MedicineToday 2011; 12(3): 29-38

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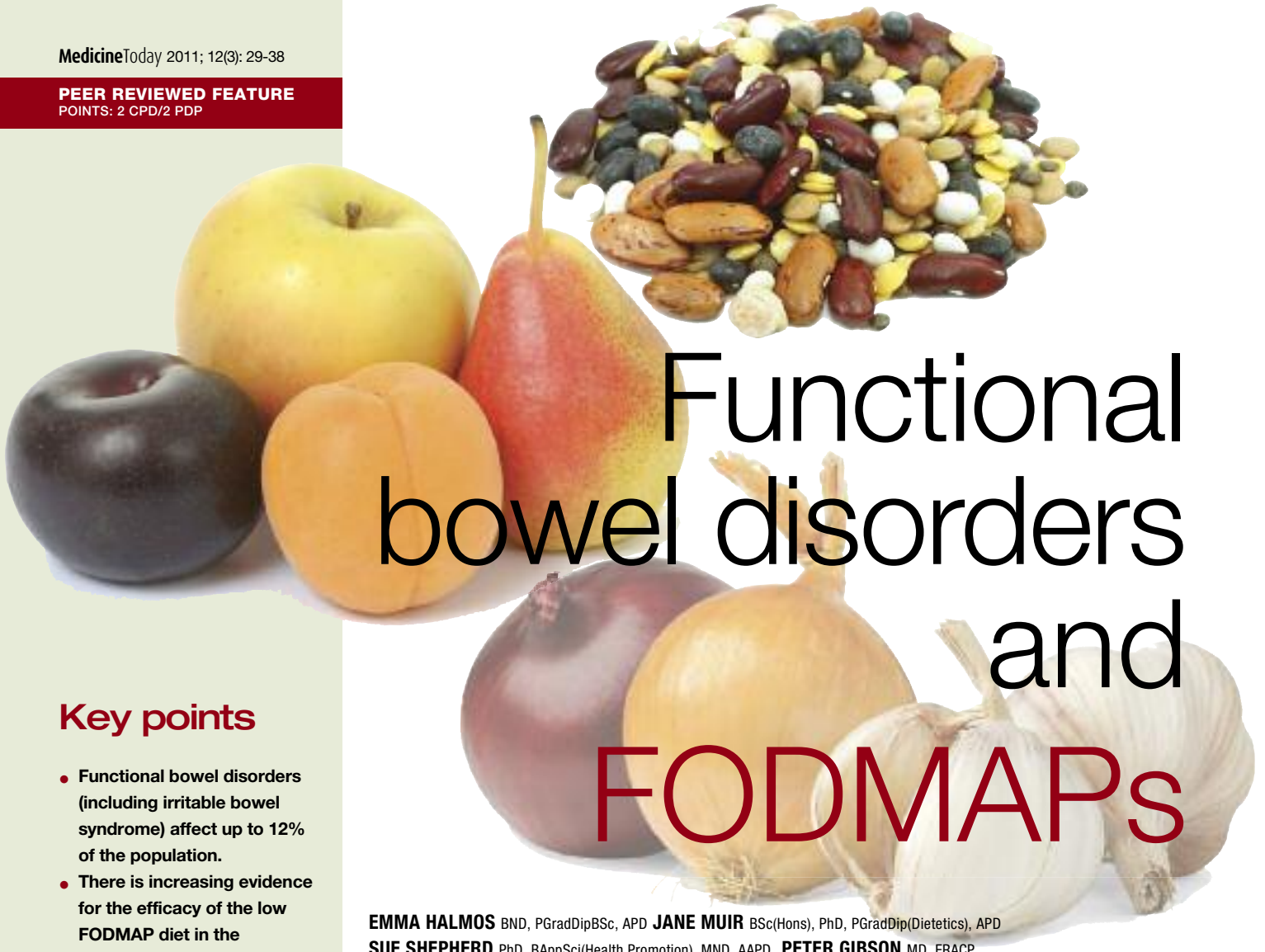

Functional bowel disorders and FODMAPs

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Key points

- **Functional bowel disorders (including irritable bowel syndrome) affect up to 12% of the population.**
- **There is increasing evidence for the efficacy of the low FODMAP diet in the management of functional bowel disorders.**
- **Breath hydrogen testing for fructose and lactose malabsorptions may help to tailor the low FODMAP diet to the individual patient.**
- **A negative result for breath testing does not exclude the diagnosis of functional bowel disorder or the use of the low FODMAP diet as restriction of the other FODMAPs may still be therapeutic.**
- **Liberalisation of the diet after symptom control has been achieved is possible in most patients.**
- **Dietary restriction through the guidance of a dietitian is recommended for patients with functional bowel disorders.**

A diet that is low in FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) has been shown to be effective in reducing gastrointestinal symptoms in patients with functional gut disorders.

Functional gastrointestinal symptoms are common and their management is often a difficult clinical problem. There is a link between food intake and symptom induction and it has been shown that restricting the intake of rapidly fermentable, short-chain carbohydrates (FODMAPs – fermentable oligosaccharides, disaccharides, monosaccharides and polyols) reduces these symptoms.[1,2] FODMAPs are widespread in the diet and their ingestion results in delivery of readily fermentable substrate and more water to the distal small intestine and proximal colon, where this osmotic load and the gas produced are likely to induce luminal distension and functional gut symptoms.[3,4]

Dietary management strategies for functional bowel disorders have changed markedly since the recent introduction of the low FODMAP diet.

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FUNCTIONAL BOWEL DISORDER

The term 'functional gastrointestinal disorder' (FGID) refers to gastrointestinal symptoms in the absence of a clear pathologically evident cause. Because FGID can originate from anywhere in the gastrointestinal tract, a symptom-based classification comprising eight categories, the Rome III classification, was developed by a consensus body.[5] Categories include chronic symptoms derived from the proximal gut (functional heartburn and functional dyspepsia) and from the small and large intestine (irritable bowel syndrome, functional bloating, functional constipation and functional diarrhoea).

Although this system is useful to ensure homogeneity in classifying patients for trials (particularly those that are multi-centred), in clinical practice there is a large degree of overlap between categories, and it is not uncommon for patients to move from one category to another over time.[6] The classification therefore has yet to be a good guide for therapeutic decision-making.

This review will use the term 'functional bowel disorder' (FBD) to cover symptoms originating from the intestine. The term includes the categories of irritable bowel syndrome, functional bloating, functional constipation and functional diarrhoea.

Why should we be interested in FBD?

Reasons for being concerned about FBD include those listed below.

- FBD is very common, with a prevalence of 5 to 12% in Western countries.[7,8]
- The various disorders are chronic conditions of unknown cause and cannot be 'cured'. Approaches therefore need to focus on strategies to manage symptoms.
- Patients with FBD commonly seek attention from medical and other health professionals. FBD accounts for up to 10% of consultations for general practitioners and 50% of referrals to gastroenterologists.[9]
- FBD can lead to substantial reduction in quality of life. The impact on quality of life is comparable with that of depression and of chronic renal failure and is associated with increased use of health care resources.[7,8,10]
- Treatment has had limited success. The therapeutic approach is 'palliative' and, until recently, therapeutic interventions have been frustratingly unreliable and targeted at treating only one symptom. For example, a patient who has IBS with constipation as the predominant bowel function can be treated with laxatives, but this often has little effect on the bloating and abdominal discomfort that are commonly found in conjunction. Furthermore, benefit of therapeutic interventions is usually marginal, with some studies reflecting no efficacy over placebo.[11]

Understanding the pathogenesis of FBD

Although the aetiologies of the various conditions included in FBD are not known, there is increasing understanding of the pathophysiological basis for the genesis of symptoms. A central underlying problem appears to reside in the enteric nervous system, which is estimated to have as many neurones and synapses as the brain and is arranged with immense complexity that includes complex reflex pathways and memory. The enteric nervous system has been called the 'gut brain'.

The two major characteristics of the enteric nervous system that are seen in patients with FBD are discussed below.

- **Visceral hypersensitivity.** Patients with FBD have a low sensory threshold. This is best demonstrated by barostat studies where distension of the gut (of the rectum by a balloon, or the stomach by liquid) leads to the sensation of distension or pain.[12] The sensory threshold of patients with FBD is often much lower than would usually be encountered. This might be due to sensitisation of the stretch receptors of the gut, which provide a major sensory input, or from altered signalling from colonic mucosa to the enteric nervous system.[12,13] The enteric nervous system may also be stimulated by bioactive molecules in the diet; this mechanism has been implicated in, for example, salicylate sensitivity. Immune stimulation in the gut in response to dietary proteins (allergic or hypersensitivity mechanisms) may also stimulate the enteric nervous system via, for example, serotonin or histamine release.[14,15]
- **Changed motility response to sensory stimulation.** This has been best demonstrated by gas distension studies where some patients with FBD do not develop the normal propulsive response (hence expelling the gas) and the gas remains within the lumen.[16]

The enteric nervous system is connected to the central nervous system, suggesting the brain also plays a substantial role in the genesis of symptoms via the 'gut–brain axis'. There is little doubt that the brain can influence motility patterns and visceral sensitivity, as shown by the commonly observed occurrence of diarrhoea and/or abdominal pain in times of stress, such as sitting for an examination.

There is also a large body of data indicating the brain's involvement in the genesis of FBD symptoms. Information sent to the brain via the enteric nervous system will cause regional patterns of stimulation that are different in patients with FBD from those in healthy controls, leading to a different interpretation of the sensory input. Thus, in patients with FBD, a similar sensory input from the enteric nervous system may be interpreted as, for example, abdominal pain rather than mild discomfort.[17] Thus, the brain is an important modifying

factor in symptom genesis and in symptom interpretation.

Current therapeutic strategies for FBD

Ideally, the best way of dealing with FBD is to 'retune' the enteric nervous system or the central nervous system, just as the best approach to food allergy or hypersensitivity is to stop the immune reaction to the inciting protein. The ability to do this is poor. Hence, other more palliative approaches to dealing with the symptoms are needed. Approaches to controlling FBD symptoms include the following:

- minimising stimulation of the enteric nervous system: potential targets for therapy include stimuli that may be present in food (having effects either directly or secondarily) and possibly also in the gut microbiota
- treating specific symptoms: pharmacological or other approaches to deal with specific symptoms, such as analgesics for pain, laxatives and prokinetics for constipation, and hypomotility agents for diarrhoea
- managing the gut–brain axis: pharmacological and psychological therapies, such as anxiolytics, antidepressants, cognitive behaviour therapy and hypnotherapy, to modulate symptom perception and the distress it causes.

Dietary change as a therapeutic tool in FBD

Until recently, dietary management of FBD has included reduction of fat (on the basis that fat influences motility); avoidance of caffeine (presumably related to its bioactive nature); alteration of dietary fibre intake (to improve the quality of colonic contents); and elimination of suspected dietary triggers such as milk and wheat, with a purely trial-and-error approach.[9,18,19]

Many other dietary regimens, including low salicylate, gluten-free and very low carbohydrate diets, have been reported in the medical literature or are discussed on the internet. Although many of these diets may produce some improvement in symptoms, the evidence for efficacy is anecdotal only and there is no rational way of matching an appropriate dietary modification to individual patients. Another problem is that protocols using strict elimination diets followed by fixed rechallenges are labour-intensive and require seriously devoted patients to carry them out. Such protocols also give no opportunity to assess dosing of dietary triggers in the induction of symptoms.

Dietary management strategies for FBD have changed markedly since the recent introduction of the low FODMAP diet. This dietary approach is based on known and validated pathophysiological principles and has a high level of evidence for efficacy.[2,4,20] Moreover, this treatment appears effective across many centres and is supported by detailed food composition analysis.[21,22]

TABLE 1. FODMAPS AND GASTROINTESTINAL SYMPTOMS IN PATIENTS WITH FUNCTIONAL BOWEL DISORDERS

Property of FODMAPs	Outcome
Poorly absorbed in the small intestine	Delivery to the colon of fructans, galacto-oligosaccharides and polyols, and in some people, fructose and lactose
Osmotically active	A laxative effect by increasing liquidity in luminal contents, thereby affecting gut motility
Rapidly fermented by colonic bacteria	Production of gas causing luminal distension

FODMAPS

The acronym FODMAPs refers to fermentable short-chain carbohydrates that are poorly absorbed and may then have effects on the gastrointestinal tract – fermentable oligosaccharides, disaccharides, monosaccharides and polyols.[1] The specific carbohydrates are:

- oligosaccharides: fructans (including fructo-oligosaccharides [FOS] and inulin) and galacto-oligosaccharides (GOS)
- disaccharide: lactose
- monosaccharide: fructose (in excess of glucose, see later under 'Small intestinal absorption of FODMAPs')
- polyols: including sorbitol and mannitol.

The effects of the various FODMAPs are additive and these carbohydrates should be viewed together as a group rather than individually.

The concept of reducing dietary FODMAPs to control gastrointestinal symptoms in patients with FBD relates to their physiological effect. FODMAPs are poorly absorbed in the small intestine and have the same physiological effects in the distal small bowel and proximal colon, namely luminal distension with solids, liquids and gas. FODMAPs contribute particularly to luminal distension by liquids and gases: liquids because of the large osmotic load they cause due to their small molecular size, and gases because of their rapid fermentation by intestinal bacteria (rapid due to their short chain length).[4] They have a smaller contribution to the solids content, through their fibre content and the microbial mass expansion associated with their fermentation.

The increased water content of the colon exerts a natural laxative effect, and the fermentation of the unabsorbed FODMAPs by intestinal bacteria leads to the production of short-chain fatty acids as well as gas.[3,4] In small amounts, some FODMAPs (fructans and GOS) may exert

beneficial prebiotic effects by promoting differential expansion of bacterial populations, which will suppress potential colonic pathogens.[23]

Conversely, the increased water load and gas production caused by FODMAPs can lead to laxation and distension of the bowel that results in symptoms of bloating, pain and secondary motility disturbances.[4] Such effects are exaggerated in patients with FBD because of the presence of visceral hypersensitivity and abnormal motility responses. This explains why FODMAPs exacerbate gastrointestinal symptoms in patients with FBD, with studies confirming this hypothesis.[3,4] Furthermore, studies have shown that reducing the intake of dietary FODMAPs in this population group has been successful in reducing gastrointestinal symptoms.[2,20] The features unique to FODMAPs that explain this impact are listed in Table 1.

Small intestinal absorption of FODMAPs

The various FODMAPs are digested and adsorbed in the small intestine to differing extents. There are three patterns of digestion and absorption, as described below.

- **Totally malabsorbed: the fructans and GOS.** As the human body does not possess enzymes to hydrolyse these oligosaccharides, they cannot be absorbed and their fate is to be fermented by bacteria in the distal small intestine and, particularly, the colon.[4,24]
- **Partly malabsorbed in all people: the polyols.** Absorption of polyols is considered to be by passive diffusion, which is inefficient (because of the presence of the attached alcohol group). Thus, there will usually be some malabsorption of polyols; indeed, on average, about 70% of sorbitol (the most commonly found dietary polyol) will be malabsorbed.[25]
- **Partly malabsorbed in only some people: fructose and lactose.** In the presence of equimolar glucose, fructose is rapidly absorbed by the proximal small intestine due to glucose-mediated activation of a high capacity transporter. However, fructose in excess of glucose ('free fructose') is transported by a slow and low capacity transporter that acts all along the small intestine.[24] This transporter varies in its capacity across individuals and can be readily overloaded by too much free fructose or by too rapid a transit along the small intestine.[24] Lactose, on the other hand, is a disaccharide (galactose plus glucose) and requires splitting into its component molecules (by lactase in the small intestinal brush border) to be absorbed. The activity of lactase varies across individuals according to ethnicity and other factors. Thus, only some people malabsorb fructose and/or lactose.

THE LOW FODMAP DIET

Determining FODMAP absorptive capacity

As the aim of the low FODMAP diet is to restrict the intake

of short-chain carbohydrates that are poorly absorbed and delivered to the colon, it would be helpful to know whether fructose and/or lactose is completely absorbed in an individual because this may permit a less restrictive diet. Fructose and lactose absorption may be explored through dietary trials, but experience has shown the inaccuracy of this method. It is preferable to determine an individual's absorptive capacity by performing breath hydrogen tests.

Breath hydrogen tests are based on the principles that the only source of hydrogen in the breath is from bacterial fermentation of carbohydrates in the intestine and that a rise in breath hydrogen following the ingestion of a specific carbohydrate implies malabsorption of that carbohydrate. A relatively large dose of lactose (50 g) or fructose (35 g) is generally given to determine whether complete absorption is achieved. Results showing absorption of the load given indicate that restriction of that particular short-chain carbohydrate is not necessary. Interpretation of the test can be improved by also performing a test with lactulose (15 g), which is not absorbed in anyone, as the control.

Breath methane can also be measured, but results are not as easily interpreted as breath hydrogen responses and the test should only be used if there is a poor hydrogen response (often reflected in the lactulose control breath test).

Interpreting breath hydrogen tests

There has been a tendency to misinterpret or overinterpret the results of breath testing. Important points to note include those listed below.

- Carbohydrate malabsorption is present in all populations, not just those with FBD. It is physiologically normal to have fructose or lactose malabsorption, both of which occur with similar frequency in healthy individuals and patients with FBD.[26] Therefore, breath testing does not indicate visceral hypersensitivity and is not a diagnostic test for FBD. Breath testing is therefore indicated only in symptomatic patients who would consider adopting dietary modification to treat their symptoms.
- Breath testing does not provide information as to whether the low FODMAP diet should be used; rather, it gives information on the design of the diet, tailoring it to the individual. After all, fructans and GOS, and to a lesser extent polyols, are malabsorbed by all people. Their restriction forms the core of the diet for symptomatic patients.
- The presence of malabsorption does not give an indication of the proportion of ingested sugar malabsorbed, so cannot be used to predict therapeutic reductions in food sources.
- It is difficult to interpret symptoms that are generated during (or after) breath testing because the predictive value of symptoms relates to usual intake of the sugar from dietary sources. Also, dietary FODMAPs are seldom consumed alone

TABLE 2. TOP EIGHT SOURCES OF FODMAPS AND THEIR ALTERNATIVES[21,22]

High FODMAP food	FODMAP present	Low FODMAP alternatives
Onions (all varieties) and garlic	Fructans	Chives, green section of spring onions, garlic-infused oils, ginger, chilli, fresh herbs
Wheat- and rye-based breads, pastas and cereals	Fructans	Rice, gluten-free breads and pastas, oats, cornflakes
Legumes (e.g. lentils, chickpeas, baked beans, kidney beans)	Fructans and GOS	Tofu, eggs, meat, poultry, fish
Honey	Fructose	Golden syrup, maple syrup, sugar, jam (with no added fructose)
Apples and pears	Fructose and sorbitol	Bananas, grapes, citrus fruits, strawberries
Stone fruit	Sorbitol	Bananas, grapes, citrus fruits, strawberries
Mushrooms, cauliflower	Mannitol	Broccoli, green beans, capsicum, carrot, potato, pumpkin, spinach
Milk, yoghurt, ice-cream	Lactose	Lactose-free milks, yoghurts and cheese, rice milk

and different FODMAPs have additive effects on the bowel.

Instituting the low FODMAP diet

The low FODMAP diet is a dietitian-taught diet. As with all conditions requiring dietary restriction, nutritional adequacy and, therefore, degree and length of time of restriction are important in tailoring the low FODMAP diet to the individual patient.

A dietitian with a good knowledge in implementing the low FODMAP diet will assess the patient and adapt the diet to accommodate the greatest possible food variety while achieving good symptom control, to improve the patient's quality of life. This will often involve replacing foods rich in FODMAPs (high FODMAP foods) with appropriate alternatives that are poor in FODMAPs (low FODMAP foods). Detailed lists of foods are available in the article *J Gastroenterol Hepatol* 2010; 25: 252-258 and in the literature available on the Monash University Eastern Health Clinical School's website (see the box on page 37).[23] The most common high FODMAP foods and suitable low FODMAP alternatives are shown in Table 2.[25,26]

The best procedure in finding this balance of food restriction and symptom control is to over-restrict all potential FODMAPs (while considering breath test results, if completed) until the best symptom control is achieved and maintained. This usually takes six to 12 weeks, depending on the patient's dietary compliance and symptoms.

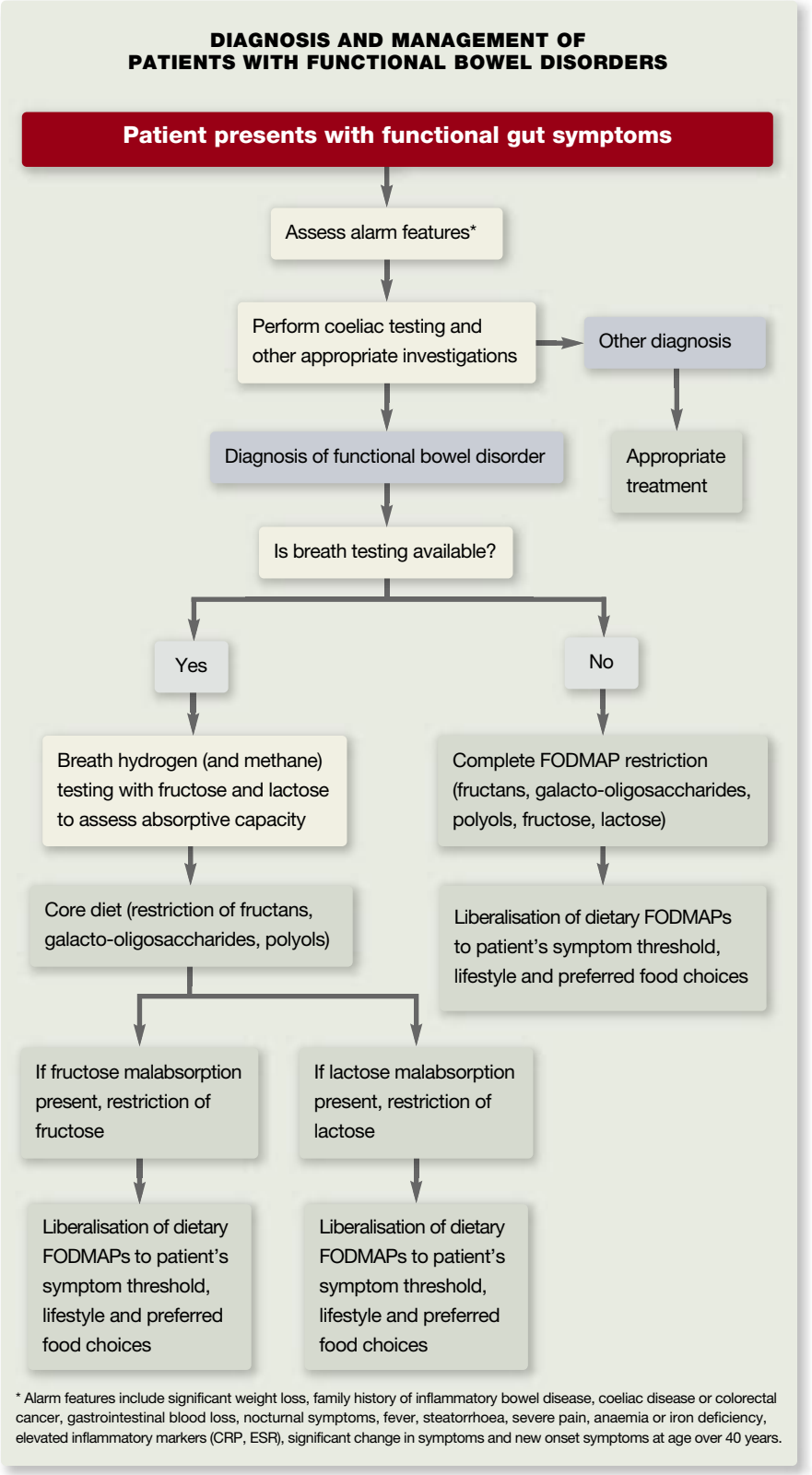
Once symptom relief is achieved, controlled doses of foods are reintroduced to assess the patient's absorptive capacity for the partly absorbed FODMAP (if breath testing has not been performed) as well as the amount of malabsorbed FODMAPs that may still be tolerated. Improvement of FBD symptoms is relatively rapid (some improvement is usually seen within days), but clinical improvement has been noted to occur over a few weeks, particularly in patients with constipation-predominant IBS who may require more time to rid the effects of luminal distension.

On review, the dietitian will aim to widen the patient's knowledge of the FODMAP-content of foods, so that the patient may best predict the role of FODMAPs in inducing symptoms. Dietary restriction may then be in the control of the patient, and be flexible enough to suit the patient's lifestyle and preferred food choices. If there is no improvement in symptoms from the low FODMAP diet and adherence to the regimen is confirmed, another symptom management strategy must be considered.

The flowchart on page 36 summarises the diagnosis and management of patients with FBD.

Modified approaches to the low FODMAP diet

Educating patients about the concept of the low FODMAP diet to reduce FBD symptoms can be quite involved. As such, the dietitian may use simplified versions or perhaps an adapted 'reduced' FODMAP diet for patients with limited education or understanding or for those with English as a second language. Elderly patients, children or patients at risk of malnutrition may also require a less restrictive approach to ensure adequate intake from a greater variety of food sources. Furthermore, a patient with a diet history that is very high in FODMAPs may require only a reduction in the amount of FODMAPs in the diet, rather than a diet that is low in FODMAPs. Conversely, patients with extreme visceral hypersensitivity may require a very low FODMAP diet with strict ongoing adherence.



Costs associated with implementing the strict low FODMAP diet are ideally short-term. Consultation with the dietitian usually only requires two consultations. Extra food costs mainly relate to specialty breads and pastas. Most low FODMAP alternatives are commonly found in the supermarket with no additional costs. Nonetheless, socioeconomic status and food availability should also be taken into account on assessment.

Success of the low FODMAP diet

In clinical practice, the low FODMAP diet is very successful in assisting the management of FBD. The diet is now widely practiced across Australia and New Zealand, in the United Kingdom and to a lesser extent in North America. Three out of four patients report significant reduction of symptoms and improvement in quality of life.[2,20]

Although patients with extreme visceral hypersensitivity may not achieve complete symptom resolution, improvement is usually enough to motivate continued compliance. Combination therapy with pharmacological agents or psychological therapies may be advantageous in this select patient group.

Long-term strategies of the low FODMAP diet

The most important aspect in restricting FODMAPs in patients with FBD is that there is a dose-dependent relation to symptom induction. Often small amounts of FODMAPs are well tolerated. Therefore, after symptom control is achieved, liberalisation of the diet to the individual threshold of the patient is encouraged to gain the health benefits of FODMAP-containing foods and to continue good food variety while still managing symptoms.

Furthermore, the extent of dietary restriction should accommodate the individual patient's satisfaction of symptom control, food knowledge, understanding of his or her condition and ability to interpret information.

OTHER USES FOR FODMAP MANIPULATIONS

In addition to reducing gastrointestinal symptoms in patients with FBD, reducing FODMAP ingestion also appears effective in other illness situations. It is effective in controlling functional gut symptoms in patients with quiescent inflammatory bowel disease and in reducing the frequency of bowel actions in patients with an ileal pouch.[27,28] It may be also be useful in decreasing effluent volume in patients with high output ileostomies, and the selecting of low FODMAP enteral solutions might be effective in preventing diarrhoea in patients receiving enteral nutrition.[3,29]

Conversely, increasing FODMAP intake may possibly improve general gastrointestinal health. FODMAPs may exert a prebiotic effect, with the putative health benefits of this. Because FODMAPs assist in laxation (as outlined above), they may play an important role in bowel function in patients with visceral sensitivity or other physiological abnormalities associated with FBD. The fermentation of FODMAPs in the large bowel yields short-chain fatty acids that appear to be anticarcinogenic. For these reasons, a low FODMAP diet should not be considered a 'diet for good health', but rather a therapeutic diet for those individuals troubled by functional gut and possibly other symptoms.

OTHER STRATEGIES FOR TREATING FBD SYMPTOMS

The low FODMAP diet is the first dietary approach proven to be successful in treating FBD symptoms.[2] Unfortunately, well-designed clinical trials investigating other dietary approaches have not yet been completed. These other approaches are discussed below.

Food chemical sensitivities

Salicylates, amines and glutamate are food chemicals hypothesised to induce gastrointestinal symptoms in some patients with food chemical sensitivities. Patients will often present with non-gastrointestinal symptoms as well, including skin, respiratory and 'systemic' symptoms (e.g. 'fuzzy head', extreme fatigue and headaches).

As foods included in the low FODMAP diet are often still high in food chemicals, patients who are food chemical-sensitive can usually be identified. Currently, the protocol recommended for assessing food chemical sensitivities requires a very restrictive diet and must be carried out in conjunction with a dietitian.

Non-coeliac gluten intolerance

Gluten restriction to aid FBD symptoms in patients thought to be 'non-coeliac gluten intolerant' is frequently promoted by naturopaths and other alternative health practitioners. Evidence for its basis is presently being investigated;[30] while its existence is likely, how commonly it occurs

USEFUL RESOURCES

Article

Gibson PR, Shepherd SJ. Evidence-based dietary management of functional gastrointestinal symptoms: the FODMAP approach. *J Gastroenterol Hepatol* 2010; 25: 252-258.

Website

Monash University Eastern Health Clinical School: www.med.monash.edu.au/ehcs/research/ index.html

CONSULTANT'S COMMENT

The authors of this article are to be congratulated for their practical overview of the functional bowel disorders, of which irritable bowel syndrome is the best known, and a relatively novel, effective and accessible non-drug therapy for them.

As reviewed by Halmos and colleagues, functional bowel disorders are extremely common and lead to decreased quality of life for patients and substantial costs to the health care system. To date, as there have not been highly effective therapies to either cure the disorder or modify symptoms, there has been a degree of therapeutic nihilism among patients and doctors alike, with a high degree of frustration for all.

The discovery and development of the low FODMAP diet and the proof of both its efficacy and the pathological reasons for it have been a real step forward for patients with functional bowel disorders. The discovery and implementation of this dietary approach by this group represents a highly successful collaboration among clinicians, dietitians and scientists and shows what can be achieved when patient observations (here specifically about food intolerances) are listened to and subjected to rigorous evaluation without prejudice.

Using this diet is well worthwhile for patients with these common gastrointestinal complaints, with a low number-needed-to-treat for benefit. A practical way to do so is set out in this article. Readers are encouraged to discuss this approach with their patients and local dietitians in an attempt to improve patient quality of life without recourse to medications.

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and who should be offered a gluten-free diet, is unknown.

Before recommending a gluten-free (or reduced gluten) diet, it is crucial that coeliac disease is properly excluded. As a minimum, coeliac serology should be per - formed while the patient is on a gluten-containing diet. Ideally, all patients in whom coeliac disease is suspected should have a small bowel biopsy before adopting a gluten-free diet. *HLA-D* gene status can exclude coeliac disease if neither *HLA-DQ2* nor *HLA-DQ8* are present, but the presence of either or both does not make the diagnosis and so genetic testing is only useful for excluding the condition.

Probiotics

There is a body of research on the role of probiotics in controlling symptoms in patients with FBD. Unfortunately, studies have not generally shown much benefit, although there have been some notable exceptions.[31] It is clear, however, that results from one probiotic cannot be extrapolated to another. The recommending of probiotics generally has no negative implications, but their use should be ceased if symptoms remain unchanged.

SUPPORTIVE RESOURCES

Essential in the successful implementation of the low FODMAP diet is the provision of supporting written information, food lists, recipes and other advice such as suitable websites to access accurate information. Refer to www.med.monash.edu.au/ehcs, www.shepherdworks.com.au or www.dietsolutions.net.au for useful resources including information booklets, cookbooks, supportive journal articles, and dietitians specialising in FBD.

The importance of good education resources, quantity of free-time and level of education was evident in a study of patients with functional gut symptoms and quiescent inflammatory bowel disease.[27] It was found that the best predictors of improved response included the use of low FODMAP cookbooks, working less than or equal to 35 hours per week and having a post-secondary school qualification.[27]

CONCLUSION

FBDs are common conditions and until recently therapies have been largely unsuccessful. There is increasing evidence that the implementation of the low FODMAP diet offers a new and effective strategy to symptom management. Through dietetic guidance, an appropriate balance between maintainable dietary restriction and symptom management may be achieved in most patients. MT

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A list of references is available on request to the editorial office.

COMPETING INTERESTS: Ms Halmos and Dr Muir: None. Dr Shepherd is the author of five low FODMAP cookbooks and a low FODMAP shopping guide. Dr Shepherd and Professor Gibson are co-owners of the FODMAP trademark.

Medicine Today 2011; 12(3): 29-38

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