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ADVANCES IN CLINICAL PRACTICE

Evidence-based dietary management of functional gastrointestinal symptoms: The FODMAP approach

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Key words
dietary therapy, fermentable carbohydrates, fructans, fructose, galactans, irritable bowel syndrome, lactose, polyols.

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Conflicts of interest: SJS has published five cookbooks directed towards issues of celiac disease and low FODMAP diet. The authors have registered the term 'FODMAPs' as a trademark.

Abstract

Background and Aim: Functional gastrointestinal symptoms are common and their management is often a difficult clinical problem. The link between food intake and symptom induction is recognized. This review aims to describe the evidence base for restricting rapidly fermentable, short-chain carbohydrates (FODMAPs) in controlling such symptoms.

Methods: The nature of FODMAPs, their mode of action in symptom induction, results of clinical trials and the implementation of the diet are described.

Results: FODMAPs are widespread in the diet and comprise a monosaccharide (fructose), a disaccharide (lactose), oligosaccharides (fructans and galactans), and polyols. Their ingestion increases delivery of readily fermentable substrate and water to the distal small intestine and proximal colon, which are likely to induce luminal distension and induction of functional gut symptoms. The restriction of their intake globally (as opposed to individually) reduces functional gut symptoms, an effect that is durable and can be reversed by their reintroduction into the diet (as shown by a randomized placebo-controlled trial). The diet has a high compliance rate. However it requires expert delivery by a dietitian trained in the diet. Breath hydrogen tests are useful to identify individuals who can completely absorb a load of fructose and lactose so that dietary restriction can be less stringent.

Conclusions: The low FODMAP diet provides an effective approach to the management of patients with functional gut symptoms. The evidence base is now sufficiently strong to recommend its widespread application.

Functional gastrointestinal disorders (FGID) are very common and present as major challenges for clinicians, particularly as pharmaceutical therapies offer little more than mild palliation in the vast majority of patients. The symptoms can markedly interfere with quality of life and rank second in the causes of absence from work or school.[1] While the predominant underlying cause of symptoms appears to reside in the enteric nervous system, manifesting as visceral hypersensitivity and/or motility disturbances, multiple other factors contribute to symptoms generation, including psychological factors and diet. Consequently, treatment has spanned multiple modalities and has involved a variety of health professionals, including medical practitioners, psychologists, hypnotherapists, dietitians and naturopaths, each bringing a different perspective. A major limitation has been the limited evidence base for many therapies, not helped by the considerable placebo response seen in these disorders. However, dietary therapy, specifically the low FODMAP diet (see below for explanation), has now emerged as a key player with a well-substantiated mechanism of action and evidence-based efficacy. This review will describe the theoretical basis for the diet, the evidence for efficacy and its implementation, and it will address unanswered questions.

Mechanistic basis for dietary intervention

The physiological basis for the genesis of many functional gut symptoms is luminal distension. Evidence for this comes from barostat and gas infusion studies.[2,3] Luminal distension not only induces the symptoms of pain, the sensation of bloating and visible abdominal distension, but may also lead to secondary motility changes. Thus, minimizing the consumption of dietary factors that can distend the intestine would theoretically lead to improvement in global symptoms that characterize FGID. In the case of two of the most common types of FGID involving the intestine, irritable bowel syndrome (IBS) and functional bloating, the distal small and proximal large intestine would be the target regions of the gut.

The intestinal lumen can be distended by solids, liquids and gas. Solids can be altered in the proximal large intestine by changing the dietary fiber content both directly and indirectly via expansion or contraction of the bacterial mass. The liquid content in the distal small intestine will be dictated by the osmotic load in the lumen, and in the proximal large intestine by the osmotic load and the absorptive ability of the epithelium. The gas content will

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include a component of swallowed nitrogen, but the majority will be locally produced by bacterial fermentation. The volume that the gas creates will depend upon the number of molecules and its diffusion capacity across the epithelium and into the circulation. Dietary components that will putatively lead to luminal distension in the regions of interest will therefore be poorly absorbed in the proximal small intestine, will be small molecules (i.e. osmotically active), will be rapidly fermented by bacteria (with the potential to be fermented by small intestinal as well as cecal bacteria and to expand the bacterial population), and will be associated with hydrogen rather than methane production. Dietary FODMAP are the best fit for these principles.

FODMAPs

The acronym, 'FODMAP'—Fermentable Oligo-, Di- and Monosaccharides and Polyols—was coined to describe a previously-unrelated group of short-chain carbohydrates and sugar alcohols (polyols).[4] They comprise fructose, lactose, fructo- and galacto-oligosaccharides (fructans, and galactans), and polyols (such as sorbitol, mannitol, xylitol and maltitol) all of which putatively have three common functional properties:

- *Poorly absorbed in the small intestine:* Poor absorption occurs by virtue of slow, low-capacity transport mechanisms across the epithelium (fructose), reduced activity of brush border hydrolases (lactose), lack of hydrolases (fructans, galactans), or molecules being too large for simple diffusion (polyols).
- *Small and therefore osmotically-active molecules:* This effect has been demonstrated with, for example, a synthetic FODMAP, lactulose, which exerts a laxative effect when given in sufficient dose by increasing the liquidity of luminal contents and subsequently affecting gut motility.[5]
- *Rapidly fermented by bacteria:* The rapidity of fermentation by bacteria is dictated by the chain length of the carbohydrate; oligosaccharides and sugars are very rapidly fermented compared with polysaccharides such as soluble dietary fibre.[6]

These functional properties have recently been confirmed in studies in which diets high and low in FODMAPs (rather than pure individual FODMAPs) have been fed to volunteers. In a study of 10 ileostomates, changes in dry-weight ileostomy effluent could be explained entirely on the basis of dietary FODMAPs and the effluent volume increased by a mean of 22% on the high FODMAP diet.[7] Fermentation of FODMAPs in the small intestine was suggested by the recovery of only 34% of FODMAPs consumed in the ileostomy effluent, although some fermentation in the ileostomy bag *ex vivo* also would have contributed. When the diets were fed to healthy volunteers, breath hydrogen production, a marker of gas production in the intestine, was markedly elevated throughout the day.[8] Furthermore, in methane-producers, high FODMAP intake favored production of hydrogen over methane, which occupies a smaller volume per hydrogen molecule generated. Thus, all the putative functional properties have been confirmed to occur *in vivo* in association with dietary intake of FODMAPs.

There is considerable evidence that individual FODMAPs induce abdominal symptoms. Acute provocation tests with lactose,[9] fructose[9–11] fructo-oligosaccharides (FOS)[12,13] or sorbitol[9,14–17] cause abdominal symptoms such as bloating, pain, nausea and disturbed bowel habit (diarrhea and/or constipation) in many people, especially those with IBS.[15] The role of lactose and polyols in the induction of gut symptoms has been well-described in clinical practice; the dietary regimen for the management of lactose malabsorption has been comprehensively addressed[18] and mandatory declaration of 'excess consumption may have a laxative effect' is in place for food products containing polyols. Increased flatulence and change of bowel habits after consuming 'windy vegetables', such as lentils and baked beans, are common knowledge although identification of galactans, in addition to resistant starch, as the culprit molecules may not be. Additive effects fructose and sorbitol[10,19,20] and lactose and fructans[21] on abdominal symptoms are also well-described.

The FODMAP concept in the management of functional gut symptoms

There are two key components to the FODMAP concept.

- *The dietary approach restricts FODMAP intake globally, not individually.* Restriction of individual FODMAPs has been used with varying success in the management of functional gut symptoms for a long time. The best example is restriction of dietary lactose in patients with hypolactasia. Restriction of fructose, with or without sorbitol, has also been reported. However, such approaches have not become widespread in their application, perhaps in part related to their limited success. Restricting one FODMAP in isolation ignores the likelihood that there is potentially a range of FODMAPs in the diet, all of which have similar end-effects in the bowel. The innovation in the FODMAP concept is that global restriction should have a far greater and more consistent effect than limited restriction. Thus, the central focus is to reduce the intake of all poorly absorbed short chain carbohydrates to be more effective in reducing luminal distension than merely concentrating on one of these. Such a global approach to restricting carbohydrates that have similar actions (high osmotic effect and rapid fermentation) should optimize symptom control in patients with IBS.
- *FODMAPs do not cause the underlying FGID, but represent an opportunity for reducing symptoms.* This concept is important as it steers away from the more traditional concepts of lactose 'intolerance' versus 'malabsorption' and fructose 'intolerance' versus 'malabsorption'. The reason the symptoms are triggered by the ingestion of lactose or fructose in the individual is the response of the enteric nervous system to luminal distension (due to visceral hypersensitivity, excessive gas production due to the nature of the resident microbiota, or motility problems with clearance of the fluid/gas) not because the malabsorption of the sugar is abnormal or a 'condition'. After all, delivery of dietary FODMAP to the distal small and proximal large intestine is a normal phenomenon, one that will generate symptoms if the underlying bowel response is exaggerated or abnormal.

FODMAPs in the diet

While all FODMAPs are potentially important in the genesis of symptoms (summary of food sources of FODMAP are listed in Table 1), the relative contribution of different subgroups of FODMAPs varies across ethnic and dietary groups due to the dose delivered in the diet. In North American and Western European diets, fructose and fructans are by far the most widespread in the

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Table 1 Food sources of FODMAPs (where FODMAPs are problematic based on standard serving size) and suitable alternatives

FODMAP	Excess fructose	Lactose	Oligosaccharides (fructans and/or galactans)	Polyols
Problem high FODMAP food source	*Fruits:* apples, pears, nashi pears, clingstone peaches, mango, sugar snap peas, watermelon, tinned fruit in natural juice	*Milk:* cow, goat and sheep (regular & low-fat), Ice cream	*Vegetables:* artichokes, asparagus, beetroot, Brussels sprout, broccoli, cabbage, fennel, garlic, leeks, okra, onions, peas, shallots.	*Fruits:* apples, apricots, cherries, longon, lychee, nashi pears, nectarine, pears, peaches, plums, prunes, watermelon
		Yoghurt (regular & low-fat)		*Vegetables:* avocado, cauliflower, mushrooms, snow peas
	Honey	*Cheeses:* soft & fresh (e.g. ricotta, cottage)	*Cereals:* wheat & rye when eaten in large amounts (e.g. bread, pasta, couscous, crackers, biscuits)	*Sweeteners:* sorbitol(420), mannitol(421), xylitol(967), maltitol (965), isomalt (953) & others ending in '-ol'
	Sweeteners: fructose, high fructose corn syrup		*Legumes*: chickpeas, lentils, red kidney beans, baked beans	
	Large total fructose dose: concentrated fruit sources; large serves of fruit, dried fruit, fruit juice		*Fruits:* watermelon, custard apple, white peaches, rambutan, persimmon	
Suitable alternative low-FODMAP food source	*Fruit:* banana, blueberry, carambola, durian, grapefruit, grape, honeydew melon, kiwifruit, lemon, lime, mandarin, orange, passionfruit, paw paw, raspberry, rockmelon, strawberry, tangelo.	*Milk:* lactose-free, rice milk	*Vegetables:* bamboo shoots, bok choy, carrot, celery, capsicum, choko, choy sum, corn, eggplant, green beans, lettuce, chives, parsnip, pumpkin, silverbeet, spring onion (green only), tomato	*Fruits:* banana, blueberry, carambola, durian, grapefruit, grape, honeydew melon, kiwifruit, lemon, lime, mandarin, orange, passionfruit, paw paw, raspberry, rockmelon
		Cheese: 'hard' cheeses including brie, camembert		*Sweeteners:* sugar (sucrose), glucose, other artificial sweeteners not ending in 'ol'
		Yoghurt: lactose-free		
		Ice cream substitutes: gelati, sorbet		
	Honey substitutes: maple syrup, golden syrup	*Butter*	*Onion/garlic substitutes:* garlic-infused oil	
	Sweeteners: any except polyols		*Cereals:* gluten-free & spelt bread/cereal products	

diet and therefore the ones to which nearly all patients with IBS are exposed in their everyday diet. In addition, fructose is important because its absorption in the small intestine varies widely, its significance in dietary intervention will consequently vary widely among different people, and because it is often accompanied in food by sorbitol. An understanding of fructose and fructans are therefore critical to appropriate implementation of the diet.

Fructose is presented to the intestinal lumen as a free hexose in foods or following hydrolysis of sucrose. It is present in fruits, honey, and high fructose corn syrup. It is absorbed across the small intestinal epithelium via two mechanisms (reviewed in detail elsewhere[22]). First, free fructose is taken up by a facultative transporter, GLUT-5, that is present throughout the small intestine. This is of low capacity. Secondly, when present with glucose, fructose is taken up more efficiently, a response that is believed to be related to the insertion of GLUT-2 into the apical membrane of the enterocyte. Thus, fructose malabsorption manifests when free fructose (i.e. in excess of glucose) is in the lumen. This is the reason why fructose supplied in the form of sucrose is only malabsorbed if sucrase activity is diminished.

The ability to absorb free fructose varies widely across individuals. If fructose absorption is efficient in an individual, then dietary restriction of foods rich in free fructose should be unnecessary. It is therefore desirable to identify those who *completely* absorb a load of fructose. This is effectively done by breath hydrogen testing, preferably with a moderately high dose of fructose (35 g), although the evidence base for the dose that should be tested is minimal.[23,24]

Fructans are linear or branched fructose polymers and are the naturally occurring storage carbohydrates of a variety of vegetables, including onions, garlic and artichokes, fruits such as bananas, and in cereals.[25,26] Wheat is a major source of fructans in the diet, and contains 1–4% fructans on solid matter.[27] Additional sources of fructans are inulin (mostly as a long-chain fructan) and FOS, which are increasingly being added to foods for their putative prebiotic effects. Because the small intestine lacks hydrolases capable of breaking fructose-fructose bonds, and fructans cannot be transported across the epithelium, they are not absorbed at all. Formal examination of this has confirmed that 34–90% of ingested fructans can be recovered from small intestinal output in subjects with an ileostomy.[7,26,28–30] Lower yields, particularly of the short-chain fructans, are likely to be due to microbial degradation by the microflora colonizing the distal small intestine.[7,28]

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More than just fructose and fructans

Lactose is a disaccharide naturally-occurring in mammalian milk, including that from cows, sheep and goats. Human digestion of lactose requires the enzymic action of lactase to hydrolyse the disaccharide to its constituent monosaccharides, glucose and galactose, which are then readily absorbed. As recently reviewed,[29] the activity of lactase is deficient in a proportion of adults and children, varying with ethnicity. Malabsorption of lactose (which can be detected by breath hydrogen testing, a lactose tolerance test, or lactase activity associated with small bowel biopsy) indicates that lactose should be considered a FODMAP in that individual.

Legumes, including lentils, chickpeas, and red kidney beans, are significant dietary sources of *galactans*. Vegetarians often consume large amounts of galactans due to increased consumption of legumes—these are commonly utilized as an important source of protein in the vegetarian diet, particularly those following vegan vegetarian diets. Also, people consuming cuisines that are based on these foods, such as dahl, many curries and soups from the Indian sub-continent, and chilli con carne and refried beans from Mexico, are also likely to have a greater intake of galactans.

Polyols are relatively underexplored as FODMAPs but are found widely in foods. Polyols do not have associated active transport systems in the small intestine and are probably absorbed by passive diffusion. The rate of absorption is related to three factors. First, the diffusion occurs through 'pores' in the epithelium and therefore depends upon molecular size.[30] For example, erythritol, a 4-carbon polyol, is well-absorbed in the jejunum but mannitol, a 6-carbon polyol, is not.[31] Secondly, there is variation of pore size along the small intestine with larger pores proximally. Thus, erythritol is less well-absorbed in the ileum.[32] The rapidity of transit through the jejunum will therefore influence the degree of absorption. Finally, pore size is affected by mucosal disease; pore size reduces in celiac disease where erythritol is poorly absorbed.[32] It is not surprising then that limited studies performed on the absorption of sorbitol and mannitol have yielded considerable individual variation and that the amount available for fermentation varies with dose taken.[33,34] Polyols are present in food (for example, sorbitol is often found in food rich in free fructose, mannitol is found in mushrooms) and are used as artificial sweeteners, being identified by the following additive numbers on food packages: sorbitol (420), xylitol (967), mannitol (421), maltitol (965), and isomalt (953). Sorbitol has also been marketed as a laxative and warnings have been placed on candies, especially sugarless chewing gum, that polyols used as an artificial sweetener can have a laxative effect.

Efficacy of the low FODMAP diet

Efficacy of restricting dietary fructose and/or sorbitol has been reported in several observational studies[35–38] and the benefits appeared to be durable.[36] The nature of the diets used, however, was generally poorly defined. Using a well-defined diet, restriction of fructose and fructans, together with general avoidance of other FODMAPs led to impressive global symptoms response in three out of four patients with IBS and fructose malabsorption in a retrospective study.[39] Efficacy was durable and was closely related to dietary compliance. That the efficacy of the low FODMAP diet was due to restriction of fructose and/or fructans in the diet was convincingly shown in a subsequent double-blinded randomized quadruple-arm placebo-controlled re-challenge trial.[40] Further evaluation of the diet in other groups with functional gut symptoms has shown consistent benefit in patients with quiescent inflammatory bowel disease and ileal pouch. An evaluation of patients who did not have a breath hydrogen test supported the efficacy of the diet in those with complete fructose absorption.[41] Of importance is that efficacy is not restricted to patients with diarrhea-predominant IBS, but applies equally to any bowel habit. Thus, the evidence base for efficacy of the diet is now substantial.

The ability of those instructed in the low FODMAP diet to adhere to it is remarkably good. More than 75% of patients were judged to be completely or mostly compliant with the diet in a retrospective review median 14 months (range 2–40 months) after implementation of the diet. In patients with inflammatory bowel disease, dietary compliance and efficacy of the diet were associated with more time availability, higher education status, and the use of specific cookbooks. These findings suggested that an understanding of the dietary principles and allocation of time to work on applying the diet were important to ensure success. These findings are not surprising.

Limitations of tables of the FODMAP content of foods

There are limitations in developing tables of FODMAP-rich and FODMAP-poor foods. This review paper provides a summary that is incomplete and is useful as a guide only.

- Published lists of foods are generally limited in the description of FODMAP content. This limitation has been assisted by the development of methodologies to measure FODMAP content, together with a systematic examination of fruits, vegetables and cereals.[25,26]
- The cut-off levels of FODMAP content, which dictates whether it is classified as 'high' or not, are not clearly defined. This is further complicated by the fact that the total of FODMAPs ingested (not the individual FODMAPs) at any one meal is a major factor in determining whether symptoms will be induced or not. In the original description of the diet,[39] cut-off values were based on careful clinical observation, which included obtaining feedback from patients regarding foods that they identified as triggers for symptoms. The foods reported by patients as being troublesome were examined for trends in the pooled food composition table. Foods and beverages containing > 0.5 g fructose in excess of glucose per 100 g, > 3 g fructose in an average serving quantity regardless of glucose intake (termed a 'high fructose load'), and > 0.2 g of fructans per serve were considered at-risk of inducing symptoms. The concept of a 'high fructose load' has not been evaluated in terms of its importance in the success of the diet.

The low FODMAP dietary strategy

The pre-dietary workup is important and is outlined in Figure 1. Breath hydrogen testing, to define who can completely absorb a load of fructose and/or lactose is very useful as it can reduce the breadth of dietary restriction that is necessary. It is not strictly necessary—the fully restricted diet can be initiated—but altering diet carries the risk of nutritional compromise and it is a good principle not to restrict foods if not necessary.

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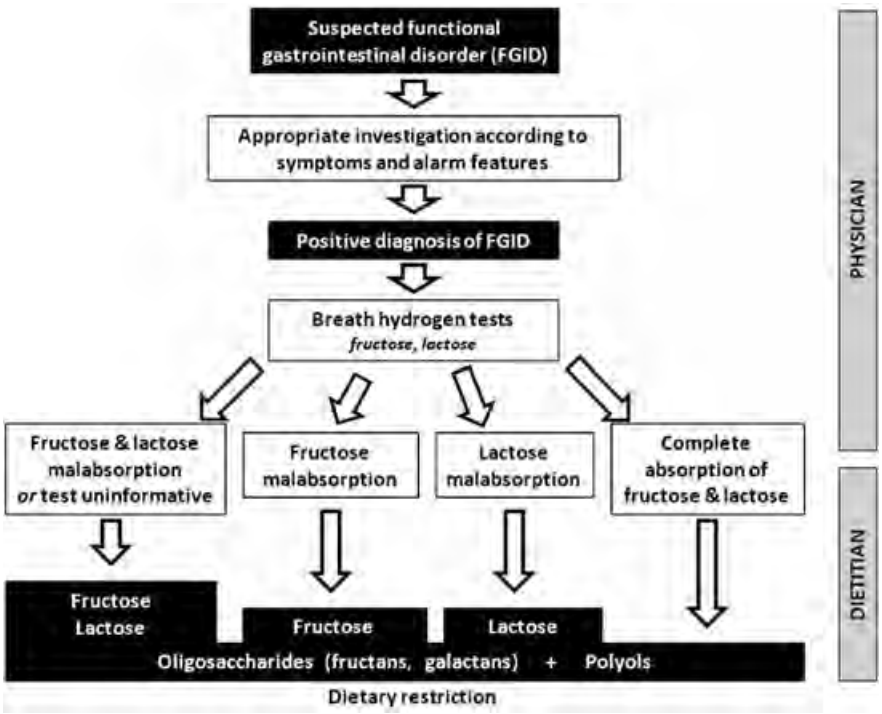


Figure 1 A bi-disciplinary approach to the patient with functional gastrointestinal disorder (FGID), especially irritable bowel syndrome or functional bloating. Breath hydrogen tests determine the degree of dietary restriction necessary by defining who can completely absorb fructose and/or lactose. Other FODMAP (oligosaccharides and polyols) are malabsorbed by all.

The low FODMAP diet has only been evaluated as a dietitian-delivered diet.[39,42] This has mostly been achieved in a one-to-one setting, but group education sessions have also been used with apparent success. The ability of written literature only to achieve efficacy has not been studied and clinicians should be cautious in undertaking such an approach. Patients often only select the parts of any diet that appeal to them and ignore the rest. This defeats the goals the diet is designed to achieve.

The strategy used at the first consultation is as follows:

- Define qualitatively the typical eating practices and style of the patient. It is important to understand the likely FODMAPs to which the patient has daily exposure. Pre-completed food recording diaries and direct questioning of the patient during the consultation can be useful methods to obtain such information. This enables individualized dietary advice to be given. For example, if a patient already omits lactose-containing foods from their diet, then this potential FODMAP would not be contributing to ongoing symptoms.
- The physiological framework for the dietary approach (i.e. the scientific basis of FODMAPs and their malabsorption and subsequent fermentation) is explained to the patient. This is pertinent as it provides the basis for a better understanding of food choice and may increase the likelihood of durable adherence (although this has not been evaluated).
- Specific dietary instructions are given to the patient:
  - Avoid foods that contain significant free fructose in excess of glucose, unless complete fructose absorption was demonstrated on breath hydrogen testing;
  - Encourage choice of foods where fructose and glucose are 'in balance', or where glucose is in excess of fructose;
  - Co-ingestion of free glucose to 'balance' excess free fructose problematic foods.
  - Limitation of dietary fructose load (in the form of free fructose or sucrose) at any one meal; and
  - Avoidance of foods that are a substantial source of fructans and galactans.
  - Restrict lactose-containing foods, if lactose malabsorption was demonstrated on breath hydrogen or lactose tolerance testing.
  - Avoidance of polyols in, for example, stone fruits and mushrooms.
- Literature providing food lists and reinforcing instructions are provided.
- Patients are provided with positive food messages emphasizing suitable food alternatives. To assist in this, verbal descriptions or visual props using packages of commercially available food alternatives are provided, together with suggestions for their use or application, and information regarding retail outlets likely to stock such foods. Several suggestions are provided to cater for 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 is also provided.
- Techniques for handling situations where control of food preparation is limited, such as eating away from home (such as restaurants, school camps and eating at friend's homes) are discussed.

As it is the total dose that will dictate the contribution to symptoms, the accumulated intake of FODMAPs over several days is critical in defining how strict an individual needs to be. In order to ensure symptoms are well-controlled, a strict trial of the low FODMAP diet is warranted for the first 6–8 weeks. On the dietetic review, assessment of symptom response will lead to discussions of individual tolerance, keeping the total FODMAP load in mind. In practice, many patients will manage, for example, occasional

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ingestion of wheat or rye breads, garlic as a minor ingredient and small serves of broccoli or cauliflower. Testing of tolerance is a vital stage of the dietetic process to ensure maximum variety in the diet.

If the symptomatic response is inadequate, specific questioning is required to determine the adherence to the dietary principles and any deficiencies corrected. If adherence was strict, attention may be needed to modify intake of resistant starch and insoluble and soluble fiber. Other dietary triggers such as food chemicals may need to be considered, as should potential factors such as caffeine, fat, meal size and regularity.

More than just FGID

There is considerable evidence to point to a strong association of functional gut disorders with inflammatory bowel disease; FGID appears to be about two- to threefold more common in than in the general community.[43,44] Functional gut symptoms in patients with quiescent inflammatory bowel disease appear to respond just as well to the low FODMAP diet.[45] Other applications include patients with troublesome frequency of bowel actions in the presence of an ileal pouch. In a pilot study, the frequency of pouch emptying was reduced when the low FODMAP diet was instituted, particularly in those who had a high dietary intake of FODMAPs, although there was little evidence of any benefit in those with pouchitis.[46] Likewise, high ileostomy output might respond to reducing dietary FODMAP intake, although the study was not performed in patients who considered their ileostomy output troublesome.[7]

Limitations and potential concerns

The diet is not a panacea for patients with FGID.[47] It provides good relief of symptoms in about 75% of patients, but has little benefit in some. Studies have yet to identify predictive factors of benefit apart from dietary adherence. Intermittent symptoms remain, albeit at a now tolerable level, in many patients since the underlying FGID is not directly addressed by the diet. Patients should not be given expectations of a 'cure'. Symptomatic hyperresponsiveness to the reintroduction of FODMAPs in the diet has been anecdotally described, although this aspect has not been formally studied. The mechanism for this has also not been evaluated. However, in rats fed fructose-poor diets, GLUT-5 expression falls as does the ability to absorb fructose from the small intestine.[48] Whether this occurs in humans warrants further investigation.

Restriction of FODMAP intake might potentially have a down side. It does mean restriction of dietary components with prebiotic effects.[49] This might potentially be detrimental to large bowel health (such as the promotion of colorectal carcinogenesis), although no studies have addressed this issue to date. The restriction of wheat-based products may lead to reduced fiber intake, but part of dietary counseling is to ensure continuing adequate intake of resistant starch and non-starch polysaccharides. This should be addressed during the dietary consultation. One study vaguely suggested that restricting FODMAPs in patients with ileal pouch, to reduce the frequency of pouch emptying, might increase the risk of pouchitis.[46] Ileostomates who have a low output from the ileostomy might depend upon the osmotic effects of FODMAPs and such patients may risk functional bowel obstruction if these are strictly reduced.[7] While these suggestions are all unsubstantiated, they do provide a reminder that this dietary intervention is established for those with functional gut symptoms and is not a diet for otherwise healthy people.

Conclusions

The low FODMAP diet provides an effective approach to the management of patients with functional gut symptoms, with an increasing evidence base. It is a dietitian-delivered diet that achieves a high degree of compliance. It provides relief of global symptoms in the majority of patients with IBS and offers improvement in functional gut symptoms in patients with inflammatory bowel disease. It warrants widespread application.

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