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RESEARCH PAPER

Comparison of symptom response following advice for a diet low in fermentable carbohydrates (FODMAPs) versus standard dietary advice in patients with irritable bowel syndrome

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Abstract

Background: Emerging evidence indicates that the consumption of fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) may result in symptoms in some patients with irritable bowel syndrome (IBS). The present study aimed to determine whether a low FODMAP diet is effective for symptom control in patients with IBS and to compare its effects with those of standard dietary advice based on the UK National Institute for Health and Clinical Excellence (NICE) guidelines.
Methods: Consecutive patients with IBS who attended a follow-up dietetic outpatient visit for dietary management of their symptoms were included. Questionnaires were completed for patients who received standard (n = 39) or low FODMAP dietary advice (n = 43). Data were recorded on symptom change and comparisons were made between groups.
Results: In total, more patients in the low FODMAP group reported satisfaction with their symptom response (76%) compared to the standard group (54%, P = 0.038). Composite symptom score data showed better overall symptom response in the low FODMAP group (86%) compared to the standard group (49%, $P < 0.001$). Significantly more patients in the low FODMAP group compared to the standard group reported improvements in bloating (low FODMAP 82% versus standard 49%, P = 0.002), abdominal pain (low FODMAP 85% versus standard 61%, P = 0.023) and flatulence (low FODMAP 87% versus standard 50%, P = 0.001).
Conclusions: A low FODMAP diet appears to be more effective than standard dietary advice for symptom control in IBS.

Introduction

Irritable bowel syndrome (IBS) is a chronic debilitating disorder that is notoriously difficult to treat. Medication (antispasmodics, bulking agents, laxatives or antidepressants), diet and lifestyle adjustment have been the principal therapeutic options for treatment. Most patients are interested in the role of diet in IBS (Locke *et al.*, 2000; Monsbakken *et al.*, 2006; Halpert *et al.*, 2007) and perceive that some of their symptoms are related to food (Monsbakken *et al.*, 2006) and, consequently, many restrict their intake of certain foods to control symptoms (Halpert *et al.*, 2007).

Numerous approaches to dietary management of IBS have been investigated. Modification of dietary fibre has been a mainstay of IBS management for many years (Brandt *et al.*, 2002; Bijkerk *et al.*, 2004; Ford *et al.*, 2008). Identification and management of lactose intolerance is often also considered [National Institute for Health and Clinical Excellence (NICE), 2008; Reeves & Lomer, 2008].

Although there are no randomised controlled studies supporting the use of adjusting dietary components such as caffeine and fat (Heizer *et al.*, 2009), these also often form the basis of general dietary advice to patients. Meanwhile, there is emerging but conflicting evidence for the use of probiotics in IBS (Parkes *et al.*, 2010).

NICE have published guidelines for dietary intervention in IBS (Reeves & Lomer, 2008). They cover both general dietary advice (e.g. regular meal pattern, adjusting fibre intake and reducing alcohol and caffeine) and symptom specific dietary advice for wind and bloating, diarrhoea, and constipation. Information is provided on reducing resistant starches, based on evidence that high intakes can cause symptoms in healthy individuals (Muir *et al.*, 2004; Storey *et al.*, 2007), although there is a lack of randomised controlled data. In addition, the resource provides information on addition of linseeds, which may improve constipation and abdominal symptoms (Tarpila *et al.*, 2004) and the use of probiotics. These resources are available from NICE and are widely used across the UK.

The restriction of poorly absorbed carbohydrates may improve symptoms of IBS (Jain *et al.*, 1985; Rumessen & Gudmand-Hoyer, 1988; Dear *et al.*, 2005). An innovative approach to the treatment of IBS has recently been developed that comprises a reduction in fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) in the diet (Barrett & Gibson, 2007; Shepherd *et al.*, 2008). These short-chain carbohydrates have common functional properties in that they are poorly absorbed, osmotically active (Barrett *et al.*, 2010) and rapidly fermented by bacteria (Ong *et al.*, 2010). A low FODMAP diet appears to be associated with a reduction in IBS symptoms in retrospective studies (Shepherd & Gibson, 2006; Gearry *et al.*, 2009) and others have reported similar durable outcomes with dietary restriction of one or more of these carbohydrates (Fernandez-Banares *et al.*, 2006; Choi *et al.*, 2008). More recently, a randomised placebo-controlled trial demonstrated that, in patients with IBS whose symptoms improved on a low FODMAP diet, recurrence of symptoms occurred on rechallenge with fructose, fructans and a combination of the two, but not with placebo (Shepherd *et al.*, 2008). Although initial reports of the success of a FODMAP-modified diet are encouraging, they all originate from a single centre in Australia. The clinical effectiveness of the low FODMAP diet and the ability of patients to follow it have not been formally evaluated outside of Australia. Furthermore, the utility of the low FODMAP diet has not been compared with standard dietary advice used for IBS patients.

The present study aimed to compare, in an IBS outpatient service, the clinical effectiveness of the low FODMAP diet with the standard NICE guidelines for dietary therapy for IBS.

Materials and methods

Dietetic service

A low FODMAP dietetic service was established with the assistance of an experienced dietitian who had implemented the diet in Australia for several years. Numerous written and visual resources were developed and tailored to the UK context. This required extensive review of food availability and composition of processed foods in the UK. Greater availability of prepacked foods, particularly convenience meals, frequent addition of high FODMAP ingredients (e.g. oligofructose) to packaged foods and medications, and differences in the availability and consumption of fruit and vegetables resulted in the need for significant adaptation of existing patient information.

Examples of differences in Australian and UK food supply and diet of relevance to the low FODMAP diet include differences in names of fruits and vegetables (e.g. eggplant in Australia is the same as aubergine in the UK), removal of foods from 'safe food lists' that are not widely available in the UK (e.g. durian), and the addition of some fruits and vegetables to the resource where UK varieties were considered to be equivalent (e.g. clementine equivalent to tangelo). More detail was provided on label reading, particularly for added fructose, oligofructose and inulin. Lists of suitable and unsuitable foods, meal plans and recipes required adaptation, and lists of suitable supermarket foods required formulation based on UK availability and composition. Dietitians within the department, working in primary and secondary care, attended training sessions and became experienced in delivering low FODMAP dietary advice.

Study population

Consecutive adult patients with IBS ($n = 82$) using NICE criteria (abdominal pain or discomfort or bloating or change in bowel habit for at least 6 months; NICE, 2008) who returned for a follow up dietetic outpatient visit for dietary management of their symptoms were included over the course of the 9-month evaluation. The NICE criteria, which are based on consensus opinion, were used to define IBS because this study was on patients who were referred by gastroenterologists as part of normal clinical practice. The Rome III criteria were not used because they are generally used as a research tool rather than in the clinical setting. All patients had been first diagnosed with IBS by their primary care physician or gastroenterologist; second, had been referred for dietary advice; and, third, had been seen by a dietitian within

the previous 2–6 months for management of their symptoms.

Dietary advice

Symptom and dietary assessment was carried out on all patients. A detailed diet history was taken to assess nutritional adequacy and establish current eating habits (meal pattern, meal location etc.), in line with routine clinical practice. Careful consideration of these parameters helped with individualisation of dietary advice. Consultations were undertaken by the same series of dietitians, irrespective of which dietary advice group patients were in, with a similar level of motivation and appointment duration.

Patients who had been seen by a dietitian prior to June 2009 (i.e. before implementation of the low FODMAP service) received standard dietary advice based on the general NICE guidelines ('standard' group). Depending on symptoms, and where appropriate, certain patients received specific NICE-based advice (e.g. for lactose malabsorption, the use of probiotics and exclusion diets).

Patients seen after the implementation of the low FODMAP service were advised on reducing dietary FODMAP intake ('low FODMAP' group). Fructooligosaccharides (e.g. wheat, onion, garlic), galactooligosaccharides (e.g. legumes) and polyols (e.g. sugar free gums and some fruits and vegetables) were restricted in all patients. Clinical judgement and hydrogen breath test results, where available, dictated whether fructose and/or lactose restriction occurred. The nature of the written information provided at the initial consultation was specific to the dietary advice given, and consisted of a two-page written resource for the standard group and a colour booklet for the low FODMAP group.

Questionnaire

At the follow-up appointment, all patients were asked to complete a 16-point questionnaire. Information on demographics, predominant symptoms and dietary treatment provided at the initial dietetic consultation was extracted using information from the dietetic record card and patients provided verbal responses for the remainder of the questionnaire. The patients rated symptom changes for bloating, abdominal pain/discomfort, flatulence/wind, diarrhoea, constipation, nausea and energy levels using a seven-point Likert scale taken from the validated IBS Global Improvement Scale (substantially worse, moderately worse, slightly worse, no change, slightly improved, moderately improved, substantially improved, or never had the symptom) (Gordon *et al.*, 2003). Details of the symptom data collection method are available as Supporting information (Appendix S1).

A further four statements relating to satisfaction with symptom response and dietary advice were also included. These were 'Overall, I am satisfied with the improvement in my symptoms', 'I found the diet easy to follow', 'I found the written information easy to understand' and 'I would be interested in further changing my diet to improve my symptoms'. These statements were scored using a five-point Likert scale (strongly disagree, disagree, neutral, agree, strongly agree). All data were anonymous and confidential. Patients were encouraged to answer questions honestly to reduce response bias. Ethical approval was not required for the study because it was an evaluation of the dietetic service.

Statistical analysis

All data were analysed using SPSS, version 18 (SPSS Inc, Chicago, IL, USA). Demographic data and baseline symptom comparisons and types of standard dietary advice administered were analysed descriptively where appropriate and differences between groups for symptom response and satisfaction were determined using the chi-squared test. To provide clinically meaningful data, and robust data distribution to warrant chi-squared analysis, symptom responses were collapsed into a dichotomous response set (improved, not improved) and a magnitude of improvement response set (worsened/no change, slightly improved, moderately improved, substantially improved). Satisfaction responses were collapsed into agree and disagree responses. $P < 0.05$ (two-tailed) was considered statistically significant.

Results

A total of 82 consecutive patients were studied: 39 in the standard group and 43 in the low FODMAP group. Demographics of the study population are detailed in

Table 1 Demographic information and baseline symptoms

Characteristic	All patients (n = 82)	Standard (n = 39)	FODMAP (n = 43)	P
Female, n/N (%)	58 (71)	30 (77)	28 (65)	0.241
Age, mean (SD)*	38.1 (12.8)	38.5 (12.2)	37.8 (13.5)	0.235
Symptoms on initial visit, n/N (%)				
Bloating	58 (71)	26 (67)	32 (74)	0.441
Abdominal pain	45 (55)	21 (54)	24 (56)	0.858
Diarrhoea	49 (60)	22 (56)	27 (63)	0.556
Constipation	33 (40)	17 (44)	16 (37)	0.556

*Chi-squared was used to test for differences between groups except where an independent samples t-test was used.
FODMAP = low FODMAP.

Table 1. There were no significant differences between groups with regard to age and gender. Importantly, there were no differences in the prevalence of each symptom between the groups before dietary intervention. Bloating was the most frequently reported symptom (71%) and diarrhoea (60%), abdominal pain (55%) and constipation (40%) were also reported.

Patients were seen in secondary care (n = 73, 89%) or primary care (n = 9, 11%). Comparisons of the effectiveness of advice given between these groups were not performed because of the low numbers recruited from primary care.

Standard dietary advice predominantly consisted of the general dietary advice recommended in the NICE guidelines (74%). Others in the standard group were provided with more specific advice based on the NICE guidelines, such as reducing lactose intake (12%), increasing or decreasing total fibre intake (8%) or an exclusion diet (avoidance of one or two suspect trigger foods, e.g. wheat, milk; 5%).

Symptom change

A higher proportion of the low FODMAP group reported improvement for each symptom assessed compared to the standard group, as represented by symptom response data in Table 2. There were significantly more patients in the low FODMAP group who reported improvement in bloating (low FODMAP 82% versus standard 49%, P = 0.002), abdominal pain (low FODMAP 85% versus standard 61%, P = 0.023) and flatulence (low FODMAP 87% versus standard 50%, P = 0.001). When the magnitude of symptom response was assessed for bloating, the low FODMAP group were less likely to report a deterioration or lack of improvement in symptoms than the standard group (low FODMAP 18% versus standard 51%, P = 0.026), as was the case for flatulence (low FODMAP 13% versus standard 50%, P = 0.01). There was a trend for more patients in the low FODMAP group to report symptom improvement for diarrhoea than in the standard group, although this was not statistically significant (low FODMAP 83% versus standard 62%, P = 0.052).

When the results were analysed for the magnitude of improvement in symptoms of diarrhoea, there was a significant difference between groups (P = 0.017). Fewer patients in the low FODMAP group reported diarrhoea to be 'slightly improved' compared to the standard group (low FODMAP 8% versus standard 24%), although more patients in the low FODMAP group reported moderate improvement (low FODMAP 28% versus standard 7%) or substantial improvement (low FODMAP 47% versus standard 31%).

When all symptoms were combined into a composite symptom score, there was a significant difference between groups, with more patients in the low FODMAP group having an improvement in score (low FODMAP 86% versus standard 49%, $P < 0.001$). The difference in degree of improvement was predominantly driven by a strong trend for fewer patients in the low FODMAP group

Table 2 Symptom response of standard and FODMAP groups

Symptom	Group	Improved	P*	No change or worse	Slightly improved	Moderately improved	Substantially improved	$P^{\dagger}$
Bloating	Standard	17/35 (49)	0.002	18/35 (51)	3/35 (9)	6/35 (17)	8/35 (23)	0.026
	FODMAP	32/39 (82)		7/39 (18)	5/39 (13)	11/39 (28)	16/39 (41)	
Abdominal pain/discomfort	Standard	20/33 (61)	0.023	13/33 (40)	7/33 (21)	4/33 (12)	9/33 (27)	0.014
	FODMAP	29/34 (85)		5/34 (15)	3/34 (9)	13/34 (38)	13/34 (38)	
Flatulence/wind	Standard	14/28 (50)	0.001	14/28 (50)	7/28 (25)	4/28 (14)	3/28 (11)	0.01
	FODMAP	33/38 (87)		5/38 (13)	15/38 (40)	7/38 (18)	11/38 (29)	
Diarrhoea	Standard	18/29 (62)	0.052	11/29 (38)	7/29 (24)	2/29 (7)	9/29 (31)	0.017
	FODMAP	30/36 (83)		6/36 (17)	3/36 (8)	10/36 (28)	17/36 (47)	
Constipation	Standard	10/22 (45)	0.161	12/22 (55)	6/22 (27)	0/22 (0)	4/22 (18)	0.007
	FODMAP	10/21 (67)		7/21 (33)	1/21 (5)	7/21 (33)	6/21 (29)	
Nausea	Standard	4/14 (29)	0.04	10/14 (71)	1/14 (7)	2/14 (15)	1/14 (7)	0.155
	FODMAP	10/15 (67)		5/15 (33)	4/15 (27)	2/15 (13)	1/15 (27)	
Energy levels	Standard	11/30 (37)	0.042	19/30 (63)	4/30 (13)	5/30 (17)	2/30 (7)	0.235
	FODMAP	20/32 (63)		12/32 (37)	6/32 (19)	10/32 (31)	4/32 (13)	
Composite score	Standard	19/39 (49)	<0.001	20/39 (51)	8/39 (21)	7/39 (18)	4/39 (10)	0.002
	FODMAP	37/43 (86)		6/43 (14)	9/43 (21)	16/43 (37)	12/43 (28)	

Data are presented as n/N (%). Chi-squared was used to test for differences between groups.
*$P < 0.05$, statistical difference between groups in proportion of patients having any symptom improvement.
$^{\dagger}P < 0.05$, statistical difference between groups in proportion of patients for degrees of symptom improvement.
FODMAP = low FODMAP.

reporting deterioration or no change in symptoms (low FODMAP 14% versus standard 51%).

There were no significant differences in the proportion of patients reporting improvement in constipation between groups. Analysis of the magnitude of the effect revealed that there were differences between groups, driven by a higher proportion of patients reporting moderate improvement in the low FODMAP group (low FODMAP 33% versus standard 0%, $P = 0.007$).

Significantly more patients on the low FODMAP diet had an improvement in nausea (low FODMAP 67% versus standard 29%, $P = 0.04$) and energy levels (low FODMAP 63% versus standard 37%, $P = 0.042$). However, there was no significant difference when analysed according to the magnitude of the response.

Satisfaction with symptom response and dietary advice

In total, 32/42 (76%) patients in the low FODMAP group reported satisfaction with their symptom response compared to 20/37 (54%) in the standard group ($P = 0.038$). There were no differences between the groups when reporting ease of understanding the written information (low FODMAP 100% versus standard 94%, $P = 0.116$) or ease of following the diet (low FODMAP 70% versus standard 85%, $P = 0.112$). More patients in the low FODMAP group were interested in implementing further change to their diet to improve their symptoms (low FODMAP 25% versus standard 5%, $P = 0.014$).

A subgroup ($n = 36/43$, 84%) of the low FODMAP group were asked about their level of compliance to the diet and time to symptom improvement. Most patients reported following the diet strictly (23/36, 64%) or at least 50% of the time (11/36, 30%). In the 10 patients who were asked, the mean (median, range) time taken for symptom resolution was 3.5 (2, 2–8) weeks. Correlation between compliance and symptom response was not analysed because of the small numbers.

Discussion

Irritable bowel syndrome causes significant morbidity and the available treatments have variable clinical effectiveness. Dietary treatments are considered important by patients, although there is limited high quality evidence to support their use and the effectiveness of standard NICE dietary guidelines for IBS has not been formally examined. FODMAPs have been shown to trigger IBS symptoms in a randomised, blinded, controlled re-challenge study (Shepherd *et al.*, 2008) but data comparing a low FODMAP diet with other commonly used dietary approaches are lacking. The present data suggest that, although standard dietary advice is effective in IBS, low FODMAP dietary advice implemented by trained dietitians is more successful in improving composite symptoms and that patient satisfaction with symptoms is also greater. The magnitude of response to the low FODMAP diet supports previous Australian data in patients with similar IBS symptom profiles (Shepherd & Gibson, 2006). This is likely to be a result of a reduction in FODMAPs rather than a change in other components of the diet or placebo, as discussed previously (Shepherd *et al.*, 2008). However, it is not possible to completely exclude the fact that avoidance of non-FODMAP components in the diet, such as gluten, might have been in part responsible for its success. That said, there is only preliminary evidence of gluten as a cause of symptoms in a subset of patients with IBS (Biesiekierski *et al.*, 2011).

The present study group were representative of typical IBS patients seen in primary and secondary care. The prevalence of bloating, diarrhoea or constipation before dietary intervention was consistent with a large cross-sectional European study, although abdominal pain was less common (present study 54% versus European study 80%) (Hungin *et al.*, 2003).

Excessive gas production may be associated with bloating, pain and flatulence. Recent evidence indicates that bloating is likely to be a result of increased visceral sensitivity and abnormal gas handling (Gunnarsson & Simrén, 2009). Hence, therapy that reduces intestinal gas production is theoretically likely to improve bloating. Abdominal pain has been strongly correlated with level of abdominal bloating (Houghton *et al.*, 2006), and so interventions that reduce bloating, such as the low FODMAP diet, may also improve pain. Large samples of flatus gas collected in a laboratory setting consist almost entirely of fermentation gases (Tomlin *et al.*, 1991), indicating that a reduction in fermentation, by reducing FODMAPs, should improve flatulence. Indeed, after only 24–48 h, a low FODMAP diet reduces colonic gas production in healthy people as well as patients with IBS compared to a high FODMAP diet (Ong *et al.*, 2010). In the present study, a significant difference between groups in symptom response for bloating, pain and flatulence was therefore not unexpected.

A randomised cross-over intervention study has shown that FODMAP restriction reduces osmotic load at the terminal ileum (Barrett *et al.*, 2010), providing physiological evidence for the effectiveness of a low FODMAP diet in patients with diarrhoea. Other studies report improvements in diarrhoea in IBS patients (Shepherd & Gibson, 2006) and inflammatory bowel disease patients with functional symptoms (Gearry *et al.*, 2009). In the present study, there was a nonsignificant trend for patients on the low FODMAP diet to have an improvement in diarrhoea compared to patients in the

standard group. Furthermore, significant differences were seen in the magnitude of response; however, these findings should be interpreted with caution as a result of the low number of responses and therefore the potential for type 1 errors.

There were no differences between groups for symptom response in patients with constipation. The effectiveness of the NICE dietary guidelines on constipation has not been assessed until now. Isphagula husk has been shown to be more effective than placebo for IBS symptoms in a recent meta-analysis (Ford *et al.*, 2008). However, conclusions about the effectiveness of fibre manipulation on IBS symptoms are difficult because of the heterogeneity of studies with respect to the classification of IBS, subtypes of IBS included in studies, lack of validated tools to assess symptoms and differences in the type of fibre studied. A reduction in FODMAPs should reduce osmotic fluid transit into the gut lumen, increasing the likelihood of constipation. Reduction in the FODMAP content of the diet may also result in a simultaneous reduction in fibre intake in some patients if wholegrain wheat products or high FODMAP fruit and vegetables are not replaced with suitable low FODMAP alternatives. In clinical practice, the reintroduction of high FODMAP foods to tolerance may assist stool softening in those who become constipated on FODMAP restriction.

Dietary intake was not assessed and therefore it was not possible to determine fibre intake in the present study. Accordingly, the finding that there was a significant difference in the magnitude of response between groups for constipation, driven by a large proportion in the low FODMAP group reporting moderate improvement, is somewhat unexpected. Although this should be interpreted with caution as a result of the low number of responses, it may indicate a role for the low FODMAP diet in a subgroup of patients with constipation. It also concurs with experience from other centres (Shepherd & Gibson, 2006) and might, in theory, be explained by a decrease in methane production in patients on a low FODMAP diet (Ong *et al.*, 2010) because methanogenesis is associated with slowed intestinal transit (Pimentel *et al.*, 2006; Attaluri *et al.*, 2010).

Nausea and energy levels improved more often in the low FODMAP group than in the standard group. These data are difficult to explain mechanistically, although they are probably related to improvement in systemic symptoms driven by IBS. In addition, previous studies report similar outcomes, with less nausea and lethargy being reported in IBS patients on a low FODMAP diet compared to a high FODMAP diet (Ong *et al.*, 2010).

Dietary intervention is only effective if patients are able to comply with the diet. This relies not only on patient motivation, but also on the resources that they are provided with to support them in following the diet. It has also been speculated that breath testing may 'prime' the patient for low FODMAP dietary advice. Breath testing was conducted in a small proportion of the low FODMAP group, and the type of tests conducted and the results of the tests were not recorded for the purposes of the present study.

A low FODMAP diet is novel in the UK and some may consider it difficult to understand and follow; however, this was not found to be the case in the present study. Patients in both groups reported that the written material was easy to understand and the diet was easy to follow, indicating that verbal delivery and written resources were clear and effective. Interestingly, a higher percentage of the low FODMAP group wanted further information on how to change their diet despite the fact that the diet was effective. This may be a result of the success of the diet encouraging patients to explore further possibilities and/or the fact that patients receiving standard advice were more likely to have no improvement or worsening of symptoms and may therefore have lost faith in dietary intervention.

The time to response data is interesting and has not been described previously. Clinically, some patients report rapid symptom response within 2 weeks, whereas, in others, it may take up to 8 weeks. Where osmotic and motility changes are the only mechanisms causing symptoms then it would be expected that the response to the low FODMAP diet would be rapid, and this has been shown to occur within 24–48 h where dietary intake is controlled (Ong *et al.*, 2010). However, other potential mechanisms through which a low FODMAP diet has been suggested to improve symptoms include an effect on the gastrointestinal microbiota, which are known to be in dysbiosis in patients with IBS (Parkes *et al.*, 2008). Dietary mediated changes to the gastrointestinal microbiota may take considerably longer than 2 weeks to occur. However, the effect of a low FODMAP diet on the gastrointestinal microbiota in the context of IBS is not known, and such research is urgently required to delineate whether indeed this is a mechanism through which symptom response is mediated, as well to determine whether a reduction of FODMAPs has any effect on the number and species of the microbiota, and therefore overall gut health.

It was expected that a low FODMAP diet would be more difficult to follow in the UK than in Australia as a result of a more varied food supply, the addition of FODMAPs to many manufactured foods and the lack of UK-specific FODMAP food composition data. Nevertheless, patients reported high levels of dietary compliance compared to that reported previously (Shepherd & Gibson, 2006; Gearry *et al.*, 2009).

There are several limitations to the present study. Although the assessment of symptom response was

recorded prospectively, the questionnaire was completed at the time of review consultation, which may have introduced bias. Details regarding the types of symptoms experienced at baseline were collected from the dietetic record cards, therefore leading to the assumption of accurate record keeping. Bias regarding symptom change data was minimised as much as possible by instructing dietitians on consistent administration of the symptom questionnaire for all patients. In addition, because the data were collected in the same way for both groups of patients, any response-bias is likely to be similar in both groups. Other variables, such as use of or changes in medication usage, probiotic intake and overall dietary intake (e.g. meal frequency, caffeine, alcohol), were not recorded and could have contributed to the changes in IBS symptoms being investigated. Allocation to standard or low FODMAP dietary advice was based upon introduction of the service rather than by randomisation. However, despite this, there were no differences in demographics between groups.

In addition, patients were seen by different dietitians and therefore differences in communication and style of dietary education had the potential to affect adherence and motivation between different patients; however, this bias was applicable to both groups. It is also impossible to exclude the impact of differences in presentation and detail of the written resources on compliance and effectiveness of the dietary approaches. However, the level of detail provided in the low FODMAP booklet is necessary for proper implementation.

Importantly, it must be noted that there is some minor overlap between low FODMAP advice and standard NICE advice (Table 3). For example, the NICE guidelines recommend a reduction in sugar free products containing sorbitol, although whether this specific advice was given to each patient in the standard group was not recorded. Furthermore, the impact on the outcome of this study was likely to be minimal because sorbitol also occurs naturally in certain fruits (e.g. apricots), which were not avoided using standard NICE advice.

Therefore, there are no reasons to believe that the patients in the standard or low FODMAP groups differed in any meaningful way except for the dietary advice they received. Finally, only patients who attended a review appointment were examined in the present study, potentially leading to ascertainment bias as a result of the experience of those patients not attending for review not being available, although, again, this bias is likely to be equal between the two groups.

In summary, this preliminary study supports the use of the low FODMAP diet in patients with IBS in the UK, with symptom response being superior to standard dietary therapy. In addition, markedly fewer patients reported no change or worsening of symptoms in response to the low FODMAP diet compared to those receiving standard dietary advice. These results clearly indicate that a randomised controlled trial of low FODMAP dietary advice is required to substantiate these results further. Additionally, although the low FODMAP diet, currently based on Australian data (Muir *et al.*, 2007, 2009), has shown to be useful in this group, composition analysis of the UK food supply will further refine and optimise the diet for the UK population.

Table 3 Key aspects of the NICE dietary guidelines for IBS and the low FODMAP diet

NICE dietary guidelines for IBS
• Healthy eating principles (e.g. regular eating, taking time to eat). Limit high fat foods, ensure a good intake of non caffeinated fluids, limit fizzy drinks
• Limit insoluble fibre for diarrhoea and increase gradually for constipation
• Limit sugar free sweets and foods containing sorbitol
• Limit fruit to 3 portions a day
• Avoiding resistant starch may be useful (e.g. pulses, sweetcorn, green bananas, part-baked and reheated bread)
• Addition of oats and linseeds may be helpful
Low FODMAP diet
• Reduction in high fructan foods (e.g. wheat, onion) and substitution with wheat free and other low fructan alternatives
• Reduction in high galactooligosaccharide foods (e.g. chickpeas, lentils)
• Reduction in high polyol foods and avoid polyol-sweetened sources. Replace with suitable fruits and vegetables
• In those with lactose malabsorption, reduction in high lactose foods (e.g. milk, yoghurt) by restricting volume in one sitting or substitution with lactose free products
• In those with fructose malabsorption, reduction in excess fructose foods (e.g. honey)

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HS contributed to the design, data collection and input, statistical analysis, and write up of the final submission. KW contributed to the design, statistical analysis and editing of the final submission. PI contributed to the design and editing of the final submission. ML contributed to the design, data collection and editing of the final submission. All authors read and approved the final version of the manuscript submitted for publication.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. IBS satisfaction survey.

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