

Pilot Study on the Effect of Reducing Dietary FODMAP Intake on Bowel Function in Patients without a Colon

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Background: Poorly absorbed short-chain carbohydrates (FODMAPs) in the diet should, by virtue of their osmotic effects, increase fecal output following colectomy and ileal pouch formation or ileorectal anastomosis (IRA). The aim was to perform a proof-of-concept evaluation of this hypothesis.

Methods: Fifteen patients (13 pouch, 2 IRA) had dietary and symptomatic evaluation before and during a low FODMAP diet. Carbohydrate malabsorption was evaluated by breath tests. Pouchitis was assessed clinically/endoscopically or by fecal lactoferrin.

Results: Of 8 patients with a breath hydrogen response to lactulose, 7 had fructose malabsorption, 3 with lactose malabsorption, and 1 had lactose malabsorption alone. Five of 7 studied retrospectively improved stool frequency (from median 8 to 4 per day; $P = 0.02$), this being sustained over 0.5–3 years of follow-up. Five of 8 patients completed a prospective arm of the study. One patient had sustained improvement in stool frequency and 1 had reduced wind production. Overall, none of 8 patients who had pouchitis improved. In contrast, median daily stool frequency fell from 8 to 4 ($P = 0.001$) in the 7 without pouchitis. The degree of change in FODMAP intake also predicted response. There was a tendency for pouchitis to be associated with low baseline FODMAP intake.

Conclusions: There is a high prevalence of carbohydrate malabsorption in these patients. Reduction of the intake of FODMAPs may be efficacious in reducing stool frequency in patients without pouchitis, depending on dietary adherence and baseline diet.

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Key Words: inflammatory bowel disease, colectomy, ileal pouch,

ileorectal anastomosis, diet, short-chain carbohydrates, pouch function

Ileal pouch-anal anastomosis (IPAA) surgery is curative surgical treatment for ulcerative colitis (UC) that is medically refractory or complicated by dysplasia. Colectomy with ileorectal anastomosis is also used for the treatment of inflammatory bowel disease and familial adenomatous polyposis. IPAA significantly improves quality of life in patients with UC who require surgery.¹ However, the clinical outcome is not ideal. Even though the ileal pouch acts as a reservoir and continence is usually maintained, the average number of stools per day is 6–8 and usually with overnight stool production, and the consistency of those stools is rarely solid.² A problematic functional result might be due to inflammation of the pouch (pouchitis), the retained rectal cuff (cuffitis), or ileum proximal to the pouch (pre-pouch ileitis),³ but irritable pouch syndrome may also occur, being a syndrome of increased frequency of bowel movement with change in stool consistency, abdominal pain or cramping, and perianal or pelvic discomfort in the absence of endoscopic and histologic inflammation.⁴ Similar frequent bowel actions and unformed consistency is also associated with ileorectal anastomosis.

Management of functional problems with pouches has ranged from antibiotic therapy for pouchitis to hypomotility agents such as loperamide, to pouch excision in extreme situations.³ Dietary intervention has figured little in management recommendations, except that fiber supplementation is often recommended. However, the evidence base for this is absent, with only 1 crossover study that showed methylcellulose and pectin to be ineffective.⁵ Supplementation with inulin at a relatively high dose improved mucosal inflammation in patients with pouchitis without altering symptoms,⁶ but its effect in those without inflammation has not been reported.

One factor that will influence the functional properties of a pouch or rectum is the volume of liquid entering it from the small bowel. This is well documented for the normal large bowel, where diarrhea will result if the volume entering it from the small intestine overwhelms the desiccating ability of the large bowel mucosa. An ileal pouch or rectum will have markedly reduced desiccating ability than an intact large

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bowel. Hence, it might be anticipated that the pouch/rectum will be less able to cope with changes in small intestinal output, with such increases being readily expressed as looser and more frequent bowel actions. Two factors that potentially influence small intestinal output are the small intestinal transit⁷ and the number of osmotically active molecules in the small intestinal lumen, a factor utilized by osmotic laxatives.

We have hypothesized that the osmotic load on the pouch is significantly influenced by the amount of poorly absorbed short-chain carbohydrates in the diet. Such carbohydrates have been collectively termed FODMAPs (Fermentable Oligo-, Di-, and Mono-saccharides And Polyols) and include free fructose and lactose (if fructose and/or lactose malabsorption is/are present), fructans (oligosaccharides of fructose) that are not hydrolyzed in the small intestine, and polyols such as sorbitol, which are absorbed to a very limited extent only.^{8,9} Malabsorption of fructose and other FODMAPs has been linked to symptoms of irritable bowel syndrome including diarrhea, gas, and abdominal discomfort.^{9,10} In addition, a moderate to high fructose/sorbitol load hastens small intestinal transit.¹¹ Thus, the present study aimed to explore the concept that a change in dietary FODMAPs will influence the frequency and quality of stool output from an ileal pouch or a rectum following an IRA.

MATERIALS AND METHODS

Patients

Two cohorts of patients were studied. The first comprised 7 patients referred for the management of a dysfunctional pouch or poor results following colectomy and IRA. The results from investigation and dietary intervention were audited retrospectively.

The second group was studied prospectively using a defined protocol before and during dietary intervention. These patients were self-referred in response to advertising through the newsletter and website of the Australian Crohn's and Colitis Association (ACCA). Twenty-three patients responded to advertising. Ten were excluded since they resided interstate or overseas and 5 were not interested in participating. Eight, all of whom had an ileal pouch performed following colectomy for UC, were studied. The protocol for the prospective arm of the study was approved by the Research & Ethics Committee of Eastern Health.

Protocol

For the retrospective cohort, patients' notes were reviewed with regard to medical history, demographic data, and symptomatology prior to dietary education. The dietitian's assessment of dietary FODMAP intake was noted, as was their subsequent adherence to the dietary regimen, for which they were educated in a single one-on-one session of \approx 1 hour. Changes in symptoms and other clinical indices were noted following initiation of the diet.

For the prospective study, patients gave written, informed consent. A medical history was taken and general physical examination performed. A full dietary assessment was made by an accredited practicing dietitian and a prospective 7-day food diary using standard measures was performed by the patient during the screening period. The patients also filled out a daily symptom diary and continued this throughout the study. Fecal samples were obtained prior to commencing the diet. Breath hydrogen tests (see below) were also performed during the screening period. The patients were then educated on the low FODMAP diet (see below) and asked to strictly follow that diet for the next 6 weeks. The patient was telephoned after 3 weeks by both the dietitian and a clinician. The patient was examined and diary cards perused at the last visit. The patient was again contacted by telephone for a 6-week follow-up assessment.

Breath Hydrogen Testing

Following dietary restriction of fermentable substrates for at least 24 hours and an overnight fast, patients underwent breath hydrogen tests with the ingestion of 15 g lactulose (as positive control), 35 g fructose in 200 mL water, and 50 g lactose in 200 mL water on separate days. Breath hydrogen was measured every 15 minutes using a hand-held breath hydrogen monitor (Bedfont Gastrolyzer, Air-Met Scientific, Nunawading, Victoria, Australia, or SC Microlyzer, Quintron Instrument, Milwaukee, WI). A positive test was defined as a rise of 10 ppm in breath hydrogen above the baseline.

Evaluation of the Baseline Diet

All patients in the retrospective group had an assessment of FODMAP intake on a qualitative basis via a dietary history taken by a dietitian. For the prospective study, baseline diet was analyzed semiquantitatively using tables of known FODMAP content.¹⁰ The intake was described as average problematic serves per day; 1 serve represented a portion of food that contained >0.5 g free fructose or fructans,⁹ >4 g lactose, and any sorbitol. An assessment was also made of the total fructose load (comprising the sum of free fructose, equimolar fructose-glucose, and sucrose intake) and was expressed as the average number of sittings per day that the load exceeded 3 g.¹⁰ All evaluations were performed without knowledge of the response to dietary intervention.

Dietary Intervention

The low FODMAP diet was instructed via a 1-hour, one-on-one session with an Accredited Practicing Dietitian (M.B. or S.J.S.). The diet aimed to reduce free fructose and fructan ingestion as previously outlined in detail.⁹ If the fructose breath hydrogen test showed complete fructose absorption, total fructose ingestion was limited at any 1 meal, but foods containing free fructose were permitted in moderation. If the lactose breath hydrogen test was negative, then no dietary

restriction of lactose was advised. Patients who failed to produce hydrogen in response to lactulose were considered to malabsorb free fructose and lactose in the construction of their diet. Normal dietary habits were taken into account in the design of the dietary approach. Reinforcement of the diet and issues that arose with its use were managed at a telephone call made 3 weeks after commencing the diet. Assessment of adherence to the diet was made on the basis of cross-check questioning as previously applied.¹⁰

Fecal Lactoferrin

In the prospective group, feces were evaluated for lactoferrin content by enzyme-linked immunosorbent assay (ELISA) using a commercially available kit (TechLab, Blacksburg, VA). This was performed outside the protocol for the study, but was approved by the Eastern Health Research & Ethics Committee. The assay was performed according to the manufacturer's instructions. Lactoferrin has been previously shown to be a good marker of inflammation associated with the pouch (pouchitis, cuffitis, and pre-pouch ileitis).¹²

Data Evaluation

Data were expressed using descriptive statistics. Changes in indices were evaluated by paired or unpaired *t*-tests. Proportions were compared using a Fisher's exact test. A *P*-value of 0.05 or less was considered statistically significant.

RESULTS

Patient Characteristics

The characteristics of the 7 patients in the retrospectively studied group are shown in Table 1. Five had an ileal pouch performed because of UC at a median of 6 (range 5–17) years previously, while an ileorectal anastomosis was formed in 2 after colectomy for chronic constipation (2 years previously) and Crohn's disease (20 years previously). All had problematic increased frequency of and/or nocturnal bowel actions. Two were associated with chronic pouchitis, both of whom responded to antibiotic therapy (metronidazole or ciprofloxacin) either previously or subsequent to the study. The other 3 patients with an ileal pouch had no evidence of pouchitis on clinical grounds or on recent endoscopic examination.

All patients in the prospectively studied group had J-pouch surgery for UC with formation of the pouch a median of 7 (range 3–10) years previously. In the majority, no significant comorbidities were present, but 5 patients had been treated for pouchitis on 1 or more occasions in the past, including 1 who had chronic pouchitis.

Prevalence of Fructose and Lactose Malabsorption

As shown in Table 1, 7 of the 15 patients had no breath hydrogen response to lactulose ("nonhydrogen-producers"). Of the other 8, 4 had evidence of fructose malabsorption

alone, 1 had lactose malabsorption alone, and 3 had both fructose and lactose malabsorption.

Analysis of Usual Dietary Intake of FODMAPs

In the retrospective group, 2 patients (R4 and R5) were noted to consume foods with a very high FODMAP content, particularly of fructose, lactose, and sorbitol. One patient (R3) had a low intake of FODMAPs, although did regularly consume sources of fructose with equimolar concentrations of glucose, equating to a high fructose load. The other patients had an intake of FODMAPs that was neither high nor low.

Eight patients in the prospective arm completed a 7-day food diary and a summary of their intake of FODMAPs is shown in Table 2. Overall, the patients ate few foods containing free fructose (such as apples, pears, honey, coconut milk/cream). The major contribution to FODMAP intake came from fructans, mainly as wheat-containing breakfast cereals, breads, pasta, and onions. Most also avoided milk or used lactose-free or soy milk, but ate cheese.

Adherence to the Diet

All patients in the retrospective group had good apparent understanding of the diet, but adherence, as assessed by dietary history, was questioned in 2. Patient R2 had only partial adherence to the diet. Patient R3 had a low FODMAP intake prior to dietary change and changed her diet minimally.

In the prospective group, 5 of the 8 patients completed 6 weeks on the diet together with week-6 food diaries (Table 2). In 3 there was excellent adherence to the diet and 1 patient (P2) markedly reduced FODMAP intake, although did consume some free fructose. One patient (P5) inadvertently consumed foods high in FODMAPs, such as onion in tinned soups and sauces and wheat-containing foods at a celebratory event.

Three patients did not complete the diet. One (P8) was lost to follow-up before receiving dietary instruction, 1 (P6) abandoned the diet on development of acute chronic pouchitis shortly after commencing the diet, and the third (P7) reported practical difficulty adhering to the changes required by the diet and abandoned it within 1 week.

Effect of Diet on Symptoms

Retrospective Study

Five of the 7 patients had substantial improvement in stool frequency and consistency as shown by patient self-reporting. The median number of stools per day fell from 8 to 4 (*P* = 0.02; paired *t*-test). Benefits were sustained over longer-term follow-up of 2 (0.5–3) years. Furthermore, patients uniformly reported that reintroduction of prohibited foods worsened their symptoms. The results of breath testing, while influencing the nature of the dietary intervention, did not

TABLE 1. Patient Characteristics, Test Results, and Effect of Low FODMAP Diet on Symptoms

Patient No.	Age (y)	Sex	Anatomy	Disease	Breath Hydrogen Tests		Fecal Lactoferrin ^a ($\mu\text{g/g}$)	Early Adherence to Diet	Effect of Diet on Symptoms	Clinical Scenario	Follow-up
					Fructose	Lactose					
Retrospective Study											
R1	48	Female	J pouch	Ulcerative colitis	+	-	Not tested	Good	Improved		Durable efficacy
R2	74	Male	J pouch	Ulcerative colitis	+	+	Not tested	Partial	No change	Chronic pouchitis ^c	Not following diet
R3	42	Female	J pouch	Ulcerative colitis	+	-	Not tested	Poor	No change	Chronic pouchitis ^c	Not following diet
R4	41	Male	J pouch	Ulcerative colitis	-	+	Not tested	Good	Improved		Durable efficacy
R5	35	Female	IRA ^b	Chronic constipation	Non-H ₂ -producer		Not tested	Good	Improved		Durable efficacy
R6	49	Female	IRA	Crohn's disease	+	+	Not tested	Good	Improved	Crohn's proctitis ^c	Durable efficacy
R7	41	Female	J pouch	Ulcerative colitis	+	+	Not tested	Good	Improved		Durable efficacy
Prospective Study											
P1	37	Male	J pouch	Ulcerative colitis	+	-	9.25	Good	No change		Partial adherence
P2	41	Male	J pouch	Ulcerative colitis	Non-H ₂ -producer		1.7	Good	Improved		Durable efficacy
P3	49	Female	J pouch	Ulcerative colitis	Non-H ₂ -producer		12.4	Good	Worse	Acute pouchitis during the study ^d	Not following diet
P4	47	Female	J pouch	Ulcerative colitis	+	-	5.6	Good	Reduced wind		Partial adherence
P5	42	Male	J pouch	Ulcerative colitis	Non-H ₂ -producer		23.6	Partial	No change	Recent pouchitis and dilatation of pouch stricture	Not following diet
P6	51	Female	J pouch	Ulcerative colitis	Non-H ₂ -producer		12.1	Poor	Withdrawn	Chronic pouchitis ^c	Diet efficacious after treatment of pouchitis
P7	22	Female	J pouch	Ulcerative colitis	Non-H ₂ -producer		Not tested	1 week trial	Withdrawn		Not following diet
P8	36	Male	J pouch	Ulcerative colitis	Non-H ₂ -producer		20.5	Nil	Withdrawn	Chronic pouchitis ^d	Lost to follow-up

^a Normal value <7.25 $\mu\text{g/g}$

^b IRA, ileorectal anastomosis.

^c Proven on endoscopy and histopathology.

^d Based on clinical scenario and raised fecal lactoferrin.

TABLE 2. Quantification of FODMAP Intake According to Prospectively Completed 7-Day Food Diaries

Patient No.	Before Dietary Intervention (Numbers of Serves per Day)						During Dietary Intervention (Numbers of Serves per Day)					
	Fructose	Fructans	Lactose	Fructose Load	Sorbitol	Total FODMAPs	Fructose	Fructans	Lactose	Fructose Load	Sorbitol	Total FODMAPs
P1	1	6	1	0	0	8	0.5	1	0	0	0	1.5
P2	3	6	2	1	1	12	2	0	0	0	0	2
P3	2	6	1	1	1	11	0	0	0	0	0	0
P4	2	4	1	0	1	9	0	0	0	0	0	0
P5	3	6	1	1	1	12	1	2	1	1	0	5
P6	4	6	1	1	0	12	Noncompliant with or did not commence diet					
P7	4	7	3	1	1	16	Noncompliant with or did not commence diet					
P8	2	5	0	1	0	8	Noncompliant with or did not commence diet					

Results are shown as averaged daily intake of problematic serves. The serve sizes are free fructose 0.5 g, fructans 0.5 g, lactose >4 g, sorbitol any, and total fructose load (free fructose plus sucrose) >3 g.

appear to influence the response to the diet. Both patients who had no response to dietary change had chronic pouchitis, documented histopathologically.

Prospective Study

Only 5 patients were evaluable for the effect of diet on stool frequency (Fig. 1), with no change seen (median 6 to 5 per day; *P* = ns). One patient had considerable worsening of stool frequency and this proved to be due to acute pouchitis. As outlined in Table 1, the 3 patients who symptomatically did not improve had inflammation associated with the pouch on fecal lactoferrin and clinical criteria, while the 2 who improved did not.

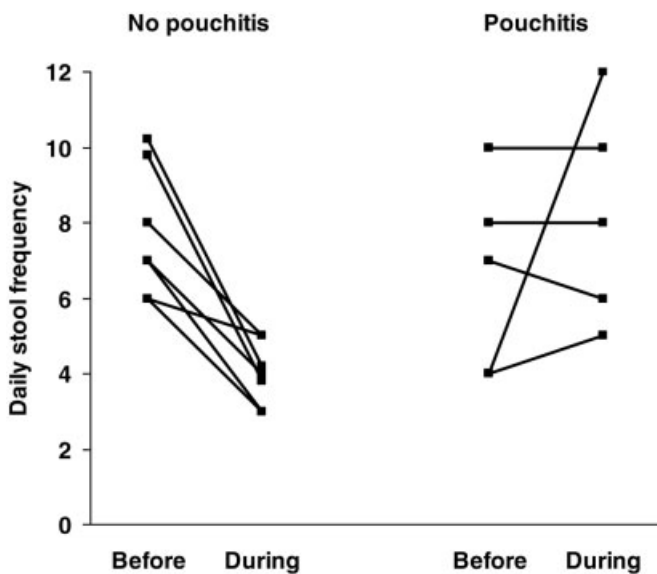


FIGURE 1. Daily stool frequency before and during the low FODMAP diet according the presence or absence of evidence of pouchitis. Changes in those without pouchitis were statistically significant (*P* = 0.001; paired *t*-test).

At follow-up by telephone call 6 weeks after cessation of the study treatment period, the 2 patients who reported benefit had continued dietary change. Patient P2 adhered to the diet most of the time and had continued improvement in stool output and consistency. Patient P4 continued with partial adherence to the diet via the avoidance of apples, pears, and apricots, as well as lactose-containing foods. The improvement was not in stool frequency but wind production had reduced. One of the patients (P1) who initially did not improve had incorporated some aspects of the dietary advice into his eating habits, such as avoiding free fructose contained in honey and apples, presumably related to symptomatic benefit. Two unresponsive patients (P3 and P5) did not continue the diet. Patient P6 who withdrew due to poorly controlled chronic pouchitis responded to ciprofloxacin therapy, and subsequent reintroduction of the diet led to significant improvement in symptoms, especially stool consistency.

Predictors of Response

Examining the 2 cohorts together, the presence or absence of evidence for pouchitis predicted the effect of dietary intervention on stool frequency. As shown in Figure 1, for the 7 patients without pouchitis median daily stool frequency fell from 8 to 4 (*P* = 0.001) compared with the 5 patients with pouchitis, where the daily stool frequency rose from 7 to 8 (*P* = ns). This represents a statistically significant median fall of 3 stools per day compared with no change in those with pouchitis (*P* = 0.007; *t*-test).

Qualitative data in the retrospective group suggested that the degree of reduction in FODMAP intake might predict symptomatic improvement. Response to dietary change was seen in both patients (R4 and R5) who had a high baseline intake of FODMAPs with strict adherence to the diet. One patient (R2) had a low baseline FODMAP intake, made few dietary changes, and did not respond. Semiquantitative as-

assessment in the prospective group showed that clear symptomatic improvement was observed in Patient P2, who reduced FODMAP intake by 10 serves per day and partial symptomatic benefit was reported by P4, who reduced by 9 serves per day. No improvement was seen in P5, who reduced by 7 serves per day and P3, who developed acute pouchitis during the study but was strictly adherent and reduced intake by 9 serves per day. Thus, these data suggest that the degree of reduction in FODMAP intake may be another factor in predicting symptomatic response.

Of the 5 patients who were found to consume a diet low in FODMAPs, 4 had evidence of pouchitis. In contrast, of the 9 patients who consumed a diet that contained moderate to high amounts of FODMAPs, only 2 had evidence of pouchitis. This difference was not, however, statistically significant ($P = 0.068$, Fisher's exact test).

DISCUSSION

Managing frequency and nocturnal bowel actions is challenging in patients following colectomy and ileoanal pouch anastomosis or IRA. Diet has figured poorly as a therapeutic tool due to inefficacy. Limiting dietary intake of FODMAPs as outlined in the present study represents the first dietary therapy that may reduce stool frequency, at least in the absence of pouchitis or other causes of pouch-associated inflammation.

The present study was designed to be a 'proof-of-concept' study in that there was no blinding or randomization. It was limited by the fact that several patients were studied retrospectively, where less precise documentation of stool frequency is almost certain to occur. Furthermore, the prospective arm was limited in the selection of patients, where volunteers responded to advertisements to participate. This method of recruitment attracted those with problematic pouches in that at least 5 of the 8 patients had pouchitis, or other cause for inflammation such as cuffitis or pre-pouch ileitis, as shown by elevated fecal lactoferrin concentrations, compared with only 2 of 7 in the retrospective study, who were offered the dietary therapy when seen in routine clinical practice. Nevertheless, this study did permit important observations to be made.

Nearly 50% of patients failed to produce hydrogen in response to a load of lactulose compared with less than 10% across healthy populations and patients with functional gut disorders.⁸ This was similar to the observations of Santavirta,¹³ where 32% of subjects had no hydrogen response to lactulose in the only previous study describing hydrogen production in patients with an ileal pouch. It may well relate to the length of time the lactulose is in contact with bacteria or to the type of bacteria present in the pouch or rectum. It did not correlate with the presence of inflammation. In those who did produce detectable hydrogen, fructose malabsorption was common, being seen in 7/8 (88%) patients with a J pouch or IRA. Likewise, lactose malabsorption was found in 4 patients

(50%). These observations are of great importance since they offer an opportunity to reduce the osmotic load to the pouch or rectum via dietary modification. If attention is also paid to minimizing intake of poorly absorbed oligosaccharides, most importantly fructans, and of polyols, particularly sorbitol, perhaps the fluid load on the pouch might be significantly altered. Indeed, semiquantitative assessment of dietary FODMAP intake showed a generally modest intake of free fructose, with most of the FODMAP intake deriving from fructans contained in onions and wheat products.

The main outcome measure was the daily stool frequency. In the retrospective group, this was significantly reduced by dietary change, but such a finding was not confirmed in the prospective study. However, it was apparent that the responders and nonresponders differed in a clear way. The presence of inflammation in the gastrointestinal tract, as demonstrated by endoscopic examination of the pouch or by the measurement of fecal lactoferrin,¹² was strongly associated with failure to change daily stool frequency on the low-FODMAP diet. In contrast, a highly significant reduction in daily stool frequency was observed in patients where no evidence of pouchitis was found. Reasons for the lack of response in patients with inflammation are not clear. There may be a significant secretory element to diarrhea in association with inflammation. Intestinal transit might also be accelerated in response to inflammation, although studies examining pouch dysfunction have not linked pouchitis with the rapidity of transit.¹¹ Both of these factors, however, would render pouch output and function less responsive to changes in the osmotic load. Additionally, it is likely that increased irritability of the pouch in pouchitis may have contributed to stool frequency independently of the volume in the pouch.

A second possible predictive factor was the degree of reduction in FODMAP intake. Those with high baseline intake of dietary FODMAPs and with good adherence to the diet responded, while those with a low baseline intake and/or partial adherence did not. These observations would be anticipated from the postulated mechanism for dietary efficacy and, in that way, they support the hypothesis.

A limiting factor of the efficacy of any dietary change for a chronic problem is the ability of patients to follow dietary instruction and to maintain adherence in the long term. We have previously found that long-term adherence can be achieved in at least 3 out of 4 patients with functional gut disorders.¹⁰ Patients who had responded to the diet in the retrospective group did, on self reporting, adhere to the dietary guidelines in the longer term. Reasons for such success include the perception of continuing efficacy and the intermittent reinforcement associated with a relapse of symptoms when excluded foods were again consumed. The fact that the majority of patients remain adherent to the diet most of the time is in itself a testament to the diet's efficacy. Long-term follow-up in the prospective group was not possible, but it

was interesting that 6 weeks after the completion of the formal treatment part of the study the patient with excellent improvement of symptoms continued on the diet and that 2 patients who had some or little efficacy reported during the treatment period had continued with some of the dietary restrictions, presumably since they had now recognized and experienced direct precipitation of increased or more frequent pouch output in response to specific FODMAP-rich foods.

There may be potential risks of reducing dietary intake of FODMAPs. The prebiotic effect of fructose and fructans is well demonstrated.¹⁴ One study has provided evidence that dietary supplementation with a large dose of inulin (24 g/day) reduced pouch inflammation in patients with pouchitis.⁶ Inulin is a long-chain fructan and is different in this respect to the short-chain (oligosaccharide) fructans that make up the majority of dietary fructans. However, it does exhibit similar prebiotic effects to fructose and fructo-oligosaccharides.¹³ This raises the possibility that reduction in dietary fructans as well as fructose might lead to a less favorable spectrum of bacteria in the pouch. Indeed, 1 patient in the study had worsening symptoms on the low FODMAP diet, although fecal lactoferrin level was elevated prior to commencing the diet. There was also a tendency for patients with pouchitis to consume a low amount of FODMAPs in their usual diet. Further studies are required to determine whether the association of low FODMAP intake with pouchitis is indeed real.

In conclusion, the present study suggests that reduction of dietary FODMAPs in patients with ileal pouch or IRA may be efficacious in reducing stool frequency in patients who do not have active pouchitis or other inflammation associated with the pouch, especially in those who consume at least modest quantities of FODMAPs in their usual diet. The high prevalence of fructose and lactose malabsorption indicates that reduction of the intake of free fructose and lactose should be integral to the dietary approach. Taken together with the observation that 1 in 2 patients are nonhydrogen producers, the performance of breath hydrogen tests probably contributes little to dietary design. The application of the FODMAP approach to reducing the osmotic load on the ileal pouch or rectum represents the first dietary strategy likely to have a

positive impact on the quality of pouch function in such patients. Further prospective evaluation of this approach in a larger population of patients without pouchitis, in whom baseline dietary intake is evaluated, is needed to determine whether the diet indeed has an important role in improving symptoms in these patients.

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