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## Clinical Ramifications of Malabsorption of Fructose and Other Short-chain Carbohydrates



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Symptoms of pain, bloating, wind, constipation and/or diarrhea are commonly present in various gastrointestinal disorders and are often attributed to a functional gut disturbance. Dealing with such symptoms is currently unsatisfactory and is minimally featured in conventional medical literature. Fructose is receiving increasing attention as a factor in the diet that, when malabsorbed, may induce these symptoms. However, fructose is only one of many poorly absorbed, short-chain carbohydrates (Fermentable Oligo-, Di- and Mono-saccharides And Polyols or FODMAPs) in the diet. Others include fructans, lactose (in hypolactasic individuals), polyols and galactans. FODMAPs are theoretically attractive targets for dietary change due to their high osmotic activity and rapid fermentability, leading to luminal distension and the potential for subsequent symptom induction in those with less adaptable bowels and/or visceral hypersensitivity. A global approach to the reduction of dietary FODMAPs is proposed and current evidence supports its efficacy in relieving symptoms in the majority of patients with functional gut disorders.

### INTRODUCTION

**F**ructose is a hexose that is present widely in the diet as free fructose, in the disaccharide, sucrose, and in fructans, which are chains of fructose with

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a terminal glucose. Fructans are also known as inulins (when the number of fructose molecules, or degree of polymerization or DP, is  $\geq 10$ ), or fructo-oligosaccharide (FOS) or oligofructose (when the DP is  $< 10$ ). Fructose has created much interest because of its possible role in contributing to the obesity epidemic in the USA (1) and for its postulated role in inducing functional gut symptoms.

Symptoms such as bloating, abdominal distension, discomfort, pain, and altered bowel habits are often

described as “functional” gut symptoms since their cause is usually related to alterations in the function of the gut and enteric nervous system rather than being manifestations of structural abnormalities. Indeed, functional gut disorders (FGD) are very common, affecting at least 15% of the community across the world. These disorders have been classified into entities such as “irritable bowel syndrome” (IBS), “functional diarrhea,” “functional bloating,” and “functional dyspepsia” according to the symptom complex (2). While the definition of such syndromes is useful in the design of clinical trials, they are somewhat artificial as their overlap is considerable and they are all believed to involve similar mechanisms of visceral hypersensitivity and disorders of the gut-brain axis. Functional gut disorders also commonly exist in association with untreated celiac disease (a common presentation) and inflammatory bowel disease (IBD) where such symptoms are two-to-threefold more common than in the community at large (3,4).

Management of functional gut symptoms is unsatisfactory, as outlined in scholarly reviews (5–7). Diet has figured poorly in such reviews, mainly because of the paucity of high level evidence for efficacy of dietary change. The relationship between the ingestion of food and abdominal symptoms is well recognized by patients and health professionals. The internet and bookshops overflow with dietary remedies for FGD, but the scientific basis of most of these is tenuous at best. The increasing attention to fructose and its malabsorption in the small intestine as a possible major dietary trigger has led to excitement that an effective dietary approach to patients with FGD, supported by solid physiological principles, might now be recognized and accepted. Furthermore, fructose is only one of a family of short-chain poorly absorbed carbohydrates (termed FODMAPs—see below) in the diet. The purpose of this review is to outline the theoretical basis and evidence of efficacy for the low FODMAP diet and to describe how it can be instituted.

### FRUCTOSE MALABSORPTION

Fructose malabsorption (FM) is defined as the incomplete absorption in the small intestine of dietary free fructose, with subsequent delivery of fructose to the distal small bowel and colon. FM is generally detected

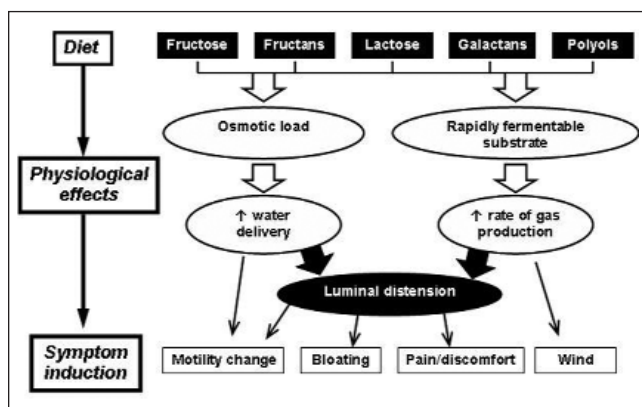
by breath hydrogen testing after an oral load of fructose, since bacteria present in those regions will rapidly ferment the malabsorbed fructose with subsequent hydrogen generation and appearance in the breath. In reality, FM represents the presentation of fructose to luminal bacteria before it is absorbed. This can be the result of three interdependent mechanisms—a low absorptive capacity of the small intestinal epithelium, rapid transit through the small bowel, and bacterial overgrowth in the distal small bowel (SBBO) (8). Differentiation of these mechanisms is not easy in the individual patient, even with the concomitant use of lactulose or glucose breath testing.

The ability of the human small intestine to absorb fructose is limited, especially when compared with the rapidity and completeness of glucose absorption. Thus, when 50 g fructose is ingested without other food, up to 80% of people will incompletely absorb the fructose, as demonstrated by breath hydrogen testing (9–12). Absorption is enhanced by co-ingestion with glucose, since glucose uptake stimulates additional transport pathways for fructose absorption in the small intestinal epithelial cell. Because of this, the fructose released from the hydrolysis of sucrose is generally completely absorbed.

There appears to be a spectrum of absorptive capacity of free fructose (that is, without glucose present) across populations from very poor to efficient, although reasons for the differences are complex and not completely understood. An important concept is that FM is probably a physiologically normal phenomenon, not specifically associated with a disease state (8). Whether it is more common in patients with FGD or IBS specifically remains uncertain, since prevalence studies have been limited by the number of healthy, asymptomatic controls tested, and heterogeneity of breath test methodologies. What is clear, however, is that the presence of FM in a person who has symptoms such as abdominal pain, bloating, wind, altered bowel habit and lethargy offers an opportunity for dietary manipulation to help reduce those symptoms, whether the underlying gastrointestinal condition is primarily a FGD such as IBS, secondary to SBBO, or, secondarily associated with IBD or celiac disease (11,13–16).

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**Figure 1.** The theoretical basis for the relationship between FODMAPs and the genesis of functional gut symptoms.

### PHYSIOLOGICAL TARGETS FOR DIETARY MANIPULATION

Luminal distension is an important stimulus for inducing functional gut symptoms. Studies using barostat methodologies, where the stomach or part of the bowel is distended by balloons or fluid, have clearly shown the importance of visceral hypersensitivity in the induction of sensations of pain and bloating (17). Luminal distension may also lead to altered motility patterns. It implies an increased volume of the contents in that segment of bowel. The contents comprise solid, liquid and gas components and dietary factors can influence all of these. The liquid and gas phases would be more likely to change acutely (as mimicked by balloon distension) and are, therefore, rational targets for dietary manipulation.

The likely regions of the gut that generate most symptoms are the distal small intestine and proximal colon. The volume of liquid is dependent upon the osmotic load in that segment of bowel, since intraluminal osmolality is kept within strict limits. Thus, small molecules in the diet that are poorly absorbed are candidates for change. Intraluminal gas is largely produced by fermentation of carbohydrates by bacteria. Hydrogen, carbon dioxide and, in some, methane are the major gases formed and they are readily absorbed into the circulation to be released in the lungs. Their rapid production, however, will potentially cause increased luminal pressure and distension. Thus, target dietary factors will be those that are rapidly fermented.

Candidate dietary components that fulfill both an osmotic effect and rapid fermentation are short-chain carbohydrates that are poorly absorbed in the small intestine (Figure 1). These comprise fructo- and galacto-oligosaccharides, sugar alcohols (polyols) and, in some patients, fructose and lactose. Since there is no collective term for these related molecules, we have termed them “FODMAPs” (Fermentable Oligo-, Di- and Mono-saccharides And Polyols) (18).

### EVIDENCE BASE FOR FODMAPS AS DIETARY TRIGGERS

The potential for FODMAPs to induce abdominal symptoms has been well documented in acute challenge studies where fructose and sorbitol (alone or in combination), lactose, and fructo-oligosaccharides have induced bloating, abdominal pain, diarrhea and wind (9–11,19,20). Withdrawal of individual components from the diet have been reported to have beneficial effects on the symptoms of some patients, although the most widely studied FODMAP, lactose in hypolactasic people, has a poor record of sustained benefit (21). Limitation of the intake of fructose and fructans specifically, but with associated minimization of other FODMAPs, led to sustained and substantial relief of all the symptoms of IBS in a series of 46 of 62 patients (74%) so treated (15). Efficacy was also related to adherence to the dietary regimen. In further support, worsening of symptoms with reintroduction of dietary fructose and fructans in a randomized controlled manner (22) conclusively indicates that restriction of these components is the most likely mechanism for the observed symptom relief in these patients while on the low FODMAP diet. Similar symptomatic benefits in patients with IBD (Crohn’s disease and ulcerative colitis) have been described in a brief report (16).

### THE LOW FODMAP DIET

#### Principle of the Diet

The key underlying principle to this diet is that FODMAPs exert additive effects since their actions (osmotic and fermentative) are shared. Therefore, opti-

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**Table 1**  
**FODMAP Checklist**

**Check the food items you eat regularly:**

- Fruit:** apple, pear, guava, honeydew melon, mango, nashi fruit (Asian pear), pawpaw/papaya, quince, star fruit (carambola), watermelon
- Stone fruits:** apricots, peaches, cherries, plums, nectarines
- Fruits with high sugar content:** grapes, persimmon, lychee
- Dried fruit**
- Fruit juice, canned packing juice**
- Dried fruit bars**
- Fruit pastes and sauces:** tomato paste, chutney, relish, plum sauce, sweet and sour sauce, barbecue sauce
- Fruit juice concentrate**
- Fructose** as an added sweetener
- High fructose corn syrup or corn syrup solids including:** Fruit drinks, carbonated drinks, pancake syrups, catsup, jams, jellies, pickle, relish, etc. and/or liquid cough remedies and liquid pain relievers, etc
- Honey**
- Coconut:** milk, cream
- Fortified wines:** sherry, port, etc
- Vegetables:** Onion, leek, asparagus, artichokes, cabbage, Brussel sprouts, beans
- Legumes:** Baked beans, kidney beans, lentils, black eye peas, chickpeas, butter beans
- Wheat or white bread**
- Wheat pasta, noodles**
- Wheat-based breakfast cereal**
- Wheat-based cakes, cookies, crackers**
- Chicory-based coffee-substitute beverages**
- Artificial sweeteners:** sorbitol, mannitol, isomalt, xylitol
- If lactose malabsorption:** milk, ice-cream, yoghurt

\*FODMAPs consumed on a regular basis are potential triggers for functional gut symptoms and a trial of limiting them should be undertaken. Please note, this checklist has not undergone evaluation for its usefulness in clinical practice.

only one or two FODMAPs (such as lactose or fructose with or without sorbitol). In order to implement such a diet, the content of FODMAPs in food is required. Tables of fructose and fructan content, as current data permit, have recently been published (15). However, the tables are incomplete and further analyses to fill the gaps are ongoing (23) and will assist refinement of the diet. In clinical practice, a checklist such as that shown in Table 1, may be useful to identify FODMAP-rich food items individuals consume regularly.

In order to understand the low FODMAP diet, individual FODMAPs need to be understood and they are discussed below.

### Fructose

**Physiological basis:** Fructose is normally absorbed in the small intestine via two routes: a low capacity facilitated diffusion via the GLUT5 transporter and a glucose-activated more rapid diffusion via insertion of GLUT2 into the apical membrane (24). Thus, fructose is well absorbed in the presence of equimolar glucose in the proximal small intestine (25), whereas free fructose is slowly absorbed and such absorption occurs right along the length of the small intestine. Fructose malabsorption will occur when the activity of GLUT5 is impaired (24), or when small intestinal bacteria are able to ferment the fructose before it can be absorbed (26). Fructose malabsorption, as defined by the appearance of hydrogen in the breath after an oral load of fructose, is common. The frequency is dependent upon how much fructose is given; 53% will incompletely absorb a 25 g load, increasing to 73% with a 50 g load (9). Because fructose malabsorption is so common, the presence of fructose malabsorption is a poor predictor of who might have IBS, as its prevalence appears to be similar to that of normal controls (10). In other words, fructose malabsorption is a normal physiological state, but, in the presence of visceral hypersensitivity, the effect of malabsorbed fructose on intestinal distension induces symptoms.

**Problematic foods:** Since fructose is absorbed efficiently in the presence of an equimolar concentration with glucose, people with fructose malabsorption need to avoid foods high in free fructose, but can manage those with balanced concentrations of fructose and

mal efficacy in minimizing distension of the distal small bowel and proximal large bowel thereby improving symptoms will be achieved by a global approach in restricting the intake of all FODMAPs (Figure 1). This is in contrast to previous approaches that have restricted

**Table 2**  
Foods with high fructose content \*

<i>Food</i>	<i>Average serving</i>	<i>Glucose (g)</i>	<i>Fructose (g)</i>	<i>Excess fructose (g)</i>
Watermelon	1 large slice, 4 oz	1.13	1.5	0.44
Pineapple	1 slice, 3 oz	1.25	1.69	0.45
Orange	1 whole, 5 oz	2.08	2.73	0.78
Honeydew melon	1 large slice, 4 oz	1.63	2.38	1
Custard apple	1 whole, 6 oz	12.4	13.5	1.1
Honey	1 tablespoon	6.8	8.2	1.4
Mandarin	1 whole, 6 oz	2.59	3.4	1.8
Peach, clingstone	1 whole, 5 oz	1.16	2.76	1.9
Carambola, star fruit	1 whole, 3 oz	0.86	2.76	1.9
Asian Pear	1 whole, 4 oz	1.62	7.13	4.0
Mango	1 whole, 7 oz	1.85	5.95	4.1
Apple	1 whole, 6 oz	4.29	10.4	8.1
Pear	1 whole, 6 oz	2.64	10.73	8.4

\*Their composition has been expressed per average serving in terms of the total fructose and glucose in addition to the fructose in excess of glucose. Fructose and high fructose corn syrup/corn syrup solids are often used as sweeteners in the commercial food industry. Patients are encouraged to check food labels for these sugars.

glucose (or a greater concentration of glucose). Using our FODMAP database, we have determined which foods contain excess fructose and compiled a list of such problem foods (Table 2). The use of fructose and high fructose corn syrup (HFCS) is increasing, particularly in the United States. HFCS is used as a sweetener in food manufacturing, a cheaper alternative to sucrose. This has resulted in a significant increase in fructose intake over the past few decades with the rise in consumption of HFCS known to have been >1000% between 1970 and 1990 (9). For those people suffering IBS symptoms secondary to fructose malabsorption, this is bad news.

In addition to excess fructose foods, those high in the balanced sugars may also be problematic when consumed in large amounts as these provide a high fructose load (Table 3). For example, an orange is in equal balance of fructose and glucose so is safe to eat, however, orange juice is concentrated, with one glass containing up to six oranges, so this should be limited to only one-third glass in one sitting (equivalent to the juice from one serving of fruit).

## Lactose

**Physiological basis:** Lactose, a disaccharide of glucose and galactose, is hydrolyzed by lactase in the brush border of the proximal small intestine. Reduced lactase activity is present in 1%–95% of the population, depending on ethnicity and environmental factors (27). The consequence of lactose malabsorption depends

**Table 3**  
Typical foods that deliver high fructose loads\*

<i>Food</i>	<i>Average serving</i>	<i>Per 100 g (foods) or 100 mL (fluids)</i>	
		<i>Glucose (g)</i>	<i>Fructose (g)</i>
Lychee	104 g	8.22	7.9
Orange juice	1 cup	6.24	8.19
Figs	100 g	9.6	8.2

Note: All dried fruits and juices are potentially high fructose foods—100 mL (one-third glass) of “safe” fruit juices (i.e. with no excess fructose) or a tablespoon of dried fruit is the maximum to be consumed in one sitting.

**Table 4**  
**Lactose Content of Common Dairy Products**

<i>Product</i>	<i>Serving Size</i>	<i>Lactose Content (g)</i>
Milk (whole, lowfat, or nonfat)	1 cup	11
Buttermilk	1 cup	10
Acidophilous milk	1 cup	11
Yogurt, whole milk	1 cup	10–12
Yogurt, low fat	1 cup	5–19
Cheese (such as cheddar, American, Swiss, parmesan)	1 oz	0.4–3
Cottage cheese	1/2 cup	3–4
Ice Cream	1/2 cup	5–6
Sherbet, orange	1/2 cup	2
Whipped cream	1/2 cup	3
Sour cream	1/2 cup	4
Cream cheese	1 oz	1
Half & Half / cream	2 tbsp	1
Butter / margarine	1 tbsp	Trace

Used with permission from the University of Virginia Health System Nutrition Support Traineeship Syllabus (33)

upon how severe the hypolactasia is and, as for fructose malabsorption, the response of the bowel to the luminal distension and increased osmotic load. Small amounts of lactose in the diet (up to 7 g in one sitting) are usually well tolerated (28).

**Problematic foods:** Typical lactose-containing foods and their lactose content are shown in Table 4. Without appropriate education, people often make the mistake of presuming the treatment for lactose intolerance requires a dairy free diet. Consequently, poor calcium and protein intake result, increasing the long-term risk of osteoporosis. Formal education by a dietitian will ensure that the client is aware of the availability of lactose-free milk and yogurt (regular cow’s milk/yogurt treated with lactase enzyme), alternatives to lactose containing foods as well as their ability to tolerate small amounts of cream, soft cheeses and chocolate and the lactose-free nature of hard cheeses and butter (Table 5). We encourage lactose free cow’s milk products rather than soy, as in our experience we have a high number

**Table 5**  
**Lactose-containing foods and best alternatives\***

<i>Lactose-containing foods</i>	<i>Best alternatives</i>
Cow’s milk (full cream and low fat)	Lactose free cow’s milk, rice milk
Ice cream	Gelati/sorbet (watch fruit juices)
Custard	Custard powder made with lactose free milk Commercial lactose free custard
Evaporated milk	No suitable alternative Purchase lactase enzyme and formulate own
Condensed milk	No suitable alternative Purchase lactase enzyme and formulate own
Yogurt	Lactose free yogurt
Soft cheeses (ricotta, cottage, cream)	Use safely in small amounts

\*Lactase enzyme can be purchased and used to formulate lactose free products. Other dairy products not listed here contain insufficient lactose to cause symptoms in people with lactose intolerance due to lactose malabsorption.

of clients who react with gastrointestinal symptoms to the fermentable galactans in soy products.

### Oligosaccharides

**Physiological basis:** Oligosaccharides with fructose chains (fructo-oligosaccharides, fructans) or galactose chains (galacto-oligosaccharides or galactans) are common in the diet. The average intake is uncertain, but older data from the USA indicated an average of 10 g of fructans per day (29). This may be a considerable underestimate as tables of food fructan content are incomplete, and inulins and fructooligosaccharides are now commonly added to commercial food products. Galactan content varies considerably according to the intake of legumes. The mammalian small intestine does not contain hydrolases that can split the fructose-fructose and galactose-galactose bonds. Thus, fructans and galactans are almost entirely malabsorbed in all people. The chain length (degree of polymerization or

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**Table 6**  
Foods containing low DP (degree of polymerization) fructo-oligosaccharides

Wheat (as the main ingredient)*	Onion	Spring onion
Spanish onion	Leeks	Shallot
Garlic	Artichokes	Asparagus
Zucchini	Custard apple	Grapes, Thompson
Mango	Peach, white	Peach, yellow
Persimmon	Pineapple	Rambutan
Watermelon	Inulin	Fructo-oligosaccharides

\*Wheat is a problem in large amounts, i.e. when it is the main ingredient in a product. This would include breads, pasta, biscuits, noodles, cakes, pastries. Patients are advised not to be concerned with wheat-derived ingredients such as wheat thickener, maltodextrin and dextrose as these contain negligible amounts of fructans.

DP) varies, but oligosaccharides are considered to have a DP less than 10. Longer fructans are generally referred to as inulins. Theoretically, fructans with a short DP will have greater effects when malabsorbed due to more rapid digestion and greater osmotic effect. Indeed, most fructans in food are short. However, inulins can also induce functional gut symptoms, particularly increased wind (20).

**Problematic foods:** Wheat, members of the onion family and artichokes are the major sources of fructans in the diet, but a more complete list is shown in Table 6. Foods containing high DP fructans (such as rye) appear in clinical practice to be better tolerated and are considered less problematic. The legumes are the most common source of galactans (such as raffinose and stachyose) (Table 7). Restricting galactans can be challenging in a vegetarian diet with much discussion required about alternative protein sources that the client can safely include.

**Polyols**

**Physiological basis:** Polyols are poorly absorbed with at least 70% being recovered, with sorbitol the most common polyol in the diet. Their absorption is not accelerated by co-ingestion with glucose but seems to be worse when given concomitantly with fructose. An

**Table 7**  
Foods containing raffinose and other galacto-oligosaccharides

Baked beans	Lentils	Chickpeas
Kidney beans	Black eye beans	Borlotti beans
4 bean mix	Cabbage	Brussel sprouts
Green beans	Yellow beans	Butter beans

additive effect with fructose on symptom generation is well described (30,31).

**Problematic foods:** Sorbitol is the most commonly consumed polyol with its high presence in some fruits and its use as an artificial sweetener. Table 8 lists foods/commercial products containing polyols and artificial sweeteners.

**How the Diet is Delivered**

Dietary education is most suitably undertaken with a one-on-one consultation. This allows the dietitian to gain a broad understanding of symptoms experienced, and also ensures the information provided is personalized. A diet history is taken to screen the individual's diet for FODMAP content to determine the most likely contributors. The role of FODMAPs in symptom generation is explained, and each FODMAP is discussed with lists of problem foods provided. Education can be tailored to the individual so there is little focus on

**Table 8**  
Foods containing polyols

Fruits	Artificial sweeteners*
Apples	Sorbitol
Pears	Mannitol
Apricots	Isomalt
Peaches	Xylitol
Plums	
Cherries	
Nectarines	

\*Found especially in artificially sweetened gums, mints and lollies. In addition, toothpastes, liquid cough or other medications may contain them as well.

FODMAPs that are never consumed. Lists of suitable foods are vital to assist the patient in considering what they are able to safely eat. The skilled dietitian will encourage appropriate alternatives to improve compliance and palatability. They are also provided with a sample meal plan to help with meal and snack ideas.

For those that have shown evidence of fructose malabsorption and/or lactose intolerance on breath testing, group sessions are available. This is primarily because of the demand for dietary education in this patient group and an inability to see all patients individually due to time constraints. Other FODMAPs (e.g. sorbitol and raffinose) may be discussed briefly at these sessions, but usually an individual consultation is undertaken at a later date to assess progress, discuss additional potential triggers, and refine dietary management as required.

## FREQUENTLY ASKED QUESTIONS ON LOW FODMAP DIET

### *What is the role of breath hydrogen tests?*

Not all sufferers of functional gut symptoms poorly absorb fructose and lactose. The results of breath hydrogen testing enable the dietitian to know whether to exclude/include fructose and/or lactose as a problem FODMAP and will potentially ensure that dietary restrictions are imposed only when necessary. Undergoing the tests also primes the patient to be receptive of dietary advice and may facilitate the educative process. However, these tests are not mandatory. A skilled dietitian will be able to screen their diet for FODMAPs and a dietary trial for six-to-eight weeks limiting those FODMAPs in significant amounts is commenced. A dietary challenge follows to determine which FODMAPs cause the majority of symptoms.

### *How do FODMAPs relate to small bowel bacterial overgrowth (SBBO)?*

In one study, FM was reversed in many patients by the use of antibiotics (26) suggesting that SBBO was a major factor in the incomplete absorption of fructose. This is consistent with the concept that FM has multiple contributing factors and that it reflects more than just reduced absorption due to impaired function of fructose absorptive mechanisms. Fructose is like a

“fast food” for bacteria and its presence in the lumen of the distal small bowel may itself lead to increased bacterial populations. Furthermore, fructose is converted to fructans by many bacteria and this process promotes adherence of bacteria to teeth in the pathogenesis of dental caries. If such an effect occurs in the small intestinal biofilm, luminal fructose will further enhance the likelihood of SBBO. In other words, FM and SBBO may be interdependent factors and dealing with one might affect the other. Thus, antibiotics reduce FM (26) and an elemental diet (i.e. nutritional formula not requiring digestion, where bacterial substrates are diminished) reduces SBBO (32). Alternative options exist, therefore, for managing patients with functional gut symptoms and FM with antibiotics or dietary reduction of FODMAPs. Since the problem is chronic, dietary change is a lot more attractive than long term or recurrent antibiotics as therapy. However, comparative studies have not been performed and it is unknown whether the dietary FODMAP restriction and antibiotics have an additive effect.

### *How long do you wait to assess the response?*

Experience indicates that response to the diet usually occurs within the first week, but there is a clear increase in efficacy over the first eight weeks. It is recommended that six-to-eight weeks strict adherence to the diet occur prior to declaring it a failure.

### *How long do patients need to continue with the diet?*

Patients in whom symptoms have improved significantly are then encouraged to challenge their diet with individual FODMAPs (such as a challenge of sorbitol with stone fruits). If they have not had breath testing, they will also be encouraged to trial fructose and lactose (each trial a minimum of two weeks apart). Fructans and raffinose are not challenged, as it is known that they will be malabsorbed in all cases. However, when symptoms have improved, individuals may trial a reintroduction of small amounts of fructans and/or raffinose to determine their tolerance level for these carbohydrates.

If after eight weeks there is no improvement in symptoms, there seems little to be gained in continuing the diet. However, many non-responders report that



although their symptoms continue, when they do consume high FODMAP foods they notice immediate exacerbation of symptoms. For this reason, such patients usually choose to follow the diet despite insufficient improvement in symptoms. In responders, the diet should continue as long as it is needed.

#### ***How well do patients understand the diet?***

A key to sustained adherence to the diet is adequate understanding by the patient of the concepts upon which the diet is based. From a dietitian's perspective, fructose malabsorption can be a difficult condition to describe. It is important that the physiology behind the fructose malabsorption diet is explained, as it does not require a "fructose free" diet. The fructose malabsorption education session must also include a discussion regarding the fact that the fructose-glucose absorptive capacity can be saturated. Medium to long-term evaluation of knowledge of and adherence to the diet when educated by a dietitian is very good (15).

#### ***What is the effect of cooking and food preparation on FODMAP content?***

There are few data on whether the FODMAP content of foods changes sufficiently to enable inclusion in a patient's diet under certain conditions. Research is currently underway to assess this, particularly in garlic and onions as patients often find these foods the most difficult to avoid due to their wide use in restaurants and commercial products.

#### ***Can patients tolerate the diet in the longer term and maintain nutrition?***

The low FODMAP diet includes limiting several different carbohydrates and can appear quite restrictive, however, the multitude of wheat free products available and the fruits with balanced glucose and fructose concentrations ensure that the diet is nutritionally adequate. The main concern the dietitian faces is whether fiber intake is sufficient. If all the suitable alternative foods are consumed, such as safe fruits, multigrain gluten-free bread and brown rice, then fiber is of no concern, but difficulties are faced with patients who dislike the alternatives. Such patients are encouraged to trial a variety of breads such as rye bread and include at least two servings of safe fruits and five servings of vegetables daily.

#### ***Is dietitian-led instruction necessary or can the diet be administered with a dietary sheet?***

The education session for a FODMAP-restricted diet is paramount to the success of the therapy. A skilled dietitian is required to spend considerable time examining the patient's diet for potential triggers, explaining the physiology of the malabsorption, the safety of glucose and fructose in balance, and portion sizes of high fructose load foods. It is also important that the patients are given sufficient food alternatives to maintain nutritional adequacy, as well as cooking tips and quick meal and snack ideas. Compliance will undoubtedly be low without such information, leaving the dietary therapy too difficult to adhere to and therefore unlikely to succeed. Current information available on the Internet is inaccurate and confusing. A patient attempting his or her own research into the condition is unlikely to find appropriate information, resulting in a loss of faith in the therapy. This makes it very difficult for a dietitian in the future to convince this patient to retrial the FODMAP approach. As such, all patients complaining of IBS-like symptoms who have had all other gastrointestinal disorders excluded must see a dietitian for appropriate FODMAP education. Educating the patient via a diet sheet alone, while attractive, has not been validated. It is likely that there would be selective uptake of aspects of the diet with reduced efficacy.

## **CONCLUSION**

After years of unsatisfactory dietary manipulation, it is evident that a low FODMAP diet is successful in providing relief from functional gut symptoms, whether in the setting of a FGD or IBD. More research is required into the FODMAP content of foods around the world, as differences in food composition are known to vary. Refinement in the dietary advice given will improve the diet, especially with regard to completion of the analysis of all foods for their FODMAP content. Further studies are required to correct the limitations associated with current food composition tables, to refine this dietary approach, and to better explore its application in patients with irritable bowel syndrome and other disorders such as inflammatory bowel and celiac disease. ■

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