BDA The Association of UK Dietitians
THE OFFICIAL JOURNAL OF THE BRITISH DIETETIC ASSOCIATION
Journal of Human Nutrition and Dietetics

PROFESSIONAL GUIDELINE

British Dietetic Association systematic review and evidence-based practice guidelines for the dietary management of irritable bowel syndrome in adults (2016 update)

Y. A. McKenzie,[1] R. K. Bowyer,[2] H. Leach,[3] P. Gulia,[4] J. Horobin,[5] N. A. O'Sullivan,[6] C. Pettitt,[7] L. B. Reeves,[8] L. Seamark,[9] M. Williams,[9] J. Thompson,[10] M. C. E. Lomer[6,11] (IBS Dietetic Guideline Review Group on behalf of Gastroenterology Specialist Group of the British Dietetic Association)

[1]Nuffield Health The Manor Hospital, Oxford, UK
[2]Department of Nutrition and Dietetics, Great Western Hospitals NHS Foundation Trust, Swindon, UK
[3]Department of Nutrition and Dietetics, Southampton NHS Foundation Trust, Southampton, UK
[4]Dr Ashok Ayurveda Clinic, Birmingham, UK
[5]Department of Nutrition and Dietetics, North Middlesex University Hospital NHS Trust, London, UK
[6]Faculty of Life Sciences and Medicine, Diabetes and Nutritional Sciences Division, King's College London, London, UK
[7]Faculty of Medicine, Imperial College London, London, UK
[8]Allergy Services, Oxford University Hospitals NHS Foundation Trust, Oxford, UK
[9]Specialist Gastroenterology Community Dietetic Service, Somerset Partnership NHS Foundation Trust, Bridgwater, UK
[10]Calm Gut Clinic, Todmorden, UK
[11]Department of Nutrition and Dietetics, St Thomas' Hospital, Guy's and St Thomas' NHS Foundation Trust, London, UK

Keywords
alcohol, caffeine, diet, dietary fibre, dietary habits, elimination diets and food hypersensitivity, fat, fermentable carbohydrates, fluid, gluten, guidelines, healthy eating, low FODMAP diet, milk and dairy, probiotics, spicy food, systematic review.

Correspondence
Y. A. McKenzie, Nuffield Health The Manor Hospital, Oxford, Beech Road, Headington, Oxford OX3 7RP, UK.
Tel.: +44 7966 878758
Fax: +441865 307788
E-mail: yvonne@digestiblenutrition.co.uk

How to cite this article
McKenzie Y.A., Bowyer R.K., Leach H., Gulia P., Horobin J., O'Sullivan N.A., Pettitt C., Reeves L.B., Seamark L., Williams M., Thompson J. & Lomer M.C.E. (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*.
doi:10.1111/jhn.12385

Abstract

Background: The first British Dietetic Association (BDA) guidelines for the dietary management of irritable bowel syndrome (IBS) in adults were published in 2012. Subsequently, there has been a wealth of new research. The aim of this work was to systematically review the evidence for the role of diet in the management of IBS and to update the guidelines.
Methods: Twelve questions relating to diet and IBS were defined based on review of the previous guideline questions, current evidence and clinical practice. Chosen topics were on healthy eating and lifestyle (alcohol, caffeine, spicy food, elimination diets, fat and fluid intakes and dietary habits), milk and dairy, dietary fibre, fermentable carbohydrates, gluten, probiotics and elimination diets/food hypersensitivity. Data sources were CINAHL, Cochrane Register of Controlled Trials, Embase, Medline, Scopus and Web of Science up to October 2015. Studies were assessed independently in duplicate using risk of bias tools specific to each included study based on inclusion and exclusion criteria for each question. National Health and Medical Research Council grading evidence levels were used to develop evidence statements and recommendations, in accordance with Practice-based Evidence in Nutrition Global protocol used by the BDA.
Results: Eighty-six studies were critically appraised to generate 46 evidence statements, 15 clinical recommendations and four research recommendations. The IBS dietary algorithm was simplified to first-line (healthy eating, provided by any healthcare professional) and second-line [low FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) to be provided by dietitian] dietary advice.
Conclusions: These guidelines provide updated comprehensive evidence-based details to achieve the successful dietary management of IBS in adults.

Introduction

Irritable bowel syndrome (IBS) in adults is a global problem, with prevalence rates of 7–21% [1]. It is a chronic gastrointestinal disorder characterised by fluctuating abdominal pain or discomfort associated with an altered bowel habit in the absence of organic disease [2]. Subtypes of IBS are classified as diarrhoea-predominant (IBS-D), constipation-predominant (IBS-C), both diarrhoea and constipation (IBS-M) or unspecified (IBS-U) [3]. Dietary triggers are common, with up to nine out of 10 individuals reporting that food generates symptoms [4,5]. Two-thirds of individuals with IBS initiate dietary restrictions to improve symptoms [6], and so dietary management is an important option within medical treatment. The first British Dietetic Association (BDA) guidelines for the dietary management of IBS were developed several years ago [7] but lacked comprehensive critical appraisal for all dietary management strategies, notably within first-line approaches on healthy eating and lifestyle. Evidence on the efficacy of a low fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAP) diet has rapidly developed [8–10], has been incorporated into other IBS guidelines [2,11], and needs further review. In addition, the potential for gluten to initiate gastrointestinal symptoms has become of interest [12,13] and so the research on the role of gluten in IBS needs appraisal.

The aims of these BDA updated guidelines were (i) to systematically review the evidence on diet in IBS in adults (>16 years), incorporating risk of bias assessment, in relation to symptom generation and management and (ii) to update the previous guidelines with comprehensive evidence-based guidelines and a clinical dietary pathway with international scope and application.

Methods

Guideline development was in accordance with guidance from Practice-based Evidence in Nutrition (PEN) Global (Table 1) and the BDA. An IBS dietetic guideline review group (IBS-DGRG) was formed consisting of 12 registered dietitians belonging to the BDA Gastroenterology Specialist Group. All members developed critical appraisal skills to competently use the assessment tools described; more experienced members teamed up with less experienced members to develop equality in competence.

Questions were developed based on research literature, changing clinical practice and gaps in the evidence base, and included the topics: healthy eating and lifestyle (alcohol, caffeine, spicy food, fat, fluid and dietary habits), milk and dairy, dietary fibre, fermentable carbohydrates, gluten, probiotics and elimination diets/food hypersensitivity. For probiotics, a systematic review of systematic reviews was undertaken, for which details are provided elsewhere [15].

Table 1 Practice-based evidence in nutrition (PEN) evidence grading [14]

Level A – the conclusion is supported by good evidence
The evidence consists of results from studies of strong design for answering the question addressed. The results are both clinically important and consistent with minor exceptions at most. The results are free of any significant doubts about generalisability, bias, and flaws in research design. Studies with negative results have sufficiently large samples to have adequate statistical power
Level B – the conclusion is supported by fair evidence
The evidence consists of results from studies of strong design for answering the question addressed, but there is some uncertainty attached to the conclusion because of inconsistencies among the results from the studies or because of minor doubts about generalisability, bias, research design flaws, or adequacy of sample size. Alternatively, the evidence consists solely of results from weaker designs for the question addressed, but the results have been confirmed in separate studies and are consistent, with minor exceptions at most
Level C – the conclusion is supported by limited evidence or expert opinion
The evidence consists of results from studies of strong design for answering the question addressed, but there is substantial uncertainty attached to the conclusion because of inconsistencies among the results from different studies or because of serious doubts about generalisability, bias, research design flaws, or adequacy of sample size. Alternatively, the evidence consists solely of results from a limited number of studies of weak design for answering the question addressed. Finally, the support for a particular opinion may consist of statement of informed, respected authorities based on their experiences, descriptive studies of reports of expert panels
Level D – evidence is limited
The evidence is limited studies that are either such poor quality or too conflicting that no conclusions can be made. No evidence from either authoritative sources or research involving humans was found

Generic and question-specific inclusion and exclusion criteria were based on Population, Interventions, Comparisons, Outcome measures and Types of study (PICOT) (see Supporting information, Table S1). Search terms for each question are also described in the Supporting information (Table S2).

A systematic literature search using databases CINAHL, Cochrane Register of Controlled Trials, Embase, Medline, Scopus and Web of Science was conducted and relevant studies were identified from January 1985 to October 2015. Studies prior to 1985 were excluded as a result of inadequate definitions of IBS, as well as insufficiently described methodology and outcomes. Only full papers (i.e. not abstracts) in the English language were eligible. Reference lists of included studies were cross-searched for other studies of potential relevance. Each title/abstract of

any potentially relevant study was screened against question-specific inclusion and exclusion criteria by at least one member independently. Reasons for excluding studies are provided in the Supporting information (Table S3). At least two members independently critically appraised each study and assessed risk of bias methodology [16,17] (see Supporting information, Table S4). In line with the hierarchy of research evidence, PEN Global and the BDA guidance, more weighting was given to systematic reviews over randomised controlled trials (RCTs) over case–control studies and finally cross-sectional studies. Critical appraisals and risk of bias assessments were agreed by the question-specific members. Where applicable, measurement of dietary intake was considered as an important aspect of study design.

There are no biological markers to assess symptom response in IBS and so there is much reliance on patient-reported symptom assessment to measure outcomes [18]. Traditional binary and 50% improvement symptom assessment tools have been shown to be reliable, valid and appropriate for use in IBS subtypes [18]. Thus, for the purpose of these guidelines, interpretation of outcomes from different studies have been assumed as being comparable.

Using Australian Government National Health and Medical Research Council (NHMRC) guidance [19], an evidence statement matrix was developed for each question and included considered judgement on the evidence base, consistency, clinical impact and generalisability, which enabled members to link the evidence statements to clinical practice and research recommendations. SIGN grading (2008) for evidence statements from the first guidelines were updated to PEN Global evidence grades (A–D) [14] (Table 1). IBS-DGRG consensus agreement was used throughout the process to resolve any issues. Finally, the IBS dietary algorithm was revised.

Results

The comprehensive literature search identified a potential 3170 papers. Eighty-six of these met the inclusion criteria and included nine systematic reviews, 67 RCTs, six case–control studies and four cross-sectional cohort studies (see Supporting information, Table S5). Risk of bias assessments for all questions except probiotics are shown in Table 2 and probiotics are presented elsewhere [15].

Forty-six evidence statements led to the development of 15 recommendations (Table 3). A recommendation related to fluid intake was not developed as a result of insufficient evidence. Four research recommendations were made. The IBS algorithm for clinical practice was updated and simplified to include only first-line and second-line advice; third-line advice was removed (Fig. 1).

Healthy eating and lifestyle

Individuals with IBS often report that alcohol, caffeine, spicy food and fatty food trigger gastrointestinal symptoms [6,20]. Alcohol affects gastrointestinal motility, absorption and intestinal permeability [21,22]. Caffeine increases gastric acid secretion and colonic motor activity in healthy subjects [23,24] and coffee has also been found to rapidly increase rectosigmoid motor activity [25]. Capsaicin is the active component in hot peppers and, in spicy food, this compound is responsible for accelerating gastrointestinal transit via the transient receptor potential vanilloid-1 (TRPV) causing abdominal pain and burning sensations in healthy individuals [26]. Increased TRPV receptors have been found in individuals with visceral hypersensitivity [27–29]. Fat stimulates the gastrocolic reflex and, when delivered directly into the duodenum, the response is prolonged and exaggerated in individuals with IBS [30,31]. Moreover, fat affects small intestinal motility [32]. Some of these mechanisms may explain why these food components exacerbate IBS symptoms.

The evidence for healthy eating and lifestyle was not systematically reviewed in the first guidelines [7] and so forms part of this update.

1a What effect does alcohol have on IBS symptoms?

Included studies and evidence statements. Four level III case–control studies [33–36] and one level III cross-sectional study [37] fulfilled the inclusion criteria and were evaluated (Table 4). These observational studies reported the perceived effects of alcohol intake with respect to symptom development in individuals with IBS compared to controls. One case–control study reported alcohol induced symptoms [34], whereas two more noted specific symptom associations: loose stools [36]; abdominal pain, nausea, indigestion and diarrhoea in binge drinking (more than four drinks) in women but not in men [35]. The cross-sectional study reported that beer and wine induced symptoms [37]. One case–control study did not find alcohol induced symptoms [33]. There was a high risk of bias providing limited evidence that up to one-third of patients found that alcohol induced or worsened IBS symptoms [34,36,37].

1a-i Alcohol can induce or worsen IBS symptoms [34–37] **C.**

Practical considerations. Assess alcohol intake in relation to symptoms to determine whether a reduction may relieve symptoms and ensure intake is within recommended safe limits.

Table 2 Risk of bias assessment for all studies except probiotics included in the systematic review

Topic and reference	Risk of bias*							
	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8
Healthy eating and lifestyle								
Alcohol								
Böhn *et al.* (2013)[37]	–	?	+	+	?	?	?	+
Faresjo *et al.* (2010)[33]	+	+	?	?	–			
Hayes *et al.* (2013)[34]	+	–	+	–	–			
Reding *et al.* (2013)[35]	–	+	–	+	+			
Simren *et al.* (2001)[36]	?	+	–	?	–			
Caffeine								
Faresjo *et al.* (2010)[33]	+	+	?	?	–			
Hayes *et al.* (2014)[34]	+	–	+	–	–			
Reding *et al.* (2013)[35]	–	+	–	+	+			
Simren *et al.* (2001)[36]	?	+	–	?	–			
Spicy food								
Agarwal *et al.* (2002[42]	+	+	–	–	–			
Böhn *et al.* (2013)[37]	–	?	+	+	?	?	?	+
Bortolotti *et al.* (2011)[40]	?	?	+	+	–	+	–	
Faresjo *et al.* (2010)[33]	+	+	?	?	–			
Gonlachanvit *et al.* (2009)[41]	?	?	+	?	?	+	–	
Hayes *et al.* (2014)[34]	+	–	+	–	–			
Simren *et al.* (2001)[36]	?	+	–	?	–			
Fat								
Böhn *et al.* (2013)[37]	–	?	+	+	?	?	?	+
Faresjo *et al.* (2010)[33]	+	+	?	?	–			
Hayes *et al.* (2014)[34]	+	–	+	–	–			
Serra *et al.* (2002)[43]	?	–	?	+	+	+	+	
Simren *et al.* (2001)[36]	?	+	–	?	–			
Simren *et al.* (2007)[44]	+	+	–	–	–			
Fluid								
No reviewed papers								
Dietary habits								
Guo *et al.* (2015)[47]	–	–	?	–	?			
Kang *et al.* (2011)[46]	–	–	–	–	?	?	–	–
Khadamolhosseini *et al.* (2011)[48]	+	+	–	–	–	?	–	–
Miwa *et al.* (2012)[49]	+	+	–	–	–	?	–	–
Milk and dairy products								
Böhmer *et al.* (1996)[55]	+	+	+	+	–	?	?	
Böhmer *et al.* (2001)[52]	+	–	+	+	–	?	?	
Bozzani *et al.* (1986)[53]	+	+	?	?	?	?	–	
Parker *et al.* (2001)[54]	+	+	+	?	–	+	–	
Vernia *et al.* (1995)[56]	+	+	–	?	–	–	–	
Dietary fibre								
Aller *et al.* (2004)[66]	?	?	–	?	?	+	–	
Arffmann *et al.* (1985)[68]	?	?	–	–	+	+	–	
Bijkerk *et al.* (2009)[67]	+	+	+	+	–	–	+	
Cockerell *et al.* (2012)[76]	+	?	–	+	–	+	–	
Fowlie *et al.* (1992)[74]	–	–	+	?	–	+	–	
Hebden *et al.* (2002)[69]	+	?	+	+	+	+	–	
Kruis *et al.* (1986)[70]	?	?	–	–	+	+	–	
Lucey *et al.* (1987)[71]	?	?	+	+	–	+	–	
Rees *et al.* (2005)[72]	?	?	–	+	+	–	–	
Snook and Shepherd (1994)[73]	+	?	+	+	+	+	+	
Tarpila *et al.* (2004)[75]	?	?	+	+	+	+	–	
Fermentable carbohydrates								
Berg *et al.* (2013)[87]	+	–	–	?	+	+	–	
Böhn *et al.* (2015)[93]	+	+	+	+	+	+		
Olesen *et al.* (2010)[89]	+	+	+	+	+	+	–	
Halmos *et al.* (2014)[88]	+	?	? +	+	+	+	+	
Pedersen *et al.* (2014)[94]	+	+	–	+	?	?	–	
Shepherd *et al.* (2008)[90]	+	+	+	+	+	+	+	
Silk *et al.* (2009)[92]	+	?	–	?	–	–	–	
Staudacher *et al.* (2012)[91]	+	+	–	?	+	+	+	
Gluten								
Biesiekierski *et al.* (2011)[104]	+	+	+	+	+	+	–	
Biesiekierski *et al.* (2013)[105]	+	?	+	?	+	?	+	
Shahbazkhani *et al.* (2015)[103]	?	+	–	+	–	–	–	
Vazquez-Roque *et al.* (2013)[102]	+	+	?	?	+	+	–	
Food hypersensitivity								
No reviewed papers								

*The content and number of risk of bias questions are different for different study designs (see Supporting information Table S4 for details).

Not applicable + Low bias – High bias ? Unclear bias

Table 3 Clinical practice recommendations

Recommendation	PEN Grade[14]	
1 Healthy eating & lifestyle		
Alcohol	Assess intake and screen for signs of binge drinking. Ensure alcohol intake is in keeping with safe national limits (2016)	C
Caffeine	Insufficient evidence to make a recommendation (2016)	D
Spicy food	If related to symptoms assess spicy food intake and trial restriction (2016)	C
Fat	If related to symptoms during or after eating, assess fat intake and ensure it is in line with national healthy eating guidelines (2016)	C
Fluid	No evidence to make a recommendation (2016)	
Dietary habits	Insufficient evidence to make a recommendation (2016)	D
2 Restricting milk and dairy products	In individuals with IBS where sensitivity to milk is suspected and a lactose hydrogen breath test is not available or appropriate, a trial period of a low lactose diet is recommended. This is particularly useful in individuals with an ethnic background with a high prevalence of primary lactase deficiency (2012)	D
	Use a low lactose diet to treat individuals with a positive lactose hydrogen breath test (2012)	D
3 Dietary fibre modification	Avoid using dietary supplementation of wheat bran to treat IBS. Individuals should not be advised to increase their intake of wheat bran above their usual dietary intake from (2012)	C
	For individuals with IBS-C, try dietary supplementation of linseeds of up to 2 tablespoons/day for a 3 month trial. Improvements in constipation, abdominal pain and bloating from linseed supplementation may be gradual (2016)	D
4 Fermentable carbohydrates	For individuals with IBS, consider a low FODMAP diet to improve abdominal pain, bloating and/or diarrhoea for a minimum of 3 [88] or 4 weeks [87,91]. If no symptom improvement occurs within 4 weeks of strict adherence to the diet, then the intervention should be stopped and other therapeutic options considered (2016)	B
	There may be individual tolerance levels to FODMAPs. A planned and systematic reintroduction challenge of foods high in FODMAPs will identify which foods can be reintroduced to the diet and what individual tolerance levels are (2016)	D
5 Gluten	At this time no recommendation can be made to treat IBS symptoms with a gluten-free diet (2016)	D
6 Probiotic products to improve IBS symptoms	Advise that probiotics are unlikely to provide substantial benefit to IBS symptoms. However, individuals choosing to try probiotics are advised to select one product at a time and monitor the effects. They should try it for a minimum of 4 weeks at the dose recommended by the manufacturer (2016)	B
	Taking a probiotic product is considered safe in IBS (2016)	B
7 Elimination diets/ food hypersensitivity	Non-specific elimination diets are no longer valid to improve IBS symptoms (2016)	D

FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides and polyols; IBS, irritable bowel syndrome; IBS-C, IBS – constipation-predominant; PEN, Practice-based Evidence in Nutrition.

1b What effect does caffeine have on IBS symptoms?

Included studies and evidence statements. Four level III case–control studies were included [33–36]. Two related IBS symptoms to coffee and tea consumption [33,36] (Table 4), and one specifically to caffeine intake [34]. One study noted hard stools for tea, as well as gastro-oesophageal reflux, dyspepsia, abdominal pain, loose stools for coffee [36]. Three studies with high risk of bias provided limited evidence for an association with caffeine or coffee consumption on symptoms [33,34,36] and one study with a high risk of bias found no association with caffeine [35].

1b-i Caffeine can induce or worsen IBS symptoms [33,34,36] **C.**

Practical considerations. Assess caffeine intake and, if related to symptoms, consider reducing intake. Daily caffeine intakes up to 400 mg day^{-1} do not raise any safety concerns in the general population, apart from in pregnancy where 200 mg day^{-1} is the current maximum [38]. Behavioural changes (e.g. irritability, nervousness or anxiety) have been reported in caffeine intakes of 5 mg day^{-1} body weight [39].

1c What effect does spicy food have on IBS symptoms?

Included studies and evidence statements. Two level II RCTs [40,41], four level III case–control studies [33,34,36,42] and one cross-sectional level III study [37] met the inclusion criteria and were evaluated (Table 4). Four studies relate to the ingestion of hot or spicy food [33], hot spices [36], cayenne/red pepper or chilli/Tabasco [37], spicy food, curry, chilli [34]. One study compared chilli powder (2 g day^{-1}, 1.87 mg of capsaicin) in a meal with capsule supplementation in IBS-D [41]. In two studies, supplementation was given as four enteric coated tablets per day for 6 weeks (600 mg of red pepper powder per

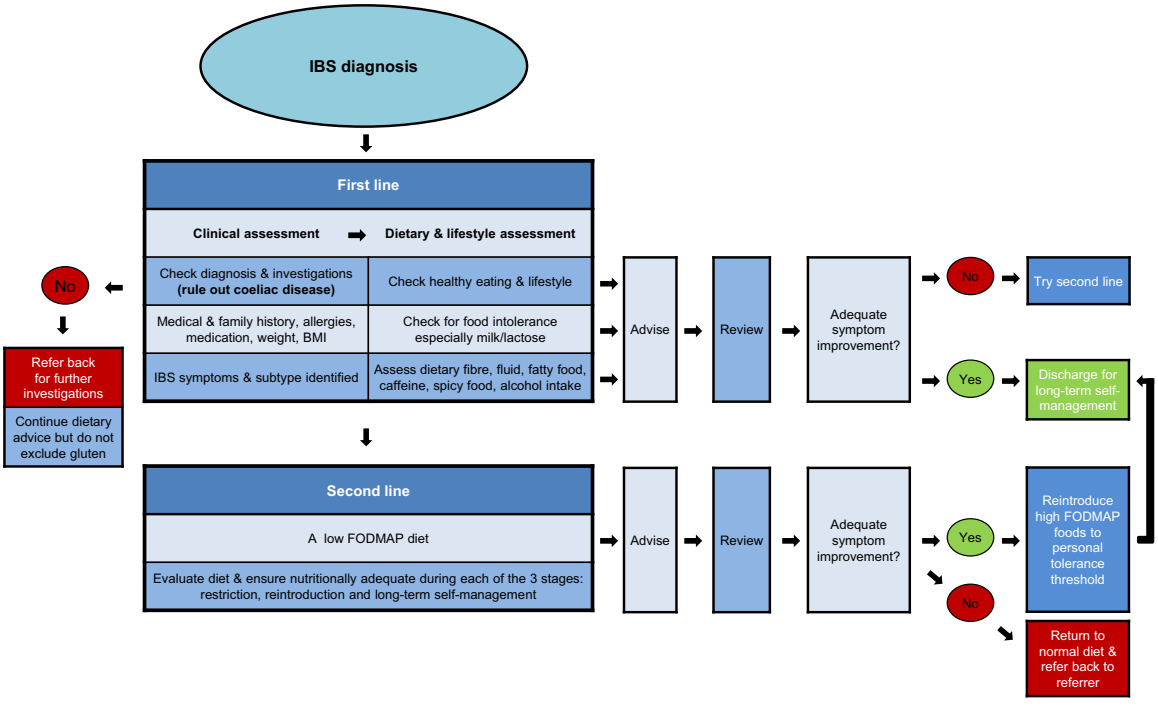

Figure 1 Irritable bowel syndrome (IBS) dietary algorithm: if first-line dietary assessment and interventions indicate that further dietary changes are necessary to improve symptoms, consider second-line dietary intervention, a low fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAP) diet delivered by a dietitian; if this is not appropriate, refer back to the referring clinician as diet may not be the most effective management.

day, 2 mg of capsaicin) [40] and a one-off dose of 10 capsules (10 g of red chilli powder, 14 mg of capsaicin) [42]. One study showed positive effects of high dose chilli powder supplementation on abdominal pain threshold in IBS [40], whereas the other studies demonstrated negative effects of spicy food, hot spices and smaller quantities of chilli powder on symptom induction. Two studies noted specific symptom onset: abdominal pain and oral burning [41], as well as abdominal pain and gastro-oesophageal reflux [36]. The evidence was limited with a high risk of bias.

1c-i Spicy food induced symptoms in IBS [34,36,37], specifically in men [33], and IBS-D [41] **C.**

1c-ii Oral administration of red pepper tablets (2 mg day^{-1} capsaicin) improved abdominal pain threshold but not abdominal pain or bloating compared to placebo when taken for 6 weeks [40] **C.**

Practical considerations. It is useful to assess other components of spicy meals that may contribute to symptoms (e.g. FODMAPs in onion and garlic).

1d What effect does fat have on IBS symptoms?

Included studies and evidence statements. One level II RCT [43], four level III case–control studies [33,34,36,44] and one level III cross-sectional study [37] met the inclusion criteria and were evaluated (Table 4). Four observational studies assessed patient perceived effects of dietary fat on symptom development in individuals with IBS, with three of these including controls [33,34,36] and one having no control [37]. The effect of duodenal lipid infusion was assessed in one RCT using 6.7 g of fat over 2 h [43] and one case–control study using a single dose of 20 g over 1 h [44] on the development of symptoms in individuals with IBS versus controls. One study reported abdominal pain, dyspepsia and flatulence [36]. The evidence was limited with a high risk of bias.

1d-i Fat increased IBS symptoms [33,34,36,37,43,44] **C.**

Practical considerations. A decrease in fat intake may be beneficial in relieving IBS symptoms, in particular meal-related abdominal pain and discomfort associated with visceral hypersensitivity.

1e What effect does fluid intake have on IBS symptoms?

Included studies and evidence statements. There were no eligible studies and so no evidence statements were developed.

Practical considerations. Despite the lack of evidence, a gradual increase in fluid intake is recommended (aim for

Table 4 Characteristics and summary of symptom outcomes of included studies relating to alcohol, caffeine, spicy food and fat

Study (Year) Country	Study design and *N*	Intervention related to question	Outcome	Risk of bias
Agarwal *et al.* (2002) [42] India	RCT 29/35 male IBS 21/23 male healthy controls	10 g of red chilli powder (14 mg of capsaicin), in a single dose of 10 capsules	Chilli did not alter small bowel or colonic transit in IBS patients compared to controls Chilli induced symptoms of nausea, abdominal distension and rectal discomfort more often in IBS than controls Chilli had no beneficial or deleterious effect on overall symptoms	High
Böhn *et al.* (2013) [37] Sweden	Cohort 197 IBS	Questionnaire to assess dietary triggers related to wine/beer, hot spices/Tabasco, fatty food	31% reported wine or beer induced symptoms 28% reported gastrointestinal symptoms related to chilli/tabasco Fried and fatty foods were the most commonly reported foods to induce symptoms (52.3%)	High
Bortolloti and Porta (2011) [40] Italy	RCT 50 IBS 17/23 intervention 25/27 healthy controls	150 mg of red pepper powder (0.50 mg of capsaicin) or placebo in two enteric coated tablets, twice a day for 6 weeks	6 withdrawn from intervention due to intense abdominal pain so dose reduced to 50% 50% red pepper dose reduced abdominal pain and bloating more than placebo	High
Faresjo *et al.* (2010) [33] Sweden	Case–control 347 IBS with new GP diagnosis 2509 healthy controls	Questionnaire to assess dietary triggers related to alcohol, tea/coffee, hot/spicy food, fatty food	Outcomes are adjusted for age No significant differences for symptom induction between IBS and controls in relation to alcohol Females with IBS limited coffee intake. Significantly more females with IBS reported tea and coffee induced symptoms compared with controls (21% versus 11%; $P = 0.004$) but not for males ($P = 0.13$) Significantly more males with IBS reported hot or spicy food induced symptoms more than controls (9% versus 3%; $P = 0.01$) but not for females ($P = 0.53$) Significantly more IBS patients than controls reported high-fat food induced symptoms in males (15% versus 4%; $P < 0.004$) and females (24% versus 9%; $P < 0.001$)	High
Gonlanchavit *et al.* (2009) [41] Thailand	CO RCT 20 IBS-D 38 healthy controls	Spicy meal (standard meal mixed with 2 g of chilli) 2 g of chilli capsules with standard meal Control (standard meal)	IBS patients reported significant abdominal pain, diarrhoea and rectal burning up to 2 h after spicy food or chilli capsules ($P < 0.05$) Controls reported mild abdominal burning ($P < 0.05$)	High

Table 4. Continued

Study (Year) Country	Study design and *N*	Intervention related to question	Outcome	Risk of bias
Guo *et al.* (2015) [47] China	Case–control 78 IBS 79 healthy controls	Food frequency questionnaire including eating and lifestyle habits	IBS patients compared to controls reported increased irregular meals (65.4% versus 36.7%; $P < 0.001$) but no differences in time taken to eat or being a picky eater	High
Hayes *et al.* (2014) [34] Ireland	Case–control 135 IBS 111 healthy controls	Questionnaire to assess dietary triggers related to alcohol, caffeine, spicy food and fatty food	Significantly more IBS patients than controls reported alcohol (14.1% versus 2.7% $P < 0.01$), caffeine (11.9% versus 1.8%; $P < 0.01$), spicy food (39.3% versus 18.9%; $P < 0.01$) and fatty foods (35.6% versus 18.9%; $P < 0.01$) induced symptoms	High
Kang *et al.* (2011) [46] Korea	Cohort 89 IBS army cadets (male = 73) army nurses ($n = 16$)	Lifestyle modification for 9 weeks	63% of IBS patients reported their symptoms improved with lifestyle modification	High
Khademolhosseini *et al.* (2011) [48] Iran	Cohort 215 IBS patients 1978 healthy controls	Questionnaire to assess lifestyle factors that may be associated with IBS	More subjects with IBS consumed fast food (33%) versus subjects without IBS (25%; $P = 0.007$) and less subjects with IBS ate fruit and vegetables (92%) versus subjects without IBS (96%; $P = 0.027$)	High
Miwa (2012) Japan [49]	Cohort 2547 FD or IBS	Questionnaire to assess lifestyle factors that may be associated with IBS	Less subjects with FD or IBS compared to healthy subjects ate meals regularly (70% versus 78%; $P < 0.01$), always had an appetite (45% versus 28%; $P < 0.05$), liked meat (79% versus 84%; $P < 0.01$), and thought their vegetable consumption was insufficient (52% versus 58%; $P < 0.01$)	High
Reding *et al.* (2013) [35] USA	Case–control 166 female IBS	Diary to assess dietary triggers and symptoms related to alcohol and caffeine	Binge drinking (>4 drinks day^{-1}) in IBS was significantly associated with next day diarrhoea ($P = 0.01$), nausea ($P < 0.001$), stomach pain ($P = 0.002$) and indigestion ($P = 0.002$) No association found for light/moderate drinking or caffeine intake	High
Serra *et al.* (2002) [43] Spain	RCT 30 IBS 45 healthy controls, only 30 in data presented	Duodenal fat infusion 6.7 g in 2 h 15 IBS and 15 healthy controls per group: 0 kcal min^{-1} (saline) 0.5 kcal min^{-1}	IBS patients were hypersensitive to the small lipid infusion, reporting crampy pain and bloating with significant gas retention, abdominal symptoms, and distension versus healthy controls ($P < 0.05$)	High

Table 4. Continued

Study (Year) Country	Study design and *N*	Intervention related to question	Outcome	Risk of bias
Simren *et al.* (2001) [36] Sweden	Case–control 330 IBS 80 healthy controls	Questionnaire to assess dietary triggers related to alcohol, coffee, spicy and fatty food	Alcohol induced symptoms in 33% of IBS subjects Alcohol was second most common trigger of loose stools Symptoms associated with tea and coffee were reported in 13% and 39% of IBS patients, respectively. Coffee was seventh most common trigger of symptoms 45% of IBS reported clinically significant symptoms with hot spices IBS versus controls fatty foods problematic ($P < 0.0001$)	High
Simren *et al.* (2007) [44] Sweden	Case–control 61 IBS 20 healthy controls	Duodenal fat infusion of Calogen 120 mL (1.5 kcal mL^{-1} at 2 mL min^{-1} 20 g of fat in 1 h)	Duodenal fat infusion significantly increased pain, measured by change in sensory pressure threshold (8.6 ± 9.1 versus 1.3 ± 5.5 mmHg; $P = 0.001$) and discomfort (7.3 ± 9.8 versus 2.5 ± 5.0 mmHg, $P = 0.006$) in IBS patients versus healthy controls	High

CO, cross-over; DB, double-blind; FD, functional dyspepsia; GP, general practitioner; IBS, irritable bowel syndrome; IBD-D, IBS – diarrhoea-predominant; RCT, randomised controlled trial.

a total intake of 1.5–3.0 L day^{-1}) to improve stool frequency and decrease the need for laxatives in IBS-C [45].

1f What effect do dietary habits have on IBS symptoms?

Included studies and evidence statements. Three level IV cross-sectional studies and one case–control study were included (Table 4) [46–49]. The outcomes were not specific to only IBS patients; one study included functional dyspepsia [49]. Irregular meal pattern, lower consumption of fruit and vegetables and higher fast food consumption may be related to worsening IBS symptoms, although the studies had heterogeneous comparison groups [46–49]. There was limited evidence with a high risk of bias.

1f-i There was inadequate evidence that dietary habits are associated with IBS symptoms **D.**

Practical considerations. Despite the lack of evidence, assess dietary habits and provide advice on how to achieve a healthy balanced diet with a regular meal pattern (breakfast, lunch and evening meal with snacks as appropriate). Good eating lifestyle includes taking time over meals, sitting down to eat, chewing food thoroughly and not eating late at night [2].

What is the effectiveness of restricting milk and dairy products to improve IBS symptoms?

Many individuals with IBS restrict milk and/or dairy products resulting in low calcium intakes [50,51]. To avoid unnecessary exclusion and potential dietary deficiencies, it was important to review the evidence for restricting milk and dairy products, including lactose restriction, and to evaluate its effect on symptoms.

Included studies and evidence statements

No new studies were identified and the five original studies from the first guidelines were re-evaluated (Table 5) [52–56]. In a minority of individuals, dairy exclusion can induce anaphylactic food hypersensitivity reactions when reintroduced [57,58] and so one previous evidence statement was removed. Five remaining evidence statements were still valid and have been updated. The evidence was limited with a high risk of bias.

2i Using a hydrogen breath test with a lactose load of between 25–50 g, the incidence of lactose malabsorption was higher in individuals with IBS compared to individuals without IBS from a white, Caucasian, Northern European background [54,55] **C.**

2ii The incidence of lactose malabsorption was higher in individuals with IBS from ethnic groups with an increased prevalence of primary lactase deficiency [53,56] **C.**

2iii In individuals with IBS and a positive diagnosis of lactose malabsorption using a hydrogen breath test, a low lactose diet improved abdominal symptoms in the short and long-term [53,55,56] **C.**

2iv No specific IBS symptom profiles were associated with lactose intolerance or responded better to a low lactose diet (<9 g day^{-1}) [54,55] **C.**

2v Lactose intolerance is a recognised condition in itself and should be ruled out before the diagnosis of IBS is made, especially in those from an ethnic background where the incidence of primary lactase deficiency is high [55,56] **C.**

Practical considerations

In individuals with IBS, lactose restriction in isolation may only provide marginal symptom benefits. Therefore, lactose restriction is generally considered as part of a low FODMAP diet (see section on fermentable carbohydrates further below).

If individuals wish to follow a milk-free diet, they should be informed that there is no high-quality evidence for this to improve their IBS symptoms. Cow's milk protein elimination in atopic individuals (i.e. eczema, asthma or hay-fever) should only be conducted by appropriately allergy-experienced dietitians. This is a result of the very small risk associated with dangerous anaphylactic reactions on food reintroduction following extended elimination of foods [57,58].

Which type of dietary fibre improves IBS symptoms?

The Scientific Advisory Committee on Nutrition (SACN) and the European Food Safety Authority (EFSA) define dietary fibre as nonstarch polysaccharides, all resistant starches, all nondigestible oligosaccharides with three or more monomeric units and other nondigestible but quantitatively minor components that are associated with the dietary fibre polysaccharides, especially lignin. This incorporates total dietary fibre as measured using the AOAC method 2009.01 and that used in EU nutritional labelling of packaged foods [59–61]. The report advised that the terms insoluble and soluble fibre are phased out because they often co-exist in intact plant cell walls and solubility does not always predict physiological function [61].

Diets rich in dietary fibre are associated with a lower incidence of cardiovascular diseases, coronary events, stroke, type 2 diabetes and colorectal cancers [61]. Dietary fibre has a beneficial effect on the gastrointestinal microbiota and fermentation by-products [62]. The SACN recommends that dietary fibre intake should increase to 30 g day^{-1}, which is consistent with other recommendations of >3 g MJ^{-1} (25–35 g day^{-1}) and 25 g day^{-1} (women) to 30 g day^{-1} (men) [63,64]. High intakes of dietary fibre have

Table 5 Characteristics and summary of symptom outcomes of included studies relating to milk

Study (Year) Country	Study design and N	Intervention	Outcome	Risk of bias
Böhmer *et al.* (1996) [55] Holland	DB non-RCT 70 with IBS	Low lactose diet (<9 g day^{-1}) for 6 weeks 17/70 LHBT +ve 53/70 LHBT −ve	Symptom scores: baseline to 6 weeks LHBT +ve: 13.5–4; ($P < 0.001$) LHBT −ve: 13–11; ($P > 0.05$)	High
Böhmer and Tuynman (2001) [52] Holland	Non-RCT 16 with IBS and LHBT +ve	Low lactose diet (unquantified) for 5 years	Symptom scores: baseline to 5 years 13.5–5.1; $P < 0.001$	High
Bozzani (1986) [53] Italy	Non-RCT 40 with IBS and LHBT +ve	Lactose-free diet (<9 g day^{-1}) for 4 months	Symptoms assessment at 4 months: 3 symptom-free, 21 improved and 16 no change ($P > 0.05$)	High
Parker *et al.* (2001) [54] UK	Non RCT 33 with IBS and LHBT +ve	Low lactose diet (<1 g day^{-1}) for 3 weeks	9 improved versus 14 did not improve* 10 withdrawals/lost to follow-up	High
Vernia *et al.* (1995) [56] Italy	Non-RCT 110 with IBS and +ve LHBT	Lactose-free diet for 3 months	48 remission, 43 partial improvement and 17 no improvement, two not unaccounted for*	High

CO, cross-over; DB, double-blind; IBS, irritable bowel syndrome; LHBT, lactose hydrogen breath test; RCT, randomised controlled trial; +ve, positive; −ve, negative.
**P* value not reported.

been associated with symptom generation in IBS [2,65] and so the risk factors and benefits of specific foods high in dietary fibre need to be taken into consideration.

Included studies and evidence statements

One new and 10 previously evaluated level II RCTs met the inclusion criteria (Table 6) [66–76]. The new study was a parallel open-label, multicentre study assessing linseed (i.e. flaxseed) supplementation and showed no benefit of taking whole or ground linseeds over placebo [76]. There were no new studies on other types of fibre from food, or fibrous foods, such as oats, oat bran, corn or resistant starches.

Dietary fibre intake was reported at baseline and post intervention in five RCTs [66,72–74,76], at baseline only in three RCTs [67,71,75] and four RCTs provided no data [68–70,72].

A placebo containing potentially confounding components was used in seven RCTs [66,68,69,71–74] and the placebo constituents were not described in one study [70]. No placebo was used in two RCTs [75,76].

Five evidence statements were developed and/or updated. The evidence was limited with a high risk of bias.

3i Wheat bran fibre (10–40 g day^{-1} supplementation) did not improve IBS symptoms [67,69–73] **C.**

3ii Increasing dietary fibre intake from cereals and fruit did not improve IBS symptoms [66,74] **C.**

3iii Ground linseeds (6–24 g day^{-1}) relieved constipation, abdominal discomfort and bloating in IBS-C gradually over 3 months [75] **C.**

3iv Ground and whole linseeds were well tolerated in IBS as a dietary fibre supplement [76], although there is conflicting evidence for their effectiveness on symptoms [75,76] **C.**

3v There was insufficient evidence for dietary supplementation with psyllium husk (6–24 g day^{-1}) for up to 3 months to improve symptoms of IBS and IBS-C [67,75] **C.**

Practical considerations

Evidence is lacking on whether the recommended 25–30 g day^{-1} dietary fibre intake for the general population [61] is applicable to individuals with IBS, especially because population data demonstrate inadequate dietary fibre intakes [77]. Consider the symptom profile prior to assessment of dietary fibre intake to determine whether the current intake is optimal for that individual. Check dietary fibre intake from all potential sources (cereals, grains, fruit, vegetables, nuts, seeds, pulses and mycoprotein). If an increase is applicable, encourage a wide variety of high fibre starchy foods (e.g. oats and oat bran, brown rice, rice bran, wholemeal/seeded/granary bread, wholegrain pasta, wholegrain couscous, rye-based bread, potatoes with skin, quinoa). A wide variety is important and takes into account any other dietary restrictions.

Linseeds are a useful source of dietary fibre providing 22.8 g of dietary fibre per 100 g of whole seeds. Start with 4–12 g day^{-1} linseeds and increase up to 24 g day^{-1} (1 tbs = 12 g); the full benefit may take up to 6 months. Ensure linseeds are always consumed with fluid (150 mL fluid tbs^{-1}) [78]. Linseeds can be added to other food (e.g.

yoghurt, breakfast cereal, porridge, homemade bread, casserole, soup, salad). It does not matter whether linseeds are golden or brown, whole or ground. Individuals with co-existing diverticular disease often avoid whole seeds as they may irritate diverticulitis [79]; however, there is no evidence to suggest that seeds, nuts, corn or popcorn induce diverticulosis [80].

Encourage noncaffeinated and non-alcoholic fluids when fibre-rich foods are consumed to enhance the beneficial effects of dietary fibre on transit time.

What are the effects of altering the intake of fermentable carbohydrates to improve IBS symptoms?

Fermentable carbohydrates include FODMAPs, resistant starch and prebiotics. There is some overlap between prebiotics and FODMAPs because some can be considered to meet both definitions. FODMAPs can lead to increased small intestinal luminal fluid and gas production via colonic microbial fermentation [81–83]. Both of these mechanisms can result in functional bowel symptoms in susceptible individuals. Prebiotics are nondigestible, fermentable food components that result in the 'selective stimulation of growth and/or activity of one or a limited number of microbial genera/species in the gastrointestinal microbiota that confer health benefits to the host' [84].

The restriction of individual FODMAPs (e.g. lactose, fructose and sorbitol) has been of interest for a long time; however, reducing their dietary intake has only had a marginal effect on gastrointestinal symptoms, as reviewed elsewhere [85,86]. In recent years, there has been increasing evidence that a diet low in FODMAPs improves IBS symptoms [8,9,85,86]. This update has broadened its scope to appraise dietary restriction of FODMAPs and supplementation of prebiotics on all IBS symptoms, and is not just limited to abdominal bloating as in the previous guidelines.

Included studies and evidence statements

Eight level II RCTs [87–94] were included and summarised in Table 7. One study from the previous guidelines was excluded because it only had 15 IBS patients [95].

One study compared a fructose restricted diet containing less than 2 g of fructose per meal with an 'IBS' diet (details not provided); however, fructose intake was not measured in either arm [87]. One study assessed the low FODMAP diet (3.05 g FODMAPs day^{-1}) versus a typical Australian diet (23.7 g FODMAPs day^{-1}) in a feeding study [88]. Two studies assessed the low FODMAP diet versus habitual diet [91,94]: one reported FODMAP intakes of 17.7 g day^{-1} (low FODMAP diet) versus 29.6 g day^{-1} (habitual diet) [91], whereas the other did not measure FODMAP intake [94]. The latter also compared the low FODMAP diet with a probiotic *Lactobacillus rhamnosus GG* [94], although this was not part of the criteria for comparison in these guidelines. One study assessed the low FODMAP diet (3.8 g FODMAPs day^{-1}) versus traditional National Institute for Health and Care Excellence (NICE)/BDA dietary advice (15.8 g FODMAPs day^{-1}).

Two studies assessed the symptom effect of challenging with 10–20 g day^{-1} FOS [89] or 14–50 g day^{-1} fructose and 7–19 g day^{-1} fructans [90]. One study assessed 3.5–7 g day^{-1} *trans*-galacto-oligosaccharide (β-GOS) supplementation [92]. These guidelines include six new and two updated evidence statements, with good evidence from three RCTs with low or unclear risk of bias indictating that dietitian-led low FODMAP education with up to 6 weeks of FODMAP restriction improves symptoms in IBS, IBS-D and IBS-M. There is currently no evidence to support the low FODMAP diet being nondietitian delivered [88,91,93,94].

4i A low FODMAP diet for 3 [88], 4 [91,93] or 6 [94] weeks improved overall symptoms in IBS and the subtypes IBS-D and IBS-M [88,91,93,94] but not IBS-C [94], abdominal pain [88,93], bloating [88,91,93], flatulence, satisfaction with stool consistency [88], borborygmi, urgency [91] and life interference [93] in IBS **B.**

4ii A fructose restricted diet for 4 weeks improved abdominal pain, bloating and stool frequency in IBS [87] **C.**

4iii A low FODMAP diet improved overall symptoms for the subtypes IBS-D and IBS-M [88,91,93,94] but not IBS-C [94] **B.**

4iv A low FODMAP diet reduced dissatisfaction in stool consistency and reduced bowel frequency in IBS-D [88] **C.**

4v A low FODMAP diet reduced dissatisfaction in stool consistency in IBS-C [88] **C.**

4vi A low FODMAP diet had similar efficacy as NICE/BDA dietary advice for overall symptoms in IBS [93]. Symptom improvement was similar for all IBS subtypes for a low FODMAP diet but less for IBS-C than IBS-D or IBS-M for NICE/BDA dietary advice [93] **C.**

4vii A low FODMAP diet had similar efficacy as a probiotic *L. rhamnosus* GG for overall symptoms in IBS, IBS-D and IBS-M [94] **C.**

4viii A high dose of fructans (>19 g day^{-1}) and fructose (>14 g day^{-1}) in IBS with fructose malabsorption [90], and trans-GOS (7 g day^{-1}) in IBS [92], induced abdominal pain, bloating and flatulence **C.**

4vix Trans-GOS at 3.5 g day^{-1} for 4 weeks improved bloating and flatulence but not pain in IBS. At 7.0 g day^{-1}, bloating worsened, whereas flatulence improved [92] **C.**

Practical considerations

A low FODMAP diet with a restriction phase for 3–6 weeks is efficacious in the treatment of IBS when

Table 6 Characteristics and summary of symptom outcomes of included randomised controlled trials relating to dietary fibre

Study (Year) Country	Study design and *N*	Intervention (*n*/*N* completed)	Outcome	Risk of bias
Aller *et al.* (2004) [66] Spain	SB RCT 56 IBS	30.5 g† as high fibre diet (28/28) versus control: 10.4 g† as **low fibre diet** (28/28) 3 months	No significant difference for symptoms between groups. Pain, bowel and bloating improved in both groups ($P < 0.05$)	High
Arffmann *et al.* (1985) [68] Denmark	DB CO RCT 20 IBS-C or IBS-M	30 g† of wheat bran versus placebo: 30 g† coloured **breadcrumbs** (n = 18/20 per group) 6 weeks washout not stated	No significant difference for symptoms between groups Wheat bran significantly increased stool mass ($P < 0.02$) and reduced transit time ($P < 0.01$)	High
Bijkerk *et al.* (2009) [67] Holland	DB RCT 3 arm 275 IBS	4 g† of wheat bran (n = 54/97) versus 4 g† of psyllium (n = 54/85) versus placebo: white rice (n = 56/93) 12 weeks	Symptom severity was significantly less for psyllium than placebo: (−34% versus −18%, P = 0.03). No significant difference for symptoms between wheat bran and placebo	High
Cockerel *et al.* (2012) [76] UK	Open label RCT 40 IBS	Whole linseeds [mean 21 g day^{-1}, up to 7 g of NSP‡] (10/14) versus ground linseeds [mean 18 g day^{-1}, up to 6 g of NSP‡] (12/13) versus control: no supplement to diet 4 weeks (9/13)	No significant differences for overall symptoms between groups. Abdominal pain severity (P = 0.011) and days of pain (P = 0.042) improved for whole linseeds but not ground linseeds or control. Bloating severity improved for ground linseeds (P = 0.028) and control (P = 0.018) but not whole linseeds (P = 0.138)	High
Fowlie *et al.* (1992) [74] UK	Non-RCT 49 IBS-C	4.1 g† of cereal and fruit fibre in 5 tablets (n = 15/25) versus placebo: starch†, $CaPO_4$, **lactose** in 5 tablets (n = 17/24) 3 months	No significant difference for symptoms between groups	High
Hebden *et al.* (2002) [69] UK	DB CO RCT 12 IBS	30 g† of wheat bran versus placebo: 30 g† of **plain biscuits** (12/12 per group) 2 weeks	The wheat bran increased pain and bloating compared to placebo ($P < 0.02$)	High
Kruis *et al.* (1986) [70] Germany	DB RCT 80 IBS	15 g (7.95 g)† of wheat bran versus **placebo**† 16 weeks	No significant difference for symptoms between groups	High
Lucey *et al.* (1987) [71] UK	DB CO RCT 38 IBS	12 (15.6 g)§ of wheat bran biscuits versus placebo: 12 (2.76 g)§ **plain biscuits** (28/38 per group) 16 weeks	No significant difference for symptoms between groups. Global symptoms improved for both groups ($P < 0.01$)	High
Rees *et al.* (2005) [72] UK	SB RCT 28 IBS-C or IBS-M	10–20 g (3.64–7.28 g)‡ of wheat bran (12/14) versus placebo: low fibre **crispbread** (0.22–0.44 g)‡ (10/14) 12 weeks	No significant difference for symptoms between groups. Stool weight increased from 95 g to 123 g for wheat bran and decreased from 135 g to 125 g for placebo ($P < 0.02$)	High
Snook and Shepherd (1994) [73] UK	DBCO RCT 80 IBS	40 g of wheat bran (12 g)§ (71/80) versus placebo: **wheat** and rice flour (negligible)§ (71/80) 7 weeks with 2-week washout	No significant difference for symptoms between groups. Flatulence increased for wheat bran compared to placebo ($P < 0.001$)	Unclear
Tarpila *et al.* (2004) [75] Finland	SB RCT 55 IBS-C	Ground linseeds (≤24 g, 10.6 g)† (26/26) versus psyllium (≤24 g, 13.5 g)† (29/29) 3 months	Ground linseeds improved constipation (P = 0.05, NNT = 2.1) and abdominal symptoms (P = 0.001, NNT 2.4) compared to psyllium	High

CO, cross-over; DB, double-blind; NNT, number need to treat; RCT, randomised controlled trial.
†Undefined dietary fibre measurement.
‡Englyst dietary fibre measurement.
§Southgate dietary fibre measurement.
Bold signifies an inappropriate control/placebo.

delivered by a dietitian with expertise in FODMAP education [88,90,91,96]. Successful adherence and symptom management are achieved by the provision of detailed verbal and written information on avoidance of high FODMAP foods and the inclusion of suitable alternatives to ensure a nutritionally adequate diet [97]. Two studies showed that the low FODMAP diet has similar efficacy as traditional dietary advice based on NICE/BDA [93] or a probiotic *L. rhamnosus* GG [94], without any specific symptom profiling. Thus, where adherence may be compromised, it may be applicable to consider other treatment options.

Restriction of FODMAPs reduces the short-chain oligosaccharide components of dietary fibre by 4 g day^{-1} compared to habitual diet [91]. An increase in low FODMAP high fibre foods may be warranted to meet dietary fibre healthy eating recommendations. From a safety perspective, evidence shows that a low FODMAP diet alters the microbiota [91,98] and reduces calcium intake [91] in the short-term. Therefore, following satisfactory symptom improvement (3–6 weeks) [87,88,91], reintroduction of individual FODMAPs to personal tolerance using dietitian-delivered systematic food challenges is necessary. Recent long-term data indicate that calcium intakes meet nutrient recommendations using the above described dietitan-led delivery [99]. However, there are no long-term microbiota data. FODMAP reintroduction verifies effective treatment and individual tolerance to specific foods at the same time as increasing dietary variety and enabling long-term self-management.

What effect does gluten have on IBS symptoms?

Gluten is the main structural protein complex of wheat, rye and barley and a gluten-free diet is the primary treatment for coeliac disease [100]. There is increasing research suggesting a possible link between the ingestion of gluten and the development of functional bowel symptoms in noncoeliac individuals. However, many of the gluten-responsive individuals in these studies are positive for the human leucocyte antigens (HLA) DQ2 or DQ8, which are present in 98% of coeliac disease and 25% of the normal population [100], and so it is unclear whether a proportion of these individuals may actually be exhibiting sero-negative coeliac disease. Furthermore, a gluten-free diet reduces fructan intake, FODMAPs and potentially toxic components within grains such as wheat amylase trypsin inhibitors, which may be responsible for symptom improvement [101]. Therefore, whether gluten is directly involved in the exacerbation of IBS-type symptoms is unclear.

Included studies and evidence statements

Four level II RCTs with conflicting evidence met the inclusion criteria and are summarised in Table 8 [102–105]. One RCT included only IBS-D patients and compared a gluten containing diet (amount not specified) with a gluten-free diet for 4 weeks [102]. The other studies included all IBS subtypes. One study assessed a gluten-free diet with 100 g of gluten-containing powder (52% gluten) compared to a gluten-free diet with 100 g of gluten-free powder in patients whose symptoms had responded to a gluten-free diet [103]. One feeding study assessed a gluten containing diet (16 g gluten day^{-1}) compared to a gluten-free diet (0 g gluten day^{-1}) [104]. All of these studies showed that symptoms did increase in response to gluten [102–104]. A second cross-over feeding study controlled for FODMAP intake and compared high gluten (16 g gluten day^{-1}) with low gluten (2 g gluten day^{-1}) and a control (0 g gluten day^{-1}) for 1 week with a 2-week washout but showed no gluten-specific response [105]. The evidence is limited with an unclear risk of bias for one RCT [105] and a high risk of bias for the other three [102–104].

5i A gluten-free diet improved some IBS symptoms; specifically, abdominal pain, satisfaction with stool consistency and tiredness at 6 weeks [104] and stool frequency in IBS-D at 4 weeks [102], which was more pronounced in individuals with a positive HLA-DQ2 or HLA-DQ8. **C.**

5ii When dietary confounders (e.g. FODMAPs) were controlled for, there was insufficient evidence that gluten induced IBS symptoms [105]. **C.**

Practical considerations

If individuals wish to follow a gluten-free diet, they should be informed that the current evidence for its use is conflicting. The diet should be assessed for nutritional adequacy in line with healthy eating recommendations. The long-term effects of a gluten-free diet in IBS are unknown. In coeliac disease, a gluten-free diet is used as a life-long treatment and impairs quality of life [106].

Which strain-specific probiotic products improve IBS symptoms?

Probiotics are one of the most investigated treatment options for IBS and have generated great interest amongst individuals with IBS and healthcare professionals. There are many probiotics available as single or multi-strain products in a variety of formulations (e.g. capsules, powders, fermented milks and yoghurts). None have had their health claims approved by European Food & Health Safety Authority (EFSA) [107].

Table 7 Characteristics and summary of symptom outcomes of included studies relating to fermentable carbohydrates

Study (Year) Country	Study design and *N* (*n*/*N* completed)	Intervention	Outcome	Risk of bias
Fermentable carbohydrate reduction				
Berg *et al.* (2013) [87] Norway	RCT 202 IBS 88/101 intervention 94/101 control	2-week run-in: IBS diet Intervention: fructose reduced diet + IBS diet Control: IBS diet 4 weeks	Fructose reduced diet significantly reduced bloating ($P < 0.0005$), abdominal pain ($P < 0.0005$), stool frequency ($P = 0.001$) but not stool consistency (ns) compared to IBS diet alone. No IBS subtype analysis	High
Böhn *et al.* (2015) [93] Sweden	RCT 75 IBS 33/38 intervention 34/37 control	Dietary advice study. Probiotics at same dose allowed. Low lactose allowed Intervention: low FODMAP Control: NICE/BDA (avoid onion, cabbage, beans), good eating behaviour, small meals 4 weeks	Change in IBS-SSS 77 (110) low FODMAP inappropriate 65 (84) NICE/BDA was similar between groups ($P = 0.62$) 19 (50%) responders in low FODMAP 17 responders in NICE diet $P = 0.72$ Low FODMAP diet improved overall symptoms ($P < 0.001$) abdominal pain frequency ($P = 0.008$), severity of distension ($P < 0.001$) Life interference improved ($P < 0.001$). NICE/BDA diet improved overall symptoms ($P < 0.001$) abdominal pain frequency ($P < 0.001$), severity of distension ($P = 0.003$), dissatisfaction with bowel habit ($P = 0.01$). Life interference improved ($P = 0.002$). IBS subtype analysis for reduction in IBS-SSS was similar for low FODMAP diet ($P = 0.76$) but IBS-C did not respond as well as IBS-D or IBS-M for NICE/BDA ($P = 0.03$)	Low
Halmos *et al.* (2014) [88] Australia	SB CO RCT 30 IBS 8 healthy controls	Feeding study: Intervention: Low FODMAP Control: typical Australian diet 3 weeks of each diet with ≥3-week washout	Low FODMAP diet had significant improvements in global symptoms, abdominal pain, bloating and dissatisfaction with stool consistency compared to control diet for IBS (all $P < 0.001$). For IBS subtypes significant improvements in dissatisfaction with stool consistency were observed for IBS-D ($P = 0.038$) and IBS-C ($P = 0.037$) compared to control but sample size was too small to show any difference for IBS-M	Unclear
Pedersen *et al.* (2014) [94] Denmark	RCT 108/123 IBS Low FODMAP 34/42 LGG 37/41 37/40 control (delayed low FODMAP diet)	Dietary advice or probiotics study: Intervention 1: Low FODMAP Intervention 2: probiotics (LGG)† Control: normal Danish diet 6 weeks	Adjusted changes in IBS-SSS for baseline covariates showed statistically significant reduction of IBS-SSS for low FODMAP diet versus control at 6 weeks (75; 95% CI 24–126; $P < 0.01$) but not for LGG versus control (32; 95% CI 18–80 $P = 0.2$). The mean difference in IBS-SSS between low FODMAP and LGG did not reach significance (43.8; 95% CI 8.1–95.8; $P = 0.09$) For IBS subtypes IBS-D improved for all treatment groups, low FODMAP diet ($P < 0.01$), LGG and control ($P = 0.01$) and IBS-M improved for low FODMAP diet ($P = 0.01$) and LGG ($P = 0.04$) but not for control ($P = 0.12$). IBS-C did not improve for any treatment	High

Table 7. Continued

Study (Year) Country	Study design and *N* (*n*/*N* completed)	Intervention	Outcome	Risk of bias
Staudacher *et al.* (2012) [91] UK	RCT 41 IBS with diarrhoea and/or bloating 16/19 intervention 19/22 control ITT analysis	Dietary advice study: Intervention: low FODMAP Control: habitual diet 4 weeks	ITT: Low FODMAP diet had significant improvements in global symptoms ($P = 0.006$), bloating ($P = 0.007$), borborygmi ($P = 0.04$), urgency ($P = 0.047$) compared to control diet but not PP for stool consistency ($P = 0.56$) or incidence ($P = 0.24$) or severity ($P = 0.34$) of diarrhoea	High
Fermentable carbohydrate challenge or supplementation				
Olesen and Gudmand-Hoyer (2000) [89] Denmark	DB RCT 96 IBS 38/50 intervention 37/46 control	2-week run-in with placebo Intervention: 10 g day^{-1} fructo-oligosaccharide (FOS) 2 weeks then 20 g day^{-1} FOS 10 weeks Control: 10 g day^{-1} placebo (glucose syrup) 2 weeks then 20 g day^{-1} placebo 10 weeks	No significant difference found	High
Shepherd *et al.* (2008) [90] Australia	DB CO RCT 25/26 IBS	Feeding study of low FODMAP diet with interventions: 14–50 g of fructose; 7–19 g of fructans; 14–50 g of fructose + 7–19 g of fructans Control: 20 g of glucose 9 days increasing dose with at least 10 days washout in between	Overall symptoms ($P < 0.02$) and bloating ($P \leq 0.0046$) were significantly increased with fructose, fructans or fructose-fructan mix versus control	Low
Silk *et al.* (2009) [92] UK	SB RCT 3 arms 60 IBS	Low dose: 3.5 g day^{-1} 14 days placebo (maltodextrin) + 3.5 g day^{-1} *trans*-GOS‡ 28 days High dose: 7 g day^{-1} placebo 14 days + 7 g day^{-1} trans-GOS 28 days Control: 7 g day^{-1} placebo 14 days + 28 days	PP 44/60 subjective global assessment improved more in low dose trans-GOS versus placebo ($P < 0.05$) but not for high dose versus placebo	High

BDA, British Dietetic Association; CO, cross-over; DB, double-blind; FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides and polyols; IBS, irritable bowel syndrome; IBS-C, IBS – constipation-predominant; IBS-D, IBS – diarrhoea-predominant; IBS-M, IBS involving both diarrhoea and constipation; IBS-SSS, IBS-Symptom Severity Score; ITT, intention to treat; LGG, *Lactobacillus rhamnosus* GG; NICE, National Institute for Health and Care Excellence; PP, per protocol; RCT, randomised controlled trial.

†*Lactobacillus rhamnosus* GG (1.2×10^{10} CFU in 2 capsules day^{-1}).

‡*Trans*-galacto-oligosaccharide powder, made from *Bifidobacterium bifidum* NCIMD 41171 containing 22% lactose made up with water as a banana or chocolate flavoured drink.

The previous guidelines only reviewed UK available probiotics [7]; however, the current guidelines included all internationally available probiotics and so all previous evidence statements and recommendations have been reviewed.

Included studies and evidence statements

Thirty-five RCTs met the inclusion criteria for this review [108–142] from nine included systematic reviews [2,143–150]. In brief, 29 different dose-specific probiotic formulations in 35 RCTs were identified and a detailed evaluation along with 19 evidence statements for probiotics are provided elsewhere [15].

Practical considerations

There are many probiotic products available in different doses and variable bacterial strains. Individuals with IBS who choose to try probiotics should be aware that some products contain other ingredients that may increase IBS symptoms (e.g. dietary fibre: oats; FODMAPs: FOS, inulin, lactose, fructose, sorbitol and xylitol). If an individual finds 4 weeks of use of a probiotic beneficial, they can continue to take it but the long-term effects are not known.

Elimination diets and food hypersensitivity to improve IBS symptoms

Food hypersensitivity is an umbrella term used to describe an adverse reaction to food and is defined as food allergy if immune-mediated or food intolerance if not [151]. Food intolerance includes pharmacological effects of food or food components, noncoeliac gluten sensitivity and enzyme/transport defects [152]. Allergic-type responses to food have been implicated in a number of studies linking mast cell mediators with altered colonic nerve activity in IBS [153–155]. However, it remains unclear what mechanism is driving this immune activation (e.g. food allergy, autoimmune disease, bacterial or viral infection) and there is no conclusive evidence that the immune system is directly implicated in IBS [154,156].

Included studies and evidence statement

No new studies met the inclusion criteria and six studies were previously evaluated [157–162]. The previous guidelines provided third-line advice on an elimination or empirical diet for individuals who had failed second-line dietary advice [7]. However, this third-line advice was based on low-quality evidence with limited effectiveness on symptom management and was not based on well-defined mechanisms. With the emergence of the low FODMAP diet, strict elimination diets that are nonspecific are no longer used in IBS symptom management and the mechanisms underlying their role have still not been clarified. Therefore, the previous evidence statements and recommendations are no longer applicable and third-line advice has been removed.

Practical considerations

The only focused elimination-type diet that is appropriate for IBS is the low FODMAP diet (see section on fermentable carbohydrates above). Other nonspecific elimination diets do not have mechanistic evidence or clinical evidence to defend their continued use.

During medical and family history taking of individuals with IBS, it is good practice to ask about food allergy and intolerance (e.g. vasoactive amines, sulphites), although this is not related to an IBS diagnosis. Dietary advice for food allergy is not considered in these guidelines and clinical guidance should be sought elsewhere [163,164].

Discussion

The present systematic review and guidelines provide the first fully comprehensive evidence-based guidelines for the dietary management of IBS in adults using a robust methodology. The highest-quality evidence was for the low FODMAP diet for IBS and the subtypes IBS-D and IBS-M. The evidence has rapidly accumulated over recent years and is starting to become internationally recognised [165]. A new IBS algorithm has been developed to comprise first- and second-line interventions. The low FODMAP diet is considered as the only second-line intervention and the evidence to date only supports dietitian-led delivery. Evidence is lacking to support second-line dietary interventions in the management of IBS-C.

National guidelines from the UK and Japan recommend the low FODMAP diet in IBS management and weakly recommend a high fibre diet and avoiding high-fat and spicy food [2,11]. Some guidelines have not yet incorporated the low FODMAP approach [166]. Overall, there is increased awareness for more detailed dietary guidelines to be implemented into standard IBS clinical practice.

The previous guidelines did not systematically review the first-line advice in relation to healthy eating and lifestyle (alcohol, caffeine, spicy food, fat, fluid and dietary habits). The evidence identified was a high risk of bias with a lack of high quality and high quantity studies. Indirect cross-sectional evidence for modifying dietary habits to improve IBS symptoms was treated cautiously and there was insufficient evidence to make a recommendation. It is sensible to provide guidance on good eating lifestyle and a regular meal pattern considering that 60%

Table 8 Characteristics and summary of symptom outcomes of included studies relating to gluten

Study (Year) Country	Study design and N (n/N completed)	Intervention	Outcome	Risk of bias
Biesiekierski *et al.* (2011) [104] Australia	DB RCT 39 IBS with NCGS 19/20 gluten 15/19 placebo	Gluten-free diet with Intervention: 16 g day^{-1} gluten in prepared foods Placebo: 0 g day^{-1} gluten in prepared foods Up to 6 weeks	At 1 week compared to baseline of a gluten containing diet overall symptoms ($P = 0.047$), abdominal pain ($P = 0.016$), bloating ($P = 0.031$), dissatisfaction with stool consistency ($P = 0.024$), tiredness ($P = 0.0001$), significantly increased, but not for flatulence ($P = 0.053$) and nausea ($P = 0.120$). At 6 weeks the VAS score change in overall symptoms, flatulence and nausea were no different compared with placebo. Gluten-containing diet increased abdominal pain ($P = 0.02$), dissatisfaction with stool consistency ($P = 0.03$) and tiredness ($P = 0.001$). Diet not controlled for confounders e.g. FODMAPs	High
Biesiekierski *et al.* (2013) [105] Australia	DB CO RCT and rechallenge 3 arms 40 IBS with NCGS 37/40 Intervention 22/22 Rechallenge	Feeding study Run-in: 2-week gluten-free and low FODMAP Intervention: High gluten (16 g day^{-1}) versus low gluten (2 g day^{-1} + 14 g of whey) versus control (16 g of whey) for 1 week with at least 2 weeks washout Rechallenge: Gluten (16 g day^{-1}) versus whey (16 g day^{-1}) or control (no additional protein) for 3 days	Overall symptom improvement for run-in ($P = 0.001$) and symptom deterioration for intervention in all groups ($P = 0.001$). No change in symptoms for rechallenge compared to baseline. No evidence for gluten specific or gluten dose effects in IBS patients with NCGS following a low FODMAP diet	Unclear
Shahbazkhani *et al.* (2015) [103] Iran	DB RCT 72/148 IBS gluten-free diet 35/72 gluten 37/72 placebo	Patients with symptom response to gluten-free diet were randomised to gluten-free diet with: 100 g of powder 52% gluten or 100 g of gluten-free powder 6 weeks challenge	Per protocol: overall symptoms following challenge 74.3% gluten versus 16.2% placebo ($P < 0.001$). Diet not controlled for confounders e.g. FODMAPs	High
Vazquez-Roque *et al.* (2013) [102] USA	DB RCT 45 IBS-D: 22/22 gluten-containing diet 22/23 gluten-free diet	Feeding study Intervention: gluten containing diet versus gluten-free diet 4 weeks	The gluten containing diet was associated with increased stool frequency compared to the gluten-free diet ($P = 0.04$), which was greater in HLA-DQ2 or HLA-DQ8 positive patients. Diet not controlled for confounders e.g. FODMAPs	High

CO, cross-over; DB, double-blind; RCT, randomised controlled trial.

of IBS patients report symptom worsening after meals, 20% within 15 min of eating and 93% within three hours [36]. Visceral hypersensitivity, as seen in duodenal infusions with fat, and other mechanisms may explain these postprandial responses [97]. Elimination diet studies target specific foods (e.g. dairy, wheat, onion, rye and potato) that may be associated with fried and fatty foods rather than directly with fat [157–160]. However, there was no direct prospective evidence that reducing fat intake improved IBS symptoms. In clinical practice, reducing fat intake in accordance with healthy guidelines is worth exploring and it may be warranted to assess for bile acid diarrhoea because IBS is sometimes mistaken for bile salt malabsorption [167,168].

Cows' milk is sometimes blamed for symptom generation in IBS and it is assumed that lactose is the culprit; however, cows' milk protein may be implicated. Cows' milk supply typically contains A1- and A2-β-casein in a 1 : 1 ratio; however, during digestion of A1-β-casein, β-casomorphin 7 is released, which may lead to

gastrointestinal symptoms, although this has not been observed with A2-β-casein. Some preliminary research in subjects with self-reported milk intolerance has shown that these symptoms may be avoided with milk containing only A2-β-casein [169,170]; further research is warranted to determine whether these findings can be repeated in patients with IBS.

Food is a complex matrix of variable components and, depending on its solubility and fermentability, the different types of dietary fibre have a variety of actions on the gut and its environment [62]. These guidelines only included one new RCT of dietary fibre modification [76]. Bias related to dietary factors was considered carefully in respect to the study design of the dietary fibre RCTs. Inappropriate placebos were used in many of the trials, providing a lack of confidence in any effects and the potential to explain conflicting findings. Appropriate placebo with no known gut-related alterations and dietary intake should be considered during future study designs.

To date, most low FODMAP diet research has compared it to a normal diet, whether described as an habitual UK [91] or Danish [94] diet or a typical Australian diet [88]. The effectiveness of a low FODMAP diet compared to or combined with other therapies such as other dietary treatments, medication, probiotics or prebiotics is limited [93,94,171]. Two of these RCTs were assessed in these guidelines and demonstrated that the low FODMAP diet has similar efficacy as a probiotic *L. rhamnosus* GG [94] or NICE/BDA dietary advice [93] for overall symptoms in IBS. A possible explanation for the similarity for symptom outcomes between the low FODMAP diet and the NICE/BDA diet in the study by Böhn *et al.* [93] is that the FODMAP intake for the NICE/BDA arm was similar to that of the low FODMAP arm in the study by Staudacher *et al.* [91]. This provides support for further work aiming to investigate how low FODMAP intakes should be to achieve IBS symptom control. For IBS subtypes, Böhn *et al.* [93] indicated no differences in symptom response for the low FODMAP diet but IBS-C responded less well for NICE/BDA dietary advice and Pedersen *et al.* [94] demonstrated symptom improvement for IBS-D and IBS-M for both the low FODMAP diet and the probiotic. A placebo-controlled RCT is considered the gold standard study design in dietary trials [172]. The third RCT is currently in abstract form only and was not included in these guidelines [171]. The study compared the low FODMAP diet with a placebo (sham) diet, which was designed to be equivalent in nutrients and FODMAP content as a habitual diet, and showed that the low FODMAP diet is superior to a placebo diet. The study was designed as a 2×2 factorial trial to assess the combined effect of the low FODMAP diet and a multi-strain probiotic not only on symptoms, but also on the microbiota; the results of the latter are awaited. For symptoms, the effectiveness of the probiotic was equivocal [171]. Thus, although research on the low FODMAP diet is expanding, there is limited evidence for IBS subtypes. To increase the strength and applicability of the evidence, it is appropriate to develop international research collaborations for dietary trials, especially for the low FODMAP diet, in IBS. This will facilitate predictors of response for IBS-subtype targeted dietary therapy to be developed.

There is growing interest in gluten sensitivity in the general population following the popularity of a gluten-free diet promoted in the media. Thus, fairly or unfairly, gluten is sometimes blamed as a cause of IBS symptoms [101,173,174]. The findings presented here show that evidence for using a gluten-free diet in IBS is controversial and FODMAPs may be confounders. Wheat, barley and rye contain short-chain carbohydrates, particularly fructans, as well as proteins, gluten and amylase trypsin inhibitors, all of which may be involved in gastrointestinal symptom generation, although conclusive evidence is lacking [101]. Dietary exclusion of these cereals reduces the FODMAP content of the diet by 50% and may provide adequate symptom relief. Individual food challenges are appropriate to confirm whether these foods induce symptoms that will be useful for long-term self-management and nutritional adequacy. HLA-DQ2 and HLA-DQ8 tests may be warranted and negative results will rule out coeliac disease where individuals are not prepared to reintroduce wheat or rye [175]; however, positive HLA-DQ2 and -DQ8 tests will not confirm or refute coeliac disease [101,175].

Recommendations for specific probiotic products were not possible because of a lack of consistency in outcome benefits across studies on the same probiotic or only one study assessing one probiotic. Probiotics are considered as safe for use in IBS and it is likely that they will continue to be purchased by the public. Healthcare professionals can use these guidelines to inform individuals with IBS about the limited benefits of probiotics and the potential for a placebo response, which is known to be high in IBS clinical trials [176].

The previous guidelines had recommendations for food hypersensitivity and elimination diets described as third-line advice. This section has been removed because of the limited evidence without mechanistic understanding of nonspecific restrictive diets for IBS, leading to concern over the potential for nutritional inadequacy and increased symptom generation on food reintroduction.

For clinical practice, the IBS dietary algorithm has been simplified to include only first- and second-line advice (Fig. 1). The first-line advice comprises 'healthy eating and lifestyle', which could be provided by any healthcare

professional with an interest in dietary intake and management in IBS, ideally in primary care/family practice but also in secondary care/hospital where applicable [2,177]. Patient literature should be developed for first-line advice with a strong self-management approach for use by any healthcare professional interested in IBS dietary management. The second-line advice comprises 'the low FODMAP diet', which is supported only by dietitian-led counselling in addition to supportive patient literature to enhance education and understanding as they see fit [85,97,178]. Increasing awareness of the low FODMAP diet means that it may be delivered in a manner deviating from evidence-based practice and bypassing dietitian-led counselling whereby patients are given only basic information or directed to the Internet. This nondietitian-led delivery option for such a complex dietary intervention is potentially unsafe and ineffective and therefore requires investigation.

A low FODMAP diet has recently been included as a treatment option in NICE guidelines for IBS [2]. This may have implications for clinical practice because it states it should be provided by a healthcare professional with expertise in dietary management. However, without an increase in the dietetic workforce, increased dietetic referral rates warrant alternative educational options to be considered. Indeed, dietitian-led low FODMAP education groups comprise effective alternative and clinical pathways that offer a choice of groups and one-to-one appointments should also be considered [178]. Practical aspects to enhance the delivery of an effective low FODMAP service have been considered elsewhere [86,97,178].

Research recommendations

The scientific community is only just starting to unravel how diet affects gastrointestinal functions, in particular in relation to functional bowel symptoms and the gastrointestinal microbiota [179]. It appears that now is the time to re-evaluate diet as a potential confounder in IBS RCTs on medication and diet when, previously, this was rarely taken into consideration. There are few RCTs in IBS that have measured dietary intake at baseline and intervention completion and many placebos may have contained confounding ingredients. Objective markers to predict IBS symptom response in dietary trials of IBS are lacking. It was agreed that four topics are priority research areas.

Dietary fibre

Well-designed and adequately powered RCTs to assess the effects of dietary fibre intakes of at least 30 g day^{-1} in the treatment of IBS-C for at least 3 months with long-term follow-up are warranted. It is unknown whether the dietary fibre recommendations for individuals with IBS should differ from those of the general population or whether they can increase their daily dietary fibre intake to meet healthy eating recommendations.

FODMAPs

Well-designed and adequately powered RCTs on FODMAPs should focus on IBS symptom profiles, long-term follow-up with reference to symptom control, safety, microbiota and nutritional adequacy, optimal education delivery, and health economics in specific IBS-subtypes and clinical settings. High-quality RCTs should consider factors such as dietary components that affect gut motility and gut microbiota as described elsewhere [15].

The effectiveness of a low FODMAP diet compared to or combined with other therapies, such as other dietary treatments, medication, probiotics or prebiotics, is limited and needs further evaluation. Determining predictors of response for the low FODMAP diet is of paramount importance.

Dietitian-led delivery of low FODMAP education incorporates a restrictive low FODMAP diet for 4–6 weeks followed by FODMAP reintroduction using a systematic food-challenge process to determine personal tolerance thresholds to individual foods and enabling long-term self-management. The success of alternative delivery options to dietitian-led low FODMAP dietary counselling remains unknown; however, with a heightened interest in the diet, it is imperative that such alternative options are prioritised as a research need to determine their safety in terms of any effects on the gastrointestinal microbiota and nutritional adequacy, as well as their acceptability, and clinical and cost effectiveness.

Gluten

Well-designed and adequately powered RCTs are needed to clearly differentiate between gluten sensitivity and FODMAPs and to establish a clear definition of the difference in symptom profile between the two conditions. Dietary intake should be measured.

Probiotics

Well-designed and adequately powered RCTs of probiotics should consider factors such as dietary components that affect gut motility and gut microbiota and measure dietary intake.

Conclusions

In summary, these updated BDA evidence-based clinical practice recommendations provide first-line dietary and lifestyle assessment related to healthy eating and intakes

of alcohol, fat, fibre, milk, spicy foods and use of probiotics. Second-line advice should be considered where IBS symptoms persist, and recommendations include dietitian-led delivery of a low FODMAP diet. There was a lack of evidence to recommend a gluten-free diet or any non-specific food hypersensitivity interventions for treating IBS.

Acknowledgments

The authors thank Professor Kevin Whelan for advice.

Conflict of interests, source of funding and authorship

Twelve authors (YAM, RKB, HL, PG, JH, NAOS, CP, LBR, LS, MW, JT, MCEL) are practising dietitians with a professional interest in these guidelines; 10 with specialist IBS clinical experience in primary and secondary healthcare settings. These guidelines contributed to MSc dietetic projects for two of the authors (JH, NAOS). All members of the IBS-DGRG signed conflicts of interest forms during the development of these guidelines. Travel and subsistence expenses for meetings were funded by the Gastroenterology Specialist Group of The British Dietetic Association. All authors contributed to the development of the evidence statements, recommendations, practical considerations and preparation of the manuscript and tables, and agreed to the final document being submitted for publication. YAM and MCEL wrote the final manuscript, which was critically reviewed and approved by all authors prior to submission.

References

1. Lovell RM & Ford AC (2012) Global prevalence of and risk factors for irritable bowel syndrome: a meta-analysis. *Clin Gastroenterol Hepatol* **10**, e4.
2. National Institute for Health and Clinical Excellence (2015) Irritable bowel syndrome in adults. Diagnosis and management of irritable bowel syndrome in primary care. Clinical Guideline 61 Update 2015. Available at: http://www.nice.org.uk/Guidance/CG61 (accessed 6 January 2016).
3. Longstreth GF, Thompson WG, Chey WD *et al.* (2006) Functional bowel disorders. *Gastroenterology* **130**, 1480–1491.
4. Hayes PA, Fraher MH & Quigley EM (2014) Irritable bowel syndrome: the role of food in pathogenesis and management. *Gastroenterol Hepatol* **10**, 164–174.
5. Lacy BE, Weiser K & De Lee R (2009) The treatment of irritable bowel syndrome. *Ther Adv Gastroenterol* **2**, 221–238.
6. Monsbakken K, Vandvik P & Farup P (2006) Perceived food intolerance in subjects with irritable bowel syndrome – etiology, prevalence and consequences. *Eur J Clin Nutr* **60**, 667–672.
7. McKenzie YA, Alder A, Anderson W *et al.* (2012) British Dietetic Association evidence-based guidelines for the dietary management of irritable bowel syndrome in adults. *J Hum Nutr Diet* **25**, 260–274.
8. Marsh A, Eslick EM & Eslick GD (2015) 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.
9. Rao SS, Yu S & Fedewa A (2015) Systematic review: dietary fibre and FODMAP-restricted diet in the management of constipation and irritable bowel syndrome. *Aliment Pharmacol Ther* **41**, 1256–1270.
10. Fedewa A & Rao SS (2014) Dietary fructose intolerance, fructan intolerance and FODMAPs. *Curr Gastroenterol Rep* **16**, 370.
11. Fukudo S, Kaneko H, Akiho H *et al.* (2015) Evidence-based clinical practice guidelines for irritable bowel syndrome. *J Gastroenterol* **50**, 11–30.
12. Mansueto P, Seidita A, D'Alcamo A *et al.* (2014) Non-celiac gluten sensitivity: literature review. *J Am Coll Nutr* **33**, 39–54.
13. Molina-Infante J, Santolaria S, Sanders DS *et al.* (2015) Systematic review: noncoeliac gluten sensitivity. *Aliment Pharmacol Ther* **41**, 807–820.
14. Practice-based Evidence in Nutrition (PEN) Global (2015) PEN Global evidence grades. Available at: https://www.pennutrition.com/aboutpen.aspx (accessed 12 December 2015).
15. McKenzie Y, Thompson J, Gulia P *et al.* (2016) British Dietetic Association systematic review of systematic reviews and evidence-based practice guidelines for the use of probiotics in the management of irritable bowel syndrome in adults (2016 update). *J Hum Nutr Diet* doi: 10.1111/jhn.12386
16. Evidence Partners (2015) McMaster Clarity Group Risk of Bias Instruments. Available at: https://distillercer.com/resources/methodological-resources/ (accessed 12 December 2015).
17. Lundh A & Gøtzsche PC (2008) Recommendations by Cochrane Review Groups for assessment of the risk of bias in studies. *BMC Med Res Methodol* **8**, 22.
18. Spiegel B, Camilleri M, Bolus R *et al.* (2009) Psychometric evaluation of patient-reported outcomes in irritable bowel syndrome randomized controlled trials: a Rome Foundation report. *Gastroenterology* **137**, 1944–1953 e1–e3.
19. Australia Government: National Health and Medical Research Council (2009) NHMRC additional levels of

evidence and grades for recommendations for developers of guidelines. Available at: http://www.nhmrc.gov.au/guidelines-publications/cp65 (accessed 6 January 2016).
20. El-Salhy M, Ostgaard H, Gundersen D *et al.* (2012) The role of diet in the pathogenesis and management of irritable bowel syndrome (review). *Int J Mol Med* **29**, 723–731.
21. Bagyanszki M, Krecsmarik M, De Winter BY *et al.* (2010) Chronic alcohol consumption affects gastrointestinal motility and reduces the proportion of neuronal NOS-immunoreactive myenteric neurons in the murine jejunum. *Anat Rec* **293**, 1536–1542.
22. Nazer H & Wright RA (1983) The effect of alcohol on the human alimentary tract: a review. *J Clin Gastroenterol* **5**, 361–365.
23. Evatt DP & Griffiths RR (2013) Caffeine. In *Encyclopedia of Human Nutrition* [Caballero B, editor], pp. 221–227, 3rd edn. Waltham, MA: Academic Press.
24. Rao SSCA, Welcher KA, Zimmerman BB *et al.* (1998) Is coffee a colonic stimulant? *Eur J Gastroenterol Hepatol* **10**, 113–118.
25. Boekema PS, Samsom M, van Berge Henegouwen GP *et al.* (1999) Coffee and gastrointestinal function: facts and fiction: a review. *Scand J Gastroenterol* **34**(Suppl), 35–39.
26. Tomlin J & Joshi A (1992) Effect of chilli powder on gut transit. *Gut* **33**, 518.
27. Bhat YM & Bielefeldt K (2006) Capsaicin receptor (TRPV1) and non-erosive reflux disease. *Eur J Gastroenterol Hepatol* **18**, 263–270.
28. Akbar A, Yiangou Y, Facer P *et al.* (2008) Increased capsaicin receptor TRPV1-expressing sensory fibres in irritable bowel syndrome and their correlation with abdominal pain. *Gut* **57**, 923–929.
29. Chan C, Facer P, Davis J *et al.* (2003) Sensory fibres expressing capsaicin receptor TRPV1 in patients with rectal hypersensitivity and faecal urgency. *Lancet* **361**, 385–391.
30. Caldarella MP, Milano A, Laterza F *et al.* (2005) Visceral sensitivity and symptoms in patients with constipation- or diarrhea-predominant irritable bowel syndrome (IBS): effect of a low-fat intraduodenal infusion. *Am J Gastroenterol* **100**, 383–389.
31. Simren M, Abrahamsson H & Bjornsson ES (2001) An exaggerated sensory component of the gastrocolonic response in patients with irritable bowel syndrome. *Gut* **48**, 20–27.
32. Hammer HF, Phillips SF, Camilleri M *et al.* (1998) Rectal tone, distensibility, and perception: reproducibility and response to different distensions. *Am J Physiol* **274**, G584–G590.
33. Faresjö Å, Johansson S, Faresjö T *et al.* (2010) Sex differences in dietary coping with gastrointestinal symptoms. *Eur J Gastroenterol Hepatol* **22**, 327–333.
34. Hayes P, Corish C, O'Mahony E *et al.* (2014) A dietary survey of patients with irritable bowel syndrome. *J Hum Nutr Diet* **27**(Suppl 2), 36–47.
35. Reding KW, Cain KC, Jarrett ME *et al.* (2013) Relationship between patterns of alcohol consumption and gastrointestinal symptoms among patients with irritable bowel syndrome. *Am J Gastroenterol* **108**, 270–276.
36. Simrén M, Månsson A, Langkilde AM *et al.* (2001) Food-related gastrointestinal symptoms in the irritable bowel syndrome. *Digestion* **63**, 108–115.
37. Böhn L, Störsrud S, Törnblom H *et al.* (2013) Self-reported food-related gastrointestinal symptoms in IBS are common and associated with more severe symptoms and reduced quality of life. *Am J Gastroenterol* **108**, 634–641.
38. EFSA (2015) Scientific opinion on the safety of caffeine. Available at: http://www.efsa.europa.eu/sites/default/files/scientific_output/files/main_documents/4102.pdf (accessed 01/05/16).
39. Food ESCo (2014) High caffeine energy drinks and other foods containing caffeine. Available at: https://www.food.gov.uk/science/additives/energydrinks (accessed 8 April 2015).
40. Bortolotti M & Porta S (2011) Effect of red pepper on symptoms of irritable bowel syndrome: preliminary study. *Dig Dis Sci* **56**, 3288–3295.
41. Gonlachanvit S, Mahayosnond A & Kullavanijaya P (2009) Effects of chili on postprandial gastrointestinal symptoms in diarrhoea predominant irritable bowel syndrome: evidence for capsaicin-sensitive visceral nociception hypersensitivity. *Neurogastroenterol Motil* **21**, 23–32.
42. Agarwal MK, Bhatia SJ, Desai SA *et al.* (2002) Effect of red chillies on small bowel and colonic transit and rectal sensitivity in men with irritable bowel syndrome. *Indian J Gastroenterol* **21**, 179–182.
43. Serra J, Salvioli B, Azpiroz F *et al.* (2002) Lipid-induced intestinal gas retention in irritable bowel syndrome. *Gastroenterology* **123**, 700–706.
44. Simren M, Abrahamsson H & Bjornsson ES (2007) Lipid-induced colonic hypersensitivity in the irritable bowel syndrome: the role of bowel habit, sex, and psychologic factors. *Clin Gastroenterol Hepatol* **5**, 201–208.
45. Lindberg G, Hamid SS, Malfertheiner P *et al.* (2011) World Gastroenterology Organisation global guideline: constipation – a global perspective. *J Clin Gastroenterol* **45**, 483–487.
46. Kang SH, Choi SW, Lee SJ *et al.* (2011) The effects of lifestyle modification on symptoms and quality of life in patients with irritable bowel syndrome: a prospective observational study. *Gut Liv* **5**, 472–477.
47. Guo YB, Zhuang KM, Kuang L *et al.* (2015) Association between diet and lifestyle habits and irritable bowel syndrome: a case–control study. *Gut Liv* **9**, 649–656.
48. Khademolhosseini F, Mehrabani D, Nejabat M *et al.* (2011) Irritable bowel syndrome in adults over 35 years

in Shiraz, southern Iran: prevalence and associated factors. *J Res Med Sci* **16**, 200–206.
49. Miwa H (2012) Life style in persons with functional gastrointestinal disorders – large-scale internet survey of lifestyle in Japan. *Neurogastroenterol Motil* **24**, 464–471.
50. Böhn L, Störsrud S & Simrén M (2013) Nutrient intake in patients with irritable bowel syndrome compared with the general population. *Neurogastroenterol Motil* **25**, 23 e1.
51. 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 Rep* **5**, 1382–1390.
52. Böhmer CJM & Tuynman HARE (2001) The effect of a lactose-restricted diet in patients with a positive lactose tolerance test, earlier diagnosed as irritable bowel syndrome: a 5-year follow-up study. *Eur J Gastroenterol Hepatol* **13**, 941–944.
53. Bozzani A, Penagini R, Velio P *et al.* (1986) Lactose malabsorption and intolerance in Italians. Clinical implications. *Dig Dis Sci* **31**, 1313–1316.
54. Parker T, Woolner J, Prevost A *et al.* (2001) Irritable bowel syndrome: is the search for lactose intolerance justified? *Eur J Gastroenterol Hepatol* **13**, 219–225.
55. Böhmer CJ & Tuynman HA (1996) The clinical relevance of lactose malabsorption in irritable bowel syndrome. *Eur J Gastroenterol Hepatol* **8**, 1013–1016.
56. Vernia P, Ricciardi MR, Frandina C *et al.* (1995) Lactose-malabsorption and irritable-bowel-syndrome – effect of a long-term lactose-free diet. *Ital J Gastroenterol* **27**, 117–121.
57. David TJ (1984) Anaphylactic shock during elimination diets for severe atopic eczema. *Arch Dis Child* **59**, 983–986.
58. Flinterman AE, Knulst AC, Meijer Y *et al.* (2006) Acute allergic reactions in children with AEDS after prolonged cow's milk elimination diets. *Allergy* **61**, 370–374.
59. McCleary BV, DeVries JW, Rader JI *et al.* (2010) Determination of total dietary fiber (CODEX definition) by enzymatic-gravimetric method and liquid chromatography: collaborative study. *J AOAC Int* **93**, 221–233.
60. McCleary BV, DeVries JW, Rader JI *et al.* (2012) Determination of insoluble, soluble, and total dietary fiber (CODEX definition) by enzymatic-gravimetric method and liquid chromatography: collaborative study. *J AOAC Int* **95**, 824–844.
61. Scientific Advisory Committee on Nutrition (2015) Carbohydrate and health. Available at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/339771/Draft_SACN_Carbohydrates_and_Health_report_consultation.pdf (accessed 8 April 2015).
62. Eswaran S, Muir J & Chey WD (2013) Fiber and functional gastrointestinal disorders. *Am J Gastroenterol* **108**, 718–727.
63. Australia GSo (2006) Clinical update: irritable bowel syndrome. Available at: http://www.gesa.org.au/professional.asp?cid=9&id=125 (accessed 8 April 2015).
64. Nordic Co-operation (2014) Nordic nutrition recommendations. Available at: https://www.norden.org/en/theme/tidligere-temaer/themes-2014/nordic-nutrition-recommendation/nordic-nutrition-recommendations-2012 (accessed 8 April 2015).
65. Ruepert L, Quartero AO, de Wit NJ *et al.* (2011) Bulking agents, antispasmodics and antidepressants for the treatment of irritable bowel syndrome. *Cochrane Database Syst Rev* **8**, CD003460.
66. Aller R, de Luis DA, Izaola O *et al.* (2004) Effects of a high-fiber diet on symptoms of irritable bowel syndrome: a randomized clinical trial. *Nutrition* **20**, 735–737.
67. Bijkerk CJ, de Wit NJ, Muris JW *et al.* (2009) Soluble or insoluble fibre in irritable bowel syndrome in primary care? Randomised placebo controlled trial. *BMJ* **339**, b3154.
68. Arffmann S, Andersen JR, Hegnhoj J *et al.* (1985) The effect of coarse wheat bran in the irritable bowel syndrome. A double-blind cross-over study. *Scand J Gastroenterol* **20**, 295–298.
69. Hebden JM, Blackshaw E, D'Amato M *et al.* (2002) Abnormalities of GI transit in bloated irritable bowel syndrome: effect of bran on transit and symptoms. *Am J Gastroenterol* **97**, 2315–2320.
70. Kruis W, Weinzierl M, Schussler P *et al.* (1986) Comparison of the therapeutic effect of wheat bran, mebeverine and placebo in patients with the irritable bowel syndrome. *Digestion* **34**, 196–201.
71. Lucey MR, Clark ML, Lowndes J *et al.* (1987) Is bran efficacious in irritable bowel syndrome? A double blind placebo controlled crossover study. *Gut* **28**, 221–225.
72. Rees G, Davies J, Thompson R *et al.* (2005) Randomised-controlled trial of a fibre supplement on the symptoms of irritable bowel syndrome. *J R Soc Promot Health* **125**, 30–34.
73. Snook J & Shepherd HA (1994) Bran supplementation in the treatment of irritable bowel syndrome. *Aliment Pharmacol Ther* **8**, 511–514.
74. Fowlie S, Eastwood MA & Prescott R (1992) Irritable bowel syndrome: assessment of psychological disturbance and its influence on the response to fibre supplementation. *J Psychosom Res* **36**, 175–180.
75. Tarpila S, Tarpila A, Grohn P *et al.* (2004) Efficacy of ground flaxseed on constipation in patients with irritable bowel syndrome. *Curr Topics Nutraceutical Res* **2**, 119–125.
76. Cockerell KM, Watkins AS, Reeves LB *et al.* (2012) Effects of linseeds on the symptoms of irritable bowel syndrome: a pilot randomised controlled trial. *J Hum Nutr Diet* **25**, 435–443.

77. Public Health England, Food Standards Agency (2014) The National Diet and Nutrition Survey Data Assesses the Diet, Nutrient Intake and Nutritional Status of the General Population in the UK. Available at: https://www.gov.uk/government/statistics/national-diet-and-nutrition-survey-results-from-years-1-to-4-combined-of-the-rolling-programme-for-2008-and-2009-to-2011-and-2012 (accessed 22 December 2015).
78. Blumenthal M (1998) *The Complete German Commission E Monographs; Herapeutic Guide to Herbal Medicine*. Boston, MA: Integrative Medicine Communications.
79. Strate LL, Liu YL, Syngal S *et al.* (2008) Nut, corn, and popcorn consumption and the incidence of diverticular disease. *JAMA* **300**, 907–914.
80. Tarleton S & DiBaise JK (2011) Low-residue diet in diverticular disease: putting an end to a myth. *Nutr Clin Pract* **26**, 137–142.
81. 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.
82. Barrett JS, Gearry RB, Muir JG *et al.* (2010) Dietary poorly absorbed, short-chain carbohydrates increase delivery of water and fermentable substrates to the proximal colon. *Aliment Pharmacol Ther* **31**, 874–882.
83. Ong DK, Mitchell SB, Barrett JS *et al.* (2010) Manipulation of dietary short chain carbohydrates alters the pattern of gas production and genesis of symptoms in irritable bowel syndrome. *J Gastroenterol Hepatol* **25**, 1366–1373.
84. Roberfroid M, Gibson GR, Hoyles L *et al.* (2010) Prebiotic effects: metabolic and health benefits. *Br J Nutr* **104**(Suppl 2), S1–S63.
85. Staudacher HM, Irving PM, Lomer MC *et al.* (2014) Mechanisms and efficacy of dietary FODMAP restriction in IBS. *Nat Rev Gastroenterol Hepatol* **11**, 256–266.
86. Shepherd SJ, Lomer MC & Gibson PR (2013) Short-chain carbohydrates and functional gastrointestinal disorders. *Am J Gastroenterol* **108**, 707–717.
87. Berg LK, Fagerli E, Martinussen M *et al.* (2013) Effect of fructose-reduced diet in patients with irritable bowel syndrome, and its correlation to a standard fructose breath test. *Scand J Gastroenterol* **48**, 936–943.
88. Halmos EP, Power VA, Shepherd SJ *et al.* (2014) A diet low in FODMAPs reduces symptoms of irritable bowel syndrome. *Gastroenterology* **146**(67–75), e5.
89. Olesen M & Gudmand-Hoyer E (2000) Efficacy, safety, and tolerability of fructooligosaccharides in the treatment of irritable bowel syndrome. *Am J Clin Nutr* **72**, 1570–1575.
90. 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.
91. 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.
92. Silk DBA, Davis A, Vulevic J *et al.* (2009) Clinical trial: the effects of a trans-galactooligosaccharide prebiotic on faecal microbiota and symptoms in irritable bowel syndrome. *Aliment Pharmacol Ther* **29**, 508–518.
93. Böhn L, Storsrud 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.
94. Pedersen N, Andersen NN, Vegh Z *et al.* (2014) Ehealth: low FODMAP diet vs *Lactobacillus rhamnosus* GG in irritable bowel syndrome. *World J Gastroenterol* **20**, 16215–16226.
95. Symons P, Jones MP & Kellow JE (1992) Symptom provocation in irritable bowel syndrome. Effects of differing doses of fructose-sorbitol. *Scand J Gastroenterol* **27**, 940–944.
96. Staudacher HM, Whelan K, Irving PM *et al.* (2011) Comparison of symptom response following advice for a diet low in fermentable carbohydrates (FODMAPs) versus standard dietary advice in patients with irritable bowel syndrome. *J Hum Nutr Diet* **24**, 487–495.
97. Gibson PR & Shepherd SJ (2010) Evidence-based dietary management of functional gastrointestinal symptoms: the FODMAP approach. *J Gastroenterol Hepatol* **25**, 252–258.
98. 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.
99. Martin L, van Vuuren C, Seamark L, *et al.* (2015) Low FODMAP advice for patients with irritable bowel syndrome: long-term outcomes for symptoms and dietary intake. *Gut* **64**(Suppl 1), A51–A52.
100. Tack GJ, Verbeek WH, Schreurs MW *et al.* (2010) The spectrum of celiac disease: epidemiology, clinical aspects and treatment. *Nat Rev Gastroenterol Hepatol* **7**, 204–213.
101. Aziz I, Hadjivassiliou M & Sanders DS (2015) The spectrum of noncoeliac gluten sensitivity. *Nat Rev Gastroenterol Hepatol* **12**, 516–526.
102. Vazquez-Roque MI, Camilleri M, Smyrk T *et al.* (2013) A controlled trial of gluten-free diet in patients with irritable bowel syndrome-diarrhea: effects on bowel frequency and intestinal function. *Gastroenterology* **144,** 903–911: e3.
103. Shahbazkhani B, Sadeghi A, Malekzadeh R *et al.* (2015) Non-celiac gluten sensitivity has narrowed the spectrum of irritable bowel syndrome: a double-blind randomized placebo-controlled trial. *Nutrients* **7**, 4542–4554.

104. Biesiekierski JR, Newnham ED, Irving PM *et al.* (2011) Gluten causes gastrointestinal symptoms in subjects without celiac disease: a double-blind randomized placebo-controlled trial. *Am J Gastroenterol* **106**, 508–514.
105. Biesiekierski JR, Peters SL, Newnham ED *et al.* (2013) No effects of gluten in patients with self-reported non-celiac gluten sensitivity after dietary reduction of fermentable, poorly absorbed, short-chain carbohydrates. *Gastroenterology* **145**, 320–328: e1–e3.
106. See JA, Kaukinen K, Makharia GK *et al.* (2015) Practical insights into gluten-free diets. *Nat Rev Gastroenterol Hepatol* **12**, 580–591.
107. Global Alliance on Probiotics (2013) Position paper on evaluation of probiotic claims. Available at: http://www.gap-probiotics.org/wp-content/uploads/2013/02/GAP-position-paper-15.02.2013.pdf (accessed 1 May 2016).
108. Agrawal A, Houghton LA, Morris J *et al.* (2009) Clinical trial: the effects of a fermented milk product containing Bifidobacterium lactis DN-173 010 on abdominal distension and gastrointestinal transit in irritable bowel syndrome with constipation. *Aliment Pharmacol Ther* **29**, 104–114.
109. Begtrup LM, de Muckadell OB, Kjeldsen J *et al.* (2013) Long-term treatment with probiotics in primary care patients with irritable bowel syndrome – a randomised, double-blind, placebo controlled trial. *Scand J Gastroenterol* **48**, 1127–1135.
110. Enck P, Zimmermann K, Menke G *et al.* (2008) A mixture of *Escherichia coli* (DSM 17252) and *Enterococcus faecalis* (DSM 16440) for treatment of the irritable bowel syndrome – a randomized controlled trial with primary care physicians. *Neurogastroenterol Motil* **20**, 1103–1109.
111. Guyonnet D, Chassany O, Ducrotte P *et al.* (2007) Effect of a fermented milk containing Bifidobacterium animalis DN-173 010 on the health-related quality of life and symptoms in irritable bowel syndrome in adults in primary care: a multicentre, randomized, double-blind, controlled trial. *Aliment Pharmacol Ther* **26**, 475–486.
112. Hong KS, Kang HW, Im JP *et al.* (2009) Effect of probiotics on symptoms in korean adults with irritable bowel syndrome. *Gut Liv* **3**, 101–107.
113. Hong YS, Hong KS, Park MH *et al.* (2011) Metabonomic understanding of probiotic effects in humans with irritable bowel syndrome. *J Clin Gastroenterol* **45**, 415–425.
114. Jafari E, Vahedi H, Merat S *et al.* (2014) Therapeutic effects, tolerability and safety of a multi-strain probiotic in Iranian adults with irritable bowel syndrome and bloating. *Arch Iran Med* **17**, 466–470.
115. Ki Cha B, Mun Jung S, Hwan Choi C *et al.* (2012) The effect of a multispecies probiotic mixture on the symptoms and fecal microbiota in diarrhea-dominant irritable bowel syndrome: a randomized, double-blind, placebo-controlled trial. *J Clin Gastroenterol* **46**, 220–227.
116. Kim HJ, Camilleri M, McKinzie S *et al.* (2003) A randomized controlled trial of a probiotic, VSL#3, on gut transit and symptoms in diarrhoea-predominant irritable bowel syndrome. *Aliment Pharmacol Ther* **17**, 895–904.
117. Kim HJ, Vazquez Roque MI, Camilleri M *et al.* (2005) A randomized controlled trial of a probiotic combination VSL# 3 and placebo in irritable bowel syndrome with bloating. *Neurogastroenterol Motil* **17**, 687–696.
118. Ko SJ, Han G, Kim SK *et al.* (2013) Effect of korean herbal medicine combined with a probiotic mixture on diarrhea-dominant irritable bowel syndrome: a double-blind, randomized, placebo-controlled trial. *Evid Based Complement Alternat Med* **2013**, 824605.
119. Lorenzo-Zuniga V, Llop E, Suarez C *et al.* (2014) I.31, a new combination of probiotics, improves irritable bowel syndrome-related quality of life. *World J Gastroenterol* **20**, 8709–8716.
120. Ludidi S, Jonkers DM, Koning CJ *et al.* (2014) Randomized clinical trial on the effect of a multispecies probiotic on visceroperception in hypersensitive IBS patients. *Neurogastroenterol Motil* **26**, 705–714.
121. Roberts LM, McCahon D, Holder R *et al.* (2013) A randomised controlled trial of a probiotic 'functional food' in the management of irritable bowel syndrome. *BMC Gastroenterol* **13**, 45.
122. Simren M, Ohman L, Olsson J *et al.* (2010) Clinical trial: the effects of a fermented milk containing three probiotic bacteria in patients with irritable bowel syndrome – a randomized, double-blind, controlled study. *Aliment Pharmacol Ther* **31**, 218–227.
123. Sisson G, Ayis S, Sherwood RA *et al.* (2014) Randomised clinical trial: a liquid multi-strain probiotic vs. placebo in the irritable bowel syndrome – a 12 week double-blind study. *Aliment Pharmacol Ther* **40**, 51–62.
124. Sondergaard B, Olsson J, Ohlson K *et al.* (2011) Effects of probiotic fermented milk on symptoms and intestinal flora in patients with irritable bowel syndrome: a randomized, placebo-controlled trial. *Scand J Gastroenterol* **46**, 663–672.
125. Williams EA, Stimpson J, Wang D *et al.* (2009) Clinical trial: a multistrain probiotic preparation significantly reduces symptoms of irritable bowel syndrome in a double-blind placebo-controlled study. *Aliment Pharmacol Ther* **29**, 97–103.
126. Yoon JS, Sohn W, Lee OY *et al.* (2014) Effect of multispecies probiotics on irritable bowel syndrome: a randomized, double-blind, placebo-controlled trial. *J Gastroenterol Hepatol* **29**, 52–59.
127. Charbonneau D, Gibb RD & Quigley EM (2013) Fecal excretion of *Bifidobacterium infantis* 35624 and changes in fecal microbiota after eight weeks of oral supplementation with encapsulated probiotic. *Gut Microbes* **4**, 201–211.

128. Choi CH, Jo SY, Park HJ *et al.* (2011) A randomized, double-blind, placebo-controlled multicenter trial of *Saccharomyces boulardii* in irritable bowel syndrome: effect on quality of life. *J Clin Gastroenterol* **45**, 679–683.
129. Dolin BJ (2009) Effects of a proprietary *Bacillus coagulans* preparation on symptoms of diarrhea-predominant irritable bowel syndrome. *Methods Find Exp Clin Pharmacol* **31**, 655–659.
130. Ducrotte P, Sawant P & Jayanthi V (2012) Clinical trial: *Lactobacillus plantarum* 299v (DSM 9843) improves symptoms of irritable bowel syndrome. *World J Gastroenterol* **18**, 4012–4018.
131. Gade J & Thorn P (1989) Paraghurt for patients with irritable bowel syndrome. A controlled clinical investigation from general practice. *Scand J Prim Health Care* **7**, 23–26.
132. Hun L (2009) *Bacillus coagulans* significantly improved abdominal pain and bloating in patients with IBS. *Postgrad Med* **121**, 119–124.
133. Kruis W, Chrubasik S, Boehm S *et al.* (2012) A double-blind placebo-controlled trial to study therapeutic effects of probiotic *Escherichia coli* Nissle 1917 in subgroups of patients with irritable bowel syndrome. *Int J Colorectal Dis* **27**, 467–474.
134. Niedzielin K, Kordecki H & Birkenfeld B (2001) A controlled, double-blind, randomized study on the efficacy of *Lactobacillus plantarum* 299V in patients with irritable bowel syndrome. *Eur J Gastroenterol Hepatol* **13**, 1143–1147.
135. Niv E, Naftali T, Hallak R *et al.* (2005) The efficacy of *Lactobacillus reuteri* ATCC 55730 in the treatment of patients with irritable bowel syndrome – a double blind, placebo-controlled, randomized study. *Clin Nutr* **24**, 925–931.
136. Nobaek S, Johansson ML, Molin G *et al.* (2000) Alteration of intestinal microflora is associated with reduction in abdominal bloating and pain in patients with irritable bowel syndrome. *Am J Gastroenterol* **95**, 1231–1238.
137. O'Mahony L, McCarthy J, Kelly P *et al.* (2005) Lactobacillus and bifidobacterium in irritable bowel syndrome: symptom responses and relationship to cytokine profiles. *Gastroenterology* **128**, 541–551.
138. O'Sullivan MA & O'Morain CA (2000) Bacterial supplementation in the irritable bowel syndrome. A randomised double-blind placebo-controlled crossover study. *Dig Liver Dis* **32**, 294–301.
139. Pineton de Chambrun G, Neut C, Chau A *et al.* (2015) A randomized clinical trial of *Saccharomyces cerevisiae* versus placebo in the irritable bowel syndrome. *Dig Liver Dis* **47**, 119–124.
140. Sinn DH, Song JH, Kim HJ *et al.* (2008) Therapeutic effect of *Lactobacillus acidophilus*-SDC 2012, 2013 in patients with irritable bowel syndrome. *Dig Dis Sci* **53**, 2714–2718.
141. Stevenson C, Blaauw R, Fredericks E *et al.* (2014) Randomized clinical trial: effect of *Lactobacillus plantarum* 299 v on symptoms of irritable bowel syndrome. *Nutrition* **30**, 1151–1157.
142. Whorwell PJ, Altringer L, Morel J *et al.* (2006) Efficacy of an encapsulated probiotic *Bifidobacterium infantis* 35624 in women with irritable bowel syndrome. *Am J Gastroenterol* **101**, 1581–1590.
143. Ortiz-Lucas M, Tobias A, Saz P *et al.* (2013) Effect of probiotic species on irritable bowel syndrome symptoms: a bring up to date meta-analysis. *Rev Esp Enferm Dig* **105**, 19–36.
144. Moayyedi P, Ford AC, Talley NJ *et al.* (2010) The efficacy of probiotics in the treatment of irritable bowel syndrome: a systematic review. *Gut* **59**, 325–332.
145. McFarland LV & Dublin S (2008) Meta-analysis of probiotics for the treatment of irritable bowel syndrome. *World J Gastroenterol* **14**, 2650–2661.
146. Mazurak N, Broelz E, Storr M *et al.* (2015) Probiotic therapy of the irritable bowel syndrome: why is the evidence still poor and what can be done about it? *J Neurogastroenterol Motil* **21**, 471–485.
147. Hungin AP, Mulligan C, Pot B *et al.* (2013) Systematic review: probiotics in the management of lower gastrointestinal symptoms in clinical practice – an evidence-based international guide. *Aliment Pharmacol Ther* **38**, 864–886.
148. Hoveyda N, Heneghan C, Mahtani KR *et al.* (2009) A systematic review and meta-analysis: probiotics in the treatment of irritable bowel syndrome. *BMC Gastroenterol* **9**, 15.
149. Ford AC, Quigley EM, Lacy BE *et al.* (2014) Efficacy of prebiotics, probiotics, and synbiotics in irritable bowel syndrome and chronic idiopathic constipation: systematic review and meta-analysis. *Am J Gastroenterol* **109**, 1547–1561; quiz 6, 62.
150. Brenner DM, Moeller MJ, Chey WD *et al.* (2009) The utility of probiotics in the treatment of irritable bowel syndrome: a systematic review. *Am J Gastroenterol* **104**, 1033–1049.
151. Muraro A, Werfel T, Hoffmann-Sommergruber K *et al.* (2014) EAACI Food Allergy and Anaphylaxis Guidelines: diagnosis and management of food allergy. *Allergy* **69**, 1008–1025.
152. Lomer MC (2015) Review article: the aetiology, diagnosis, mechanisms and clinical evidence for food intolerance. *Aliment Pharmacol Ther* **41**, 262–275.
153. Barbara GWB & Stanghellini V (2007) Mast cell-dependent excitation of visceral-nociceptive sensory neurons in irritable bowel syndrome. *Gastroenterology* **132**, 26–37.
154. Hughes PAZH, Penttila IA, Blackshaw LA *et al.* (2013) Immune activation in irritable bowel syndrom: can neuroimmune interactions explain symptoms? *Am J Gastroenterology* **108**, 1066–1074.

155. Matricon J, Meleine M, Gelot A *et al.* (2012) Review article: associations between immune activation, intestinal permeability and the irritable bowel syndrome. *Aliment Pharmacol Ther* **36**, 1009–1031.
156. Ligaarden SCLS & Farup PG (2012) IgG and IgG4 antibodies in subjects with irritable bowel syndrom: a case control study in the general population. *Gastroenterology* **12**, 166.
157. Hawthorne B, Lambert S, Scott D *et al.* (1991) Food intolerance and the irritable bowel syndrome. *J Hum Nutr Diet* **4**, 19–23.
158. Nanda R, James R, Smith H *et al.* (1989) Food intolerance and the irritable bowel syndrome. *Gut* **30**, 1099–1104.
159. Parker TJ, Naylor SJ, Riordan AM *et al.* (1995) Management of patients with food intolerance in irritable bowel syndrome. The development and use of an exclusion diet. *J Hum Nutr Diet* **8**, 159–166.
160. Petitpierre M, Gumowski P & Girard JP (1985) Irritable bowel syndrome and hypersensitivity to food. *Ann Allergy* **54**, 538–540.
161. Piccinini G, Feliciani M, Mazzetti M *et al.* (1990) A potential diagnostic role of a disodium chromoglycate course in irritable bowel syndrome. *Int J Immunopathol Pharmacol* **3**, 107–112.
162. Stefanini G, Saggioro A, Alvisi V *et al.* (1995) Oral cromolyn sodium in comparison with elimination diet in the irritable bowel syndrome, diarrheic type. Multicenter study of 428 patients. *Scand J Gastroenterol* **30**, 535–541.
163. Anaphylaxis (2015) Food allergens. Available at: www.anaphylaxis.org.uk (accessed 12 December 2015).
164. Allergy UK (2015) Available at: www.allergyuk.org (accessed 1 May 2016).
165. Simren M (2014) Diet as a therapy for irritable bowel syndrome: progress at last. *Gastroenterology* **146**, 10–12.
166. Ford AC, Moayyedi P, Lacy BE *et al.* (2014) American College of Gastroenterology monograph on the management of irritable bowel syndrome and chronic idiopathic constipation. *Am J Gastroenterol* **109**(Suppl 1), S2–S26.
167. Camilleri M (2015) Bile acid diarrhea: prevalence, pathogenesis, and therapy. *Gut Liv* **9**, 332–339.
168. Wedlake L, A'Hern R, Russell D *et al.* (2009) Systematic review: the prevalence of idiopathic bile acid malabsorption as diagnosed by SeHCAT scanning in patients with diarrhoea-predominant irritable bowel syndrome. *Aliment Pharmacol Ther* **30**, 707–717.
169. Ho S, Woodford K, Kukuljan S *et al.* (2014) Comparative effects of A1 versus A2 beta-casein on gastrointestinal measures: a blinded randomised cross-over pilot study. *Eur J Clin Nutr* **68**, 994–1000.
170. Jianqin S, Leiming X, Lu X *et al.* (2016) Effects of milk containing only A2 beta casein versus milk containing both A1 and A2 beta casein proteins on gastrointestinal physiology, symptoms of discomfort, and cognitive behavior of people with self-reported intolerance to traditional cows' milk. *Nutr J* **15**, 35.
171. Staudacher H, Lomer M, Lindsay J *et al.* (2015) The impact of low FODMAP dietary advice and probiotics on symptoms in irritable bowel syndrome: a randomised, placebo-controlled, 2 × 2 factorial trial. *Gut* **64**(Suppl 1), A51.
172. Yao CK, Gibson PR & Shepherd SJ (2013) Design of clinical trials evaluating dietary interventions in patients with functional gastrointestinal disorders. *Am J Gastroenterol* **108**, 748–758.
173. Biesiekierski JR, Newnham ED, Shepherd SJ *et al.* (2014) Characterization of adults with a self-diagnosis of nonceliac gluten sensitivity. *Nutr Clin Pract* **29**, 504–509.
174. Catassi C, Bai JC, Bonaz B *et al.* (2013) Non-celiac gluten sensitivity: the new frontier of gluten related disorders. *Nutrients* **5**, 3839–3853.
175. Ludvigsson JF, Bai JC, Biagi F *et al.* (2014) Diagnosis and management of adult coeliac disease: guidelines from the British Society of Gastroenterology. *Gut* **63**, 1210–1228.
176. Patel SM, Stason WB, Legedza A *et al.* (2005) The placebo effect in irritable bowel syndrome trials: a meta-analysis. *Neurogastroenterol Motil* **17**, 332–340.
177. National Institute for Health and Clinical Excellence (2016). Irritable bowel syndrome in adults. Quality Standard 114. Available at: https://www.nice.org.uk/guidance/qs114 (accessed 23 February 2016).
178. 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.
179. Manichanh C, Eck A, Varela E *et al.* (2014) Anal gas evacuation and colonic microbiota in patients with flatulence: effect of diet. *Gut* **63**, 401–408.

Supporting information

Additional Supporting Information may be found online in the supporting information tab for this article:
Table S1. Generic and topic specific criteria.
Table S2. Search terms.
Table S3. Excluded studies.
Table S4. Question number and risk of bias criteria in Table 1 for different study designs.
Table S5. Summary of searches.

