

Perspectives in Practice

Fructose Malabsorption and Symptoms of Irritable Bowel Syndrome: Guidelines for Effective Dietary Management

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ABSTRACT

Dietary fructose induces abdominal symptoms in patients with fructose malabsorption, but there are no published guidelines on its dietary management. The objective was to retrospectively evaluate a potentially successful diet therapy in patients with irritable bowel syndrome and fructose malabsorption. Tables detailing the content of fructose and fructans in foods were constructed. A dietary strategy comprising avoidance of foods containing substantial free fructose and short-chain fructans, limitation of the total dietary fructose load, encouragement of foods in which glucose was balanced with fructose, and co-ingestion of free glucose to balance excess free fructose was devised. Sixty-two consecutively referred patients with irritable bowel syndrome and fructose malabsorption on breath hydrogen testing underwent dietary instruction. Dietary adherence and effect on abdominal symptoms were evaluated via telephone interview 2 to 40 months (median 14 months) later. Response to the diet was defined as improvement of all symptoms by at least 5 points on a -10- to 10-point scale. Forty-eight patients (77%) adhered to the diet always or frequently. Forty-six (74%) of all patients responded positively in all abdominal symptoms. Positive response overall was significantly better in those adherent than nonadherent (85% vs 36%; $P < 0.01$), as was improvement in individual symptoms ($P < 0.01$ for all symptoms). This comprehensive fructose

malabsorption dietary therapy achieves a high level of sustained adherence and good symptomatic response. *J Am Diet Assoc.* 2006;106:1631-1639.

Fructose is a monosaccharide found in three main forms in the diet: as free fructose (present in fruits and honey); as a constituent of the disaccharide sucrose; or as fructans, a polymer of fructose usually in oligosaccharide form (present in some vegetables and wheat) (1). Failure to completely absorb fructose in the small intestine (that is, fructose malabsorption) leads to its delivery to the colonic lumen, together with water due to its osmotic effect. Luminal bacteria rapidly ferment fructose to hydrogen, carbon dioxide, and short-chain fatty acids (2). Thus, if sufficient fructose reaches the colon, luminal distention may occur due to the osmotic load and rapid gas production, which potentially leads to bloating, abdominal discomfort, and motility changes. The osmotic load itself might also have a laxative effect similar to that utilized by the commonly used disaccharide laxative lactulose.

These symptoms are also commonly experienced by patients with irritable bowel syndrome (IBS). Provocation studies in which fructose loads are given to people with fructose malabsorption induce gas, bloating, abdominal discomfort, nausea, and disturbed bowel function much more readily in subjects with IBS than in those without it (3-5). These observations have led to the suggestion that malabsorption of dietary fructose trigger symptoms in patients with IBS, and that removal of fructose from the diet might lead to improvement in symptoms. Open studies of modifying fructose intake have strongly supported this view (6,7).

However, in standard clinical practice, the management of IBS seldom addresses dietary fructose. Several reasons may explain this. First, fructose malabsorption may be considered uncommon and as such is a separate disease distinct from IBS. However, more than one in three adults with symptoms of IBS are unable to absorb a fructose load of 25 to 50 g and, therefore, have fructose malabsorption (3,8,9). Second, fructose malabsorption is not specific to patients with IBS. The limited data available suggest that its prevalence in the IBS population is similar to that in asymptomatic controls (9). As outlined earlier, the response to fructose is exaggerated in patients with IBS compared with those without IBS. Third, there are no dietary guidelines published to construct an appropriate diet. Previous dietary intervention studies

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0002-8223/06/10610-0013\$32.00/0

doi: 10.1016/j.jada.2006.07.010

Substrate	Small intestinal handling	Dietary strategies
Fructose	Carrier-mediated facilitated diffusion of low-capacity–impaired in fructose malabsorption Facilitation of absorption by glucose Facilitation of absorption by amino acids Absorption saturable if high fructose loads	Limit “free fructose” (>0.5 g/100 g fructose in excess of glucose) Co-ingestion of glucose with high “free fructose” foods Not utilized Limit total load of fructose load (with or without glucose) at any one meal
Fructans	No small intestinal hydrolysis or absorption	Limit foods with significant (>0.5 g/serving) fructan content

Figure 1. Dietary strategies designed and utilized on the basis of the known physiology of small intestinal handling of fructose and fructans.

have used “fructose-free” diets without describing their nature (6,7). It would be unfeasible for the dietary management of the condition to require total removal of fructose from the diet, a near impossible task because of its abundant presence in our food supply. Finally, the potential contribution of fructans has been ignored, as illustrated by a recent review (9), despite the fact that they are not digested or absorbed in the small intestine, are rapidly fermented by luminal bacteria (8,10,11,12), and can induce IBS-like symptoms by themselves (13,14).

The physiological principles underlying potential malabsorption of fructose were identified from published works (9,15-20). These principles are summarized in Figure 1. As a monosaccharide, fructose does not require hydrolysis before its absorption across villous enterocytes. Sucrose, a disaccharide, is efficiently hydrolyzed by the brush border enzyme sucrase to the monosaccharides glucose and fructose. Fructose is absorbed by carrier-mediated facilitated diffusion, which seems to be of low capacity (17). In the presence of luminal glucose, fructose absorption is markedly enhanced, but the mechanism of this interaction has not been definitively ascertained. This permits efficient fructose absorption in the presence of glucose.

Some amino acids also enhance fructose absorption (21). In subjects with fructose malabsorption, fructose given as sucrose, or in equimolar combination with glucose, can be well absorbed (16-19). The amount of fructose ingested in excess of glucose is likely, therefore, to be the main determinant of fructose malabsorption. Because the fructose absorption capacity of the bowel is saturable (19), a high fructose load itself consumed at a meal/sitting may also lead to malabsorption of fructose independently of the presence or absence of facilitators of its absorption. Malabsorption of fructose when consumed as sucrose or equimolar fructose/glucose in large amounts has been documented on breath hydrogen testing in a minority of patients (15,22,23), and symptoms of IBS have been induced (4,17,18).

Fructans are oligosaccharides and polysaccharides of fructose units with a glucose terminal end, and may be either inulin (beta 1-2 bond) or levan (beta 2-6 bond) types (10). Inulin terminology is often confusing in that those with a chain length (degree of polymerization) less than 10 are generally referred to as fructooligosaccharides, whereas molecules with a degree of polymerization more than 10 are generally called inulins. Because fructans are not digested or absorbed in the small bowel (11,12), their codelivery to the colon with fructose will

provide an additional substrate that is rapidly fermented. Fructans alone induce abdominal symptoms (8,13) and exaggerate those associated with lactose malabsorption (24). Hence, fructans should also be limited in any dietary modification in patients with fructose malabsorption and IBS. Because fructans with a low degree of polymerization have a greater osmotic effect and are more rapidly fermented than those with a high degree of polymerization (8,11,25), the chain length of fructans may be an important determinant of the degree of contribution to symptoms.

The aim of the present study was to address the hypothesis that reducing the delivery of dietary fructose and fructans to the large bowel will reduce the symptoms of IBS in patients with fructose malabsorption. Adherence to and the effects on gastrointestinal symptoms of a fructose malabsorption diet were evaluated in a retrospective audit of patients with IBS and fructose malabsorption on breath hydrogen testing.

METHODS

Patient Selection

Sixty-two consecutive patients with IBS and proven fructose malabsorption were referred in a private practice setting by physicians for dietary management. Age range of subjects was 17 to 81 years (median, 50 years).

Fifteen were men (17 to 77 years, median 61 years) and 47 were women (23 to 81 years, median 46). Inclusion criteria comprised a diagnosis of IBS (made by the referring physician and fulfilling the Rome II criteria [26]). Patients with predominant diarrhea or constipation and those with alternating bowel habit were included. All had completed a breath hydrogen test (27) with the ingestion of 35 g of fructose dissolved in 200 mL water. A positive test was defined as an increase of 15 ppm in breath hydrogen more than the baseline before fructose ingestion. Celiac disease and inflammatory bowel disease had been excluded by endoscopic investigations and/or celiac serology. Patients with lactose malabsorption (on breath hydrogen criteria) were also excluded.

Development of Fructose Malabsorption Diet

Food composition data detailing fructose, glucose, and fructan content of foods was sought from published composition tables from the Australian National Food Authority (28) and the US Department of Agriculture (29), and scientific journal articles (12,30-34). On the basis of

Unfavorable Foods		Favorable Foods	
Excess free fructose ^a (fructose > glucose)	Fructose load > 3 g/serving ^a	Glucose in balance with or in excess of fructose	Glucose-rich accompaniments
<ul style="list-style-type: none"> ● Fruit: apple, pear, guava, honeydew melon, mango, nashi fruit, pawpaw/papaya, quince, star fruit (carambola), watermelon ● Honey ● Major sweetening ingredient: <ul style="list-style-type: none"> ● High-fructose corn syrup ● Corn syrup solids ● Fructose ● Fruit juice concentrate 	<p>In average serving^b quantities:</p> <ul style="list-style-type: none"> ● Dried fruit: apple, apricot, currant, date, fig, pear, prune, raisin, sultana ● Fruit juice, canned packing juice ● Fruit pastes and sauces: tomato paste, chutney, relish, plum sauce, sweet and sour sauce, barbecue sauce ● Fruits with high sugar content: cherry, grape, persimmon, lychee, apple, pear, watermelon ● >1 standard serving of fruit per sitting ● Coconut: milk, cream ● Dried fruit bars ● Honey ● Fortified wines: sherry, port, etc <p>In indulgent quantities:</p> <ul style="list-style-type: none"> ● Sucrose sweetened soft drink: >375 mL (average 40 g sucrose per 375 mL) ● Confectionery: excessive intake (average 40 g sucrose per 50 g) 	<ul style="list-style-type: none"> ● Stone fruit: apricot, nectarine, peach, plum (note these contain sorbitol) ● Berry fruit: blueberry, blackberry, boysenberry, cranberry, raspberry, strawberry ● Citrus fruit: kumquat, grapefruit, lemon, lime, mandarin, orange, tangelo ● Other fruits: ripe banana, jackfruit, kiwi fruit, passion fruit, pineapple, rhubarb, tamarillo ● Other sucrose consumed in moderation 	<ul style="list-style-type: none"> ● Glucose-sweetened energy/sports drinks ● Glucose supplements: eg, tablets and powder ● Glucose powder, tablets, syrup ● Glucose-sweetened confectionery (even if wheat-derived glucose)
<p>^aData from food compositional tables (26,27) and scientific journal articles (12,28-32). ^bAverage serving=serving in grams, as defined in Australian food composition tables, or mean weighed measures if data unavailable in food composition tables.</p>			

Figure 2. Status of foods related to fructose content, and the glucose-to-fructose ratio, for people with fructose malabsorption.

the physiological principles of fructose and fructan absorption and malabsorption (Figure 1), a list of potentially problematic foods was compiled. These were based on arbitrary cutoff values for the fructose and fructan content of individual foods and were defined as: (a) foods that have naturally occurring free fructose in excess of glucose (>0.5 g/100 g); (b) a fructose load of more than 3 g in an average serving quantity of the food or beverage; and (c) substantial food sources of fructans (>0.5 g/serving). Some foods had both an increased load and excess of fructose. If there were inconsistencies in food compositional tables, a mean value of all data was applied. These data are shown in Figure 2.

Implementation of the Fructose Malabsorption Diet

At the initial interview with a dietitian, a qualitative patient-defined typical days' dietary intake reflecting usual eating practices over the past month was recorded. Symptoms were assessed by direct questioning: ie, patients were asked about the presence and nature of abdominal pain, gas, bloating, diarrhea, constipation, and nausea. If both diarrhea and constipation were experienced, both symptoms were recorded.

Dietary education was delivered in an individual con-

sultation over a 1-hour period on one occasion. Four dietary strategies were discussed with all patients. The first was avoidance strategies. Foods to be avoided were those containing significant free fructose in excess of glucose, and foods that are a substantial source of fructans. Another strategy was positive food choices; ie, choosing foods in which fructose and glucose are "in balance" or have more glucose than fructose. Co-ingestion strategies included co-ingestion of free glucose to "balance" excess free fructose problematic foods. Co-ingestion of alanine-rich foods was not taught because it was considered a more difficult concept for patients to understand and implement and it was less practical. Limitation strategies included limiting dietary fructose load (in the form of free fructose or excessive sucrose) at any one meal.

In addition to these dietary modification strategies, other dietary recommendations included avoiding intake of foods rich in polyols, such as sorbitol and xylitol, in subjects who seemed to be symptomatic after eating such foods. (Polyols are well-documented to induce symptoms [4,5,19,35].)

Patient education involved teaching the scientific basis of malabsorption and how this helped determine the list of problem foods. Patients were then provided with posi-

tive food messages, emphasizing suitable food alternatives. To assist in this, verbal descriptions or visual props using packages of commercially available food alternatives were provided to patients, together with suggestions for their use/application and where to buy such foods. Several suggestions were provided to cater to a wide spectrum of food preferences and also to optimize variety and nutritional adequacy in the diet. A sample meal plan encompassing the dietary principles was also provided. The diet was reinforced 4 to 6 weeks later at the patient's request or if the registered dietitian was uncertain that the diet was fully understood. The patient was educated about all of the dietary strategies for the fructose malabsorption diet described. Current medications were recorded. No advice was given about medications.

Evaluation of the Fructose Malabsorption Diet

A retrospective review (of patients with proven fructose malabsorption and symptoms of IBS who had been instructed in the fructose malabsorption diet) evaluated adherence to the diet, barriers to adherence, strategies used by patients, and effects of the diet on IBS symptoms.

After a median of 14 months (range 2 to 40 months) of dietary intervention, a structured telephone interview assessed adherence to the diet and the longer-term effect on symptoms. Adherence was assessed first by patient self-evaluation and then by cross-check direct questioning of dietary history. This involved direct closed-ended questioning about actual intake of problematic foods, as described in Figure 2. Patients were classified as "adherent" or "nonadherent" and by degree of adherence, as outlined in Figure 3. After responding to specific questions regarding IBS symptoms (as discussed later), patients were asked open-ended questions about barriers to adherence that they had experienced, strategies they had used to adhere to the diet, and, if relevant, experiences of subsequent dietary indiscretions. Patient's self-reported changes in medication were documented. Individual symptoms were scored by the patient on a subjective self-assessment using a -10 to 10 scale where 0=pretreatment symptoms, 10=total improvement, and -10=extreme worsening. Overall response was defined as a score more than 5 for all IBS symptoms experienced before dietary intervention. In other words, those who improved by more than 5 in only some symptoms were not considered to have responded. Responses were also compared according to adherence to diet. The conduct of this audit of practice complied with the ethical guidelines of the National Health and Medical Research Council of Australia and was approved by the Research and Ethics Committee of Eastern Health.

Data were expressed as median and interquartile ranges, and were compared using the Mann-Whitney test. Proportions were compared using Fisher's exact test. A P value ≤ 0.05 was considered statistically significant.

RESULTS

From tables of fructose, glucose, and fructan content of foods, detailed lists of problematic and favorable foods related to fructose and glucose were compiled and are shown in Figure 2. For fructans, published detailed data

Major category	Subcategory	Details
Nonadherent	Never	Never followed the diet
	Occasionally	Followed the diet as taught for some time ^a (time specified), but now do not follow the diet
Adherent	Frequently	Followed the diet as taught immediately, but now follow it less than 50% of the time
		Followed the diet at least 50% of the time
	Always	Followed the diet as taught immediately and now follow it at all times except on some occasions
		Followed the diet as taught immediately and now follow it at all times except when eating away from home (time specified)
		Followed the diet as taught immediately, and still follow the diet totally

^a"Some time" is defined as up to 3 months' duration; ie, the diet was followed initially.

Figure 3. Definition and self-reported degree of adherence to the fructose malabsorption diet.

are more limited (12,36-39). Problematic foods have been grouped according to the likelihood of inducing symptoms and are outlined in the Table. The majority of fructan intake in a typical Western diet derives from wheat-based products (pasta, bread, breakfast cereals) and onions (36).

The majority of patients (89%) had only one educational session. Six people requested one review consultation, and one patient required three consultations. Of the 62 patients, 48 (77%) were considered adherent (39% frequently and 38% always following the dietary prescription). An additional six patients (10%) reported partial or occasional incorporation of the dietary principles, and eight (13%) did not follow the diet at all. The duration of follow-up was a median of 11 months (range 10 to 40 months) in adherent and 16 months (range 2 to 36 months) in nonadherent patients (P =not significant). In adherent patients, the consequences of occasional indiscretions varied from abdominal bloating, gas, and nausea, to diarrhea.

The limitations in complying with the diet were not unexpected. When asked via direct questioning, the patient-reported main barriers to adherence included an unwillingness to undertake dietary recommendations, difficulties accessing and increased expense of specialty wheat-free foods, and dislike of the taste of these foods. The diet was also found to be more difficult to follow when eating away from home. Nineteen patients changed medications (started, stopped, or changed) during the follow-up period, but changes in the patients who altered medications are similar to those who did not alter medication in the adherent and nonadherent groups (data not shown).

Table. Fructan content of food ordered in significance of observed impact on symptoms in clinical experience

Foods containing fructans	Serving size (g) ^a	Fructan Content ^b	
		g/100 g	g/serving ^c
Most problematic			
Wheat-based foods			
Flour	100 g	1.0-4.0	4.0
White bread	2 slices (65 g)	0.7-2.8	1.8
Pasta	1 c. cooked (165 g)	1.0-4.0	2.5
Whole-grain			
breakfast cereal	1 c. (60 g)	0.8-3.2	1.9
Breakfast muffin	1 muffin (65 g)	0.6-2.2	1.4
Crumpet	2 crumpets (90 g)	0.5-1.9	1.6
Cracker	2 biscuits (40 g)	0.8-3.4	1.2
Crispbread	2 biscuits (30 g)	1.0-3.8	1.2
Plain sweet			
cookie	2-3 biscuits (30 g)	0.5-2.0	0.6
Onion	2 T. (35 g)	1.1-10.1	2.1
Leek	1/2 c. (85 g)	3.0-10.0	5.6
Asparagus	6 spears (90 g)	1.4-4.1	2.6
Jerusalem artichoke	1/2 c. (75 g)	16.0-20.0	15.0
Potentially problematic, but largely untested			
Globe artichoke	1 medium (120 g)	2.0-6.8	5.5
Dandelion greens	1/2 c. (25 g)	12.0-15.0	3.1
Chicory roots	1/2 c. (75 g)	35.7-47.6	30.4
Chicory greens			
(witlof, Belgian endive)	1/2 c. (75 g)	NA ^d	NA
Radicchio	3 medium leaves (25 g)	NA	NA
Chicory root-based			
coffee-substitute			
beverages	1-2 tsp (7 g)	35.7-47.6	3.0
Murnong	NA	NA	NA
Yacon	NA	NA	NA
Burdock	NA	NA	NA
Scorzonera	NA	NA	NA
Well-tolerated, not problematic in moderate amounts			
Garlic	1 clove (3 g)	9.0-16.0	0.48
Barley grain	100 g	0.1-0.2	0.2
Rye			
Flour	100 g	0.5-0.9	0.9
100% rye bread	2 slices (65 g)	0.35-0.63	0.4
Rye crispbread	2 biscuits (30 g)	0.4-0.72	0.3
Banana	1 average size (90 g)	0.3-0.7	0.6
Lettuce	3 medium leaves (25 g)	NA	NA

^aServing=serving in grams, as defined in Australian food composition tables, or mean weighed measures if data unavailable in food composition tables.

^bData from references 12, 35-38, and M. Rennie, D. McKiernan, personal communication, April 2003.

^cUpper end of the range.

^dNA=data not available.

Most patients implemented the dietary strategies by self-selecting alternative foods. Patients were directly asked which foods were consumed as alternatives to problematic foods. Examples of the reported preferred food alternatives are shown in Figure 4. Fifteen percent used supplemental glucose in their diet to balance free fructose, and all reported to be symptom-free with this strategy.

In addition to abdominal discomfort and associated altered bowel habits, the predominant presenting symptoms were abdominal pain (66%), gas (85%), bloating (76%), diarrhea (64%), constipation (33%), and nausea (30%). Forty-six patients (74%) exhibited a positive response (a score >5 for all symptoms) to the dietary education. Although there were no differences in the frequency of initial symptoms between the groups (data not shown), the positive response was significantly greater in adherent (85%) than nonadherent patients (36%; $P < 0.01$, Fisher's exact test). As shown in Figure 5, adherence to the diet was associated with marked improvement in all symptoms, and this was significantly better than the symptom scores in the nonadherent group ($P < 0.01$; Mann-Whitney U test). However, there was considerable improvement in abdominal pain and gas in the patients who were nonadherent. The proportion of patients who had persisting response to individual symptoms at follow-up are shown in Figure 6 according to adherence with the diet. For every symptom, the adherent group responded significantly more than the nonadherent group.

DISCUSSION

The potential importance of malabsorbed fructose and fructans as a trigger for symptoms in patients with IBS has been generally underappreciated. The evidence that a fructose or fructan load can provoke acute gastrointestinal symptoms is substantial (3-5,8,13,14,19).

There is also support for the view that chronic symptoms of IBS can be effectively reduced in patients with fructose malabsorption by restricting dietary fructose ingestion (6,7). During the last few decades, reports conflict about whether sugar intake has increased (40,41), but a consistent finding in US studies is that the proportion of sugars made up by fructose is increasing (40,42). Contributing to this is the increased consumption of fruit juices and the use of high-fructose corn syrups (which contain 42% to 55% fructose) as sweeteners in many manufactured foods, particularly in the United States (43,44). The proportion of energy from caloric sweeteners during the last 4 decades has increased 22%, more than 80% of which can be attributed to increased consumption of soft drinks and/or sugared fruit drinks (45). The increased dietary load of fructose may more commonly exceed the absorptive capacity of the small intestine, leading to worsening or unmasking of IBS. Thus, the need for dietary guidelines for IBS seems to have increased.

The guidelines developed in the present study have attempted to address this in several ways. First, although restriction of certain foods was critical to the success of the diet, positive food choices were an integral part. Foods that contain sucrose, or glucose in equal amounts to fructose, were not considered problematic because fructose is not malabsorbed in the presence of glucose (in normal food servings). Second, successful implementation of the dietary principles was enhanced by placing an emphasis

Problematic food	Highly favorable alternative ^a	Other suitable alternative ^a
Wheat bread	Wheat-free rye bread	Gluten-free bread
Wheat pasta	Gluten-free pasta, rice	Rice noodles
Wheat-based breakfast cereal	Porridge, cornflakes, puffed rice	Gluten-free cereals low in dried fruit and honey
Wheat-based cakes	Flourless (almond-meal) cakes, cornflour sponge, friands	Gluten-free cakes
Wheat-based crackers	100% rye crispbreads, corn crispbreads, rice crispbreads and crackers	Other commercially available gluten-free savory crackers
Wheat-based cookies	Gluten-free cookies, almond macarons	
Wheat-based noodles	Rice noodles	100% buckwheat soba noodles, rice
Wheat-based pastry	Avoid consuming pastry, eat content/filling only	Gluten-free pastry, rice base (eg, quiche)
Wheat-based breadcrumbs	Gluten-free rice crumbs	Cornflake crumbs
Honey	Jam (jelly), marmalade, yeast extract spreads, peanut butter, golden syrup, treacle, maple syrup	
Onion	Cook with, but do not consume, onion (eg, in stir-fries and sauces, cut into large chunks and leave behind on plate, in soups/stews, use whole onion and remove prior to serving)	
Chicory-based coffee-substitute beverages	Tea, coffee, herbal teas/infusions	
Problem fruits	Any alternative, consumed as one serving per meal/sitting	
Problem vegetables	Any alternative not containing fructans	
Fructose sweeteners	Sucrose in moderation, glucose	

^aSuggested alternative foods that have been well tolerated by the patient group. Highly favorable foods generally relate to those that are more readily available/accessible.

Figure 4. Patient-reported acceptable alternative food preferences.

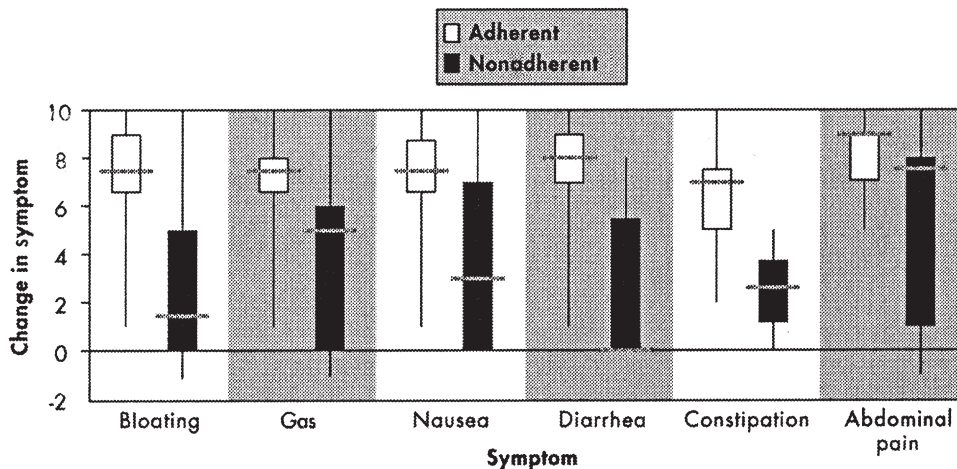


Figure 5. Change in symptom scores from before dietary instruction to the status at the time of telephone interview, a median of 14 months later. Individual symptoms were scored by the patient on a subjective self-assessment using a -10 to 10 scale where 0=pretreatment symptoms, 10=total improvement, and -10=extreme worsening for irritable bowel syndrome symptoms experienced before dietary intervention. Score >5 was defined as response. Data shown according to whether the patient was considered adherent (unshaded) using criteria shown in Figure 3. The range, median, and 25th and 75th percentiles are shown. For all individual symptoms, scores were significantly higher for adherent patients compared with those of nonadherent patients ($P<.01$, Mann-Whitney).

on suitable food alternatives rather than only focusing on the problematic foods. Third, flexibility in the choice of strategies was offered. Realistic goals were set by intake limitations rather than by absolute bans. For example, it was essential that patients did not perceive this as a “fructose-free” (and therefore a “fruit-free”) diet, or that every trace of wheat must be avoided. Balancing fructose

(but not fructans) with glucose can be achieved by supplementing foods with excess free fructose with free glucose. This dietary principle poses issues for patients with diabetes and/or obesity and for prevention of dental caries and should be considered in such settings.

Fourth, individualization of the approach is an important principle and a feeling of empowerment by the pa-

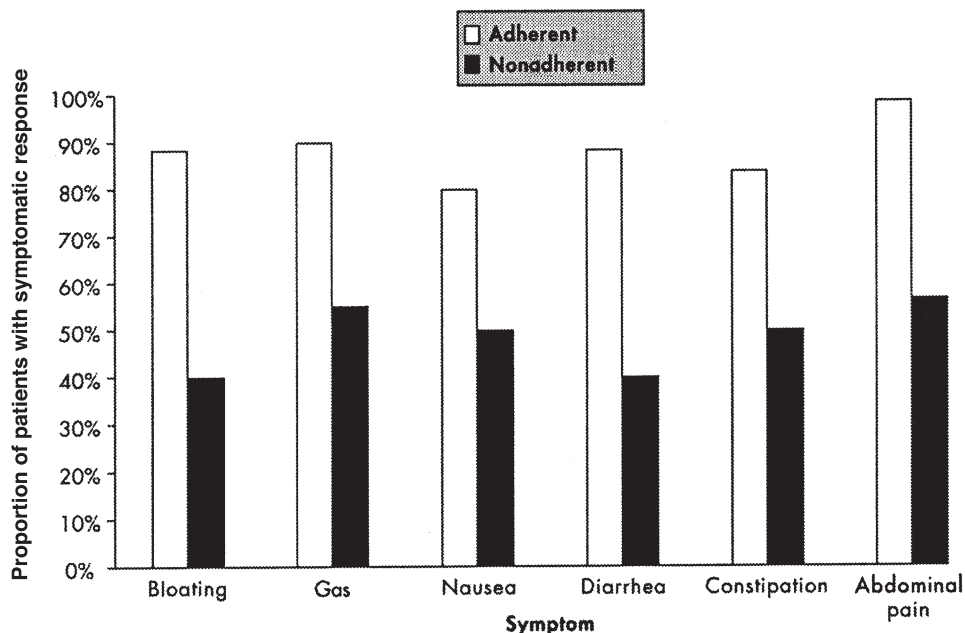


Figure 6. The proportion of patients who showed an improvement in symptoms (score >5, as defined in the Methods section) for individual symptoms according to adherence to the diet. Scores were self-rated by the patients at the time of telephone interview relative to symptoms prior to receiving dietary instruction. The proportion of patients with response in every symptom was significantly better in adherent than in nonadherent patients ($P < .01$; Fisher's exact test).

tient should be fostered. This is best achieved by appropriate education of the principles involved and provision of clear information on core knowledge of relevant food content. Patients are encouraged to apply the dietary principles discussed as required to manage their symptoms and experimentation is not discouraged. Partial adherence may be an individual's compromise to resolve the symptoms to a satisfactory level, while minimizing the social limitations that dietary restrictions can impose. Most patients reported episodes of IBS symptoms clearly related to dietary indiscretions, particularly with increased frequency of indiscretions.

Nutritional composition of the fructose malabsorption diet has not been assessed. Dietary changes were implemented through replacement strategies and therefore should not impact nutrition quality. Alternative foods are encouraged, rather than omitting whole food groups. Adequacy of micronutrient, fiber, phytochemical, and whole food groups (for example, fruit) intake would be useful to assess.

A retrospective analysis of experience with a diet is not the ideal way to determine whether efficacy was due to a placebo effect, which is notoriously high in clinical trials of patients with IBS (14). One factor in the genesis of placebo effects is believed to be the regular review and attention by interested and empathetic staff that clinical trials offer (46). This was not the case in the present study because most patients were seen only once by the registered dietitian, and then were contacted when they were not expecting a call. Furthermore, many patients did notice induction of symptoms when dietary indiscretions occurred. On the other hand, the application of educational techniques to empower the patient to control

their symptoms by dietary choices may have increased confidence and reduced anxiety levels.

Although avoidance of free fructose is clearly a major focus of the diet, as presumably has been previously applied (6,7), three novel principles have been incorporated into this new approach. First, strategies to balance free fructose with glucose-rich foods and drinks were used. Second, the total load of fructose was limited. This is irrespective of whether the food might have glucose equivalent to or in excess of fructose. Fructose, even when delivered in equimolar concentrations with glucose (or as sucrose), can induce symptoms of IBS in some people (4,18,19). This can be addressed with a number of dietary strategies. First, small amounts of these foods should be consumed at one time (eg, no more than the equivalent of one standard fruit serving at a time, or stagger fruit intake throughout the day). Second, their consumption should occur together with that of other foods, so that release from the stomach might be slower (eg, one third of a glass of orange juice consumed with, not before, breakfast), and there is co-ingestion with other food components that enhance its absorption.

The third, and perhaps the most important, innovation was modifying the intake of fructans, something that has only had limited previous attention (8). Because fructans of short degree of polymerization are osmotically active, rapidly fermented (8,11,25), and induce IBS-like symptoms (8,47), an additive effect to that of malabsorbed fructose seems likely, as has been described for sorbitol and fructose (4-6) and for fructans and lactose (15). The mean daily intake of fructans has been calculated to be as much as 12 g/day (10,12,36). This may increase with the addition of inulins and fructooligosaccharides to food for putative

health benefits (37,48,49). As a major source of fructans in the diet (36), wheat is likely to be problematic when consumed in large amounts, such as in pasta, breakfast cereals, bread, cakes, cookies, and crackers. Dietary trends in the United States and Europe indicate increasing consumption of these food items. Unlike the gluten-free diet for celiac disease in which there must be strict gluten restriction, not every trace of wheat needs to be avoided. Thus, minor sources of fructans such as wheat starches and thickeners used in commercially prepared foods (such as sauces) are not of concern. Wheat-free rye bread was not problematic for the majority (91%) of the patient group studied. Rye contains fructans (1,12,38,50), but the chain length is longer than those found in wheat (12,38,50), and may not be as osmotically active or as rapidly fermented. This could offer an explanation to their high tolerability.

The diet was not effective for all patients. This reflects the multifactorial nature of the genesis of symptoms in patients with IBS. Luminal distention may not be as important in triggering symptoms in some patients where visceral hypersensitivity (51), issues of symptom perception, and the existence of other trigger factors such as stress may be major factors (52).

The evaluation of the effect of the diet on symptoms was retrospective and uncontrolled. Such methodology has several limitations. Because the results obtained are based on subjective dietary assessment over a timeframe of 2 to 40 months, findings may not be generalizable. Patients were asked to judge retrospectively whether their symptoms had changed over an assessment time frame of 2 to 40 months; although the documented symptoms were cross-checked with those claimed to be present at the telephone follow-up interview, there may have been a bias toward overstating the improvement noted. However, all patients were contacted and a clear and statistically significant difference in scores was noted between the adherent and nonadherent groups. Some improvement was noted in many patients who self-reported nonadherence. The introduction or withdrawal of drugs or complementary medicines may have contributed to symptomatic improvement in some, although there are few drugs known to give such global benefits in symptoms of IBS. Alternatively, improvement may have been a part of the natural history of IBS. An additional factor may have been partial adherence and the introduction of some aspects of the diet. Six of the 14 nonadherent patients did follow the diet in a limited way (classified as "occasionally adherent").

In conclusion, a comprehensive new diet directed at limiting fructose and fructan intake has been developed on the basis of physiological principles and food composition tables. Application of the diet to patients with IBS and fructose malabsorption has revealed a high level of sustained adherence associated with a high rate of symptomatic improvement. Unanswered questions include whether the response is due to reduced free fructose and/or fructan intake, to some other factor in the diet, or to a placebo effect. Also unanswered are the predictors of response, particularly whether the presence of fructose malabsorption as defined by the breath hydrogen test is indeed required for response. This experience suggests that a clinical trial studying fructose malabsorption diets incorporating the successful approaches reported here should be conducted to support this approach.

Ian Willett, MB, BS, FRACP, provided consultation and advice regarding implementation of the diet. Amanda Anderson (BSci, M Nut Diet, APD) provided advice regarding implementation of the diet.

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