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Low-FODMAP Diet

The low-FODMAP diet is the most rigorously studied dietary approach for irritable bowel syndrome: the vast majority of studies have been conducted as randomised trials in humans, several with both symptom and microbiome endpoints. The most intriguing finding relates to a side effect — the diet measurably alters the gut microbiota, and the field is still debating how to manage this change.

194

STUDIES IN
TOTAL

165

IN HUMANS

110

OF WHICH
CONTROLLED

3

IN ANIMALS

2

IN THE LAB

Figures from the IHR study database · Last updated 2026-10-16

How to Read This Dossier

ESTABLISHED

measured in humans, with a comparison group

PLAUSIBLE

supported by mechanism, animal studies or observation

HYPOTHESIS

a likely connection that still needs to be tested

Low-FODMAP diet at a glance

ORIGIN:

The low-FODMAP diet temporarily restricts fermentable oligo-, di-, and monosaccharides and polyols — short-chain carbohydrates that are only partially absorbed in the small intestine and fermented in the colon. It does not add anything but is implemented in stages: a clear restriction phase, gradual reintroduction of individual groups, and an individual tolerance threshold. In the studies, this is done as dietary counselling, not as a self-experiment.

EVIDENCE BASE:

194 evaluated works · 110 controlled human studies · 55 observational studies · 8 review articles · 3 animal studies · 0 cell culture studies

STRONGEST EVIDENCE:

- Intestine and microbiome: A meta-analysis of twelve studies (nine parallel-group, three crossover trials) showed a moderate to large reduction in IBS severity compared to a control diet (SMD -0.66; 95 % CI -0.88 to -0.44).
- Intestine and microbiome: When analysing only studies using the validated IBS-SSS, the mean reduction was 45 points (95 % CI -77 to -14; I²=89 %) [25].
- Intestine: Randomised crossover trial in children with Rome III IBS — abdominal pain frequency was lower on the low-FODMAP diet (1.1 ± 0.2 vs. 1.7 ± 0.4 episodes per day; p<0.05) [4].
- Intestine: A meta-analysis of randomised controlled trials and cohort studies found a significant reduction in pain and bloating compared to traditional and high-FODMAP diets [10].
- Intestine: Randomised open-label trial in IBD patients — response in 30 of 37 (81 %) on low-FODMAP vs. 19 of 41 (46 %) on standard diet (OR 5.30; 95 % CI 1.81 to 15.55; p<0.01) [11].

LIMITATIONS:

- The comparability of studies is limited: definitions of restricted carbohydrates, duration of restriction, and assessment tools vary between studies.
- A diet cannot be blinded; some controlled studies compare against other dietary recommendations such as modified NICE guidelines rather than placebo.
- The primary endpoints are predominantly symptom diaries and questionnaires, i.e., self-reported symptoms rather than disease progression. Laboratory markers describe the state of the gut environment, not disease progression.
- Long-term, large randomised controlled trials, as well as nutrient adequacy, cost, and implementation of the diet, remain open questions [32].

ASSESSMENT:

The low-FODMAP diet is the most rigorously studied dietary approach for irritable bowel syndrome; its symptomatic effect is considered evidence-based.

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1. What the low-FODMAP Diet Is

FODMAP stands for fermentable oligo-, di-, and monosaccharides and polyols — short-chain carbohydrates that are only partially absorbed in the small intestine and fermented in the colon. The low-FODMAP diet does not add anything; it temporarily restricts this group of carbohydrates. It is implemented in stages: an initial phase of clear restriction, followed by gradual reintroduction of individual groups, and finally an individual tolerance threshold. In the studies, this is done as **dietary counselling** and not as a self-experiment; several studies explicitly examine the effect of counselling compared to other dietary advice. Dietary intake is recorded via protocols and food selection, not via the dose of a single substance.

A key consideration in evaluating all studies is that a diet cannot be blinded: those who eat fewer FODMAPs know it. Some controlled studies therefore compare not against placebo but against other dietary recommendations, such as modified NICE guidelines; the measured difference is then between two dietary approaches, not the effect of diet per se. The primary endpoints are predominantly symptom diaries and questionnaires, i.e., self-reported symptoms rather than disease progression. Parallel to this, markers are collected: faecal microbiome, cytokine profiles, inflammatory and metabolic markers. Such **laboratory values** describe the state of the gut environment, not the progression of a disease.

2. What the Studies Show in Detail

The key human studies and major reviews — grouped by application area, with population, duration, preparation, measurement, and outcome as reported in the original works. Numbers are included only where the study provides them.

Distribution of **controlled human studies** by application area — the numbers are not additive, as a single study may be assigned to multiple areas:

APPLICATION AREA	CONTROLLED STUDIES
Intestine and microbiome	81
Safety and tolerability	32
Inflammatory processes	20
Cognition and mental health	9

APPLICATION AREA	CONTROLLED STUDIES
Metabolism	8
Fatigue and stress	6
Immune system	5
Oncology	3

Intestine and Microbiome

Single-blind, randomised crossover study: 27 IBS patients and six healthy participants received two predefined diets for 21 days each, differing only in FODMAP content (mean with 95 % CI 3.05 [1.86 to 4.25] g/day vs. 23.7 [16.9 to 30.6] g/day), separated by at least 21 days of washout [1]. Stool samples from a five-day baseline period and days 17–21 of the intervention diets were pooled; faecal pH, short-chain fatty acids, and bacterial abundance and diversity were measured. On the low-FODMAP diet, faecal pH was higher (7.37 [7.23 to 7.51] vs. 7.16 [7.02 to 7.30]; $p=0.001$) and total bacterial count lower (9.63 [9.53 to 9.73] vs. 9.83 [9.72 to 9.93] log₁₀ copies/g; $p<0.001$), diversity was greater, and short-chain fatty acid concentrations remained similar. In a controlled, single-blind study in IBS patients, 20 were randomised to a low-FODMAP diet and 20 to a high-FODMAP diet for three weeks, with 37 participants (19 and 18) completing the phase [2]. The IBS-SSS decreased only in the low-FODMAP group ($p<0.001$). The lactulose breath test showed a small decrease in hydrogen production in this group, and urinary metabolic profiling differed significantly between groups after the diet ($p<0.01$), with histamine, *p*-hydroxybenzoic acid, and azelaic acid driving the separation; urinary histamine concentration was eightfold lower ($p<0.05$), and the study reports greater Actinobacteria diversity.

A 2×2 factorial, randomised, placebo-controlled study in 104 IBS patients (aged 18–65, Rome III) at two UK clinics compared four weeks of counselling for a sham diet or a low-FODMAP diet, each combined with placebo or a multi-strain probiotic; this resulted in four groups (27 sham diet/placebo, 26 sham diet/probiotic, 24 low-FODMAP/placebo, 27 low-FODMAP/probiotic) [3]. The sham diet restricted a similar number of staple and other foods and was similarly difficult to adhere to; 15 gastrointestinal symptoms, overall symptomatology, and stool frequency and consistency were recorded daily for seven days before the study and during the study. The study title notes that a probiotic restores Bifidobacterium species. A double-blind crossover study in children with Rome III IBS followed a one-week baseline period with a low-FODMAP diet and a typical American childhood diet, separated by a five-day washout; 33 children completed the study [4]. The primary endpoint, abdominal pain frequency, was lower on the low-FODMAP diet than on the comparison diet (1.1 ± 0.2 vs. 1.7 ± 0.4 episodes per day; $p<0.05$) and lower than the baseline of 1.4 ± 0.2 ($p<0.01$), while it increased on the typical American diet ($p<0.01$); responders were defined as children with at least a 50 % reduction in pain frequency solely on the low-FODMAP diet, and responders differed in baseline microbiome composition.

A randomised study in 50 IBS patients (Group I $n=23$, Group II $n=27$) began a low-FODMAP diet either immediately or after three months; Group I reintroduced selected FODMAPs after three months [16]. After three months, the IBS-SSS had decreased more in Group I (275.6 ± 63.6 to 128.8 ± 82.5 versus 246.8 ± 71.1 to 203.6 ± 70.1 ; $p<0.0002$), and quality of life had improved more (68.5 ± 18.0 to 83 ± 13.4 versus 72.9 ± 12.8 to 73.3 ± 14.4 ; $p<0.0001$); after six months, the reduction persisted in Group I (160 ± 102) and emerged in Group II (124 ± 76). Fibre intake decreased during the diet (baseline 33 ± 17 g/day). **Proven:** A meta-analysis of twelve studies (nine parallel, three crossover trials) found a moderate to large reduction in IBS severity compared with a control diet (SMD -0.66; 95 % CI -0.88 to -0.44; $I^2=54$ %) [25]; when restricted to studies using the validated IBS-SSS, the mean reduction was 45 points (95 % CI -77 to -14; $I^2=89$ %), and subgroup analyses for adherence, age, intervention duration, IBS subtype, outcome measures and risk of bias showed no significantly different results. In an analysis of 59 consecutive patients with functional dyspepsia, 40 received low-FODMAP advice and 19 standard advice; 48 of 59 (81 %) also had IBS. Epigastric symptom burden decreased more with low-FODMAP advice than with standard advice (estimated marginal mean -3.6 points) [34].

Reviews outline the mechanisms of action. MRI studies showed that short-chain fermentable carbohydrates increase small-intestinal water volume and colonic gas production, triggering functional symptoms in visceral hypersensitivity [7]; ileostomy and MRI studies revealed higher ileal water content, and breath tests showed increased colonic hydrogen production [9]. At least ten randomised controlled or randomised comparative trials report clinical response in 50 % to 80 % of IBS patients, particularly for bloating, flatulence, diarrhoea and global symptoms [7]; a review cites improvement in global and individual symptoms in up to 86 % of patients and highlights adherence and specialist dietary advice as crucial, while long-term health effects remain unclear [14]. Implementation is described as a three-step approach of restriction, reintroduction and individualisation [29]; most evidence concerns the restriction phase and relief of bloating and pain [30], with additional mechanisms discussed including gut barrier function, motility, short-chain fatty acids and metabolomics [30]. For children with functional abdominal pain disorders, a systematic review of five randomised trials found an effect on pain intensity in two studies (0–10-point VAS: MD 1.77; 95 % CI 1.23 to 2.31; $n=60$ after two months versus a gastrointestinal-protective diet; MD -1.36 cm; 95 % CI -2.38 to -0.34; $n=10$ over three days versus a typical Singaporean diet) [36]. The gut microbiome is discussed as a potential predictor of response, but studies differ widely in population, measurement methods and statistics [35]. For diarrhoea-predominant IBS, a review finds no ideal dietary protocol [27]; the reduced frequency of Bifidobacterium species often seen in IBS—linked to worse symptom profiles—and the opposing effects of probiotics versus FODMAP restriction are described as paradoxical [28]; within the microbiota–gut–brain axis, the low-FODMAP diet is framed as a microbiome-modulating treatment [33]. Open questions identified by a review include long-term, large randomised controlled trials and assessment of nutrient adequacy, costs and delivery of the diet [32].

Further Investigations

The first randomised controlled trial of a low-FODMAP diet in adults with IBS and diarrhoea in the United States compared, after a two-week screening phase, a four-week low-FODMAP diet with a diet based on modified NICE guidelines (mNICE) [5]. Ninety-two participants (65 women, median age 42.6 years) were randomised; the primary endpoint was the proportion of patients with adequate relief in at least 50 % of intervention weeks 3 and 4, with secondary endpoints including a composite of at least 30 % reduction in mean daily pain score and a reduction in mean daily Bristol stool form by at least 1 from baseline, plus individual IBS symptoms from daily diaries. The IBS-SSS reduction from baseline to week 6 was 133 ± 122 with the low-FODMAP diet, 68

± 107 with *Lactobacillus rhamnosus* GG and 34 ± 95 with the normal diet ($p < 0.01$); after adjustment for baseline covariates, the difference remained significant only versus the normal diet (IBS-SSS 75; 95 % CI 24 to 126; $p < 0.01$), not for *Lactobacillus rhamnosus* GG (32; 95 % CI 18 to 80; $p = 0.20$). Quality of life did not change significantly in any of the three groups.

PROVEN

A meta-analysis of randomised controlled trials and cohort studies found that a low-FODMAP diet significantly reduced pain and bloating compared with a traditional diet and a high-FODMAP diet; no significant difference was seen in stool consistency between treatments [10]. The paper frames IBS prevalence at 7 % to 15 % of the general population. Another meta-analysis pooled studies in adults with IBS meeting Rome II, Rome III, Rome IV or NICE criteria and compared the low-FODMAP diet with a regular high-FODMAP IBS standard diet using the IBS symptom severity score; heterogeneity led to a random-effects model, and mean differences with 95 % confidence intervals were calculated from studies reporting means, standard deviations or mean differences with standard deviations of differences and p-values [15].

A secondary analysis of two randomised controlled trials examined usual nutrient intake, diet quality and diversity, and the effect of a four-week low-FODMAP diet versus controls in 130 IBS patients at a tertiary centre in London: 63 received low-FODMAP meals, 48 a sham diet and 19 maintained their usual diet; all advice was delivered by specialist dietitians and assessed using seven-day diet diaries at baseline and after four weeks [18]. A review frames FODMAP intake via fermentation and osmotic effects as triggers of abdominal pain, bloating, wind and altered bowel habit, and contrasts FODMAP restriction with elimination diets for hypersensitivity to food chemicals and possible non-coeliac gluten sensitivity [26]. **Plausible:** If symptoms arise via osmotic load and fermentation, the effect should depend on the amount of FODMAPs ingested; direct comparisons between low-FODMAP diets and other dietary strategies in larger populations over longer periods are still lacking [15][26].

Inflammation

A single-blind randomised study in 52 patients with quiescent Crohn's disease or ulcerative colitis and persistent bowel symptoms at two large UK gastroenterology centres compared a four-week low-FODMAP diet ($n = 27$) with a control diet ($n = 25$), both delivered with dietary counselling [6]. Stool and blood samples were collected at baseline and study end, the faecal microbiome was profiled for composition and function using shotgun metagenomics, and T-cell phenotypes in blood were assessed by flow cytometry. A randomised controlled open-label trial in 89 patients with IBD in remission or mild-to-moderate activity and co-existing IBS-like symptoms per Rome III criteria compared a six-week low-FODMAP diet with normal diet; 78 patients were analysed (37 versus 41; 75 % women, median age 40 years, range 20 to 70 years) [11]. In the primary endpoint of a ≥ 50 -point reduction in IBS-SSS, 30 of 37 (81 %) on the low-FODMAP diet and 19 of 41 (46 %) on normal diet responded (OR 5.30; 95 % CI 1.81 to 15.55; $p < 0.01$); the median IBS-SSS after six weeks was 115 (IQR 33 to 169) versus 170 (IQR 91 to 288; $p = 0.02$).

In a routine-care analysis, 88 consecutive IBD patients referred for low-FODMAP dietary counselling received it for symptom control; the Gastrointestinal Symptom Rating Score and Bristol stool form scale were recorded at baseline and at least six weeks later [12]. The proportion with satisfactory symptom relief rose from 14 of 88 (16 %) to 69 of 88 (78 %; $p < 0.001$), the composite symptom score fell from 1.2 (SD 0.5) to 0.7 (SD 0.5; $p < 0.001$), and stool consistency and frequency also improved. A randomised, double-blind, placebo-controlled re-challenge trial in patients with quiescent IBD and functional gastrointestinal symptoms responsive to a low-FODMAP diet tested, in random order, three-day doses of fructan (12 g/day), galacto-oligosaccharides (6 g/day), sorbitol (6 g/day) and glucose as placebo (12 g/day), each separated by a washout [17]. Of 32 recruited patients, data were available for 29 who completed all arms (12 Crohn's disease, 17 ulcerative colitis). On the last day of fructan, 18 of 29 (62.1 %) reported adequate relief versus 26 of 29 (89.7 %) on glucose ($p = 0.033$); pain (1.1 versus 0.5; $p = 0.004$), bloating (1.3 versus 0.6; $p = 0.002$), flatulence (1.5 versus 0.7; $p = 0.004$) and urgency (0.9 versus 0.4; $p = 0.014$) were more pronounced.

A study in 20 IBS patients with diarrhoea-predominant or mixed IBS followed a low-FODMAP diet for nine weeks; after three weeks, participants were randomised and double-blind for ten days to either fructo-oligosaccharides as FODMAP or maltodextrin as placebo, followed by three weeks washout and crossover [8]. IBS symptoms improved after three weeks on the diet overall, and 80 % reported symptom relief on placebo versus 30 % on fructo-oligosaccharides. Serum interleukin-6, interleukin-8 and tumour necrosis factor alpha, faecal microbiota via 16S rRNA sequencing and short-chain fatty acids were also measured. **Hypothesis:** If functional co-symptoms improve most under a low-FODMAP diet, cytokine levels might remain unchanged; whether the measured cytokines, T-cell phenotypes and metagenomic data support this separation is the question that further analysis of these cohorts will address [6][8].

Overall Picture

At the same time, this dietary approach shifts the gut flora: lower total bacterial counts and higher faecal pH [1], alongside a reduced frequency of *Bifidobacterium* species often seen in IBS [28]. Symptom relief and microbiome change occur together, raising the question of how to weigh both effects.

Reviews themselves identify open questions about how long microbiome changes persist and their clinical significance [7][30], as well as long-term effects on nutrient intake [14][32]. **Hypothesis:** Because osmotic load and fermentation are the shared variables linking symptoms and microbial substrate, individualised reintroduction might preserve symptom relief while limiting microbial shifts; this assumption has not been tested in the reintroduction phase [16]. Whether baseline microbiome profiles predict who will respond is a testable question, but studies differ widely in population, measurement and analysis [35].

Methodology of the evidence base

Key studies include randomised controlled trials with 20 to 130 participants and intervention durations from three days in re-challenge to six months, mostly three to six weeks, conducted as parallel, crossover or factorial designs with sham diet, control diet or placebo [1][3][4][8][11][13][16][17][18]. Typical endpoints include the IBS-SSS, daily pain frequency, individual symptoms from diaries and stool pH, short-chain fatty acids, bacterial abundance and diversity, breath tests, urinary metabolomics and cytokines. Rarely are double-blind re-challenge designs used [17], and even more rarely is nutrient intake and diet quality assessed simultaneously [18]. Dietary blinding remains the core methodological challenge because dietary patterns are hard to conceal [9]. Open questions include trials that examine the reintroduction phase and predictors of response within a unified design with longer follow-up [16][35][36].

Microbiome change is part of the procedure — how to handle it remains open

The most-cited controlled work in the field describes how colonic milieu changes with dietary FODMAP content. Another randomised study links IBS symptom improvement with a decrease in Bifidobacterium species and shows that an added probiotic restores these species. This shift is not automatically harmful—it is the measurable expression of reduced substrate supply to the microbiome. The question is therefore not whether the gut flora changes, but how to manage this change.

Notably, the **REINTRODUCTION PHASE** is rarely the explicit focus in highly cited work: most studies examine the restriction phase, while the long-term individualised solution is assumed in practice. One paper describes microbiome markers as predictors of clinical response, shifting the question from diet to patient selection. What remains to be clarified is whether time-limited restriction followed by reintroduction can reverse microbiome changes. The definitive study would be a controlled trial with symptom and microbiome endpoints across both phases.

3. How Robust Is the Research Base

A large share of human-controlled studies are randomised; purely observational work forms a smaller group, and basic science and mechanistic studies an even smaller one. The dominant application area is the **gut**, followed by safety and inflammation. Cognition, metabolism, fatigue and the immune system are secondary fields with few studies, and oncological questions remain isolated cases. Strikingly, the citation hierarchy places randomised controlled trials measuring both symptoms and microbiome above review articles. The field is therefore not driven by summaries, but by primary studies.

Methodologically, the field is unusually well developed: there are randomised controlled trials combining symptom and microbiome endpoints and a meta-analysis of symptom effects in IBS. Symptom reduction in IBS can therefore be considered **proven**, supported by multiple consistent controlled studies—albeit largely based on self-reported complaints. Comparability remains limited: definitions of restricted carbohydrates, duration of restriction and outcome instruments vary between studies. Observational studies provide associations but cannot rule out expectancy effects. Animal data are still emerging and should be read as **preclinical**.

4. What Is Often Claimed — and What the Data Support

CLAIM	EVIDENCE BASE
Low-FODMAP diet reduces IBS symptoms	Several randomised controlled trials and a meta-analysis show a reduction in symptoms in irritable bowel syndrome; the endpoints are predominantly self-repo
The effect is mediated via the gut flora	Kontrollierte Studien messen Symptome und Mikrobiom zugleich. One study describes an association between microbiome markers and clinical response; this
It also helps in inflammatory bowel diseases	Patients with inactive disease were examined; symptoms, faecal microbiome, and inflammatory markers were recorded. These studies do not address the prog
Children benefit in the same way	For children with irritable bowel syndrome, there is one randomised controlled trial examining microbiome markers and clinical response. Thus, the transfer of

5. The Most Robust Studies

Ordered by evidence strength, not by prominence. The full evaluation includes significantly more studies; here, the most reliable ones are presented.

Controlled Human Studies

- ¹ Diets that differ in their FODMAP content alter the colonic luminal microenvironment (2015)
- ² FODMAPs alter symptoms and the metabolome of patients with IBS: a randomised controlled trial (2017)
- ³ A Diet Low in FODMAPs Reduces Symptoms in Patients With Irritable Bowel Syndrome and A Probiotic Restores Bifidobacterium Species: A Randomized ... (2017)

- ⁴ Randomised clinical trial: gut microbiome biomarkers are associated with clinical response to a low FODMAP diet in children with the irritable bowel ... (2015)
- ⁵ A Randomized Controlled Trial Comparing the Low FODMAP Diet vs. Modified NICE Guidelines in US Adults with IBS-D (2016)
- ⁶ Effects of Low FODMAP Diet on Symptoms, Fecal Microbiome, and Markers of Inflammation in Patients With Quiescent Inflammatory Bowel Disease in a ... (2020)
- ⁷ The low FODMAP diet: recent advances in understanding its mechanisms and efficacy in IBS (2017)
- ⁸ Effects of varying dietary content of fermentable short-chain carbohydrates on symptoms, fecal microenvironment, and cytokine profiles in patients ... (2017)
- ⁹ Mechanisms and efficacy of dietary FODMAP restriction in IBS (2014)
- ¹⁰ Low-FODMAP Diet Improves Irritable Bowel Syndrome Symptoms: A Meta-Analysis (2017)
- ¹¹ Low-FODMAP diet reduces irritable bowel symptoms in patients with inflammatory bowel disease (2017)
- ¹² Fermentable Carbohydrate Restriction (Low FODMAP Diet) in Clinical Practice Improves Functional Gastrointestinal Symptoms in Patients with ... (2016)
- ¹³ Ehealth: low FODMAP diet vs Lactobacillus rhamnosus GG in irritable bowel syndrome (2014)
- ¹⁴ Efficacy of the low FODMAP diet for treating irritable bowel syndrome: the evidence to date (2016)
- ¹⁵ Low fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAP) diet improves symptoms in adults suffering from irritable bowel ... (2017)
- ¹⁶ Long-term irritable bowel syndrome symptom control with reintroduction of selected FODMAPs (2017)
- ¹⁷ Fermentable Carbohydrates [FODMAPs] Exacerbate Functional Gastrointestinal Symptoms in Patients With Inflammatory Bowel Disease: A Randomised ... (2017)
- ¹⁸ Nutrient Intake, Diet Quality, and Diet Diversity in Irritable Bowel Syndrome and the Impact of the Low FODMAP Diet (2020)
- ¹⁹ Volatile Organic Compounds in Feces Associate With Response to Dietary Intervention in Patients With Irritable Bowel Syndrome (2018)
- ²⁰ Low fermentable oligo-di-mono-saccharides and polyols diet versus general dietary advice in patients with diarrhea-predominant irritable bowel ... (2018)
- ²¹ Low fermentable oligosaccharide, disaccharide, monosaccharide, and polyol diet in patients with diarrhea-predominant irritable bowel syndrome: A ... (2021)
- ²² Efficacy and Acceptability of Dietary Therapies in Non-Constipated Irritable Bowel Syndrome: A Randomized Trial of Traditional Dietary Advice, the ... (2022)
- ²³ A low FODMAP diet is associated with changes in the microbiota and reduction in breath hydrogen but not colonic volume in healthy subjects (2018)
- Use of the low-FODMAP diet in inflammatory bowel disease (2017)

6. Summary

Evidence: Symptom reduction in irritable bowel syndrome (IBS) is considered well-established, supported by several consistent controlled studies. A meta-analysis of twelve studies (nine parallel-group, three crossover trials) reported a moderate to large reduction in IBS severity compared with a control diet (SMD -0.66; 95 % CI -0.88 to -0.44; $I^2=54$ %); when analysing studies using the validated IBS-SSS, the mean reduction was 45 points (95 % CI -77 to -14) [25]. A further meta-analysis of randomised controlled trials and cohort studies found significant reductions in pain and bloating compared with traditional and high-FODMAP diets, while stool consistency showed no significant difference [10]. Thus, symptom reduction in IBS is the central finding of this approach.

PLAUSIBLE If symptoms arise from osmotic load and fermentation, the effect should depend on the amount of FODMAPs consumed. **Hypothesis:** Changes in the microbiome might predict response; the field remains divided on how to address this [28]. The long-term health effects remain unclear [14].

What remains to be clarified is how this dietary approach performs over longer periods and in larger populations: an overview identifies the need for long-term, large randomised controlled trials, as well as assessments of nutrient adequacy, costs, and the practical delivery of the diet [32]. Direct comparisons between low-FODMAP diets and other nutritional strategies over extended periods are still lacking [15][26]. In diarrhoea-predominant IBS, no ideal dietary protocol exists according to an overview [27]. A study prospectively assessing response based on baseline microbiome and adherence, and evaluating nutrient adequacy during the reintroduction phase, would address this question.

7. What Remains Open

- **Reintroduction:** What symptom and microbiome values emerge when FODMAP-containing foods are reintroduced after the restriction phase? This phase is rarely a standalone focus in controlled studies.
- **Prediction:** Can responders be identified before starting the diet? Microbiome markers are under investigation as predictors; no robust selection criterion has yet emerged.
- **Outcomes:** Most studies rely on self-reported symptoms. It remains to be determined which combination of symptom diaries and laboratory measures best reflects changes in disease course.

Frequently Asked Questions

How is the low-FODMAP diet implemented in studies?

In controlled trials, it is introduced as dietary counselling, usually with a phase of strict restriction followed by reintroduction. Comparators include either another dietary counselling approach, such as modified NICE guidelines, or a diet with a different FODMAP content. There is no standardised protocol across studies regarding duration or extent of restriction.

What is known about its effect on the microbiome?

Controlled studies report changes in gut milieu and Bifidobacterium species; one trial shows that co-administered probiotics restore these species. Such markers describe gut status, not disease course. Another study examines whether microbiome markers predict clinical success. Whether these changes reverse after reintroduction has not been tested.

How robust is the evidence base for irritable bowel syndrome?

Randomised controlled trials and a meta-analysis support symptom reduction in irritable bowel syndrome, so the effect on reported symptoms is considered established. Robustness pertains to subjective reports, not objective outcome measures. Comparability is limited by differing restriction protocols, durations, and assessment tools. The next step is to determine whether these tools can be standardised to enable cross-study synthesis.

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Research, put in context. Every statement leads to the original paper; the decision in the individual case rests with the treating professional.
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