immunologic endpoints, and explore strategies to optimize symptom control while preserving microbial balance during dietary interventions.

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Supplementary material

Supplementary Table 1 Prisma 2020 Checklist

Section and Topic	Item #	Checklist item	Location where item is reported
		TITLE	
Title	1	Identify the report as a systematic review.	Title
		ABSTRACT	
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Abstract
		INTRODUCTION	
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Introduction
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Introduction
		METHODS	
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Methods- Selection criteria
Information sources	6	Specify all databases, registers, websites, organizations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Methods- Search strategy
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Supplemmentary Table 2
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Methods- search strategy
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Methods- Data cextraction
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Methods-Data extraction-
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Methods-Data extraction
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Methods- Risk of bias
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Methods- data analysis
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Methods – selection criteria
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Not applicable (no meta-analysis conducted)
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Results- Table 1-5 and supplementary tables
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Methods-Data analysis
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Not applicable (no heterogeneity analysis performed)

(*Contd...*)

Supplementary Table 1 (*Continued*)

Section and Topic	Item #	Checklist item	Location where item is reported
		METHODS	
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Not applicable (no sensitivity analysis performed)
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Methods-data analysis
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Not assessed due to limited number of studies
		RESULTS	
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Results-Study selection, Figure 1
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Results, Study selection
Study characteristics	17	Cite each included study and present its characteristics.	Results-Characteristics of the included studies
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Results-Risk of bias
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Results-Characteristics of included studies and Tables 1-5
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Results- Risk of bias
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Not applicable
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Not applicable (not heterogeneity analysis performed)
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	Not applicable (no sensitivity analysis performed)
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Not assessed due to limited number of studies
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Discussion
		DISCUSSION	
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Discussion
	23b	Discuss any limitations of the evidence included in the review.	Discussion
	23c	Discuss any limitations of the review processes used.	Discussion
	23d	Discuss implications of the results for practice, policy, and future research.	Discussion-Conclusions

(*Contd...*)

Supplementary Table 1 (*Continued*)

Section and Topic	Item #	Checklist item	Location where item is reported
		OTHER INFORMATION	
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	Methods
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	Methods
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	Methods
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Funding
Competing interests	26	Declare any competing interests of review authors.	Conflicts of interest
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	Data extraction

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, *et al.* The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71. This work is licensed under CC BY 4.0. To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/

Supplementary Table 2 Search strategy

PUBMED	("Inflammatory Bowel Diseases"[Mesh] OR "Colitis, Ulcerative"[Mesh] OR "Crohn Disease"[Mesh] OR "inflammatory bowel disease*"[tw] OR "IBD"[tw] OR "Crohn*"[tw] OR "Crohn's disease*"[tw] OR "Crohn disease*"[tw] OR "ulcerative colitis"[tw] OR "colitis*"[tw]) OR "UC"[tw] OR "chronic inflammatory bowel disease"[tw] OR "colitis ulcerosa"[tw] OR "enteritis regionalis"[tw]) AND ("Polysaccharides"[Mesh] OR "Oligosaccharides"[Mesh] OR "Disaccharides"[Mesh] OR "Monosaccharides"[Mesh] OR "Trisaccharides"[Mesh] OR "low FODMAP*"[tw] OR "low-FODMAP*"[tw] OR FODMAP*[tw]) AND ("Gastrointestinal Microbiome"[MeSH] OR "Microbiota"[MeSH] OR microbiota*[tw] OR microbiome*[tw] OR "bacterial diversity"[tw] OR "microbial diversity"[tw] OR "microbial composition"[tw] OR "16S rRNA"[tw] OR "bacterial community"[tw] OR "fecal microbiota"[tw] OR dysbiosis[tw]).	2424
SCOPUS	(INDEXTERMS("Inflammatory Bowel Diseases") OR INDEXTERMS("Colitis, Ulcerative") OR INDEXTERMS("Crohn Disease") OR TITLE-ABS-KEY("inflammatory bowel disease*") OR TITLE-ABS-KEY(IBD) OR TITLE-ABS-KEY(Crohn*) OR TITLE-ABS-KEY("Crohn's disease*") OR TITLE-ABS-KEY("Crohn disease*") OR TITLE-ABS-KEY("ulcerative colitis") OR TITLE-ABS-KEY(colitis*)) OR TITLE-ABS-KEY(UC) OR TITLE-ABS-KEY("chronic inflammatory bowel disease") OR TITLE-ABS-KEY("colitis ulcerosa") OR TITLE-ABS-KEY("enteritis regionalis") AND (INDEXTERMS(Polysaccharides) OR INDEXTERMS(Oligosaccharides) OR INDEXTERMS(Disaccharides) OR INDEXTERMS(Monosaccharides) OR INDEXTERMS(Trisaccharides) OR TITLE-ABS-KEY("low FODMAP*") OR TITLE-ABS-KEY(low-FODMAP*) OR TITLE-ABS-KEY(FODMAP*)) AND (INDEXTERMS("Gastrointestinal Microbiome") OR INDEXTERMS(Microbiota) OR TITLE-ABS-KEY(microbiota*) OR TITLE-ABS-KEY(microbiome*) OR TITLE-ABS-KEY("bacterial diversity") OR TITLE-ABS-KEY("microbial diversity") OR TITLE-ABS-KEY("microbial composition") OR TITLE-ABS-KEY("16S rRNA") OR TITLE-ABS-KEY("bacterial community") OR TITLE-ABS-KEY("fecal microbiota") OR TITLE-ABS-KEY(dysbiosis)) .	1058
WOS	(TS=("Inflammatory Bowel Diseases" OR "Colitis, Ulcerative" OR "Crohn Disease" OR "inflammatory bowel disease*" OR "IBD" OR "Crohn*" OR "Crohn's disease*" OR "Crohn disease*" OR "ulcerative colitis" OR "colitis*" OR "UC" OR "chronic inflammatory bowel disease" OR "colitis ulcerosa" OR "enteritis regionalis")) AND (TS=("Polysaccharides" OR "Oligosaccharides" OR "Disaccharides" OR "Monosaccharides" OR "Trisaccharides" OR "low FODMAP*" OR "low-FODMAP*" OR FODMAP*)) AND (TS=("Gastrointestinal Microbiome" OR "Microbiota" OR microbiota* OR microbiome* OR "bacterial diversity" OR "microbial diversity" OR "microbial composition" OR "16S rRNA" OR "bacterial community" OR "fecal microbiota" OR dysbiosis))	899
EMBASE	('Inflammatory Bowel Disease'/exp OR 'Ulcerative colitis'/exp OR 'Crohn Disease'/exp OR 'inflammatory bowel disease*':ti,ab,kw OR IBD:ti,ab,kw OR Crohn*:ti,ab,kw OR 'Crohn disease*':ti,ab,kw OR 'ulcerative colitis':ti,ab,kw OR colitis*:ti,ab,kw OR UC:ti,ab,kw OR 'chronic inflammatory bowel disease':ti,ab,kw OR 'colitis ulcerosa':ti,ab,kw OR 'enteritis regionalis':ti,ab,kw) AND ('low FODMAP diet'/exp OR Polysaccharides:ti,ab,kw OR Oligosaccharides:ti,ab,kw OR Disaccharides:ti,ab,kw OR Monosaccharides:ti,ab,kw OR Trisaccharides:ti,ab,kw OR 'low FODMAP*':ti,ab,kw OR low FODMAP*:ti,ab,kw OR FODMAP*:ti,ab,kw) AND ('Intestine flora'/exp OR 'colon flora'/exp OR 'Gastrointestinal Microbiome':ti,ab,kw OR microbiota*:ti,ab,kw OR microbiome*:ti,ab,kw OR 'bacterial diversity':ti,ab,kw OR 'microbial diversity':ti,ab,kw OR 'microbial composition':ti,ab,kw OR '16S rRNA':ti,ab,kw OR 'bacterial community':ti,ab,kw OR 'fecal microbiota':ti,ab,kw OR dysbiosis:ti,ab,kw)	25

Supplementary Table 3 Papers excluded and reason for exclusion

Title of the paper	First author	Reason	Explanation
Associations between adherence to literature-derived dietary indices and pre-disease biomarkers: implications for Crohn's disease prevention	A. Neustaeter	Wrong study design	This study was on healthy FDR subjects of the Crohn's Colitis Canada- Genes, Environment, Microbial (CCC-GEM) project.
Gut microbiota composition and functional changes in inflammatory bowel disease and irritable bowel syndrome	Arnau Vich Vila	Wrong study design	This study describes the features of the gut microbiota of patients with IBD or IBS and compares them to healthy population.
Effectiveness of a novel diet in attenuation of clinical activity of disease in patients with ulcerative colitis: a randomized, clinical trial	Behnaz Narimani	Wrong outcome	The primary outcome was a significant reduction in SCCAIQ score.
Gut Microbiota in IBD Patients with IBS Before and After 6 Weeks of Low FODMAP Diet	Natalia Pedersen	Wrong publication type	Abstract
Dietary manipulation of gut microbiome in inflammatory bowel disease patients: Pilot study	Barbara Olendzki	Wrong intervention	This study uses anti-inflammatory diet not low FODMAP.
Why a Low FODMAP Diet Was Ineffective for IBS Symptoms in Quiescent Crohn's Disease?	Yoshiharu Uno	Wrong publication type	Letter
A low FODMAP diet in patients with Crohn's disease	Emma Halmos	Wrong publication type	Supplementary article
Use of the low-FODMAP diet in inflammatory bowel disease	Peter Gibson	Wrong publication type	Review
Randomized, double-blind, placebo-controlled trial of fructo-oligosaccharides in active Crohn's disease	Kentaro Eijiri	Wrong intervention	The study uses a fructo-oligosaccharide diet.
Low FODMAP diet improves functional- like gastrointestinal symptoms but reduces bifidobacteria and faecalibacterium prausnitzii in Quiescent inflammatory bowel disease: a randomized controlled trial and metagenomic analysis	S. Cox	Wrong publication type	Abstract
Symptom response following advice on a diet low in short-chain fermentable carbohydrates (FODMAPS) for functional bowel symptoms in patients with IBD.	T Joyce	Wrong publication type	Abstract
A randomized, 6-wk trial of a low FODMAP diet in patients with inflammatory bowel disease	Giorgia Bodini	Wrong outcome	The primary outcome was the effect and safety of an low FODMAP diet by assessing clinical and biochemical disease activity, as well as QoL.
Beyond Irritable Bowel Syndrome: The Efficacy of the Low FODMAP Diet for Improving Symptoms in IBD and Celiac Disease.	Anna Testa	Wrong outcome	The outcome was the clinical impact of a low FODMAP diet in patients with IBS, IBD in remission, and CD on a GFD.
Low-FODMAP diet reduces irritable bowel symptoms in patients with inflammatory bowel disease	Natalia Pedersen	Wrong outcome	The outcomes are how low FODMAP diet would alter IBS like symptoms and improve the quality of life among IBD patients.
Fermentable carbohydrates (FODMAPs) exacerbate functional gastrointestinal symptoms in patients with inflammatory bowel disease: a randomized, double-blind, placebo-controlled, cross-over, re-challenge trial.	Selina Cox	Wrong outcome	The outcome was whether individual fermentable carbohydrates exacerbate functional-like GI symptoms in patients with quiescent IBD who experience FGS.
Follow-up of patients with functional bowel symptoms treated with a low FODMAP diet	Louise Maagaard	Wrong outcome	The outcomes were long-term effects of the low FODMAP diet, dietary adherence, and dietary impact on disease courses in patients with IBS and patients with IBD and coexisting IBS.
Fermentable Carbohydrate Restriction (Low FODMAP Diet) in Clinical Practice Improves Functional Gastrointestinal Symptoms in Patients with Inflammatory Bowel Disease.	Alexis Prince	Wrong outcome	The primary outcome measure was assessment of satisfactory relief of FGS measured using the global symptom question in patients with IBD and coexisting FGS.

(*Contd...*)

Supplementary Table 3 (*Continued*)

Title of the paper	First author	Reason	Explanation
EHealth: Self-management in Inflammatory Bowel Disease and in IBD using novel Constant-Care Web Applications.	Natalia Pedersen	Wrong outcome	The primary aim was evaluate the efficacy and feasibility of novel Constant-care approaches in patients with IBD and IBS to individualize their disease management
Reduction of dietary poorly absorbed short chain carbohydrates (FODMAPs) improves abdominal symptoms in patients with inflammatory bowel disease—a pilot study	Richard Gearry	Wrong outcome	The primary outcome is determining the effect of their dietary restriction on abdominal symptoms in patients with stable IBD.
A randomized controlled trial investigating the effect of a diet low in fermentable oligosaccharides, disaccharides, monosaccharides, and polyols on the intestinal microbiome and inflammation in patients with ulcerative colitis: Study protocol for a randomized controlled trial.	Alireza Milajerdi	Wrong publication type	Study protocol

Supplementary Table 4 Risk of bias using Cochrane risk-of-bias tool for randomized trials

