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Efficacy of the Autoimmune Protocol Diet for Inflammatory Bowel Disease

Gauree G. Konijeti, MD, MPH,*,† NaMee Kim, MD,‡ James D. Lewis, MD, MSCE,§ Shauna Groven, BS,∥
Anita Chandrasekaran, MD, MPH,* Sirisha Grandhe, MD,* Caroline Diamant, MD,* Emily Singh, MD,*
Glenn Oliveira, BS,†,¶ Xiaoyun Wang, MS,† Bhuvan Molparia, MS,¶,† and Ali Torkamani, PhD¶,†

Introduction: Data suggest dietary modification can improve clinical responses in inflammatory bowel disease (IBD). The goal of this study was to determine the efficacy of an autoimmune protocol diet in patients with Crohn's disease and ulcerative colitis.

Methods: We enrolled adults with active IBD (Harvey–Bradshaw index ≥ 5 or partial Mayo score ≥3 and erosions on endoscopy and/or elevated fecal calprotectin). For the autoimmune protocol, patients underwent 6-week elimination followed by 5-week maintenance phase. Clinical indices, laboratories, and biomarkers were assessed at baseline and weeks 6 and 11. Endoscopy was performed at study completion.

Results: The final cohort included 15 patients with IBD, with mean disease duration 19 years (SD 14.6) and active biological use in 7 (47%) patients. Nutrient repletion was initiated for deficiencies in vitamin D (n = 3) and iron (n = 6). From week 0 to weeks 6 and 11, mean partial Mayo score significantly improved from 5.8 (SD 1.2) to 1.2 (SD 2.0) and 1.0 (SD 2.0) for ulcerative colitis, and mean Harvey–Bradshaw index significantly improved from 7 (SD 1.5) to 3.6 (SD 2.1) and 3.4 (SD 2.6) for Crohn's disease. C-reactive protein did not significantly change during study. Mean fecal calprotectin improved from 471 (SD 562) to 112 (SD 104) at week 11 ($P = 0.12$). Among those with follow-up endoscopy at week 11 (n = 7), improvements were noted in simple endoscopic score for Crohn's disease (n = 1), Rutgeerts score (n = 1), and Mayo endoscopy subscore (n = 4).

Discussion: Dietary elimination can improve symptoms and endoscopic inflammation in patients with IBD. Randomized controlled trials are warranted.

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Key Words: ulcerative colitis, Crohn's disease, diet, autoimmune protocol

Inflammatory bowel diseases (IBDs), which include both Crohn's disease (CD) and ulcerative colitis (UC), are complex gastrointestinal disorders that arise in a genetically susceptible host due to factors involving T-cell dysregulation, gut dysbiosis, environmental exposures, and dietary factors. More than 200 genes have been identified that may increase the risk of IBD, but account for only a modest proportion of the disease variance (~13% for CD, ~7% for UC).[1,2] Therefore, IBD is considered a complex polygenic disease that is strongly influenced by environmental factors. Among these, diet and the gut microbiome, 2 related environmental factors, are hypothesized to be particularly important risk factors and can be modified to influence disease risk and course.[3]

Despite diet being implicated in the pathogenesis of IBD,[4] we have limited data to guide the use of nutritional therapy as either primary or adjunctive treatment for these conditions. Conventional medical therapy for IBD focuses on suppression of the immune system by targeting a variety of pathways, yet response rates continue to remain suboptimal. Therefore, there is an important need to study dietary factors that may not only help improve response to conventional treatment but also potentially be used as primary therapy or maintenance therapy for patients with IBD. A Western diet, high in refined carbohydrates, omega-6 fatty acids, saturated fat, low in fiber, vitamins, and generally nutrient dense foods, are associated with an increased risk of IBD.[4] Recent albeit limited data

suggest that a semivegetarian diet[5] (allowing milk and eggs, fish once per week, and other meat once every 2 weeks), specific carbohydrate diet[6–8] (removal of all grains, most dairy products, and sweeteners except for honey), or anti-inflammatory diet[9] (modified carbohydrate and fatty acid intake, and increased prebiotic/probiotic ingestion) can be associated with improved rates of achieving or maintaining clinical response.

The autoimmune protocol (AIP) diet is an extension of the Paleolithic diet[10] and incorporates some of the dietary changes previously studied in IBD, including avoidance of gluten and refined sugar. The AIP diet focuses on an initial elimination phase of food groups including grains, legumes, nightshades, dairy, eggs, coffee, alcohol, nuts and seeds, refined/processed sugars, oils, and food additives.[10,11] The rationale is to avoid foods, additives, or medications (e.g., nonsteroidal anti-inflammatory drugs) that can trigger intestinal inflammation, dysbiosis, and/or symptomatic food intolerance.[10,12–14] It also emphasizes consumption and preparation of fresh, nutrient dense foods, bone broth, and fermented foods, while addressing factors that are known to associate with disability due to IBD, such as sleep and sleep hygiene, stress management, forming a support system, and physical activity.[15] The elimination phase is followed by a maintenance phase, the duration of which can vary by individual, until they achieve a measurable improvement in their symptoms and overall well-being. Staged reintroduction of food groups is then initiated gradually, as patients identify unique foods or food groups that may contribute to symptoms while liberalizing their diet.[10,11]

Based on increasing evidence suggesting an impact of diet on clinical disease activity and IBD, and our clinical experience with patients pursuing the AIP diet for their symptomatic IBD, we performed a prospective study to evaluate the potential efficacy of the autoimmune protocol (AIP) diet in patients with active CD and UC.

METHODS

We conducted a single-center, open-labeled uncontrolled study designed to determine potential efficacy of an AIP diet in patients with active CD and UC. Eligible patients were enrolled through the Scripps Clinic Medical Group Division of Gastroenterology (La Jolla, CA). Study participants provided written informed consent under a protocol approved by the Institutional Review Board of Scripps. Eligible patients included adults ages 18 and over with either symptomatic CD (defined as Harvey–Bradshaw index [HBI] ≥ 5) or UC (partial Mayo clinic score ≥ 3). Objective evidence of active disease was also required for study inclusion either by endoscopy (colonoscopy or flexible sigmoidoscopy) or imaging (video capsule endoscopy or enterography) within 7 months of enrollment, or elevated calprotectin (>50 μg/g) within 1 month of enrollment. Additional inclusion criteria included an established Facebook account and being comfortable with the use of internet-based surveys and email. Exclusion criteria included pregnant or breast-feeding women, patients with known celiac disease or history of positive TTG antibody, evidence of untreated infection (e.g., Clostridium difficile), presence of stoma or J pouch, bowel surgery

within 12 weeks before enrollment or deemed likely during study period, use of tube or enteral feeding, elemental diet, or parenteral alimentation within 4 weeks of study initiation. Dosage of medications being used to treat IBD before enrollment was advised to remain stable during the study period with the exception of tapering of corticosteroids. As a pilot study examining efficacy of the AIP diet in active IBD, no minimum duration of medication use was required before study enrollment.

Dietary Intervention

The AIP dietary intervention consisted of a 6-week elimination phase (staged elimination of grains, legumes, nightshades, dairy, eggs, coffee, alcohol, nuts and seeds, refined/processed sugars, oils, and food additives) followed by a 5-week maintenance phase (during which no food group reintroduction was allowed), using the "SAD to AIP in 6" diet transition program.[16] All participants began the study on September 5, 2016, and completed the study on November 18, 2016. As an initial pilot study of the AIP diet for active IBD, we did not formally examine reintroduction of food groups after the maintenance phase. The study incorporated a certified health coach, who conducted the dietary intervention, and a registered dietician to provide one-to-one feedback to participants. The program also counseled participants on forming a support system, grocery shopping and food preparation, sleep and sleep hygiene, education regarding nutrient density and fermented foods, stress management, incorporation of bone broth and physical activity, and avoidance of nonsteroidal anti-inflammatory drugs. Health and group-based coaching and dietary counseling were provided through individual email and a private Facebook group accessible by invited members only. Because participants began the study at the same time, they could communicate with one another through the Facebook group, but study investigators and staff were not part of this group. During the study, participants were asked to record dietary intake, which was communicated through email and reviewed by the health coach and dietician. Participants also completed quality of life assessment during the study using the Short Inflammatory Bowel Disease Questionnaire (SIBDQ).

Before the study, participants received 2 books on AIP diet and recipes: "The Paleo Approach: Reverse Autoimmune Disease and Heal Your Body" by Ballantyne, published 2014 and "The Autoimmune Paleo Cookbook: An Allergen-Free Approach to Managing Chronic Illness" by Trescott, published 2014.

Office visits (at baseline before study start, and end of study) and laboratories (baseline, week 6, and week 11) were conducted at Scripps Clinic. Endoscopy, radiology, and/or biomarker assessment were performed at baseline and at study completion to assess for mucosal healing. At the end of the study, participants were provided guidance on any continuation of maintenance phase and staged reintroduction of food groups.

Outcome Measures

The goal of this study was to evaluate the potential efficacy of the AIP diet on for patients with active CD and UC. The

primary outcome was to examine the proportion of patients achieving clinical remission for CD and UC at study completion (11 weeks). Clinical remission was defined as HBI < 5 for CD and partial Mayo score (sum of individual scores for stool frequency, rectal bleeding, and physician global assessment) ≤ 2 for UC.[17] Secondary measures included achievement of clinical outcome measures at week 6 (end of elimination phase), changes in biomarkers and endoscopic disease activity from baseline to weeks 6 and 11, changes in steroid use (among those with active use at baseline), and examination of any adverse events during the follow-up period. Analyses of clinical outcomes were performed for those patients with follow-up data at both weeks 6 and 11, whereas analyses of biomarker (C-reactive protein [CRP] or fecal calprotectin [FC]) were performed for those with follow-up data at weeks 6 and/or 11. Only 1 patient with CD was lost to follow-up during the study before assessments were made at week 6 or 11. Secondary analyses of clinical outcomes carrying forward this patient's baseline data were conducted to minimize bias due to loss of follow-up (see "Results").

Statistical Analysis

Analyses were conducted for participants with CD or UC and for the entire cohort. Paired *t* test was used to examine differences between baseline and week 6 or 11 outcomes among those with follow-up data at those time points. Fisher's exact test was used to examine whether IBD type was associated with the primary outcome.

RESULTS

We enrolled 18 adult patients. Three patients withdrew before study start because of inability to commit to dietary change. The final cohort included 9 participants with CD and 6 with UC (Table 1). Among the 9 patients with CD, 5 (56%) had postoperative recurrence of CD (3 ileal and 2 ileocolonic). Before study start, 8 of the 9 participants with CD had active disease on endoscopic evaluation (with visible erosions or ulcers in diseased regions), 5 of whom had elevated FC (>50 µg/g). One participant with Crohn's enrolled in the setting of only an elevated FC (79 µg/g), with colonoscopy within 1 month of study showing no active erosion or ulceration. At baseline, CRP was elevated (>10 mg/L) in 33% participants (5/15), with mean CRP 7.3 (SD 10.7) and mean FC was 392 µg/g (SD 448 µg/g) (Table 1). All 6 participants with UC had active disease on baseline endoscopy, with Mayo endoscopic subscores (MESs) of 1 ($n = 3$), 2 ($n = 2$), and 3 ($n = 1$). Five of the 6 patients with UC had elevated FC at baseline (mean 447 µg/g, SD 426 µg/g).

Mean IBD duration was 19 years (SD 14.6) and active biological use in 7 participants (Table 1). Types of therapy among those with active biological use ($n = 7$) included infliximab monotherapy ($n = 2$), adalimumab monotherapy ($n = 2$), vedolizumab monotherapy ($n = 1$), and vedolizumab combination therapy with thiopurine ($n = 1$) or methotrexate ($n = 1$). Mean duration of biological use before study was 1.8 years (SD 2.5). Two patients started infliximab or vedolizumab within 3 months of study start; 1 patient with severe UC initiated infliximab monotherapy 20 days before study start and 1 patient with ileocolonic CD with postoperative recurrence and anastomotic stricture on oral methotrexate initiated vedolizumab therapy (in addition to methotrexate) 75 days before study start. Excluding these 2 patients, mean duration of biological use before study start was 2.5 years (SD 2.7). Nutrient repletion was initiated for deficiencies in vitamin D ($n = 3$) and iron ($n = 6$).

TABLE 1. Characteristics of Study Participants

	CD (n = 9)	UC (n = 6)	Total Cohort (n = 15)
Age (yr), mean (SD)	45 (22)	41 (15)	44 (19)
Female, n (%)	7 (78)	4 (67)	11 (73)
IBD duration (yr), mean (SD)	21.4 (15.0)	15.3 (14.6)	19.0 (14.6)
IBD location	Ileal (n = 4)	Rectum (n = 1)	n/a
	Colonic (n = 2)	Left side (n = 2)	
	Ileocolonic (n = 2)	Pancolitis (n = 3)	
	Ileocolonic w/perianal disease (n = 1)		
Tobacco use			
Never, n (%)	5 (56)	6 (100)	11 (73)
Current, n (%)	0 (0)	0 (0)	0 (0)
Former, n (%)	4 (44)	0 (0)	4 (27)
IBD medication use			
Mesalamine, n (%)	2 (22)	5 (83)	7 (47)
Immunomodulator, n (%)	2 (22)	0 (0)	2 (13)
Biological, n (%)	6 (67)	1 (17)	7 (47)
Systemic steroid, n (%)	1 (11)	2 (33)	3 (20)
FC (µg/g), mean (range)	404 (0–1269)	376 (25–1177)	392 (0–1269)
CRP (mg/L), mean (SD)	7.6 (13.0)	6.7 (6.9)	7.3 (10.7)

Outcome Measures

Clinical remission was achieved at week 6 by 11/15 (73%) study participants (6 CD and 5 UC), and all 11 maintained clinical remission during the maintenance phase of the study. Mean total SIBDQ scores significantly improved from 46.5 (SD 12.5) at baseline to 53.3 (SD 10.9) at week 6 ($P = 0.017$) and 60.5 (SD 4.8) at week 11 ($P = 0.045$). One patient with postoperative recurrence of ileal CD (without stricture) continued to have mild-moderately active CD (HBI range, 7–11) throughout the study, although noted resolution of joint pains. Two other patients with CD were either lost to follow-up or withdrew from the study before week 6 (see "Adverse Events"). One patient with left-sided UC continued to have mildly active symptoms (partial Mayo score remained at 5).

Among participants with UC with complete follow-up data at weeks 6 and 11 (n = 6), from week 0 to 6, mean partial Mayo score improved from 5.8 (SD 1.2) to 1.2 (SD 2.0) ($P < 0.01$), and this was sustained through week 11 (partial Mayo score 1.0, SD 2.0, P value for comparison between week 11 and 0 was <0.01) (Table 2).

Among participants with CD with complete follow-up data at weeks 6 and 11 (n = 7), mean HBI score improved from 7 (SD 1.5) to 3.6 (SD 2.1) ($P < 0.01$) at week 6, and at week 11 was 3.4 (SD 2.6) (P value for comparison between week 11 and 0 was <0.01). The 1 participant with a baseline FC of 79 μg/g and normal colonoscopy had a baseline HBI of 7 that improved to 2 by week 6 and remained at 2 at week 11. Sensitivity analyses excluding this patient, and thus only including patients with CD with active erosion or ulceration on endoscopy or imaging, did not alter the primary results: mean HBI score improved from 6.7 (SD 1.6) to 3.5 (SD 1.9) ($P < 0.01$) at week 6 and to 3.7 (SD 2.7) ($P = 0.01$) at week 11.

Achievement of clinical remission did not differ significantly between CD and UC ($p_{exact} = 0.60$). Secondary analyses conducted including the 1 participant with CD who was lost to follow-up did not alter the primary results. Mean HBI score improved from 7 (SD 1.6) to 4 (SD 2.6) ($P < 0.01$) at week 6 and at week 11 was 4.1 (SD 3.1) ($P < 0.01$).

Sensitivity analyses excluding the 2 participants (1 with CD and 1 with UC) initiating biological therapy within 3 months of study start did not alter the primary outcome results. Excluding these patients, mean HBI improved from 6.7 (SD 1.5) to 3.3 (SD 1.8) ($P < 0.01$) at week 6 and at week 11 was 3.4 (SD 2.6) ($P < 0.01$). Mean partial Mayo score improved from 5.4 (SD 0.5) to 1.4 (SD 2.2) ($P = 0.02$) at week 6 and at week 11 was 1.2 (SD 2.2) ($P = 0.02$).

Among those with laboratories completed at baseline and week 6 or week 11, mean CRP and FC decreased, but the results were not statistically significant (Table 3). Among those with a baseline FC >50 μg/g, mean FC decreased from 701 (SD 563) to 139 (SD 113) ($P = 0.09$). Among the 11 participants achieving clinical remission, 6 participants provided stool samples at baseline and week 11, with mean FC decreasing from 471 (SD 562) to 111 (SD 104), although not statistically significant ($P = 0.12$). No statistically significant differences were observed for hemoglobin, hematocrit, albumin, transaminases, bilirubin, creatinine, total cholesterol, high-density lipoprotein, triglycerides, or low-density lipoprotein between week 0 and 6 or 11. There was no significant change in patient weight from baseline to study completion ($P = 0.30$).

Among those with follow-up endoscopy at week 11 (n = 7), improvements were noted in simple endoscopic score for Crohn's disease (n = 1, rectum 8, left colon 5 → rectum 4, left colon 0), Rutgeerts score (n = 1, i3 → i1, this patient did not achieve clinical remission by HBI at week 6 or 11), and Mayo endoscopy subscore (n = 4). Specifically, among those with UC, MES improved by 1 point in 3 participants (MES 2 → 1 [n = 1] or 1 → 0 [n = 2]), 3 points in 1 participant (MES 3 → 0), and no change in 1 participant (MES 1). Among participants with IBD achieving mucosal improvement, mean FC improved from 390 (SD 4812) at baseline to 110 (SD 94) at week 11 ($P = 0.26$), and mean CRP improved from 4.6 (SD 5.2) at baseline to 2.8 (SD 6.3) at week 11 ($P = 0.60$).

TABLE 2. Effect of AIP Diet on Clinical IBD Activity

	Week 0	Week 6	*P* (week 6 versus 0)	Week 11	*P* (week 11 versus 0)
CD (n = 7 respondents at weeks 0, 6 and 11)					
HBI, mean (SD)	6.7 (1.5)	3.3 (1.8)	0.001	3.4 (2.6)	0.004
Abdominal pain, mean (SD)	0.6 (0.5)	0.4 (0.5)	0.604	0.6 (0.8)	1
Bowel movement frequency, mean (SD)	3.4 (2.2)	2.4 (0.8)	0.156	2.4 (1.3)	0.134
General well-being, mean (SD)	1.6 (0.5)	0.3 (0.8)	0.022	0.3 (0.8)	0.022
Complications, mean (SD)	1.1 (1.1)	0.1 (0.4)	0.018	0.4 (0.8)	0.14
UC (n = 6 respondents at weeks 0, 6 and 11)					
Partial Mayo score, mean (SD)	5.8 (1.2)	1.2 (2.0)	0.01	1.0 (2.0)	0.007
Stool frequency, mean (SD)	2.0 (0.9)	0.2 (0.4)	0.012	0.2 (0.4)	0.012
Rectal bleeding, mean (SD)	1.8 (0.8)	0.5 (0.8)	0.025	0.3 (0.8)	0.017
Physician global assessment, mean (SD)	2.0 (0)	0.5 (0.8)	0.007	0.5 (0.8)	0.007

TABLE 3. Effect of AIP Diet on Fecal and Serum IBD Biomarkers

Week 0 versus 6 Results	n	Week 0	Week 6	P
FC (μg/g), mean (SD)	8	267 (367)	157 (251)	0.45
Baseline FC >50 μg/g, mean (SD)	5	412 (406)	196 (317)	0.36
CRP (mg/L), mean (SD)	11	8.3 (11.5)	7.0 (14.5)	0.46
Albumin (g/dL), mean (SD)	11	3.9 (0.4)	3.9 (0.4)	0.82

Week 0 versus 11 Results	n	Week 0	Week 11	P
FC (μg/g), mean (SD)	6	471 (562)	112 (104)	0.12
Baseline FC >50 μg/g, mean (SD)	4	701 (563)	139 (113)	0.09
CRP (mg/L), mean (SD)	9	3.9 (5.2)	3.4 (5.3)	0.82
Albumin (g/dL), mean (SD)	10	4.1 (0.4)	3.9 (0.4)	0.36

Although we advised no medication change before study start, 1 participant discontinued oral mesalamine therapy but achieved clinical remission by week 6 (partial Mayo clinic score decreased from 6 at baseline to 0 at week 6). Another participant self-discontinued oral mesalamine but continued mesalamine suppository, and also noted a decrease in partial Mayo score from 5 to 0 by week 6. Two of the 3 participants were able to discontinue steroid therapy (in both, partial Mayo clinic score decreased to 0 by week 6). One patient on systemic steroids was lost to follow-up because of insurance change (see "Adverse events").

Adverse Events

One participant with postoperative recurrence of ileal CD with known ileocolonic anastomotic stricture required hospitalization for partial small bowel obstruction approximately 3 weeks after study start. This was attributed to a significant increase in raw vegetables, salad, and meat consumption. This participant did not communicate with health coach or dietician regarding dietary changes in the setting of known stricture. Participant's symptoms resolved overnight with the use of intravenous steroids, and patient was discharged on rapid steroid taper. This patient was subsequently lost to follow-up, and we were unable to make assessments at week 6 or 11.

Another participant with ileal CD with ileocecal valve stricture withdrew before the end of elimination phase because of worsening symptoms. Week 6 assessment was not made. Notably, this participant experienced increase in biomarkers from week 0 to 6, with FC increasing from 449 to 758 μg/g, and CRP increasing from 36 to 41.2 mg/L.

Although there were no significant differences in lipid subtypes between weeks 0 and 6 or weeks 0 and 11, some participants did experience small rises in triglycerides or low-density lipoprotein that prompted dietary modification by the registered dietician and health coach.

DISCUSSION

Increasing evidence suggests that dietary modification can modulate inflammation and improve clinical responses in IBD. Our prospective observational study indicate that an AIP diet, involving an elimination phase followed by a maintenance phase, demonstrates preliminary efficacy in patients with active IBD. We also identified improvements in FC along with endoscopic improvements in the mucosal appearance in most patients undergoing follow-up endoscopy.

Our results support the use of dietary modification as an adjunct to IBD therapy. Clinical remission was achieved by week 6 by 11/15 (73%) of study participants, and all 11 maintained clinical remission during the maintenance phase of the study. We did not hypothesize, a priori, that clinical remission would be achieved so early (week 6). Indeed, this proportion of participants with active IBD (HBI ≥ 5 or partial Mayo clinic score ≥3, and objective evidence of active inflammation) achieving clinical remission by week 6 rivals that of most drug therapies for IBD; importantly, our dietary study was performed as an adjunct to medical therapy, and almost 50% of patients in our study were on biological therapy. Therefore, our results suggest that dietary modification can be used as an adjunct to conventional IBD therapy, even among those with moderate-to-severe disease.

Importantly, the 2 participants with CD with ileal strictures (native or anastomotic) developed either worsening disease activity or partial small bowel obstruction. Therefore, although dietary elimination can be helpful, consideration should be given to anatomical variation and requires counseling and close follow-up.

The premise of the AIP diet, as a whole, involves a staged elimination of food groups that may be associated with immune stimulation and intolerance, maintenance of the eliminated foods, followed by staged reintroduction of certain foods or food groups over time. The purpose of our study was to examine the potential efficacy of the AIP diet for IBD, and as such we focused on the elimination phase and a minimum 1-month maintenance phase. Our study design was adapted from our health coach's online program, which focuses on the 6-week elimination phase.[16] The maintenance phase, in practice, can occur for participants anywhere from 30 to 90 days, although some continue it even longer, before starting to reintroduce food groups. The protocol emphasizes healthy food behaviors aimed at increasing the nutrient density of the diet, incorporating fresh fruits and vegetables, healthy sources of fats, lean proteins, fermented foods, and, for our study, modifying intake according to IBD phenotype (e.g., strictures). The protocol also recommends avoiding nonsteroidal anti-inflammatory drugs and low–nutrient-density processed foods, which may also contain emulsifiers or additives that can perpetuate inflammation, while improving lifestyle behaviors including sleep and sleep hygiene, stress management, and physical activity. Given the associations of stress with IBD flare[18] and low physical activity or nighttime sleep with risk of IBD,[19,20] addressing these aspects seemed logical, although they were not formally studied.

As such, it was not possible to determine how much the apparent effectiveness of the dietary/lifestyle intervention was due to these nondietary aspects.

An important goal of therapies for IBD is to reduce inflammation. Although not statistically significant, we did note decreases in FC, particularly in the patients with UC. Furthermore, rectal bleeding was significantly reduced by week 6 and 11 among patients with UC. The baseline CRP was also within normal limits (<10 mg/L) in most (66%) participants, and no statistically significant differences were noted between baseline and follow-up CRP measurements. Among those with endoscopic improvements in mucosal inflammation, we noted decreases in FC, consistent with studies demonstrating better correlation of mucosal findings with FC than clinical symptoms or indices.[21] Our study, although small, suggests dietary modification has the potential to reduce inflammation, as measured by FC and endoscopy.

The strengths of our study include a prospective design and focus on patients with not only clinically active IBD but also objective evidence of inflammation. Nutrient repletion was also initiated for patients with baseline deficiencies in vitamin D or iron, which would have helped to reduce confounding influences of micronutrient depletion on disease activity, but whose repletion may have also impacted improvements in general well-being noted during the study. Through our novel study design incorporating social media, study participants also had a unique ability to communicate with one another and our health coach and dietician, through a secret and private Facebook group. Poststudy follow-up visits suggest that online engagement, sharing of similar experiences and recipes, and convenience of communicating with health professionals directly were viewed favorably by participants.

Limitations of the study include small size, lack of a randomized trial design involving a control group, lack of blinding, and potential for selection bias among those enrolling in the study. We included 1 participant with active CD by HBI and FC, with no objective evidence of active inflammation by colonoscopy. However, analyses excluding this patient did not alter the primary outcome, and indicated significant improvement in HBI among patients with CD. Two patients in our study (1 CD and 1 UC) had initiated biological therapy within 3 months of study start, which could potentially confound the results of our study. However, secondary analyses excluding these 2 patients also did not alter the primary outcome results for CD or UC. Although endoscopic improvement was noted in 6 of the 7 participants completing endoscopic reassessment at study completion, it is important to acknowledge that 8 participants did not pursue endoscopic reassessment, and as such we could not perform statistical analysis to examine effect of the dietary intervention on mucosal healing. Another limitation includes lack of detailed assessment of the impact other recommendations such as physical activity, sleep and sleep hygiene, social support, and stress management had on their clinical course.

In conclusion, our study demonstrates that dietary modification focused on elimination of potentially immunogenic or intolerant food groups has the potential to improve symptoms and endoscopic inflammation in patients with IBD. Dietary change can be an important adjunct to IBD therapy not only to achieve remission but perhaps improve the durability of response and remission. Perhaps for a subset of patients, dietary and lifestyle modification alone may be sufficient to control underlying luminal inflammation. Patients wishing to incorporate dietary therapy should be counseled on options assessed for micronutrient deficiencies and monitored routinely. Integrating and coordinating care with health coaches and registered dieticians can allow for effective education and implementation of dietary modification over time, in accordance with unique patient goals as well. Larger randomized trials are needed to validate these findings and examine the long-term course of patients during reintroduction.

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