The Nutrition Society Winter Meeting was held at the Royal Society of Medicine, London on 2–4 December 2019

Conference on 'Diet and digestive disease'
Symposium 1: Nutrition, inflammation and pathobiology

Challenges of the low FODMAP diet for managing irritable bowel syndrome and approaches to their minimisation and mitigation

Bridgette Wilson, Selina R. Cox and Kevin Whelan*
Department of Nutritional Sciences, King's College London, London, UK

Dietary restriction of fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAP) is clinically effective and a commonly utilised approach in the management of functional symptoms in irritable bowel syndrome. Despite this, the low FODMAP diet has a number of challenges: it can alter the gut microbiota; impact nutrient intake and diet quality; is complex to understand; requires the patient to be adequately supported to follow the diet accurately and safely; and lastly, not all patients respond to the diet. The current review highlights the evidence for the clinical effectiveness of the low FODMAP diet, but focusses on the challenges associated with the diet to the patient, health professionals and the wider healthcare service. Finally, the review discusses research findings and practical guidance for how these challenges can be minimised and mitigated. The low FODMAP diet is a useful management strategy for irritable bowel syndrome, with data from clinical trials suggesting a 50–80 % response rate, and when administered appropriately, the challenges to implementing the diet can be overcome so that these outcomes can be realised effectively and safely in clinical practice.

Low FODMAP: Irritable bowel syndrome: Microbiota: Nutrient intake: Dietitian

Irritable bowel syndrome and the low FODMAP diet

Irritable bowel syndrome (IBS) is a chronic and debilitating functional gastrointestinal (GI) disorder characterised by abdominal pain and altered bowel habit. Global prevalence is reported to be in the region of 5·7–11·2 % of the population, depending upon definition and region[1,2]. The Rome criteria used to define functional bowel disorders recently updated the characteristics used to define IBS. Specifically, IBS is diagnosed when abdominal pain that is related to defaecation (i.e. related to increased or decreased stool frequency or consistency) is experienced at least one day per week. This update in definition removes ambiguity in the former classification that included

both abdominal pain 'or discomfort' and at a lower frequency of symptoms[2].

Within the Rome IV IBS classification, patients can be subtyped as: diarrhoea predominant (IBS-D, >25 % loose stool and <25 % hard stool); constipation predominant (IBS-C, >25 % hard stool and <25 % loose stool); mixed type (IBS-M, >25 % loose stool and >25 % hard stool); or un-subtyped (IBS-U, <25 % loose stool and <25 % hard stool)[3]. In addition to classifying IBS by predominant stool form, patients can be classified as post-infectious IBS (PI-IBS) where the symptoms began with or shortly after a GI infection and have not resolved within 6 months, inferring chronicity[3].

National Institute for Health and Care Excellence (NICE) guidelines for managing IBS recommend first-line

---

Abbreviations: FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides and polyols; GI, gastrointestinal; GOS, galacto-oligosaccharides; IBS, irritable bowel syndrome; LFD, low FODMAP diet; NICE, National Institute for Health and Care Excellence; RCT, randomised controlled trial.
*Corresponding author: Kevin Whelan, email kevin.whelan@kcl.ac.uk

dietary advice (healthy eating advice focussing on meal frequency, size, composition and intake of fat, spicy foods, caffeine, fluids and fibre), and if this fails to resolve symptoms, second-line dietary advice should be followed (the low fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAP) diet (LFD))[4]. The LFD involves the restriction of fermentable oligosaccharides, disaccharides, monosaccharides and polyols, including fructans (e.g. wheat, onion, garlic), galacto-oligosaccharides (GOS, e.g. lentils, beans, peas, cashews), lactose (e.g. milk), fructose in excess of glucose (e.g. mango, honey), sorbitol (e.g. avocado, apple, broccoli) and mannitol (e.g. celery)[5].

The LFD consists of three stages. Stage 1 (restriction) involves the restriction of all FODMAP sources and is recommended for a minimum of 4 weeks followed by the evaluation of symptom response. Stage 2 (reintroduction) involves challenge with increasing doses of individual FODMAP while continuing restriction of all other FODMAP. Specific challenges include individual foods containing fructans, GOS, lactose, fructose, sorbitol and mannitol, to identify the classes and amount of FODMAP that trigger symptoms in that patient. Stage 3 (personalisation) involves patients consuming liberalised types and amounts of FODMAP-containing foods that were well tolerated during stage 2, while those inducing significant GI symptoms continue to be excluded, with the aim of managing GI symptoms in the long term.

One of the first reports of the role of some FODMAP in IBS symptom induction was a double-blind, quadruple cross-over, placebo-controlled, re-challenge trial in twenty-six patients with IBS who had previously achieved adequate relief of their symptoms by following an LFD[6]. Fewer patients reported adequate relief of abdominal symptoms when challenged with fructans, fructose or a mixture of both, compared to glucose (control). Both fructans and the mixture of fructans and fructose led to significantly greater severity of abdominal pain, bloating and flatulence than placebo and fructose led to significantly worse pain and bloating severity than placebo[6].

Mechanistically, ingestion of fructose leads to an increase in small bowel water peaking at 75 min after ingestion and fructans lead to increased colonic gas peaking at 3–5 h after ingestion[7]. The effect of fructose on small bowel water can be ameliorated by glucose co-administration that leads to increased fructose absorption in the small bowel via a glucose–fructose transporter complex (GLUT2). However, when trialled in IBS, the addition of glucose to fructose solutions did not prevent symptom induction despite reducing breath hydrogen production compared with fructose alone[8]. Further, fructan-induced colonic gas production leads to similar gas production in IBS and healthy controls, despite a peak in symptom intensity in IBS[9] suggesting hypersensitivity to gas production is responsible for IBS symptoms, rather than greater gas production *per se*. The altered hypersensitivity may be due to varied microbial signalling, disordered tight junction proteins or heightened GI immune signalling and subsequent inflammatory response or a combination of all these components[10].

The first randomised controlled trial (RCT; *n* 41) of LFD advice found that it resulted in significantly higher adequate relief of IBS symptoms compared with habitual diet[11]. Subsequently, numerous studies have confirmed that the LFD alleviates IBS symptoms compared to habitual diet[12], typical diet[13,14], a high FODMAP diet[15] or a sham diet[16] (Table 1).

There are now numerous systematic reviews and meta-analyses of clinical trials of the LFD showing improvements in global symptoms (relative risk = 0·69; 95 % CI 0·54, 0·88; $P = 0.003$)[21], overall GI symptoms (standardised mean difference = −0·62; 95 % CI −0·93, −0·31; $P = 0.0001$), abdominal pain (standardised mean difference = −0·50; 95 % CI −0·77, −0·22; $P = 0.008$)[22] and bloating (OR 1·75, 95 % CI 1·07, 2·87; $P = 0.03$)[23].

Therefore, the LFD is an evidence-based dietary therapy that can be used when first-line healthy eating approaches have failed to reduce symptoms. However, there are several challenges associated with the use of this diet. The aim of this review is to critically discuss the challenges of the LFD faced by patients, health professionals and the wider healthcare service and to discuss research findings and practical guidance for how these challenges can be minimised and mitigated, so that the clinical effectiveness of the LFD can be realised in practice.

Challenge 1: the low FODMAP diet affects the luminal microbiota

Despite the beneficial effects of the LFD on global and specific symptoms, modifications in the GI microbiota have been identified. The first RCT of the LFD demonstrated it lowered bifidobacteria compared with habitual diet[11], whilst two further RCT have demonstrated a lower absolute abundance of bifidobacteria when comparing the LFD with placebo diets[16,24] and a fourth RCT did not report the LFD lowered bifidobacteria but rather that a high FODMAP diet increased both Bifidobacteriaceae and some genera within the Lachnospiraceae family[15].

In a cross-over feeding study comparing an LFD to typical Australian diet, a number of microbial effects between the two diets were observed including greater *Clostridium* cluster XIVa following a typical Australian diet, although given that diet was higher in FODMAP than participants' habitual diet, at least some of the differences may be attributed to a prebiotic effect of increasing FODMAP in the typical Australian diet control group[24]. In comparison to the habitual diet, the same study reported the LFD reduced absolute abundance of total bacteria, *Clostridium* cluster IV, *Faecalibacterium prausnitzii* and *Bifidobacterium*. However, there was no difference between habitual diet and LFD for the abundance of total bacteria[24]. In a comparison of the LFD with a traditional IBS diet (first-line advice from NICE[4]), the LFD but not traditional IBS diet was shown to specifically reduce *Bifidobacterium*, *Mycoplasma hominis* and the phylum Actinobacteria from baseline, suggesting that it is FODMAP restriction that impacts the gut microbiota[25]. The consistently reported effect of the LFD in reducing

Table 1. Randomised controlled trials and randomised comparative trials of the low FODMAP diet in irritable bowel syndrome (IBS) and the effect on global and individual symptoms

Clinical trial	Sample size	Comparison group	Outcome in the low FODMAP group *v.* comparison group at the end of the trial						
			Adequate relief or number of responders	Global symptom score	Abdominal pain	Bloating	Flatulence	Diarrhoea	Faecal urgency
Staudacher et al.[11]	41	Habitual diet	Greater (adequate relief)	Lower overall symptoms (GSRS)	Reduced incidence and severity (GSRS)	Reduced incidence and severity (GSRS)	Reduced incidence and severity (GSRS)	No difference (GSRS)	No difference (GSRS)
Halmos et al.[13]	33	Typical Australian diet	–	Lower overall symptoms (VAS)	Reduced severity (VAS)	Reduced severity (VAS)	Reduced severity (VAS)	–	–
Bohn et al.[17]	75	Traditional IBS diet	–	No difference (IBS-SSS)	No difference (IBS-SSS)	No difference (IBS-SSS)	–	–	–
Eswaran et al.[18]	92	Modified NICE guidelines	No difference (adequate relief ≥50% of last 2 weeks of trial)	No difference (composite symptom score)	More with >30% reduction in severity	Greater reduction in severity (Likert)	–	–	Greater reduction in severity (Likert)
McIntosh[15]	40	High FODMAP diet	–	Lower (IBS-SSS)	Reduced incidence and severity (IBS-SSS)	No difference (IBS-SSS)	–	–	–
Hustoft et al.[19]	20	LFD plus 16 g/d fructans	Greater (≥50 point reduction in IBS-SSS)	Lower (IBS-SSS)	Reduced incidence and severity (IBS-SSS)	Reduced severity (IBS-SSS)	Reduced (VAS)	–	–
Harvie et al.[12]	50	Habitual diet	–	Greater reduction (IBS-SSS)	Greater reduction in incidence LFD	No difference IBS-SSS	–	–	–
Staudacher et al.[16]	104	Sham diet	No difference (adequate relief)	Lower (IBS-SSS)	Reduced severity (GSRS)	Reduced severity (GSRS)	Reduced severity (GSRS)	No difference (GSRS)	Reduced severity (GSRS)
Patcharatrakul et al.[20]	66	Commonly recommended IBS diet	Greater (≥30% decrease in daily worst abdominal pain)	Lower (VAS)	No difference (VAS)	No difference (VAS)	–	–	No difference (VAS)

LFD, low FODMAP diet; GSRS, gastrointestinal symptom rating scale; VAS, visual analogue scale; IBS, irritable bowel system; IBS-SSS, IBS severity scoring system.

the abundance of bifidobacteria could be considered a potentially negative consequence, although no data are available on the health consequences of the lower bifidobacteria resulting from the LFD nor on the impact on bifidobacteria in the long term following FODMAP reintroduction.

Restricting fermentable substrate for saccharolytic bacteria, and therefore reducing saccharolytic fermentation should logically reduce SCFA production. This may be an issue as SCFA lower colonic pH thereby inhibiting pathogenic colonisation, and because butyrate provides a substrate for colonocytes and has anti-inflammatory properties[26]. In particular, the LFD restricts oligosaccharides, namely fructans and GOS that consist of fructose or galactose monomers (respectively) linked by glycosidic bonds that are not hydrolysed by human enzymes, and which when supplemented to the diet have been demonstrated to stimulate bifidobacteria numbers in the gut[27]. Therefore, fructans and GOS are major sources of microbiota-accessible carbohydrate in the diet and restriction is likely to have a greater effect on those microbes that specialise in carbohydrate fermentation. Bifidobacteria in particular dedicate about 8 % of their total genome to carbohydrate metabolism, approximately 30 % more than most other gut microbiota[28] and may explain why bifidobacteria are so affected by FODMAP restriction.

Several strategies have been investigated to mitigate against the impact of the LFD on the GI microbiota. A recent clinical trial of the LFD supplemented with a multi-strain probiotic (that included three strains of bifidobacteria) found the probiotic maintained absolute abundance of bifidobacteria during the LFD and therefore may be a useful adjunct to the LFD. Clinically, the number of patients that responded to the diet (29/51, 57 %) or the probiotic (30/53, 57 %) was greater than the number that responded to a sham diet (20/53, 38 %, $P = 0.051$) or placebo (19/51, 37 %, $P = 0.048$)[16]. Apart from flatulence, for which severity was reduced ($P = 0.033$), the probiotic did not significantly improve individual symptom severity compared with placebo, whereas the LFD reduced numerous symptoms including abdominal pain, bloating, flatulence and overall symptoms (Table 1). Therefore, it should be explained to patients that if they wish to take a probiotic with the LFD, depending upon the probiotic it may or may not improve symptoms but may prevent a reduction in bifidobacteria. Although probiotics would not maintain the native population of bifidobacteria, they may maintain overall numbers of bifidobacteria through exogenous supplementation[16], and so could be useful in preventing the native bifidobacteria being at a competitive disadvantage once FODMAP are reintroduced. Probiotic co-supplementation is therefore one approach to the maintenance of bifidobacteria during the LFD; however, other benefits of fermentable fibre consumption, especially SCFA production and immune modulation, may not be affected by probiotic co-supplementation. One RCT reports the addition of a prebiotic β-GOS to the LFD, which although improved symptoms, it was not effective at maintaining luminal bifidobacteria or SCFA[29].

Finally, it is possible that the detrimental effects of the LFD on the microbiota are reversed during the reintroduction and personalisation stage of the diet, but evidence of this is currently lacking in the literature and therefore long-term studies of the impact of all three stages of the LFD on both symptoms and gut microbiome are warranted. Until more is known about the effect of reintroduction, clinicians should address the effect on the microbiota when providing guidance on the LFD (Table 2).

Challenge 2: the low FODMAP diet may impact nutrient intake and diet quality

The LFD requires significant dietary changes for most patients. Foods are excluded from most of the commonly described food groups, including cereals/starchy foods (e.g. wheat-containing breads, pasta, pastries), fruit (e.g. apples, cherries, plums), vegetables (e.g. onion, cauliflower, celery) and dairy (many milk-containing products). Therefore, in theory, the nutrients most likely to be compromised during the LFD are calcium (restriction of lactose in dairy products), iron (restriction of iron-fortified breakfast cereals) and fibre (restriction of some grains, fruit and vegetables). Additionally, overall energy intake may be compromised, particularly considering the restriction of staple foods such as bread and pasta. However, the impact of the LFD on nutrient intake and dietary quality has not been extensively investigated.

Patients with IBS following LFD advice for 4 weeks were reported to have lower calcium intakes compared to patients following the habitual diet[11], and lower energy, carbohydrate and fibre intakes compared to patients following traditional IBS dietary advice (NICE diet)[17]. More recently, in ninety-five patients with IBS, calcium intake was not different between the LFD and sham diet groups at the end of the trial, although fewer patients in the LFD group at the end of the trial reached the calcium dietary reference value compared with baseline habitual diet indicating that calcium replacement should be addressed during LFD dietary counselling[30]. More recently, a comparison of traditional IBS dietary advice (modified NICE diet) and LFD revealed both diets reduced overall energy intake, specifically a reduction in carbohydrate intake, but micronutrients, when correcting for the percentage of overall energy intake, were not affected with the exception of riboflavin[31]. However, when comparing within groups (i.e. comparing 4-week data to baseline data), the LFD but not 'modified NICE diet' significantly reduced the intake of some micronutrients including calcium, supporting earlier findings. Finally, one publication combined the results of two earlier RCT and reported low-fibre intakes in baseline habitual diet in 130 patients with IBS, with a mean intake of only 18 g/d and only six (5 %) achieving the dietary reference value for fibre intake (30 g/d)[32]. Macronutrient, calcium and fibre intakes following the LFD were not different compared to sham diet or habitual diet. However, the LFD marginally reduced diet quality, a measure of how closely the diet aligns with the World Health Organization dietary guidelines for

Table 2. Clinical take-home messages to minimise and mitigate some of the challenges of the low FODMAP diet in clinical practice

---

Challenge 1: the low FODMAP diet adversely affects the luminal microbiota

The restriction stage of the low FODMAP diet reduces the abundance of a number of genera and species in the gut microbiota, including bifidobacteria.

A bifidobacteria-containing probiotic may prevent a decline in luminal bifidobacteria during the restriction stage of the low FODMAP diet, but prebiotics may not.

Ensure patients do not remain on the low FODMAP diet long term and undergo structured reintroduction and personalisation stages.

Challenge 2: the low FODMAP diet may impact nutrient intake and diet quality

Any dietary restriction can compromise nutrient intake, and during the restriction stage of the low FODMAP diet some studies show fibre, iron and calcium intakes may be at risk.

Clinicians should educate and support patients to optimise intakes of calcium (e.g. calcium-fortified, lactose-free products) and iron (e.g. meats, low FODMAP meat alternatives) during the restriction stage of the low FODMAP diet.

Clinicians should educate and support patients to optimise dietary fibre intake without exacerbating gut symptoms by recommending low FODMAP cereals, fruits and vegetables that are high fibre.

Challenge 3: the low FODMAP diet is complex to follow

The low FODMAP diet can be complex to follow, can require extensive label reading, be expensive and limit social activities relating to food.

NICE dietary advice should be the first-line dietary advice in IBS and only where unsuccessful should the low FODMAP diet be attempted with advice from a suitably-trained registered dietitian.

Patients should be provided with practical advice on naturally FODMAP-free ingredients to reduce the need for purchasing specialist products and provided with ideas for suitable options to replace habitual meals and for dining out.

Challenge 4: patients require support to follow the low FODMAP diet effectively

Wherever possible referral to a trained dietitian should be sought as best practice in providing advice regarding a low FODMAP diet.

Approaches to alternative delivery can be trialled including group sessions with dietitians or online support through webinars.

Recipe books, diet sheets and mobile applications are available to support patients, but at the present time these should be considered supplementary to dietetic advice.

Challenge 5: not all patients respond to the low FODMAP diet

The low FODMAP diet is effective in 50–80% of patients when followed correctly with guidance from a trained dietitian, but in order to manage expectations patients should therefore be informed that 20–50% do not respond.

In the future, it may be possible to predict non-responders in order to prevent the need for them to attempt a low FODMAP diet.

Studies indicate that measuring faecal markers of bacterial metabolism or fermentation at baseline may help identify those likely to respond to the low FODMAP diet.

No tests are currently available in routine clinical practice to determine the likelihood of response to the low FODMAP diet.

---

the prevention of chronic disease, compared to control diets[33].

The impact of the LFD on vitamin intake is reported in two studies. In one report comparing the effect of a 4-week LFD on nutrient intake, a significant reduction in thiamine and riboflavin compared to baseline intake was shown, possibly due to the restriction of lactose-containing dairy products[31], there was no effect on the intake of other vitamins. More recently, an assessment of 130 individuals randomised in two RCT to either a low FODMAP, habitual or sham diet for 4 weeks showed that vitamin B_{12} and selenium intakes were higher during the LFD compared to sham diet, and vitamin B_{12} was also higher during LFD compared to the habitual diet. Increases in intakes of these micronutrients may indicate a higher meat and potato consumption in view of some staple carbohydrates being restricted (e. g. bread, pasta, wheat-based cereal). Despite these differences in actual intake, there were no differences in the proportion meeting the dietary reference values for either vitamin B_{12} or selenium between the three diets[32]. Finally, there are currently no studies investigating the impact of the LFD on vitamin status (e.g. serum concentrations, etc.), and such investigations are required.

These data report the effect of stage 1 restriction of the LFD on nutrient intake and diet quality. However, the long-term effects of the LFD on nutrient intake were investigated in a study of patients 6–18 months after receiving FODMAP reintroduction advice[34]. At the long-term follow-up, 82% continued to follow an adapted FODMAP diet (i.e. stage 3 personalisation), while 18% had returned to their habitual diet. In the adapted FODMAP group, total FODMAP intakes were significantly lower while nutrient intakes were similar to the habitual diet group. Another study demonstrated that fibre intake declined during stage 1 FODMAP restriction but returned to normal following FODMAP reintroduction and personalisation[12]. These findings suggest that FODMAP reintroduction and personalisation may resolve nutritional inadequacies that may occur with FODMAP restriction; however, both studies used a FFQ to estimate nutrient and FODMAP intakes, which may lack accuracy compared to prospective food records[35,36]. In general, clinicians should provide guidance on how to optimise nutrient intake and maintain diet quality while following the LFD (Table 2).

Challenge 3: the low FODMAP diet is complex to follow

The LFD is complex to follow for numerous reasons. First, the LFD restricts foods across a wide range of

smartphone app (63 %) and a similar reduction in symptoms from baseline measured using the IBS severity scoring system, although this may be a type II error due to study power. The provision of a leaflet alone was significantly less effective than 1:1 dietetic advice for symptom improvement indicating patients require more support than this[47]. Larger, adequately powered clinical trials of optimal dietary delivery methods are required.

In conclusion, due to the complex nature of altering the whole diet, patients should be supported using the best available resource during this process to increase the likelihood of symptom improvement ([Table 2]).

Challenge 5: not all patients respond to the low FODMAP diet

Research data suggest that 50–80 % of patients will experience a positive symptom response to the LFD. Put another way, this means that 20–50 % of IBS patients do not respond symptomatically to the LFD despite having similar symptoms and demographics to those that do. The reason for non-response is under increasing investigation in order to optimise the selection of patients most likely to respond to the LFD, and to avoid unnecessary dietary restriction in those unlikely to respond. While adherence to the diet has been suggested as one reason for no response to the LFD[15,22], more recently, data have emerged suggesting measurable variation in both microbiota and faecal metabolites that differentiate those who go on to respond to the diet compared to those who do not.

A clinical trial randomised thirty-one patients with IBS to the LFD and thirty patients to NICE dietary advice, and stool samples were analysed at baseline using a commercially available GA-map dysbiosis test that uses fifty-four bacterial probes to identify ≥300 bacteria across different levels of phylogeny (Genetic Analysis AS, Oslo, Norway)[25]. The nineteen (61 %) responders to the LFD (defined as a reduction of ≥50 points on the IBS-severity scoring system) had lower bacterial probe signal intensities for thirteen bacterial targets at baseline than the twelve (39 %) non-responders to LFD. Bacterial groups that were lower at baseline in responders were *Bacteroides stercoris*, *Pseudomonas*, *Acinetobacter*, *Desulfitispora*, *Parabacteroides*, *Bacillus*, *Salmonella* (*Citrobacter*, *Cronobacter*, *Enterobacter*), *Corea*, *Ruminococcus gnavus*, *Clostridium*, Firmicutes (*Clostridia*) and *Streptococcus*. Multivariate orthogonal partial least-squared discriminant analysis of bacterial profiles was able to reliably distinguish between responders and non-responders to the LFD at baseline ($Q^2 = 0.54$)[25].

In a further uncontrolled study, sixty-one adult patients with IBS followed the LFD for 4 weeks[48], with the thirty-two (52 %) of responders (defined as a reduction of ≥50 points on the IBS-severity scoring system) had different baseline microbiota compared to the twenty-nine (48 %) non-responders. A group of five bacteria (*Bacteroides fragilis*, *Acinetobacter*, *Ruminiclostridium*, *Streptococcus*, and *Eubacterium*) were higher and five bacteria (Clostridia/Negativicutes/Bacilli, Actinomycetales, *Anaerotruncus*, Clostridiales and *Shigella/Escherichia*) were lower in responders than non-responders at baseline when measured using a dysbiosis test. From these ten bacteria, the authors developed a response index, and patients with a positive responder index score were five times more likely to have responded to the LFD (OR = 5·05, 95 % CI 1·58, 16·10) although this algorithm has yet to be tested prospectively.

While these results regarding microbiota differences from these two studies are promising, the findings differ between the studies despite both using the same intervention, similar selection criteria and test for assessing microbiota. There was only one common differentiating genus between the studies (*Streptococcus*); however, at baseline, it was lower in responders in one trial and higher in responders in the other, indicating there is still much to be discovered about how baseline microbiota may relate to symptom response to the LFD.

Some studies have examined faecal metabolites as a marker of bacterial activity in relation to predicting response to the LFD. An RCT in which forty-six adults with IBS were randomised to the LFD and forty-seven to a sham diet demonstrated that baseline faecal volatile organic compounds differed between the thirty-seven (80 %) LFD responders (defined as a reduction of ≥50 points on the IBS-severity scoring system) and nine (20 %) LFD non-responders with a high level of accuracy (97 %)[49]. Preliminary data from a more recent study identified that the urine metabolome and faecal propionate may predict response to the LFD and confirmed that faecal volatile organic compound differs between responders and non-responders[50]. Further adequately powered studies are required to confirm these findings and identify the factors associated with response, or lack of response, to the LFD. Importantly, in the current absence of a widely-available predictive test, it is important to manage patients' expectations when initiating the LFD ([Table 2]).

Discussion

Although there is now a wide range of evidence to support the use of the LFD in clinical practice for IBS, it is important to acknowledge the challenges with regards to long-term safety, implementation and likelihood of response to the diet. These issues should be raised and discussed with patients when advice on the LFD is provided.

Importantly, all health professionals should acknowledge the challenges of the LFD discussed here and provide guidance on where to access reliable information on the diet. Unguided use of the LFD may lead to unnecessary long-term restriction and may contribute to nutritional deficiencies and exacerbate food phobia, especially when patients are later encouraged to reintroduce foods. In addition, the LFD may be less likely to be effective when not explained with sufficient detail as subtleties could be missed or inaccurate information about the foods to restrict could be provided. This could lead to patients feeling they have tried the

diet and it did not work, whereas it may have been effective if sufficient guidance was provided and if such advice was followed correctly. It should be acknowledged that an appointment to undertake the full assessment of individual symptoms, bowel habit, current diet and to provide dietary counselling on a whole dietary change should last approximately 45–60 min[5]. Tailored advice on how to optimise the quality of the current diet and suitable swaps to make the diet low in FODMAP as well as nutritionally adequate should be provided. As the LFD restricts food with both laxative and gas-forming potential[7], it should be used with caution in patients with constipation (either IBS-C or IBS-M) and additional advice on maintaining suitable fibre and fluid intake should always be provided.

To date, the research evidence for using the LFD in IBS is supportive of a clinical benefit on symptoms and is now included as the second-line dietary approach (after NICE dietary advice) in some national guidelines[4,51]. However, all RCT of the diet have been delivered using advice from a registered dietitian and evidence for the effectiveness of the diet when provided in less structured formats or by other health professionals is limited. The training and clinical expertise of dietitians mean they are well-placed to assess habitual diets and advise on the dietary changes most likely to benefit patients. There is not a one size fits all approach and advice may include a combination of fibre, probiotics and other dietary and lifestyle approaches including the LFD. Therefore, wherever possible, assessment and dietary counselling should be provided by health professionals with expertise in diet and gastroenterology.

Conclusion

When provided by an adequately trained health professional, the LFD is a clinically effective treatment option for IBS. Challenges of the LFD should be acknowledged and discussed with the patient during the first consultation, and where possible referral to an appropriately trained dietitian should be made.

Financial Support

Conflicts of Interest

Authorship

References

1. Lovell RM & Ford AC (2012) Global prevalence of and risk factors for irritable bowel syndrome: a meta-analysis. *Clin Gastroenterol Hepatol* **10**, 712–721.
2. Palsson OS, Whitehead WE, Van Tilburg MA *et al.* (2016) Development and validation of the Rome IV diagnostic questionnaire for adults. *Gastroenterology* **150**, 1481–1491.
3. Lacy BE, Mearin F, Chang L *et al.* (2016) Bowel disorders. *Gastroenterology* **150**, 1393–1407.
4. National Institute for Health and Care Excellence (2017) NICE (2017 update): irritable bowel syndrome in adults: diagnosis and management (NICE guideline CG61) [NIfHC Excellence, editor]. https://www.nice.org.uk/guidance/cg61/resources.
5. Whelan K, Martin LD, Staudacher HM *et al.* (2018) The low FODMAP diet in the management of irritable bowel syndrome: an evidence-based review of FODMAP restriction, reintroduction and personalisation in clinical practice. *J Hum Nutr Diet* **31**, 239–255.
6. Shepherd SJ, Parker FC, Muir JG *et al.* (2008) Dietary triggers of abdominal symptoms in patients with irritable bowel syndrome: randomized placebo-controlled evidence. *Clin Gastroenterol Hepatol* **6**, 765–771.
7. Murray K, Wilkinson-Smith V, Hoad C *et al.* (2014) Differential effects of FODMAPs (fermentable oligo-, di-, mono-saccharides and polyols) on small and large intestinal contents in healthy subjects shown by MRI. *Am J Gastroenterol* **109**, 110–119.
8. Tuck CJ, Ross LA, Gibson PR *et al.* (2017) Adding glucose to food and solutions to enhance fructose absorption is not effective in preventing fructose-induced functional gastrointestinal symptoms: randomised controlled trials in patients with fructose malabsorption. *J Hum Nutr Diet* **30**, 73–82.
9. Major G, Pritchard S, Murray K *et al.* (2017) Colon hypersensitivity to distension, rather than excessive gas production, produces carbohydrate-related symptoms in individuals with irritable bowel syndrome. *Gastroenterology* **152**, 124–133.
10. Deiteren A, de Wit A, van der Linden L *et al.* (2016) Irritable bowel syndrome and visceral hypersensitivity: risk factors and pathophysiological mechanisms. *Acta Gastro-enterologica Belgica* 1996–2007 **79**, 29–38.

11. Staudacher HM, Lomer MC, Anderson JL et al. (2012) Fermentable carbohydrate restriction reduces luminal bifidobacteria and gastrointestinal symptoms in patients with irritable bowel syndrome. *J Nutr* **142**, 1510–1518.

12. Harvie RM, Chisholm AW, Bisanz JE et al. (2017) Long-term irritable bowel syndrome symptom control with reintroduction of selected FODMAPs. *World J Gastroenterol* **23**, 4632–4643.

13. Halmos EP, Power VA, Shepherd SJ et al. (2014) A diet low in FODMAPs reduces symptoms of irritable bowel syndrome. *Gastroenterology* **146**, 67–75.

14. Chumpitazi BP, Cope JL, Hollister EB et al. (2015) Randomised clinical trial: gut microbiome biomarkers are associated with clinical response to a low FODMAP diet in children with the irritable bowel syndrome. *Aliment Pharmacol Ther* **42**, 418–427.

15. McIntosh K, Reed DE, Schneider T et al. (2017) FODMAPs alter symptoms and the metabolome of patients with IBS: a randomised controlled trial. *Gut* **66**, 1241–1251.

16. Staudacher H, Lomer M, Farquharson F et al. (2017) Diet low in FODMAPs reduces symptoms in patients with irritable bowel syndrome and probiotic restores Bifidobacterium species: a randomized controlled trial. *Gastroenterology* **153**, 936–947.

17. Böhn L, Störsrud S, Liljebo T et al. (2015) Diet low in FODMAPs reduces symptoms of irritable bowel syndrome as well as traditional dietary advice: a randomized controlled trial. *Gastroenterology* **149**, 1399–1407.

18. Eswaran SL, Chey WD, Han-Markey T et al. (2016) A randomized controlled trial comparing the low FODMAP diet vs. modified NICE guidelines in US adults with IBS-D. *Am J Gastroenterol* **111**, 1824–1832.

19. Hustoft T, Hausken T, Ystad S et al. (2017) Effects of varying dietary content of fermentable short-chain carbohydrates on symptoms, fecal microenvironment, and cytokine profiles in patients with irritable bowel syndrome. *Neurogastroenterol Motil* **29**, [Epublication 16 October 2016].

20. Patcharatrakul T, Juntrapirat A, Lakananurak N et al. (2019) Effect of structural individual low-FODMAP dietary advice vs. brief advice on a commonly recommended diet on IBS symptoms and intestinal gas production. *Nutrients* **11**, 2856.

21. Dionne J, Ford AC, Yuan Y et al. (2018) A systematic review and meta-analysis evaluating the efficacy of a gluten-free diet and a low FODMAPs diet in treating symptoms of irritable bowel syndrome. *Am J Gastroenterol* **113**, 1290–1300.

22. Schumann D, Klose P, Lauche R et al. (2018) Low fermentable, oligo-, di-, mono-saccharides and polyol diet in the treatment of irritable bowel syndrome: a systematic review and meta-analysis. *Nutrition* **45**, 24–31.

23. Marsh A, Eslick E & Eslick G (2016) Does a diet low in FODMAPs reduce symptoms associated with functional gastrointestinal disorders? A comprehensive systematic review and meta-analysis. *Eur J Nutr* **55**, 897–906.

24. Halmos EP, Christophersen CT, Bird AR et al. (2015) Diets that differ in their FODMAP content alter the colonic luminal microenvironment. *Gut* **64**, 93–100.

25. Bennet SMP, Bohn L, Storsrud S et al. (2018) Multivariate modelling of faecal bacterial profiles of patients with IBS predicts responsiveness to a diet low in FODMAPs. *Gut* **67**, 872–881.

26. Gibson GR, Probert HM, Van Loo J et al. (2004) Dietary modulation of the human colonic microbiota: updating the concept of prebiotics. *Nutr Res Rev* **17**, 259–275.

27. So D, Whelan K, Rossi M et al. (2018) Dietary fiber intervention on gut microbiota composition in healthy adults: a systematic review and meta-analysis. *Am J Clin Nutr* **107**, 965–983.

28. Pokusaeva K, Fitzgerald GF & van Sinderen D (2011) Carbohydrate metabolism in Bifidobacteria. *Genes Nutr* **6**, 285–306.

29. Wilson B, Rossi M, Kanno T et al. (2020) β-Galactooligosaccharide in conjunction with low FODMAP diet improves irritable bowel syndrome symptoms but reduces fecal Bifidobacteria. *Am J Gastroenterol* (In the Press).

30. Staudacher H, Ross FS, Briscoe ZM et al. (2015) Advice from a dietitian regarding the low FODMAP diet broadly maintains nutrient intake and does not alter fibre intake. *Gut* **64**, A143–A144.

31. Eswaran S, Dolan RD, Ball SC et al. (2020) The impact of a 4-week low-FODMAP and mNICE diet on nutrient intake in a sample of US adults with irritable bowel syndrome with diarrhea. *J Acad Nutr Diet* **120**, 641–649.

32. Staudacher HM, Ralph FS, Irving PM et al. (2020) Nutrient intake, diet quality, and diet diversity in irritable bowel syndrome and the impact of the low FODMAP diet. *J Acad Nutr Diet* **120**, 535–547.

33. Huijbregts P, Feskens E, Räsänen L et al. (1997) Dietary pattern and 20 year mortality in elderly men in Finland, Italy, and The Netherlands: longitudinal cohort study. *Br Med J* **315**, 13–17.

34. O'keeffe M, Jansen C, Martin L et al. (2018) Long-term impact of the low-FODMAP diet on gastrointestinal symptoms, dietary intake, patient acceptability, and healthcare utilization in irritable bowel syndrome. *Neurogastroenterol Motil* **30**, [Epublication 14 July 2017].

35. Bingham SA, Gill C, Welch A et al. (1994) Comparison of dietary assessment methods in nutritional epidemiology: weighed records v. 24 h recalls, food-frequency questionnaires and estimated-diet records. *Br J Nutr* **72**, 619–643.

36. Prentice RL, Mossavar-Rahmani Y, Huang Y et al. (2011) Evaluation and comparison of food records, recalls, and frequencies for energy and protein assessment by using recovery biomarkers. *Am J Epidemiol* **174**, 591–603.

37. Guadagnoli L, Mutlu EA, Doerfler B et al. (2019) Food-related quality of life in patients with inflammatory bowel disease and irritable bowel syndrome. *Qual Life Res* **28**, 2195–2205.

38. Bohn L, Storsrud S, Liljebo TM et al. (2015) A randomized, controlled trial comparing a diet low in fodmaps with traditional dietary advice in patients with IBS. *Gastroenterology* **148**, S654.

39. Eswaran S, Chey WD, Jackson K et al. (2017) A diet low in fermentable oligo-, di-, and monosaccharides and polyols improves quality of life and reduces activity impairment in patients with irritable bowel syndrome and diarrhea. *Clin Gastroenterol Hepatol* **15**, 1890–1899.

40. de Roest RH, Dobbs BR, Chapman BA et al. (2013) The low FODMAP diet improves gastrointestinal symptoms in patients with irritable bowel syndrome: a prospective study. *Int J Clin Pract* **67**, 895–903.

41. Mazzawi T, Hausken T, Gundersen D et al. (2013) Effects of dietary guidance on the symptoms, quality of life and habitual dietary intake of patients with irritable bowel syndrome. *Mol Med Report* **8**, 845–852.

42. Ostgaard H, Hausken T, Gundersen D et al. (2012) Diet and effects of diet management on quality of life and symptoms in patients with irritable bowel syndrome. *Mol Med Report* **5**, 1382–1390.

43. Tuck CJ, Reed DE, Muir JG *et al.* (2020) Implementation of the low FODMAP diet in functional gastrointestinal symptoms: a real-world experience. *Neurogastroenterol Motil* **32**, [Epublication 30 September 2019].

44. Trott N, Aziz I, Rej A *et al.* (2019) How patients with IBS use low FODMAP dietary information provided by general practitioners and gastroenterologists: a qualitative study. *Nutrients* **11**, 1313.

45. Whigham L, Joyce T, Harper G *et al.* (2015) Clinical effectiveness and economic costs of group versus one-to-one education for short-chain fermentable carbohydrate restriction (low FODMAP diet) in the management of irritable bowel syndrome. *J Hum Nutr Diet* **28**, 687–696.

46. Chen J, Lieffers J, Bauman A *et al.* (2017) The use of smartphone health apps and other mobile health (mHealth) technologies in dietetic practice: a three country study. *J Hum Nutr Diet* **30**, 439–452.

47. Dimidi E, Whelan K, Lomer MC (2020) Investigating optimal education regarding the low FODMAP diet in functional bowel disorders: a feasibility randomised controlled trial of leaflet vs mobile application vs dietetic consultation. *Proc Nutr Soc* **79** (OCE1), E7.

48. Valeur J, Småstuen MC, Knudsen T *et al.* (2018) Exploring gut microbiota composition as an indicator of clinical response to dietary FODMAP restriction in patients with irritable bowel syndrome. *Dig Dis Sci* **63**, 429–436.

49. Rossi M, Aggio R, Staudacher HM *et al.* (2018) Volatile organic compounds in feces associate with response to dietary intervention in patients with irritable bowel syndrome. *Clin Gastroenterol Hepatol* **16**, 385–391.

50. Wilson B, Rossi M, Kanno T *et al.* (2020) Low FODMAP diet & prebiotic β-galactooligosaccharides improve irritable bowel syndrome and response to low FODMAP is predicted by urine and faecal metabolites: a randomised controlled trial. *Proc Nutr Soc* **79** (OCE1), E19

51. McKenzie YA, Bowyer RK, Leach H *et al.* (2016) British dietetic association systematic review and evidence-based practice guidelines for the dietary management of irritable bowel syndrome in adults (2016 update). *J Hum Nutr Diet* **29**, 549–575.