

Review

Dietary and Medical Management of Small-Intestinal Bacterial Overgrowth: A Narrative Review

Daniel J. Griffith ^{1,2,*} , Stephen Ardouin ^{1,2}, Laura Cramp ¹  and Sheldon C. Cooper ^{2,3} 

¹ Dietetics Department, Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham B15 2GW, UK

² Intestinal Failure Team, Department of Gastroenterology, Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham B15 2GW, UK

³ School of Infection, Inflammation and Immunology, University of Birmingham, Birmingham B15 2TJ, UK

* Correspondence: dan.griffith@uhb.nhs.uk

Abstract

Small-intestinal bacterial overgrowth (SIBO) is defined by an excessive microbial presence in the small intestine and is associated with a range of gastrointestinal symptoms. Its management remains complex due to diagnostic limitations and high recurrence rates following treatment. A narrative review was conducted using MEDLINE (PubMed) and Cochrane databases to identify relevant studies published between 1984 and 2024. Search terms included small intestinal bacterial overgrowth SIBO, FODMAP, probiotics, prebiotics, synbiotics, and low-carbohydrate diets. Reference lists were also screened for additional studies. Antibiotics, particularly rifaximin, are commonly used for SIBO treatment but recurrence is frequent. Dietary strategies, such as low-FODMAP and low-carbohydrate diets, may help reduce symptoms, especially in patients with complications like D-lactic acidosis. Evidence for biotic agents (probiotics, prebiotics, synbiotics) is mixed, with limited high-quality studies and inconsistent outcomes. Some probiotic strains show symptom improvement, but effects on breath-test results are variable. A tailored, multidisciplinary approach combining dietary and medical therapies may offer optimal symptom control in SIBO. However, heterogeneity in study designs and limited evidence highlight the need for further research to inform standardised, evidence-based clinical practice.

Keywords: small intestinal bacterial overgrowth; SIBO; antibiotics; FODMAPS; prebiotics; probiotics



Academic Editor: Bahram H. Arjmandi

Received: 6 October 2025

Revised: 17 November 2025

Accepted: 27 January 2026

Published: 6 February 2026

Copyright: © 2026 by the authors.

Licensee MDPI, Basel, Switzerland.

This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.

1. Introduction

Small-intestinal bacterial overgrowth (SIBO) is characterised by an abnormal increase in the population of bacteria in the small intestine—a deviation from the normal microbial balance that can result in a spectrum of gastrointestinal and systemic symptoms [1–3]. The condition has been linked to various underlying disorders, and its pathophysiology is complex, involving both disrupted motility and changes in the luminal environment that favour bacterial proliferation [1–3]. A significant clinical challenge in SIBO management is establishing a reliable diagnosis due to overlapping symptoms with other gastrointestinal conditions, as well as the limitations of existing diagnostic tests.

In recent years, considerable attention has been focused on the interplay between dietary factors and the gut microbiota. Specific dietary interventions, such as biotic agents (pre- and probiotics), low-Fermentable Oligosaccharides, Disaccharides, Monosaccharides and Polyols (FODMAP), and low-carbohydrate regimens, have been investigated for their

potential to reduce bacterial fermentation and alleviate clinical symptoms [4,5]. Furthermore, complications such as D-lactic acidosis, which can arise from excess carbohydrate fermentation [6], underscore the importance of tailored dietary strategies in susceptible patient populations, particularly those with short-bowel syndrome (SBS) or who have undergone bariatric surgery. Alongside dietary management, the medical treatment of SIBO, predominantly through antibiotics, is being revisited with emerging evidence supporting the adjunctive use of prebiotics, probiotics, and synbiotics to restore microbial balance and ameliorate symptoms [7–9].

This review aims to provide a comprehensive overview of current strategies in the dietary and medical management of SIBO. Through an extensive literature search and analysis of both clinical studies and case reports, the effectiveness and limitations of these interventions, with particular focus on how they influence nutritional status, recurrence risk, and symptom control, was explored. Given the marked heterogeneity in diagnostic criteria, study populations, and intervention protocols, a narrative review approach was chosen to allow a broad, clinically oriented synthesis rather than a narrowly defined systematic review or meta-analysis.

2. Materials and Methods

All authors searched for the relevant research conducted in patients with SIBO. The online databases MEDLINE (Pubmed) and Cochrane were searched for eligible studies. Key search terms, including medical subject headings (MeSH) terms, used were small intestinal bacterial overgrowth, SIBO, bacterial translocation, blind-loop syndrome, FODMAP, probiotics, prebiotics, synbiotics, fermented foods, d-lactic acidosis, and low carbohydrate. Publications published between 1984 and 2025 were included, and we last completed a literature search in August 2025. Each author was assigned a research section of this article, depending on their expertise, and articles were not cross-checked by the other authors. All studies underwent a preliminary screening by reviewing their titles and abstract. Full-text articles were then reviewed prior to inclusion. Additional papers were included through identifying relevant titles in included studies' reference lists to ensure appropriate studies were sourced and included. The literature quality was considered but due to a small body of research, the authors included any relevant papers to summarise the evidence.

3. Results

3.1. Diagnosis of SIBO

Establishing a diagnosis of SIBO originates with clinical suspicion leading to investigations. Unfortunately, there is no pathognomonic symptom for SIBO, and it overlaps commonly with many gastrointestinal disorders. Such symptoms may include bloating, increase flatus, change in bowel habit to diarrhoea, abdominal discomfort, and, rarely, malabsorption can be detected, but this may be associated with other underlying conditions predisposing individuals to SIBO; for example, structuring active Crohn's disease. An index of suspicion will be raised with certain comorbidities such as small-bowel diverticulosis, systemic sclerosis, altered gastrointestinal anatomy post-surgery (classically Billroth gastrectomy, but any surgery leading to a blind loop), and diabetes mellitus to name some [1,2,10,11].

The current 'gold-standard' diagnostic test is the culture of small-bowel aspirates. Some guidance will recommend a diagnosis if $\geq 10^5$ colony-forming units (CFU)/mL, but this is only commonly seen in blind-loop syndromes such as Billroth gastrectomy [10]. Many studies and Delphi agreements would accept $\geq 10^3$ CFU/mL [1–3,10]. This test, however, is not always available and is invasive in nature, requiring stringent techniques to prevent false-positives [3,12]; it also has the potential for false-negative results if the

SIBO is in the distal small bowel rather than proximal and thus is beyond the reach of the endoscope. Breath tests such as hydrogen- and methane breath-tests are less invasive, and (preferred over lactulose due to improved sensitivity and diagnostic yield) a rise in hydrogen or methane above a set threshold would indicate the presence of SIBO. There is a false negative rate with such tests, and altered anatomy can make graphic interpretation more challenging. The purpose of this paper is not to focus on the protocols or combinations of breath-testing [11,12]. The use of 16S rRNA sequencing and developing a fundamental knowledge of the interaction of not only host and microbiota but also between microbes will be key in the future understanding of SIBO and its pathophysiology and management [13].

For the purposes of this review, we use the term “SIBO” to encompass studies that defined overgrowth either by jejunal aspirate culture (typically $\geq 10^3$ colony-forming units/mL, with higher thresholds reserved for blind-loop syndromes) or by a positive hydrogen- and/or methane breath test according to the criteria used in each study. This inclusive approach reflects current practice but also recognises important heterogeneity, as cut-off values, choice of substrate (glucose versus lactulose), sampling intervals, and interpretation criteria vary between centres. These limitations should be considered when comparing studies and when interpreting treatment outcomes in routine practice.

3.2. Medical Management of SIBO

Medical management usually focuses on a reduction in the bacterial load with antibiotic therapy. The choice of antibiotic is, in part, driven by those that will work on the bacteria commonly found in excess in the gut, but also by the side-effect profile, the systemic absorption, the recurrence of overgrowth, and the risk of resistance. Thus, many guidance documents recommend the use of rifaximin as a non-absorbed antibiotic which thus has a significantly reduced side-effect profile compared with other antibiotics [1,10]. Others, such as ciprofloxacin, are no longer recommended as a first-line therapy due to significant side-effect risks such as tendon rupture [14]. Culture and sensitivity of aspirates may direct therapy. A repeated need for antibiotics is not uncommon, and the rotation of antibiotics may reduce the development of resistance. Antibiotic stewardship is the responsibility of all, so seeking other measures is important. PPI use is associated with the development of SIBO; the stopping of proton-pump inhibitors may confer an advantage with the return of gastric acid to destroy any ingested bacteria, but in itself may not be enough to treat and remains without “concrete evidence” [10]. Hence, dietary solutions are sought.

3.3. Dietary Management of SIBO

The presence of SIBO may lead to food avoidance and dietary restrictions from the concern of exacerbating symptoms. Ultimately, this can have an impact on nutritional status and result in weight loss, micronutrient deficiency, and malnutrition [15].

There is limited available evidence to determine the most effective diet, or nutritional therapy for patients with SIBO. Clinicians should consider the underlying cause of SIBO, for instance, anatomy, medications, or dysmotility, and then manage the root cause. Dietary advice should be provided in addition to medical management, as there is no evidence to suggest that nutritional therapy alone will treat SIBO. Patients should be closely monitored by a dietitian to limit unnecessary dietary restrictions, dysbiosis, and nutritional deficiency. An overview of nutritional interventions in the management of SIBO is discussed below.

3.3.1. Low-FODMAP Diet

A diet low in FODMAPs may be recommended to patients with SIBO as a second-line therapy [16]. FODMAPs are a collection of short-chain carbohydrates that are poorly absorbed in the small intestine, with subsequent fermentation by gut bacteria in the large

bowel. This process leads to gastrointestinal symptoms through an increase in fluid influx into the bowel and gas production. The low-FODMAP diet is designed for patients with irritable bowel syndrome (IBS), and there are no UK guidelines recommending the low-FODMAP diet for SIBO. The low-FODMAP diet may provide symptomatic relief from gas, bloating, abdominal pain, and diarrhoea. Indeed, a large meta-analysis reported 50 to 86 per cent symptomatic relief for patients [17]. The low-FODMAP diet involves the hydrolysis of carbohydrate bonds; the more fermentable a carbohydrate, the more carbon bonds that are present, leading to increased luminal water and gas, causing abdominal pain, diarrhoea, and bloating. However, as the low-FODMAP diet relies on symptom reporting, this is subject to bias from the placebo effect. Furthermore, the low-FODMAP diet should be followed under the supervision of a qualified dietitian [18] and should not be followed long-term due to the risk of dysbiosis secondary to a reduction in bifidobacteria [5].

Theoretically, the low-FODMAP diet could be applied to patients with short-bowel syndrome with a colonic anastomosis. This anatomy increases the transit time of food into the colon, reducing the time for carbohydrate digestion. When partially digested carbohydrates enter the colon and microbial fermentation occurs, flatulence, bloating, and diarrhoea can increase. The low-FODMAP diet reduces the amount of fermentable carbohydrates digested and, in turn, microbial fermentation. In theory, this may provide symptomatic relief.

Slow-transit constipation is multifactorial, but a low-FODMAP diet may be beneficial where there is an abundance of methane production because of intestinal fermentation [19,20]. Methane is produced by small-bowel anaerobic archaea in 30 to 62 per cent of humans because of fermentation [21]. Although archaea are single-celled organisms and not bacteria, they may stimulate bacterial overgrowth by contributing to increased transit time in the small bowel. There is evidence to support increased transit time contributing to the development of SIBO in up to 76 per cent of cases [22].

FODMAPs are fermented more rapidly due to their short chain length, yielding larger quantities of gas over a short period of time [23], which can drive methane production from hydrogen and carbon dioxide. However, no significant difference in methane-gas production between the low-FODMAP diet or high-FODMAP diet was observed in a small study [24].

A low-FODMAP diet has been suggested, as theoretically it can reduce short-chain fatty-acid production, leading to increased colonic transit time; this could counteract the benefits of reduced methane production [22]. More research is needed in this area to assess our understanding of methane production, transit, and the low-FODMAP diet.

A reduction in gas production could indicate a reduction in bacterial overgrowth. A controlled, single-blind study compared hydrogen breath-tests on a low-FODMAP diet at three weeks versus a high-FODMAP diet at three weeks [25]. The hydrogen breath test showed a minor decrease in hydrogen production, but no reduction in methane production. The participants in the study met the Rome III criteria for IBS but were not tested for SIBO. Despite this, research suggests up to 78 per cent of patients with IBS have SIBO [4]. Therefore, a reduction in hydrogen- and methane production would be expected if the low-FODMAP diet was to treat SIBO [20].

3.3.2. Elemental Feeding

Elemental feeding is a dietary treatment method in which nutrients are provided in a pre-digested liquid form that can be taken by mouth or via the enteral route. This therapeutic dietary treatment contains no fermentable carbohydrates and can modulate the gut microbiome. Hence, this has attracted recent research attention in the management of SIBO. One study of 93 participants with IBS and abnormal breath-tests had a nutritionally

complete elemental, low-fat liquid-based feed, 6% Kcal from fat (Nestle® Vivonex plus), for 14 days [26]. The quantity administered was based on individual nutritional caloric requirements, and after 2 weeks of this treatment, 80% had a normalised lactulose breath-test. There was a statistically significant improvement in bowel symptoms of those who normalised their breath-test result in comparison to those who did not. Another study included 30 participants with a positive lactulose breath test and/or intestinal methanogen overgrowth [27]. Participants had two weeks of exclusive oral elemental nutrition (mBiota Elemental, 2.5% kcal from fat). After the two weeks of this treatment, 73% had normalised their lactulose breath-tests, and adequate global relief of symptoms was reported in 83%.

There are known limitations of an elemental therapeutic diet, including palatability and compliance. Given only a few studies are reported, the routine use of this diet for SIBO cannot be determined.

3.3.3. Low-Carbohydrate Diet

D-lactic acidosis (DLA) is a rare but significant metabolic disorder observed in patients with SIBO, particularly those with SBS or after bariatric surgery [6]. D-lactate-producing bacteria include *Lactobacillus* and *Streptococcus* species, which ferment unabsorbed carbohydrates into D-lactate. Unlike L-lactate, which is efficiently metabolised in humans, D-lactate accumulates and contributes to neurotoxicity, leading to symptoms such as ataxia, slurred speech, confusion, and encephalopathy [28,29]. The clinical presentation is typically similar to alcohol intoxication, manifesting as ataxia, dysarthria, aggression, difficulty concentrating, memory and coordination disturbances, confusion, reduced consciousness, and even coma. Episodes can recur, with each lasting from a few hours to several days [29].

An earlier review of DLA identified only 32 case reports in the literature, and not all of these discuss dietary manipulation [6]. As DLA is rare, dietary treatment is based off this limited, case-based evidence, rather than larger controlled trials, so bias is inherently high.

Dietary carbohydrate restriction is typically recommended for treatment of DLA. Studies indicate that a high-carbohydrate diet exacerbates symptoms by increasing D-lactate production in the gut [30]. Several case studies have confirmed this. For example, the precipitation of confirmed DLA in a patient with intestinal bypass surgery occurred by providing a significant amount of calories (6000 kcal), containing 54% carbohydrates [31]. In a paediatric case study with short-bowel syndrome, nutrition support was provided through nasogastric feeding in which the carbohydrate source of the complete enteral feed was glucose oligosaccharides [32]. Once energy provided through this enteral feed exceeded 30% of nutritional requirements, DLA was induced [32].

Conversely, a low-carbohydrate diet (CHO constituting <10% of total caloric intake) significantly reduced D-lactate levels in both serum and urine, thereby mitigating acidosis and neurological symptoms in an adult case study with SBS [28]. In the same case study, a fasting regimen was also effective in reducing systemic D-lactate accumulation. This has been replicated in a more recent case study whereby a patient with confirmed DLA following bariatric surgery was initially restricted in carbohydrate intake. It is not clear as to what level restriction was applied, although the authors recommend restricting to <10% of intake [33]. Interestingly, after a week of concomitant medical therapy (IV sodium bicarbonate and ciprofloxacin 500 mg twice daily for a week due to pyrexia and increased stool frequency), the patient was provided with 1485 kcal per day in divided meals with almost 50% carbohydrates (22.6% proteins, and 27.9% fats), and no episodes of encephalopathy were observed in the week following this meal plan [33].

Recent studies have explored the efficacy of replacing fermentable sugars with fructose to mitigate D-lactate production. Another case study in a paediatric patient trialled substituting 25% of enteral nutrition with a fructose-based formula and as a result eliminated

recurrent DLA episodes [29]. This is attributed to the distinct metabolic pathway of fructose absorption. Fructose is absorbed via passive diffusion whereas glucose is absorbed via active transport through sodium–glucose co-transporters [29]. It has been hypothesised that fructose may be better absorbed because bacterial overgrowth in the small intestine can lead to deconjugation of bile acids, causing inflammation and damage to the intestinal mucosa. This impairs active transport mechanisms, which results in poor absorption of glucose and sucrose, which then reach the colon and undergo bacterial fermentation into lactate [29]. As fructose is absorbed through facilitated diffusion, it bypasses this issue [29]. This is the only case report, to the authors' knowledge.

Long-term dietary management of DLA is not well-explored. However, it has been postulated that high-fat, low-carbohydrate diets may shift gut microbiota composition away from lactate-producing bacteria [34]. However, in patients with SBS with a colon in continuity, an increased risk of calcium oxalate renal stones occurs from a high-fat diet [35], and, therefore, careful consideration of this dietary strategy should be approached on an individual basis. Due to the lack of robust clinical trials, it is unclear which dietary strategy is best to treat DLA. The optimal percentage of dietary intake of carbohydrates, type of carbohydrate, feeding route, and duration are all unclear and have varied in all the identified case studies.

3.4. *Prebiotics, Probiotics, and Synbiotics*

Another dietary intervention for SIBO is the use of biotic agents; prebiotics, probiotics, and synbiotics. This group of agents may be able to oppose antibiotic-driven dysbiosis and bacterial overgrowth by modulating gut microbiota and alleviating symptoms [7,8]. They are well-defined elsewhere, although the precise mechanisms of action remain uncertain [8,36,37].

3.4.1. *Prebiotics*

Prebiotics are not widely researched as a treatment for SIBO. An outdated review alluded to the scarcity of evidence for their use, whether administered alone or with a probiotic (a synbiotic) [36]. The American College of Gastroenterology in 2020 released a clinical guideline which stated that prebiotics should be avoided in the management of SIBO as the diet-manipulation strategy needs a reduction in fermentable products [10].

Few clinical studies are available, and results remain inconclusive. In a group of 77 patients with a positive glucose breath-test, rifaximin and partially hydrolysed guar gum that was administered for 10 days resulted in a significantly higher eradication rate of SIBO (versus rifaximin alone) [38]. Clinical symptoms were, however, not statistically different. Conversely, in a smaller group of patients with positive lactulose- and glucose breath-tests, significant symptom improvements were found after six months of administration of short-chain fructo-oligosaccharide and rifaximin [9]. Nausea and daily bowel movements were, however, unchanged, and overall, the changes were not statistically different to the alternative probiotic-taking group [9]. A third study found that 6 weeks of a low-FODMAP diet and partially hydrolysed guar gum administration had no statistical impact on the hydrogen breath test in comparison to an antibiotic therapy and low-FODMAP diet [39].

3.4.2. *Synbiotics*

In the 2020 consensus statement, The International Scientific Association for Probiotics and Prebiotics (ISAPP) outlined the definition of “synbiotic” as “a mixture comprising live microorganisms and substrate(s) selectively utilized by host microorganisms that confers a health benefit on the host” [37]. In essence, they are a combination of probiotics and prebiotics.

From our review, only one paper included a synbiotic intervention. This double-blind randomised study assessed the efficacy of lactol (*Bacillus coagulans* spores and Fructo-oligosaccharides), in conjunction with antibiotics for SIBO-treatment of 30 individuals [40]. After 6 months, several symptoms had significant improvements from the intervention. Hydrogen breath-test results were, however, not significantly different (93.3% negative in intervention group vs. 66.7% in the antibiotic group) [40]. Although this is the only paper found, it employed a robust empirical approach to the intervention [7]; however, it is not yet enough to inform clinical practice.

3.4.3. Probiotics

Probiotics are by far the most researched biotic agent for SIBO treatment, although their effectiveness remains inconclusive [41,42]. All in all, the available body of research presents mixed outcome measures, small sample-sizes with varying comorbidities, and low-quality methodology from which to draw conclusions.

In an open clinical trial of 40 patients with SIBO and systemic sclerosis, *Saccharomyces boulardii* in combination with metronidazole (or as a monotherapy) more effectively eradicated SIBO than metronidazole solely [43]. Gastrointestinal symptoms were also reduced from the addition of probiotics, while remaining unchanged when no probiotic was given. Another open label trial of five patients with SIBO and IBS found significant improvements to questionnaire scores of satisfaction and IBS symptoms after 30 days of a mixed probiotic (Lactolevure) [44].

In a small double-blind study on patients with gastric- or colorectal cancer, the effect of *Bifidobacterium* triple versus a placebo on SIBO was assessed after four weeks. The intervention resulted in a reduced hydrogen breath-test and significant improvement in gastrointestinal cancer-related symptoms [45].

Interestingly, symptom alleviation and breath-test improvements are opposed in other studies. *Saccharomyces boulardii* did not significantly improve stool frequency or the hydrogen breath-test in comparison to the placebo in a small cohort [46]. Another small cross-sectional study found that a probiotic-containing yoghurt (*Lactobacillus johnsonii* La1) administered to elderly residents for four weeks had no impact on positive breath-test results [47]. *Lactobacillus fermentum* was also found to have no significant impact on the hydrogen breath-test, stool frequency, or symptoms in a double-blind cross-over study [48].

More recently, a randomised clinical trial co-administered an antibiotic therapy with *Saccharomyces boulardii*. For 6 weeks after antibiotic therapy, *Bifidobacterium longum* supplementation, L-glutamine, and a low-FODMAP diet was enforced [39]. These intervention strategies did not significantly improve the methane breath-test results in comparison to the “control” (antibiotic therapy and a low-FODMAP diet) [39].

We note several other review publications in the available body of research. A 2017 meta-analysis and systematic review of probiotics in the treatment and prevention of SIBO identified that most studies are small and lack high-quality methodology, although some results were deemed promising [49]. This notion is shared by others [7,8] whereby study limitations are outlined while there is acknowledgement of the therapeutic role of probiotics that has been found in clinical papers. Specific to the bariatric-surgery population, a systematic review and meta-analysis found no significant benefits of probiotics in the treatment of SIBO [50]. More robust large-scale research is needed to suggest routine use of probiotic administration in the management of SIBO, and no particular biotics strain is an exception.

4. Discussion

This narrative review highlights that, despite growing clinical interest, there is still no single dietary or adjunctive strategy that can be recommended as routine first-line management for SIBO. The evidence-base is characterised by small sample sizes, heterogeneous populations (e.g., IBS, SBS, post-surgical cohorts), variable diagnostic methods (culture vs. glucose/lactulose breath testing; hydrogen vs. methane endpoints), and inconsistent outcome measures (symptoms vs. test normalisation). These methodological limitations complicate comparisons across studies and likely explain the mixed findings observed across interventions.

From a medical-management perspective, antibiotics remain the most commonly used therapy, with non-absorbed agents such as rifaximin frequently preferred due to a favourable side-effect profile [1,2]. However, high recurrence rates and antimicrobial-stewardship concerns underscore the importance of complementary strategies and addressing contributory factors such as PPI exposure and dysmotility wherever possible. A pragmatic approach is likely best: use antibiotics judiciously, seek modifiable drivers (e.g., medications, anatomical issues), and integrate dietetic input to minimise unnecessary restriction and risk of nutritional deficiency and/or malnutrition.

Dietary interventions show promise for symptom control in carefully selected patients, but the data remain inconclusive. A low-FODMAP diet, well-established in IBS, has theoretical and limited empirical support for reducing fermentation-related symptoms in SIBO; however, results are mixed, methane responses are inconsistent, and long-term use risks dysbiosis [5,24]. Consequently, if trialled, it should be short-term, dietitian-led, and embedded within a broader plan that prioritises nutritional adequacy and subsequent reintroduction of FODMAP-containing foods. Elemental feeding has produced encouraging short-term normalisation of breath tests and symptom improvements in small studies [26,27], but issues of palatability, adherence, and sparse high-quality trials preclude routine-use. Targeted low-carbohydrate strategies are particularly relevant for D-lactic acidosis in SBS or post-bariatric populations, where reducing fermentable substrates may mitigate neurological episodes [28,33]. However, optimal carbohydrate thresholds, formulation choices, and long-term safety require further investigation.

When these dietary approaches are considered together, it is clear that each offers potential benefits but also important trade-offs. Low-FODMAP diets may be a pragmatic option for short-term symptom relief, particularly in patients with overlapping IBS-type features, yet current data provide limited support for sustained breath-test normalisation and prolonged restriction risks adverse changes in the gut microbiota and overall nutritional status. Elemental diets appear to have the most consistent short-term effects on both symptoms and breath-test outcomes, but there are very few studies to support this. Additionally, cost, palatability, and the need for close supervision further limit use to selected patients under specialist care. Targeted low-carbohydrate regimens are especially relevant for patients with short bowel or D-lactic acidosis, but high-fat formulations may increase the risk of enteric hyperoxaluria and renal-stone formation in certain patients [35].

Biotic agents (prebiotics, probiotics, synbiotics) illustrate the broader evidence challenges. While some trials report symptom benefits and improved breath-tests, many are underpowered, open-label, or include mixed comorbidities. Prebiotics are sparsely studied and may theoretically aggravate fermentation in active SIBO [10]. Indeed, one small trial suggests a potential adjunctive role when combined with rifaximin, but findings are not uniform [38]. Probiotics have yielded variable outcomes. Some appear to produce strain-specific benefits whereas others produce neutral results [39,41–48]. This highlights that effects are likely contingent on strain, dose, host phenotype, and concurrent therapies. A single synbiotic study reported symptom gains without clear superiority on breath

testing [40]. Collectively, current data do not support the use of biotic agents routinely; however, they may be used cautiously in some individuals on a case-by-case basis and careful selection of the patient.

Future research should address several specific gaps: (1) Adequately powered randomised controlled trials (RCTs) (or meta-analysis of smaller-scale RCT's, as the number of patients with SIBO is relatively low) comparing dietary strategies (low-FODMAP, elemental, targeted low-carbohydrate) with standard medical care; (2) standardised diagnostic and outcome frameworks that incorporate both symptom and physiological endpoints (including methane and hydrogen sulphide where relevant); (3) safety, adherence, and microbiome impacts of longer-term dietary manipulation; and (4) integration of modern microbiome profiling (e.g., 16S rRNA sequencing) to clarify mechanisms and guide personalised nutrition. Until such data are available, a multidisciplinary, individualised, and nutrition-sparing approach remains the most pragmatic pathway to optimise outcomes in SIBO.

This review has several strengths and limitations that should be acknowledged. Our focus on adult patients with SIBO or D-lactic acidosis in clinically relevant conditions (such as IBS, short-bowel syndrome, systemic sclerosis, and post-surgical states) allows a pragmatic synthesis that integrates dietary and medical management. Nevertheless, as a narrative review restricted to English-language, peer-reviewed human studies, it is subject to selection and publication bias, and the small size and heterogeneity of many studies (in terms of diagnostic criteria, breath-test protocols, and outcome measures) preclude formal meta-analysis. Rare phenotypes (for example, hydrogen sulphide SIBO) and paediatric data are under-represented. These constraints reinforce the need for cautious interpretation of the available evidence.

In clinical practice, management is best delivered via a stepwise, multidisciplinary approach that first identifies and addresses underlying contributors (including structural abnormalities, motility disorders, and medication effects), then uses short, clearly defined courses of antibiotics only where indicated, and incorporates time-limited, dietitian-led dietary trials that prioritise nutritional adequacy and planned reintroduction rather than prolonged restriction

5. Conclusions

Current evidence does not support any single dietary strategy as a routine first-line therapy for SIBO. Small trials, heterogeneous cohorts, and inconsistent diagnostic/outcome measures limit firm recommendations. Physicians should begin with identifying and addressing underlying causes (e.g., dysmotility, medications, post-surgical anatomy), using antibiotics judiciously and integrating dietetic care rather than relying on nutrition alone. Patients appear to benefit most from short, dietitian-led dietary trials (e.g., low-FODMAP, elemental, targeted low-carbohydrate in selected phenotypes) that prioritise nutritional adequacy, avoid unnecessary restriction, and are followed by planned reintroduction of any restricted foods/nutrients. Biotic agents show mixed, strain-, and context-dependent effects and should not be recommended universally. Above all, preventing and treating malnutrition outweighs any theoretical benefit of prolonged dietary restriction.

Author Contributions: Conceptualization, D.J.G. and S.C.C.; methodology, D.J.G., S.A., L.C. and S.C.C.; data curation, D.J.G., S.A., L.C. and S.C.C.; writing—original draft preparation, D.J.G., S.A., L.C. and S.C.C.; writing—review and editing, D.J.G.; supervision, S.C.C. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Data Availability Statement: No new data were created or analysed in this study. Data sharing is not applicable to this article.

Acknowledgments: We would like to thank Sophie Turigel (Dietitian) for her assistance in gathering additional research papers relating to the low-FODMAP diet.

Conflicts of Interest: S.C.C. has received an honorarium from Takeda UK. D.J.G. has received honoraria from Nutricia and Fresenius Kabi.

Abbreviations

The following abbreviations are used in this manuscript:

SIBO	Small-Intestinal Bacterial Overgrowth
FODMAP	Fermentable Oligosaccharides, Disaccharides, Monosaccharides, and Polyols
DLA	D-Lactic Acidosis
SBS	Short-Bowel Syndrome
ISSAP	The International Scientific Association for Probiotics and Prebiotics
CFU	Colony-Forming Units
IBS	Irritable Bowel Syndrome
RCT	Randomised Controlled Trials

References

1. Ghoshal, U.C.; Sachdeva, S.; Ghoshal, U.; Misra, A.; Puri, A.S.; Pratap, N.; Shah, A.; Rahman, M.M.; Gwee, K.A.; Tan, V.P.Y.; et al. Asian-Pacific consensus on small intestinal bacterial overgrowth in gastrointestinal disorders: An initiative of the Indian Neurogastroenterology and Motility Association. *Indian J. Gastroenterol.* **2022**, *41*, 483–507. [\[CrossRef\]](#)
2. Losurdo, G.; D'Abramo, F.S.; Indelicati, G.; Lillo, C.; Ierardi, E.; Di Leo, A. The Influence of Small Intestinal Bacterial Overgrowth in Digestive and Extra-Intestinal Disorders. *Int. J. Mol. Sci.* **2020**, *21*, 3531. [\[CrossRef\]](#)
3. Rao, S.S.C.; Bhagatwala, J. Small Intestinal Bacterial Overgrowth: Clinical Features and Therapeutic Management. *Clin. Transl. Gastroenterol.* **2019**, *10*, e00078. [\[CrossRef\]](#)
4. Ghoshal, U.C.; Shukla, R.; Ghoshal, U. Small intestinal bacterial overgrowth and irritable bowel syndrome: A bridge between functional organic dichotomy. *Gut Liver* **2017**, *11*, 196–208. [\[CrossRef\]](#) [\[PubMed\]](#)
5. Wielgosz-Grochowska, J.P.; Domanski, N.; Drywień, M.E. Efficacy of an Irritable Bowel Syndrome Diet in the Treatment of Small Intestinal Bacterial Overgrowth: A Narrative Review. *Nutrients* **2022**, *14*, 3382. [\[CrossRef\]](#) [\[PubMed\]](#)
6. Kowlgi, N.G.; Chhabra, L. D-Lactic Acidosis: An Underrecognized Complication of Short Bowel Syndrome. *Gastroenterol. Res. Pract.* **2015**, *2015*, 476215. [\[CrossRef\]](#)
7. Chen, W.C.; Quigley, E.M.M. Probiotics, prebiotics & synbiotics in small intestinal bacterial overgrowth: Opening up a new therapeutic horizon! *Indian J. Med. Res.* **2014**, *140*, 582–584.
8. Martyniak, A.; Wójcicka, M.; Rogatko, I.; Piskorz, T.; Tomasik, P.J. A Comprehensive Review of the Usefulness of Prebiotics, Probiotics, and Postbiotics in the Diagnosis and Treatment of Small Intestine Bacterial Overgrowth. *Microorganisms* **2025**, *13*, 57. [\[CrossRef\]](#) [\[PubMed\]](#)
9. Rosania, R.; Giorgio, F.; Principi, M.; Amoroso, A.; Monno, R.; Di Leo, A.; Ierardi, E. Effect of Probiotic or Prebiotic Supplementation on Antibiotic Therapy in the Small Intestinal Bacterial Overgrowth: A Comparative Evaluation. *Curr. Clin. Pharmacol.* **2013**, *8*, 169–172. [\[CrossRef\]](#)
10. Pimentel, M.; Saad, R.J.; Long, M.D.; Rao, S.S.C. ACG Clinical Guideline: Small Intestinal Bacterial Overgrowth. *Am. J. Gastroenterol.* **2020**, *115*, 165–178. [\[CrossRef\]](#)
11. da Silva, B.C.; Ramos, G.P.; Barros, L.L.; Ramos, A.F.P.; Domingues, G.; Chinzon, D.; Passos, M.d.C.F. Diagnosis and Treatment of Small Intestinal Bacterial Overgrowth: An Official Position Paper from The Brazilian Federation of Gastroenterology. *Arq. Gastroenterol.* **2025**, *62*, e24107. [\[CrossRef\]](#)
12. Hammer, H.F.; Fox, M.R.; Keller, J.; Salvatore, S.; Basilisco, G.; Hammer, J.; Lopetuso, L.; Benninga, M.; Borrelli, O.; Dumitrascu, D.; et al. European guideline on indications, performance, and clinical impact of hydrogen and methane breath tests in adult and pediatric patients: European Association for Gastroenterology, Endoscopy and Nutrition, European Society of Neurogastroenterology and Motility, and European Society for Paediatric Gastroenterology Hepatology and Nutrition consensus. *United Eur. Gastroenterol. J.* **2022**, *10*, 15–40. [\[CrossRef\]](#)
13. Bushyhead, D.; Quigley, E.M.M. Small Intestinal Bacterial Overgrowth—Pathophysiology and Its Implications for Definition and Management. *Gastroenterology* **2022**, *163*, 593–607. [\[CrossRef\]](#)

14. Medicines and Healthcare Products Regulatory (MHRA) Agency. Fluoroquinolone antibiotics: Must now only be prescribed when other commonly recommended antibiotics are inappropriate. *Drug Saf. Update* **2024**, *17*, 2.
15. Adike, A.; DiBaise, J.K. Small Intestinal Bacterial Overgrowth. *Gastroenterol. Clin. N. Am.* **2018**, *47*, 193–208. [\[CrossRef\]](#) [\[PubMed\]](#)
16. Lauritano, E.C.; Gabrielli, M.; Scarpellini, E.; Lupascu, A.; Novi, M.; Sottili, S.; Vitale, G.; Cesario, V.; Serricchio, M.; Cammarota, G.; et al. Small Intestinal Bacterial Overgrowth Recurrence After Antibiotic Therapy. *Am. J. Gastroenterol.* **2008**, *103*, 2031–2035. [\[CrossRef\]](#) [\[PubMed\]](#)
17. Marsh, A.; Eslick, E.M.; Eslick, G.D. Does a diet low in FODMAPs reduce symptoms associated with functional gastrointestinal disorders? A comprehensive systematic review and meta-analysis. *Eur. J. Nutr.* **2016**, *55*, 897–906. [\[CrossRef\]](#)
18. National Institute for Health and Care Excellence. Irritable Bowel Syndrome in Adults: Diagnosis and Management Clinical Guideline. 2008. Available online: <http://www.nice.org.uk/guidance/cg61> (accessed on 3 September 2025).
19. Chatterjee, S.; Park, S.; Low, K.; Kong, Y.; Pimentel, M. The Degree of Breath Methane Production in IBS Correlates with the Severity of Constipation. *Am. J. Gastroenterol.* **2007**, *102*, 837–841. [\[CrossRef\]](#)
20. Eetemadi, A.; Tagkopoulos, I. Methane and fatty acid metabolism pathways are predictive of Low-FODMAP diet efficacy for patients with irritable bowel syndrome. *Clin. Nutr.* **2021**, *40*, 4414–4421. [\[CrossRef\]](#)
21. McKay, L.F.; Eastwood, M.A.; Brydon, W.G. Methane excretion in man a study of breath, flatus, and faeces. *Gut* **1985**, *26*, 69–74. [\[CrossRef\]](#)
22. Knez, E.; Kadac-Czapska, K.; Grembecka, M. The importance of food quality, gut motility, and microbiome in SIBO development and treatment. *Nutrition* **2024**, *124*, 112464. [\[CrossRef\]](#)
23. Hernot, D.C.; Boileau, T.W.; Bauer, L.L.; Middelbos, I.S.; Murphy, M.R.; Swanson, K.S.; Fahey, G.C. In vitro fermentation profiles, gas production rates, and microbiota modulation as affected by certain fructans, galactooligosaccharides, and polydextrose. *J. Agric. Food Chem.* **2009**, *57*, 1354–1361. [\[CrossRef\]](#) [\[PubMed\]](#)
24. Ong, D.K.; Mitchell, S.B.; Barrett, J.S.; Shepherd, S.J.; Irving, P.M.; Biesiekierski, J.R.; Smith, S.; Gibson, P.R.; Muir, J.G. Manipulation of dietary short chain carbohydrates alters the pattern of gas production and genesis of symptoms in irritable bowel syndrome. *J. Gastroenterol. Hepatol.* **2010**, *25*, 1366–1373. [\[CrossRef\]](#) [\[PubMed\]](#)
25. McIntosh, K.; Reed, D.E.; Schneider, T.; Dang, F.; Keshteli, A.H.; De Palma, G.; Madsen, K.; Bercik, P.; Vanner, S. FODMAPs alter symptoms and the metabolome of patients with IBS: A randomised controlled trial. *Gut* **2017**, *66*, 1241–1251. [\[CrossRef\]](#)
26. Pimentel, M.; Constantino, T.; Kong, Y.; Bajwa, M.; Rezaei, A.; Park, S. A 14-Day Elemental Diet Is Highly Effective in Normalizing the Lactulose Breath Test. *Dig. Dis. Sci.* **2004**, *49*, 73–77. [\[CrossRef\]](#)
27. Rezaie, A.; Chang, B.W.; de Freitas Germano, J.; Leite, G.; Mathur, R.; Houser, K.; Hosseini, A.; Brimberry, D.; Rashid, M.; Mehravar, S.; et al. Effect, Tolerability, and Safety of Exclusive Palatable Elemental Diet in Patients with Intestinal Microbial Overgrowth. *Clin. Gastroenterol. Hepatol.* **2025**, *23*, 2306–2317.e7. [\[CrossRef\]](#) [\[PubMed\]](#)
28. Ramakrishnan, T.; Stokes, P. Beneficial Effects of Fasting and Low Carbohydrate Diet in D-Lactic Acidosis Associated with Short-Bowel Syndrome. *J. Parenter. Enter. Nutr.* **1985**, *9*, 361–363. [\[CrossRef\]](#)
29. Travieso Suárez, L.; Quijada Fraile, P.; Pedrón Giner, C. Tratamiento dietético con fructosa en una niña de 5 años con acidosis D-láctica recurrente. *Nutr. Hosp.* **2018**, *35*, 495–498. [\[CrossRef\]](#)
30. Kang, K.P.; Lee, S.; Kang, S.K. D-Lactic Acidosis in Humans: Review of Update. *Electrolyte Blood Press.* **2006**, *4*, 53. [\[CrossRef\]](#)
31. Dahlquist, N.R.; Perrault, J.; Callaway, C.W.; Jones, J.D. d-Lactic Acidosis and Encephalopathy After Jejunioileostomy: Response to Overfeeding and to Fasting in Humans. *Mayo Clin. Proc.* **1984**, *59*, 141–145. [\[CrossRef\]](#)
32. Mayne, A.J.; Handy, D.J.; Preece, M.A.; George, R.H.; Booth, I.W. Dietary management of D-lactic acidosis in short bowel syndrome. *Arch. Dis. Child.* **1990**, *65*, 229–231. [\[CrossRef\]](#) [\[PubMed\]](#)
33. Gigante, A.; Sardo, L.; Gasperini, M.L.; Molinaro, A.; Riggio, O.; Laviano, A.; Amoroso, A. D-Lactic acidosis 25 years after bariatric surgery due to *Salmonella enteritidis*. *Nutrition* **2012**, *28*, 108–111. [\[CrossRef\]](#) [\[PubMed\]](#)
34. Bongaerts, G.P.A.; Severijnen, R.S.V.M. Arguments for a lower carbohydrate-higher fat diet in patients with a short small bowel. *Med. Hypotheses* **2006**, *67*, 280–282. [\[CrossRef\]](#)
35. Griffith, D.J.; Cooper, S.C.; Fletcher, J. Dietary and nursing management of calcium oxalate renal stones in patients with a jejunoileocolic anastomosis: A narrative review. *Gastrointest. Nurs.* **2024**, *22*, 278–285. [\[CrossRef\]](#)
36. Quigley, E.M.M.; Quera, R. Small Intestinal Bacterial Overgrowth: Roles of Antibiotics, Prebiotics, and Probiotics. *Gastroenterology* **2006**, *130*, S78–S90. [\[CrossRef\]](#)
37. Swanson, K.S.; Gibson, G.R.; Hutkins, R.; Reimer, R.A.; Reid, G.; Verbeke, K.; Scott, K.P.; Holscher, H.D.; Azad, M.B.; Delzenne, N.M.; et al. The International Scientific Association for Probiotics and Prebiotics (ISAPP) consensus statement on the definition and scope of synbiotics. *Nat. Rev. Gastroenterol. Hepatol.* **2020**, *17*, 687–701. [\[CrossRef\]](#)
38. Furnari, M.; Parodi, A.; Gemignani, L.; Giannini, E.G.; Marengo, S.; Savarino, E.; Assandri, L.; Fazio, V.; Bonfanti, D.; Infrerera, S.; et al. Clinical trial: The combination of rifaximin with partially hydrolysed guar gum is more effective than rifaximin alone in eradicating small intestinal bacterial overgrowth. *Aliment. Pharmacol. Ther.* **2010**, *32*, 1000–1006. [\[CrossRef\]](#)

39. Redondo-Cuevas, L.; Belloch, L.; Martín-Carbonell, V.; Nicolás, A.; Alexandra, I.; Sanchis, L.; Ynfante, M.; Colmenares, M.; Mora, M.; Liebana, A.R.; et al. Do Herbal Supplements and Probiotics Complement Antibiotics and Diet in the Management of SIBO? A Randomized Clinical Trial. *Nutrients* **2024**, *16*, 1083. [\[CrossRef\]](#)
40. Khalighi, A.R.; Khalighi, M.R.; Behdani, R.; Jamali, J.; Khosravi, A.; Kouhestani, S.; Radmanesh, H.; Esmaeelzadeh, S.; Khalighi, N. Evaluating the efficacy of probiotic on treatment in patients with small intestinal bacterial overgrowth (SIBO)—A pilot study. *Indian J. Med. Res.* **2014**, *140*, 604–608.
41. McFarland, L.V. Use of probiotics to correct dysbiosis of normal microbiota following disease or disruptive events: A systematic review. *BMJ Open* **2014**, *4*, e005047. [\[CrossRef\]](#) [\[PubMed\]](#)
42. Plaza-Diaz, J.; Ruiz-Ojeda, F.J.; Gil-Campos, M.; Gil, A. Mechanisms of Action of Probiotics. *Adv. Nutr.* **2019**, *10*, S49–S66. [\[CrossRef\]](#)
43. García-Collinot, G.; Madrigal-Santillán, E.O.; Martínez-Bencomo, M.A.; Carranza-Muleiro, R.A.; Jara, L.J.; Vera-Lastra, O.; Montes-Cortes, D.H.; Medina, G.; Cruz-Domínguez, M.P. Effectiveness of *Saccharomyces boulardii* and Metronidazole for Small Intestinal Bacterial Overgrowth in Systemic Sclerosis. *Dig. Dis. Sci.* **2020**, *65*, 1134–1143. [\[CrossRef\]](#)
44. Leventogiannis, K.; Gkolfakis, P.; Spithakis, G.; Tsatali, A.; Pistiki, A.; Sioulas, A.; Giamarellos-Bourboulis, E.J.; Triantafyllou, K. Effect of a Preparation of Four Probiotics on Symptoms of Patients with Irritable Bowel Syndrome: Association with Intestinal Bacterial Overgrowth. *Probiotics Antimicrob. Proteins* **2019**, *11*, 627–634. [\[CrossRef\]](#) [\[PubMed\]](#)
45. Liang, S.; Xu, L.; Zhang, D.; Wu, Z. Effect of probiotics on small intestinal bacterial overgrowth in patients with gastric and colorectal cancer. *Turk. J. Gastroenterol.* **2016**, *27*, 227–232. [\[CrossRef\]](#)
46. Attar, A.; Flourié, B.; Rambaud, J.C.; Franchisseur, C.; Ruzsniowski, P.; Bouhnik, Y. Antibiotic efficacy in small intestinal bacterial overgrowth—related chronic diarrhea: A crossover, randomized trial. *Gastroenterology* **1999**, *117*, 794–797. [\[CrossRef\]](#)
47. Schiffrin, E.J.; Parlesak, A.; Bode, C.; Christian Bode, J.; van't Hof, M.A.; Grathwohl, D.; Guigoz, Y. Probiotic yogurt in the elderly with intestinal bacterial overgrowth: Endotoxaemia and innate immune functions. *Br. J. Nutr.* **2008**, *101*, 961–966. [\[CrossRef\]](#)
48. Stotzer, P.-O.; Blomberg, L.; Conway, P.L.; Henriksson, A.; Abrahamsson, H. Probiotic Treatment of Small Intestinal Bacterial Overgrowth by *Lactobacillus fermentum* KLD. *Scand. J. Infect. Dis.* **1996**, *28*, 615–619. [\[CrossRef\]](#) [\[PubMed\]](#)
49. Zhong, C.; Qu, C.; Wang, B.; Liang, S.; Zeng, B. Probiotics for Preventing and Treating Small Intestinal Bacterial Overgrowth. *J. Clin. Gastroenterol.* **2017**, *51*, 300–311. [\[CrossRef\]](#) [\[PubMed\]](#)
50. Wagner, N.R.F.; Fernandes, R.; Teixeira Frota Reichmann, M.; Lopes, M.C.P.; Welc, L.L.S.; Campos, A.C.L. Use of Probiotics and Synbiotics in the Treatment of Small Intestinal Bacterial Overgrowth (SIBO) and Other Gastrointestinal Symptoms After Metabolic Bariatric Surgery: A Systematic Review and Meta-Analysis. *Obes. Surg.* **2025**, *35*, 312–321. [\[CrossRef\]](#)

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.