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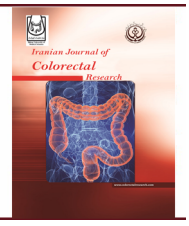

The FODMAP diet for Gut Microbiome Restoration in Patients with Irritable Bowel Syndrome: A Narrative Review

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Abstract

Irritable Bowel Syndrome (IBS) is a multifactorial, debilitating, complex, and chronic disorder of gut-brain interaction. The Gut microbiota and its metabolites play an important role in the etiology, pathogenesis, and pathophysiology of IBS. Dietary modulation of the gut microbiome to correct gut microbiota dysbiosis and restore host-microbe eubiosis is considered an effective strategy for managing IBS. Increasing evidence suggests that a diet low in fermentable oligo-, di-, and monosaccharides, and polyols (FODMAPs) can beneficially modulate the gut microbiota.

The objective of this narrative review is to elucidate how a FODMAP diet impacts the gut microbiome. Understanding how this diet influences gut microbial communities is crucial for understanding the potential long-term health consequences of this dietary regimen. This nuanced understanding can inform the development of optimal, personalized FODMAP dietary strategies tailored to the individual gut microbiome composition and function of IBS patients.

A search was conducted in the electronic databases PubMed/Medline, SCOPUS, and Web of Science for observational studies, clinical trials, systematic reviews, and meta-analyses published between 2012 and 2025. Search terms and keywords included IBS, FODMAP diet, gut microbiome, and gut microbiota.

Contrary to the prevailing belief that a diet low in FODMAPs has detrimental effects on gut health, scientific studies show that such a diet can have beneficial effects on both gut and host health.

The scientific evidence presented in this narrative review supports the FODMAP diet as a personalized precision nutrition therapy that can effectively restore, reinoculate, and rebalance the gut microbiome.

Keywords: Irritable bowel syndrome; Gut microbiota; FODMAP diet; Dysbiosis; Eubiosis; Precision nutrition

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Introduction

Irritable Bowel Syndrome (IBS) is a multifactorial, debilitating, complex, and chronic (i.e., persistent and ongoing) disorder of gut-brain interaction (DGBI) characterized by heterogeneous etiopathogenesis, pathophysiology, and clinical phenotype (1-3).

The enteropathy of IBS manifests through a constellation of symptoms, including: a) abdominal cramps, pain, and discomfort characterized by spasms, burning sensations, and tightness around the umbilicus and lower abdomen, which can be

aggravated or relieved by the emission of gas or stools; b) abnormal gas propulsion and expulsion, flatulence, abdominal bloating, and distension; c) hyperactive peristalsis, defined as the propulsive, rhythmic, wave-like muscle contractions that move food and waste through the intestines while enzymes break down the food; d) abnormalities in stool frequency, form, and/or consistency, ranging from diarrhea (stools that are too soft and/or too frequent) to constipation (infrequent, hard stools, difficult or incomplete bowel movements, straining during defecation, and sensation of incomplete evacuation); e) an urgent need to pass stools and the presence of mucus in stools (1-5).

The concept of DGBIs recognizes the significant array of physiological, microbial, and immune abnormalities involved in the pathophysiology of IBS. It is now well established that the human gut microbiome plays a vital role in the pathogenesis, etiology and pathophysiology of IBS (6-11). A growing body of evidence suggests an association between a perturbed gut microbiome, gut dysbiosis, and disturbances of the diet-microbiome-metabolome axis with the onset, exacerbation, and duration of IBS symptoms (12-15). Perturbations in the gut ecosystem can provoke chronic low-grade mucosal inflammation, epithelial barrier dysfunction, increased mucosal permeability, immune system activation, and immune dysregulation (3, 4, 13, 16, 17), which have been suggested as putative pathogenic mechanisms in IBS (14). Such disturbances can also result in food intolerance, exaggerated sensory and colonic motor responses, and visceral hypersensitivity; additionally, they may affect colonic motility through impairment of the migrating motor complex (MMC) (2, 14, 15, 18). The gut microbiota plays an important role in maintaining the structural integrity of the gastrointestinal mucosal barrier, immunomodulation, nutrient metabolism, and protection against pathogens (19, 20). Therefore, gut microbiota-targeted interventions, including the modulation of the immune response, gut microbiome composition and function, and mitigation of gut barrier dysfunction (16), could represent effective therapeutic approaches to IBS (9).

Diet is a major modulator of gut microbiota composition and structure and can significantly alter gastrointestinal functions, thereby compromising the integrity of the intestinal barrier (6, 14, 17, 21, 22). Nutrients ingested by the host can directly influence the relative and absolute abundance of different microbial communities in the gut, which in turn affect the metabolism, metabolic output, and bioactivity of food-derived compounds (14, 22). The ingested foods and their metabolites can affect gut physiology, intestinal homeostasis, and gut microbiota equilibrium, all of which are relevant to the pathogenesis of IBS (6, 9, 23, 24). Scientific evidence supports the importance of diet in IBS etiopathogenesis (9). Hence, it stands to reason that dietary therapy that rectifies the altered microbiome and metabolome and corrects gut dysbiosis may be an effective IBS management strategy to restores eubiosis, a state of homeostasis characterized by a balanced and healthy gut microbiota, featuring a diverse range of beneficial microbes that work synergistically with the host for optimal well-being (9, 25).

There is increasing evidence that dietary interventions involving fermentable oligo-, di-, and monosaccharides, as well as polyols (FODMAPs) can improve IBS symptoms, particularly through interactions with the gut microbiota (10, 13). However, the effects of a FODMAP diet on the gut microbiota and related markers of intestinal health are not completely elucidated and remain under active investigation. Therefore, the objective of this narrative review is to clarify how a FODMAP diet impacts the gut microbiome. Understanding the effects of FODMAP restriction on gut microbiota could provide valuable insights into the mechanisms underlying symptom improvement in IBS, as well as potential long-term health consequences of this dietary approach. Furthermore, understanding how this diet influences gut microbial communities is crucial for developing optimal, personalized nutritional strategies for IBS patients.

In this review article, we first elucidate the central and peripheral gut-related pathophysiological mechanisms through which FODMAPs impact the pathophysiology of IBS. We then explain the mechanisms by which the FODMAP diet reduces IBS symptoms through its effects on the gut microbiome. Next, we discuss the three-phase implementation of the FODMAP diet and the purported effects of each phase in repairing the gut and restoring microbiota equilibrium. Finally, we evaluate the evidence regarding the effect of the FODMAP diet on the gut microbiota. In doing so, we aim to alleviate concerns about potential adverse effects of the FODMAP diet on gut health. By unraveling the complex interplay between the FODMAP diet, the gut microbiome, and IBS symptomatology, this review aims to pave the way for the development of effective and sustainable personalized dietary strategies tailored to the gut microbiome composition and function of IBS patients.

Statement of Significance

This comprehensive narrative review highlights the interconnections among IBS pathophysiology, the FODMAP diet, and gut microbiota. It offers a new perspective on the complex relationship between the gut microbiome and the FODMAP diet in restoring gut health in IBS patients.

Evidence Acquisition

We searched the electronic databases PubMed/Medline, SCOPUS, and Web of Science for

observational studies, clinical trials, systematic reviews, and meta-analysis on IBS, the FODMAP diet and the effects of FODMAPs and FODMAP reduction on the gut microbiome published between 2012 and 2025. Titles, abstracts, and keywords were searched for variations and combinations of the following terms: FODMAP(s), saccharides, oligosaccharide, disaccharide, monosaccharide, galacto-oligosaccharides, fructan(s), fructose, galactans, lactose, polyol(s), sorbitol, mannitol, xylitol, maltitol, IBS, gut microbiome, and microbiota. Studies were eligible for inclusion if they met the following criteria: (1) studies explicating the mechanisms through which high-FODMAP foods impact the pathophysiology of IBS and the gut microbiome-mediated mechanisms of action of the FODMAP diet to reduce IBS symptoms;

(2) Randomized controlled trials investigating the effects of a low- and high-FODMAP diet on the gut microbiota of IBS patients, with healthy subjects serving as a control group; (3) comparing low-FODMAP diets (LFDs) with habitual or other control diets; (4) reporting outcomes related to gut microbiota composition, abundance, functions, and metabolites; (5) and including adult IBSparticipants. The exclusion criteria were as follows: children and adolescents (<18 years old); pregnant or breastfeeding women; IBS patients with significant clinical comorbidities; case studies or reports; reviews, meta-analyses, and conference abstracts; and letters to the editor.

The Mechanisms of Action of FODMAPs and the FODMAP Diet

FODMAP is an acronym that stands for *fermentable oligo-, di-, and monosaccharides, and polyols.* Fermentable means 'creates gas'. the term Oligosaccharides include: *a)* fructo-oligosaccharides (FOS) and fructans, which are linear or branched oligomers or polymers primarily composed of fructosyl-fructose units, with or without a single glucosyl unit; *b)* galacto-oligosaccharides (GOS), which are short chains of fructose, sucrose, and galactose units, such as galactans, raffinose, stachyose, verbascose, and inulin (26, 27); disaccharides (e.g., lactose, composed of glucose and galactose); monosaccharides (e.g., fructose in excess of glucose); and polyols, which are sugar alcohols, including sorbitol, mannitol, maltitol, xylitol, and isomalt (28-31). FODMAPs are poorly absorbed short-chain carbohydrates in the small intestine due to absent or insufficient hydrolysis, for example, lactose malabsorption or nondigestible oligosaccharides; lack of hydrolase enzymes required to break the bonds in fructan molecules; reduced activity of brush border hydrolases in some individuals, such as lactase deficiency; dependence on simultaneous glucose intake for adequate fructose absorption; passive diffusion of certain monosaccharides and polyols; and the molecular size of polyols being too large for simple diffusion. Additionally, slow and low-capacity transport mechanisms across the epithelium contribute to incomplete absorption, gas formation, and altered colonic motility (4, 28, 32-34). Malabsorption of FODMAPs results in an osmotic effect within the intestinal lumen, attracting water into the bowel and accelerating transit to the cecum (32, 33, 35, 36). Furthermore, FODMAPs move through the gastrointestinal tract, a semipermeable, multilayered ecosystem, and reach the large intestine (colon). There FODMAPs serve as a nutrient source for gut microbes and are rapidly fermented (digested) by gut microbiota, producing gas and other by-products (9, 14, 28, 32, 33, 37). Intestinal fermentation by gut microbes generates hydrogen (H2), hydrogen sulfide (H2S), carbon dioxide (CO2), and methane (CH4) gases (2, 3, 14). When the MMC is impaired, that is, when peristaltic waves are disrupted and the bowel's housekeeping and "cleaning" functions malfunction, food clearance becomes ineffective, allowing microbes to accumulate and proliferate on debris in the small intestine (2, 3). This condition, known as SIBO/SIFO/IMO/ISO, involves the overcolonization of aerobic and anaerobic microbes in the gastrointestinal tract, leading to disproportionate production of hydrogen, hydrogen sulfide and methane gases (2, 3, 14, 22). Consequently, this results in IBS symptoms such as bloating, abdominal distension, diarrhea, or constipation (2, 3, 14). Excessive hydrogen and hydrogen sulfide production by bacteria accelerates gut transit and is associated with diarrhea-predominant IBS-D and mixed IBS-M phenotypes (2, 19, 28, 32, 37-39). Conversely, methane gas produced by archaea slows intestinal transit and motility by augmenting segmental smooth muscle contractile activity in the intestinal wall, resulting in constipation, which is associated with the constipation-predominant IBS-C phenotype (2, 3, 24). The accumulation of liquid and gas distends (stretches) the intestine and stimulates nerves surrounding the digestive organs, which can cause the nerve network to overreact and trigger IBS symptoms. Furthermore, FODMAPs provide a substrate for microbial saccharolytic fermentation, that is, the ability of the colonic microbiota to digest carbohydrates (18) by commensal gut microbes in the digestive tract. This process yields gut microbiota-derived metabolites, including short-chain fatty acids (SCFAs), which are the end products of non-absorbed carbohydrates fermented by obligate anaerobic bacteria in the intestine (11, 28, 40). SCFAs, along with carbon dioxide, methane, hydrogen, and hydrogen sulfide create high osmotic activity, which increases intestinal secretions and water content, resulting in abnormal intestinal motility correlated with the generation and severity of IBS symptoms (31-33, 39). Finally, the effects of high-FODMAP foods in the gastrointestinal tract are exerted not only through fermentation but

also via alterations in the microbiota, metabolome, intestinal permeability, and intestinal immunity, leading to worsening IBS symptoms (13, 22). High-FODMAP diets can induce mucosal irritation, as evidenced by increased mucin excretion and visceral hypersensitivity(34). Additionally, high dietary FODMAP intake can lead to the production of lipopolysaccharide (LPS), a biomarker of intestinal barrier integrity and gut dysbiosis, and a primary driver of endotoxemia (41). Increases in LPS following the ingestion of high-FODMAP foods have been found to mediate intestinal barrier dysfunction, mucosal inflammation, and visceral nociception and hypersensitivity due to gut dysbiosis in a subset of IBS patients (41). The metabolic products of FODMAPs can trigger mast cell activation and infiltration of mucosal mast cells, resulting in secondary effects on colonic permeability and visceral sensation caused by disrupted communication between neuroendocrine signaling and effector nerves within the gastrointestinal tract (GIT) (12, 42). The hypothesized pathophysiological pathways of FODMAP-induced symptoms in individuals with IBS are illustrated in Figure 1. These mechanisms lead to IBS symptoms, including altered bowel habits and defecation patterns (diarrhea, constipation, or an alternation of these), fecal urgency, abdominal discomfort, bloating and luminal distension, flatulence, cramping, and pain associated with visceral hypersensitivity (3, 31-33, 35, 43, 44). The heterogeneity of IBS symptoms varies in extent and severity among patients, with symptoms fluctuating, waxing and waning over time, and often being unpredictable (30, 45).

The primary proposed mechanisms of action of an LFD are threefold. *First*, it reduces small intestinal malabsorption of osmotically active short-chain carbohydrates, resulting in decreased intestinal water content and a lower total colonic load of these carbohydrates. *Second*, it limits the availability of fermentable substrates for colonic fermentation and gas production, while also shifting the abundance of bacterial taxa involved in SCFA production and/or cross-feeding. *Third,* it decreases colonic gram-negative bacteria and reduces and normalizes fecal LPS levels, thereby decreasing mucosal inflammation and improving gut barrier function (41). Thus, it is conceivable that an LFD modulates visceral nociception and hypersensitivity by altering the gut microbiota and modulating intestinal inflammation.

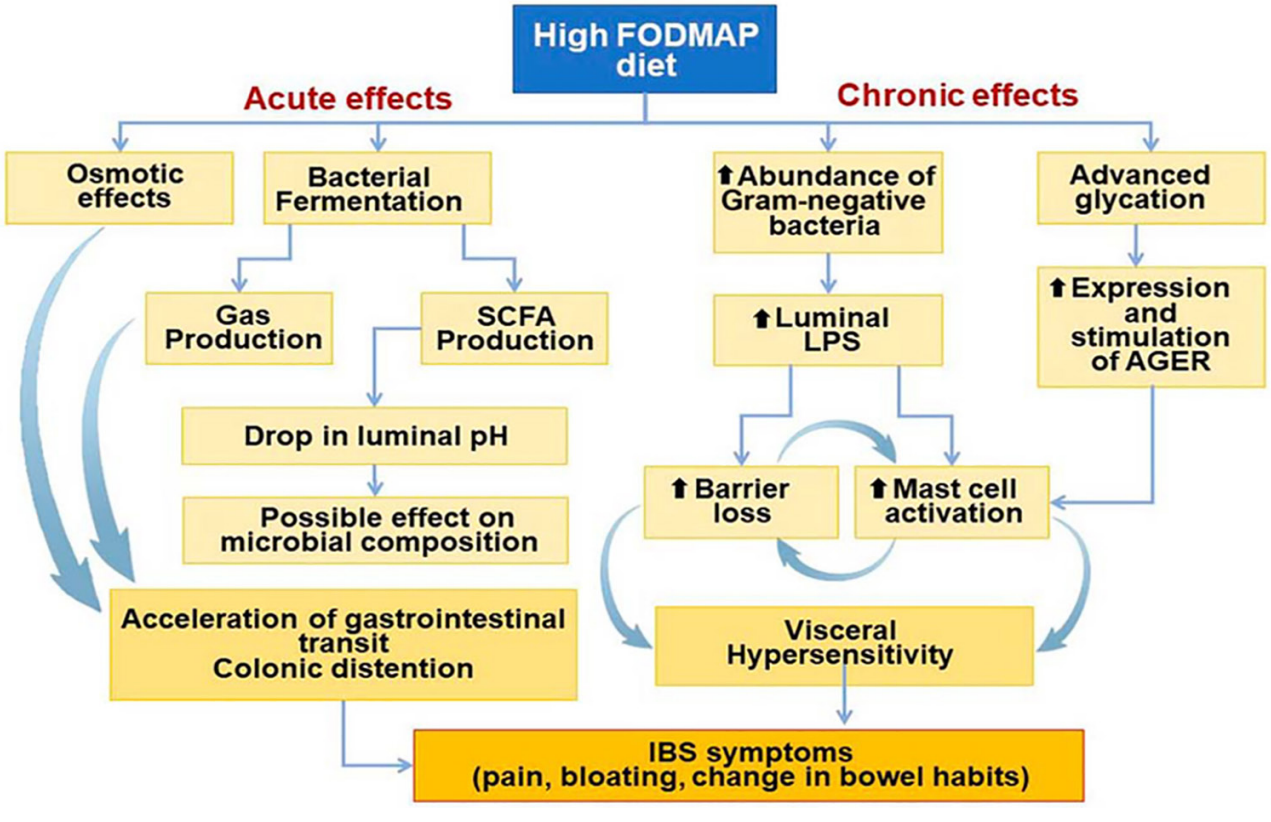

Figure 1: Pathways of FODMAP-induced symptoms in individuals with irritable bowel syndrome. Osmotic Effects: Osmosis increases the water content in the small bowel and lumen, leading to subsequent distention of the small intestine. Fermentation and Gas Production: Rapid fermentation of FODMAPs by gut microbes leads to gas production. Sensory and Motility Effects: The combined effects of increased gas and water retention stretch the intestinal walls, stimulating mechanoreceptors and triggering visceral hypersensitivity. This hypersensitivity is caused by an overreaction of the gastrocolic reflex, which involves prominent contractile and sensory responses of the colon to food ingestion. An abundance of Gram-negative bacteria increases luminal lipopolysaccharide (LPS) levels. LPS can activate mast cells, leading to increased intestinal permeability and heightened visceral sensitivity. Advanced glycation: increased expression of receptors specific to advanced glycosylation end products. Created with Biorender.com. Adopted from: [Ref. No. 40]

The reduction of osmotic activity in the intestinal lumen, reduced gas production, and correction of low-grade gut inflammation and microbiome imbalances in patients with IBS may explain the benefits of an LFD for these patients (12, 32, 35, 42, 43). Consistent with these mechanisms, the effects of an LFD, through reduced luminal osmolality, reduced gas production, and improved GI motility, correlate with a significantly higher reduction in IBS symptoms (12, 35, 42, 43). To better understand the potential mechanisms of action of the FODMAP diet in IBS pathophysiology, it is important to understand the implementation of the three-phased FODMAP diet and its impact on IBS symptoms and the gut microbiome, which will be addressed in the next section.

Implementation of the FODMAP Diet

The FODMAP diet is a complex and highly restrictive dietary intervention implemented through a three-stage, stepwise approach, as illustrated in Figure 2.

The *Elimination/Restriction phase* involves *removing* high-FODMAP foods from the diet and *replacing* them with LFD alternatives of equivalent nutritional and sensory quality for 2 to 6 weeks. This process aims to reduce or resolve the intensity and frequency of IBS symptoms (5, 23, 32, 37, 38, 46-48). The essence of this phase is to limit the food supply for gut microbes, effectively starving those that feed on fermentable carbohydrates. By eliminating high-FODMAP foods for a defined period, the gut can be reconditioned through the reduction of harmful bacterial, fungal, and methanogen overgrowth, allowing commensal and mutualistic microbes to thrive. This promotes the restoration of a balanced bowel flora composition and function, helping to re-establish a mutualistic symbiotic relationship between gut microorganisms and the host, thereby maintaining gut equilibrium.

Rechallenge and reintroduction phase: This phase helps patients identify the impact of specific FODMAPs and their doses on IBS symptoms, facilitating the reintroduction of tolerated foods into the diet (28). Tolerated FODMAP-containing foods are returned to the diet to increase dietary variety and reinoculate the gut microbiome, while keeping the type and total amount of FODMAP intake below each patient's individual tolerance threshold to maintain symptom control (4, 28, 46).

Personalization and maintenance phase: This phase involves personalization of the diet, which is essential for modulating the intestinal microbiota in a favorable manner and mitigating potential long-term adverse effects on microbiota composition, function, and diversity, thereby promoting sustained intestinal health (8, 39, 44, 47). This phase is intended to cultivate a eubiotic state within the intestinal microbial ecosystem, characterized by a predominance of beneficial microbial species that support a healthy and well-functioning gut. A FODMAP diet should be supervised by a qualified dietitian who has a thorough understanding of the interactions between the microbiota and dietary changes to achieve optimal health outcomes for patients with IBS.

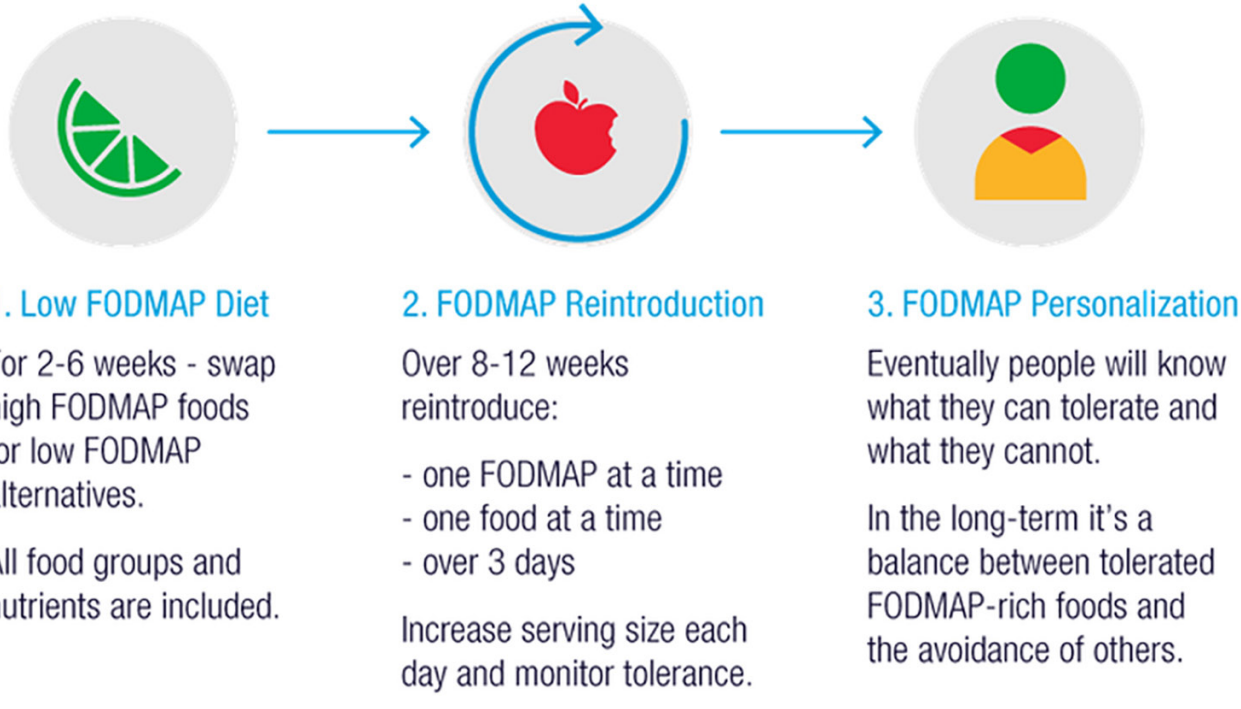

Figure 2: The three stages of the low-FODMAP diet: restriction, reintroduction, and personalization. *Phase 1:* Reduce FODMAP intake and replace with suitable FODMAP food alternatives. *Phase 2:* Rechallenge each FODMAP subgroup to assess tolerance and monitor symptoms. *Phase 3*: Personalize the diet based on responses to FODMAP foods challenges. *Source*: Monash University. What is the purpose of a FODMAP diet? Monash University. 2019. https://www.monashfodmap.com/ibs-central/i-have-ibs/starting-the-low-fodmap-diet/

Is an LFD Detrimental to the Gut Microbiome?

The restrictive FODMAP diet induces alterations in the intestinal microbiota, microbial metabolism, and metabolo me (6, 15, 22, 49). This issue has raised various concerns: The FODMAP diet is reported to adversely affect colonocytes (cells lining the colon), as well as the composition, function, diversity, and abundance of commensal intestinal microbiota. This disruption results in impaired gut homeostasis via the gut-brain-microbiome axis, negatively affecting intestinal health and overall host well-being (29, 39, 50-52). Restriction of prebiotic fermentable dietary fibre, including polysaccharides, FOS, GOS, and resistant starch, may result in decreased abundance of the SFCA butyrate producing bacterial species, including *Roseburia* (which aids butyrate production, offering anti-inflammatory effects, as well as improved gut barrier function), *Eubacterium rectale,* and *Faecalibacterium prausnitzii (*a keystone species associated with anti-inflammatory effects and improved gut barrier integrity). These bacteria are linked to higher rates of saccharolytic metabolism and are associated with positive health outcomes (11, 21). Decreased SCFA production is concerning because SCFAs provide numerous documented health benefits: they serve as an energy source for colonic epithelial cells, stimulate mucus and antimicrobial peptide production, and support mucosal homeostasis locally (e.g., epithelial function, intestinal barrier function, and immunoregulation) (11, 14, 15). SCFAs maintain mucosal integrity by reducing paracellular permeability and sustaining the expression of tight junction proteins, thereby enhancing barrier function. Furthermore, SCFAs promote the production of anti-inflammatory regulatory T-cells and cytokines in the intestinal mucosa while downregulating pro-inflammatory cytokines, thus modulating immune homeostasis and inflammation. Additionally, SCFAs contribute to the control of anaerobic conditions in the colon by limiting oxygen diffusion from colonocytes to the lumen. This mechanism contributes to the competitive exclusion of potentially pathogenic facultative anaerobes and influences immune responses and inflammation via interaction with the GALT (7-9, 53). Given these mechanisms, it is reasonable to infer that the significant reduction of SCFA-producing prebiotic fibrous carbohydrates during the elimination or restriction phase of the FODMAP diet would adversely affect intestinal health. However, while SCFAs are generally reported to be positively associated with gut health, evidence suggests that a reduction in SCFA levels following the LFD could be regarded as favourable in patients with IBS. Excessive intake of fermentable carbohydrates and the resulting elevated SCFA levels can increase osmotic load, potentially leading to diarrhea (14). Specifically, high concentrations of SCFAs, particularly acetic and propionic acid, have been associated with increased abdominal pain in patients with IBS (15). Thus, if abnormally high SCFA concentrations indicate gut dysbiosis that triggers IBS symptoms, reducing these concentrations through an LFD may be advantageous. Indeed, one study found that a decrease in the abundance of Bifidobacterium and Bacteroides following an LFD led to reduced SCFA and gas production from carbohydrate fermentation, which corresponded with alleviated IBS symptoms (18). Moreover, other studies have found that an LFD can normalize abnormal SCFA concentrations in IBS patients, thereby improving their symptoms (54, 55).

Importantly, prebiotic fibers are not the only dietary components that promote gut health. The FODMAP diet in all three phases, is rich in phytochemicals, bioactive phenolic compounds derived from the breakdown of plant substrates, collectively known as polyphenols (40, 56). Polyphenols are poorly absorbed in the small intestine; instead, they are metabolized by gut bacteria in the colon and broken down into metabolites, including SFCAs. Polyphenols serve as prebiotics and as energy sources for intestinal and immune cells. Furthermore, polyphenols enhance tight junction integrity and intestinal mucosal barrier function, increase mucus secretion, decrease intestinal permeability, protect the blood-brain barrier, and promote gut barrier function and intestinal epithelial homeostasis (3, 8, 12, 17, 35, 55). Additionally, polyphenols possess potent antibacterial, antifungal, antioxidative and anti-inflammatory properties, reducing intestinal inflammation by downregulating proinflammatory cytokines and NF-κB pathways (8, 40). Finally, polyphenols foster the growth of commensal microbes that promote resistance to colonization by harmful or pathogenic bacterial species through nutrient competition, thereby improving gut microbial diversity and resilience (3, 8, 12). Notably, both dietary phytochemicals and their microbial metabolites can promote a healthy gut microbiota (56). Therefore, it is reasonable to conclude that the interaction between phytochemicals and gut microbiota, which generates unique plant-derived microbial metabolites, can positively influence the gut health of patients with IBS consuming an LFD.

In addition to polyphenols, an LFD includes natural sources of conjugated linoleic acid (CLA). CLA is mainly formed naturally during the ruminal biohydrogenation of polyunsaturated fatty acids (PUFAs). CLAs are a mixture of positional and geometric isomers of linoleic acid (LA) with conjugated double bonds, synthesized by bacteria present in the alimentary tract of ruminant animals (57, 58). The main natural sources of CLA are milk, cheese, butter, and yogurt, as well as ruminant meats (grass-fed beef and lamb), whereas only trace amounts occur naturally in plant lipids (58). CLA is considered a functional food ingredient with prebiotic, probiotic, antioxidant, and immunomodulatory properties, providing health benefits for various

human pathologies (58). CLA has a prebiotic effect on gut microflora, particularly on Bacteroidetes/Prevotella and Akkermansia muciniphila (58). In IBS, CLA shows promise in potentially alleviating symptoms, primarily due to its anti-inflammatory and immunomodulatory properties, its ability to attenuate intestinal barrier dysfunction, and its modulation of the gut-brain axis (57). The link between CLA and IBS symptoms alleviation is mediated by gut microbes. Various strains of food-grade micro-organisms have been identified as producers of CLA. These microorganisms include Propionibacterium, Enterococcus, Clostridium, Lactococcus, Streptococcus thermophilus, Lactobacillus, and Bifidobacterium (particularly Lactobacillus acidophilus, reuteri, and bulgaricus, and Bifidobacterium lactis and breve) derived from camel, bovine, goat, and sheep (58). Bifidobacterium is considered the most significant producer of food-grade CLA due to its high bioconversion rate and enhanced production abilities (58). Lactobacillus is reported to have various roles, including stimulation of the immune system, prevention and reduction of the intensity and duration of diarrhea, and reduction of lactose intolerance (57, 59). Other properties associated with the genus Lactobacillus include the synthesis of B vitamins, enhancement of mineral and nutrient absorption, degradation of anti-nutritional factors, and modulation of intestinal physiology, and reduction of pain perception (57). Hence, CLA, through gut microbe-mediated mechanisms, can promote a gut ecosystem that supports gut health.

In summary, the LFD is nutrient-dense and contains certain food components that promote the health of the gut microbiome and the host (patients with IBS) by supporting microbial metabolite production and through mechanisms mediated by antioxidant, anti-inflammatory, and immunomodulatory signaling pathways.

Evidence from Studies Examining the Effects of an LFD on the Gut Microbiome

Various comprehensive studies on the FODMAP diet and its effects on the composition and metabolism of the gut microbiome have shown inconsistent results. Both negative and positive alterations in the intestinal microbial profile following an LFD have been found (50, 53, 60). In patients with IBS, an LFD does not appear to have consistent impacts on the colonic microbiome and microbiome metrics, including microbial composition, function, diversity, fecal SCFA concentrations, and fecal pH (61). Clinical trials and meta-analyses of randomized controlled trials examining the effect of FODMAP reduction on the gut microbiome have not detected statistically significant changes in gut microbiota composition, function, richness, and microbial α-diversity and β-diversity of taxa (13, 20, 51, 60-63). These findings are reassuring, considering that higher diversity is generally regarded as a hallmark of gastrointestinal health (61). Table 1 presents clinical trials investigating the effect of the FODMAP diet on multiple microbiome endpoints, including taxonomic changes and microbial metabolism. Several investigators have reported altered abundances of a limited number of taxa in patients with IBS (29, 61), including Bifidobacterium, Bacteroides, Faecalibacterium prausnitzii, and Actinobacteria (68). A decline in Bifidobacterium abundance, proportion, and concentration has been the most consistent finding (18, 29, 30, 45, 51, 64). The LFD reduces intake of fructose in excess of glucose, which partially explains the reduction in Bifidobacterium (21). Bifidobacteria are gram-positive bacteria and human probiotics in the intestinal tract with beneficial health properties (58). They play a crucial role in regulating the pH of the large intestine by releasing lactic and acetic acids, thereby inhibiting the proliferation of various harmful pathogens and putrefactive bacteria (22). Bifidobacteria can inhibit pathogens through the production of organic acids, antibacterial peptides, quorum-sensing inhibitors, and immune stimulation, among other mechanisms (22). Thus, the reduction in Bifidobacteria could be detrimental to host health in the long term. Consequently, the "antibifidogenic" effect of the LFD has been an area of concern (15, 61). However, Bifidobacteria levels caused by FODMAP restriction can be effectively restored through FODMAP personalization (65). In addition to reduction in Bifidobacteria, some studies have also found a loss of SCFA producers, such as Clostridiales, Bacteroides, Prevotella, and Actinobacteria (18, 50, 60). By contrast, other studies have reported an increase in the relative abundance of Actinobacteria among patients with IBS compared to healthy controls, as well as an increase in the phylum Bacteroidetes, the family Bacteroidaceae, and the genus Bacteroides (capable of fermenting large quantities of carbohydrates) after following a LFD (13). The family Ruminococcaceae has been found to be reduced with an LFD. Specifically, Ruminococcus torques was reduced after the FODMAP intervention (67). This genus has been associated with increased gut permeability and inflammation (67). Some studies have found an increase in potentially harmful species, such as Porphyromonadaceae and the non-saccharolytic taxon Bilophila (18, 50, 60). Sulfate-reducing bacteria, such as Bilophila and Desulfovibrio, are potential pathogens that compete with other lactate-utilizing bacteria, such as Anaerostipes, to produce hydrogen sulfide, which can be toxic to colonocytes (8). A high abundance of the genus Anaerostipes has been identified as a potential biomarker of a healthy core microbiota in humans, possibly mediated through the production of the SCFA butyric acid (15). The LFD has also been associated with markedly lower relative abundances of Akkermansia muciniphila (14). Akkermansia

Table 1: Clinical trial that examined the role of the FODMAP-diet and the impact on gut microbiota

Study/ references	Study design	Cohort	Results: the effect of FODMAP-diet on gut microbiome endpoints	Positive and/ or negative or neutral effect*
Staudacher et al., 2012 (53)	Randomized controlled trial. Patients with IBS were randomized to the intervention diet or habitual diet for 4 wk. The incidence and severity of symptoms and stool output were recorded for 7 d at baseline and follow-up. A stool sample was collected and analysed for bacterial groups using fluorescent in situ hybridization	41 IBS-patients in the United Kingdom.	The total luminal bacteria at follow-up did not differ between groups. Lower concentrations and proportions of bifidobacteria in the intervention group compared with controls when adjusted for baseline. Demonstrated a reduction in concentration and proportion of luminal bifidobacteria after 4 week of fermentable carbohydrate restriction.	-
Halmos et al., 2015 (29)	Single-blinded, randomised, cross-over trial. Random allocation to one of two 21-day provided diets, differing only in FODMAP content low vs Australian diet, and then crossed over to the other diet with ≥21-day washout period. Faeces passed over a 5-day run-in on their habitual diet and from day 17 to day 21 of the interventional diets were pooled, and pH, short-chain fatty acid concentrations and bacterial abundance and diversity were assessed.	27 IBS and 6 healthy subjects in Australia.	Faecal indices were similar in IBS and healthy subjects during habitual diets. The LFD was associated with higher faecal pH, similar short-chain fatty acid concentrations, greater microbial diversity and reduced total bacterial abundance compared with the Australian diet.	+
Harvie et al., 2017 (20)	A parallel design study. Group I participants received education immediately, were started on the LFD at baseline and started reintroduction of foods at three months. Group II participants were given the intervention (dietary education) in the second three month period. During the initial 3 months waiting period group II received no dietary education. Data was collected at baseline, 3 months (main comparison) and 6 months. Participants provided a stool sample at baseline, three and six months for microbiome analysis.	50 participants in New Zealand were enrolled into group I (n=23) or group II (n=27).	There was no change seen in the intestinal microbiome when participants adopted a LFD. No changes in alpha diversity.	±
Hustoft et al., 2017 (52)	A 9-week low-FODMAP diet. Cytokines (interleukin [IL]-6, IL-8, and tumour necrosis factor alpha) were analysed in blood samples, and gut microbiota composition (16S rRNA) and SCFAs were analysed in fecal samples.	20 IBS patients in Norway.	Gut bacteria, such as Actinobacteria, *Bifidobacterium*, and *Faecalibacterium prausnitzii* were significantly decreased. Serum levels of proinflammatory IL-6 and IL-8, total SCFAs, and n-butyric acid, decreased significantly on the LFD as compared to baseline. Ten days of FOS supplementation increased the level of these bacteria. Serum levels of proinflammatory IL-6 and IL-8 decreased significantly on the LFD compared with baseline, resulting in improved IBS-symptoms.	+

Study/ references	Study design	Cohort	Results: the effect of FODMAP-diet on gut microbiome endpoints	Positive and/ or negative or neutral effect*
Staudacher et al., 2017 (30)	Randomized, placebo-controlled study Duration: 4 weeks	51 IBS-patients (Rome III criteria) from the United Kingdom Sham diet/placebo (*n*=27). Low FODMAP diet/placebo (*n*=24)	LFD increased numbers of Bifidobacterium species. No change in *Lactobacilli* was observed, which increases with inulin and resistant starch intake.	+
Bennet et al., 2018 (51)	Randomization to traditional IBS (n=34) or low fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) (n=33) diets for 4 weeks	67 patients with IBS in Sweden	No difference in abundance of *Streptococcus* between responders and non-responders patients with IBS after LFD A LFD was associated with reduced *Bifidobacterium* and Actinobacteria	-
McIntosh et al. 2019 (61)	Controlled, single blind randomised study A 3-week low-FODMAP diet Low FODMAP diet (*n*=19); High FODMAP diet (*n*=18)	37 IBS-patients (Rome III criteria) in Canada.	Low vs. High FODMAP-diet Increased abundance: *Firmicutes, Actinobacteria, Clostridiales*, *Clostridiales family XIII Incertae sedis*) *Porphyromonas* Decreased abundance: *Propionibacteriaceae, Lachnospiraceae*	-
Wilson et al., 2020 (64)	Randomized, placebo-controlled, 3-arm trial Patients were randomized to a sham diet with placebo supplement (control) or LFD supplemented with either placebo (LFD) or 1.4 g/d B-GOS (LFD/B-GOS) for 4 weeks. Gastrointestinal symptoms, fecal microbiota (fluorescent *in situ* hybridization and 16S rRNA sequencing), fecal short-chain fatty acids (gas-liquid chromatography) and pH (probe).	69 adult patients with IBS from secondary care in the United Kingdom. and urine metabolites (^{1}H NMR) were analysed.	*Bifidobacterium* concentrations were not different between LFD and LFD/B-GOS but were lower in the LFD/B-GOS than in the control group. A proportion of Actinobacteria was lower in LFD and LFD/B-GOS groups than in the control group. The LFD reduced fecal Actinobacteria and butyrate: Fecal butyrate was lower in the LFD and LFD/B-GOS groups than in the control group.	+/-
Naseri et al., 2021 (13)	Clinical trial. LF-GFD intervention for 6 weeks. Fecal samples were collected at baseline and after intervention and analysed by quantitative 16 S rRNA PCR assay. The diversity of gut microbiota compared before and after 6 weeks of dietary intervention.	42 Iranian patients with IBS (Rome IV criteria)	Patients with IBS under an LF-GFD had reduced FC level following normalization of their gut microbiota composition. The relative abundance of *Bifidobacterium* and *Lactobacillus* significantly increased after the intervention, and the relative abundance of Actinobacteria was increased although not statistically significant. No difference in the relative abundance of *Enterobacteriaceae* before and after the dietary intervention. No difference in the relative abundance of *Streptococcus* after the dietary intervention compared to the baseline.	+

Study/ references	Study design	Cohort	Results: the effect of FODMAP-diet on gut microbiome endpoints	Positive and/ or negative or neutral effect*
Staudacher et al. (2021) (63)	Participants from a previously published 4-week 2x2 factorial design randomized controlled trial. Diet was assessed at four hierarchical levels and partial 16S.	95 IBS-patients from the United Kingdom rRNA gene sequencing was used to profile the microbiota.	Low FODMAP diet/placebo v sham diet/placebo Abundance increased: *Bacteroidetes, Bacteroidaceae Bacteriodes* Abundance decreased: *Actinobacteria, Firmicutes, Bifidobacteriaceae, Ruminococcaceae, Bifidobacterium* Probiotic supplementation led to higher Lactobacillus and Streptococcus abundance. The probiotic treatment buffered the impact of the LFD on *Bifidobacterium*.	+
Zhang et al. (2021) (18)	Randomized, parallel-group controlled study Duration: 3 weeks LFD (n=30); TDA (n=26). LFD vs traditional dietary advice.	56 Chinese IBS-D patients	LFD reduced carbohydrate-fermenting bacteria, such as *Bifidobacterium* and *Bacteroides*, and decreased saccharolytic fermentation activity. After FODMAP restriction. *Bifidobacterium* and *Fusobacterium* decreased significantly in the responders. *B. acidifaciens*, *B. fragilis*, and other *Bacteroides* species also showed a decreasing trend in responders. The abundance of *Bilophila and Prevotella* increased.	-
Bootz-Maoz et al. (2022) (62)	Clinical trial. Microbiome sampling throughout 6 weeks of low-FODMAP diet.	64 IBS- patients in Israel.	The post-FODMAP-diet microbiome is characterized by increased levels of Acutalibacter timonesis and Oscillibacter sp900066435 species and decreased abundance of several bacterial species, most notably Eubacterium ventriosum, Clostridium disporicum, and Bifidobacterium adolescentis	-
Staudacher et al., 2022 (65)	Short-term clinical trial. Stool samples were collected and analysed for microbiota (qPCR) and short-chain fatty acids (SCFA) from	18 IBS patients in the United Kingdom	Bifidobacteria abundance was not different between baseline and long-term personalized LFD. Lower concentrations of total SCFA, acetate, propionate, and butyrate.	+ and -
Vervier et al., 2022 (66)	Clinical trial of 4 weeks on a LFD. A detailed and carefully controlled metagenomic analysis of faecal microbiota before, during and after FODMAP restriction	41 IBS-patients in the United Kingdom.	FODMAP-diet intervention shifted the taxonomic composition of IBS^{P} cases by increasing *Bacteroides* levels (*B. cutis, B. stercorirosoris*), and decreasing pathobiont levels (including *C. difficile*, *Streptococcus parasanguinis*, *Paeniclostridium sordellii*) A shift in IBS patients' microbiota to a healthy profile was observed and appeared stable for at least 3 months.	+
Nordin et al., 2023 (67)	A double-blind, randomized, crossover study with week-long provocations of FODMAPs, gluten, and placebo.	90 participants with IBS in Sweden	A LFD produced higher levels of fecal saccharolytic bacteria, such as Anaerostipes, Bifidobacterium, Faecalibacterium, Fusicatenibacter, Agathobacter, Paraprevotella, and Oxalobacter. Lachnoclostridium, Roseburia, (Ruminoccocus) torques, Lachnospiraceae NK4A136, Lachnospiraceae NA, Hungatella, Eisenbergiella, Negativibacillus, Coprostanoligenes group NA, and Flavinofractor decreased. FODMAPs increased fecal saccharolytic bacteria, plasma phenolicderived metabolites, 3-indolepropionate, and decreased isobutyrate and bile acids	+

+ is a positive effect of the LFD on the gut microbiota; - is a negative effect; +/- is a positive and negative effect; ± is a neutral effect

muciniphila is a gram-negative, anaerobic, mucus-layer-degrading keystone bacterial species that colonizes the intestinal mucosa of humans. It plays a critical role in metabolic activities in the gut by converting fiber and polyphenols in food into metabolites such as the postbiotic short-chain fatty acids butyrate and propionate (61). Akkermansia muciniphila is involved in maintaining intestinal integrity by stimulating a thicker mucus layer and mucin rejuvenation, thereby fortifying the intestinal barrier and regulating gut permeability (69). Nonetheless, while Akkermansia muciniphila provides substantial health benefits when present at 3-5% of total gut microbes, overpopulation may disrupt the mucus barrier and increase endotoxemia due to excessive consumption of human intestinal mucus. One study observed that a typical Australian diet containing FODMAPs increased the relative abundance of Akkermansia muciniphila in patients with IBS (29). Other studies have found that an increased number of Akkermansia muciniphila in patients with IBS was correlated with episodes of abdominal pain (14, 41). It follows that lower relative abundances of Akkermansia muciniphila following an LFD might benefit IBS symptoms through reduced disruption of the mucus barrier and decreased endotoxemia.

Overall, mixed results from studies examining the effects of the FODMAP diet and alterations in the gut microbiome of patients with IBS highlights the complex and multifaceted relationship between diet, gut microbiota, the so-called diet-microbiome axis, gastrointestinal physiology, and health. The discrepancies observed across studies are likely attributable to various factors: (I) differences in study populations and sample sizes, IBS subtypes, duration of dietary intervention, levels of dietary adherence, dietary advice trials versus feeding trials, and washout periods in crossover studies; (II) baseline and follow-up data collection (biological samples, dietary data, health information, etc.) and sample processing (collection, analysis, interpretation, and data analysis); (III) a significant reduction in specific FODMAPs (fructans/GOS) versus other FODMAPs (lactose, excess fructose, and polyols); (IV) the LFD matrix, including the amount and types of FODMAPs consumed, as well as differences between animal-based and plant-based LFD; and (V) the possible presence of low abundances of certain microbiota genera at baseline. These factors collectively influence alterations in the gut microbiota and the outcomes of dietary interventions. Furthermore, these factors likely explain the lack of consistent changes in specific microbial taxa in response to the LFD, indicating a highly individualized microbial response.

Regarding concerns about potential adverse effects on the gut microbiota, some changes may be reversible with the reintroduction of FODMAPs (51). Studies have shown that after FODMAP reintroduction and FODMAP personalization, the abundance of beneficial microbes can return to baseline levels (4). Therefore, the potential harms may be overestimated, as the reintroduction phase involves gradually adding FODMAP-containing foods back into the diet, which helps repopulate the gut microbiome. Importantly, emerging research suggests that a LFD does not negatively impact the gut microbiome. Instead, the diet positively influences microbial composition and function by altering the composition and relative abundance of intestinal microbiota, leading to changes in metabolites and ultimately improving gut conditions in patients with IBS (22). Studies have found that an LFD normalizes dysbiotic microbial communities and promotes the restoration of intestinal microbiota eubiosis in patients with IBS. This effect is characterized by an expansion of commensal, symbiotic microbial species, resulting in a gut microbiome associated with improved health (13, 61, 66).

In addition to normalizing gut microbiome equilibrium, dietary FODMAP restriction has been shown to reduce intestinal hyperpermeability, immune activation (22, 51, 60), and visceral sensitivity by lowering the concentration of fecal lipopolysaccharides (15, 17). LFD also appears to exert positive effects on gut endocrine cells, which can help normalize bowel function (17). Furthermore, LFD has been shown to reduce blood concentrations of proinflammatory cytokines in patients with IBS, thus downregulating intestinal inflammation and enhancing intestinal immunity (13, 51). A clinical trial involving 47 IBS-D patients, diagnosed according to Rome IV criteria, demonstrated that at the end of the LFD, improvements in small intestinal permeability and intestinal mucosal integrity were observed (12). The study also demonstrated that levels of LPS (an intestinal mucosal integrity), IL-6 (a pro-inflammatory cytokine) and IL-10 (which inhibits NF-κB signaling and the production of pro-inflammatory cytokines) significantly decreased by the end of the LFD. These findings confirm the beneficial effects of the LFD on intestinal barrier health and its role in preventing bacterial translocation from the gut into circulation.

The findings from these studies showing that the FODMAP diet can beneficially modulate the gut microbiota and restore host-microbe eubiosis, thereby promoting gut and overall host health. These results should alleviate concerns regarding the safety of a short-term LFD in relation to the colonic microenvironment. Overall, there is insufficient conclusive evidence to support claims that the FODMAP diet adversely affects the gut, thereby refuting arguments from LFD detractors who assert that the FODMAP diet is unsafe and harmful to the gut microbiome in

individuals with IBS.

The scientific evidence synthesized in this narrative review provides exploratory justification for viewing the FODMAP diet as an effective dietary approach to shift the gut microbiome from a "pathobiome" state, characterized by amensalism (including predation, parasitism, antagonism, or competition), to a eubiotic state, characterized by commensalism, mutualism, and synergism between the human host and its microbial symbionts (70, 71). The aim is to promote the health of the holobiont, i.e., the host and its associated microbiota coevolving as a single entity to achieve gut symbiosis and homeostasis (9, 70). A well-balanced, healthy gut microbial community is essential for the host and microbiome to coexist in a mutually beneficial relationship. Currently, a substantial knowledge gap exists regarding what constitutes an optimal healthy microbiome, and an evidence-based definition of a "eubiotic" or "healthy" gut microbiome remains to be established (71). Nonetheless, certain hallmarks of a healthy gut microbiome are frequently cited (7, 40, 70, 71): high taxonomic diversity and richness; functional redundancy; stability, resistance, and resilience of microbial networks, including the ability to rebound from perturbations caused by opportunistic pathogens; maintenance of the structural integrity of the gut mucosa; and provision of immune tolerance to commensal bacteria. Additional hallmarks include the gut microbiota's ability to facilitate nutrient absorption and support proper gastrointestinal function and motility. What constitutes a healthy gut microbiome will differ for each individual with IBS, as microbiome communities are highly individualized with distinct ecological characteristics and exhibit a high degree of interindividual variation (9, 70, 71). Therefore, future large prospective cohort studies should be conducted with larger population of patients with IBS to address both inter- and intraindividual variation. These studies should use repeated measures within individuals to mitigate intraindividual variability. Such studies can elucidate the interactions between gut microbiota and host responses to nutritional interventions, such as the FODMAP diet. Furthermore, future clinical investigations are necessary to establish relationships between gut microbiome features (including diversity, specific taxa, gene clusters, metabolites, and more) and biomarkers or surrogate endpoints in patients with IBS. These biomarkers should be recognized indicators of normal biological and pathogenic processes, host physiology and function, and overall health thereby helping to determine what constitutes a healthy gut microbiome.

Limitations and Directions for Future Research

Several limitations of this review that need to be acknowledged. First, although not the primary aim, this article is not a systematic review, and does not include a quantitative evaluation or meta-analysis of the studies. Second, some relevant studies that met the eligibility criteria may have been missed due to the use of selective search terms. Nevertheless, we believe the evidence acquisition strategy was effective in identifying pertinent studies, particularly through additional methods such as citation matching and searching the reference lists of included studies. Despite these limitations, the strengths of this review lie in its comprehensiveness and its synthesis of up-to-date, high-quality published scientific evidence on the mechanisms of action of FODMAPs and the effects of the FODMAP diet on the gut microbiome of patients with IBS.

There are several implications for future research aimed at addressing the gaps in the existing literature.

To date, there is limited evidence from studies supporting the long-term efficacy of the FODMAP diet, particularly concerning the maintenance of gut homeostasis. It is important to investigate the effects of an LFD on gut microbiota and related variables during extended periods of FODMAP restriction. Additionally, examining the gut microbiota during the reintroduction and personalization phases, rather than solely after the restriction phase, is equally important.

The application of multi-omics next-generation sequencing technologies and bioinformatics (9, 10) enables the evaluation of gut microbiome composition, richness, functional activity, and metabolite profiles. These methods can identify gut bacteria, viruses, fungi, archaea, and other microbial components at the strain level, providing a more accurate and comprehensive characterization of the microbial ecosystem in IBS subjects following an LFD. This highlights the importance of precision microbiomics, which utilizes the gut microbiome as a predictive biomarker for responsiveness to specific dietary constituents (72).

Precision microbiomics and artificial intelligence-assisted multi-omics next-generation sequencing technologies offer significant potential for evidence-based personalized precision nutrition. This approach involves customized dietary interventions tailored to individual factors such as genetics, epigenetics, the gut microbiome, and lifestyle. Central to precision nutrition is the identification of biological markers and the individual gut microbiome's composition and function, enabling the design of customized and effective nutritional interventions specifically, determining which foods to avoid, limit, or include in the diet, to optimize gut health of in patients with IBS.

Gut microbiome research is an emerging field. Due to the considerable interindividual variability in gut microbiome composition, many uncertainties, unanswered questions, and debates around the role of the gut microbiome in the etiology, pathogenesis, and responsiveness

to dietary interventions in IBS. Species-level stratification of patients with IBS based on unique taxonomic microbial signatures (such as composition, function, metabolic capacity, and metabolite production) (73) holds promise for the development of novel, clinically meaningful, gut-centric personalized microbiome manipulation treatments. These interventions aim to cultivate a biodiverse gut ecosystem, thereby promoting a longer health span for individual IBS patients.

Conclusion

This narrative review elucidates the pathophysiological mechanisms by which FODMAPs elicit or exacerbate IBS symptoms, as well as the mechanisms underlying the effects of reducing FODMAPs in the diet. By exploring these mechanisms in depth, the review enhances our understanding of how the FODMAP dietary intervention can modify the gut microbiome and improve gut microbiota homeostasis. The scientific evidence synthesized in this narrative review indicates that there is inconclusive evidence that the LFD is detrimental to gut health. Several studies have reported reductions in Bifidobacteria and other SCFA producing bacteria, along with increases in potentially harmful species. However, other studies found a reduction in genus associated with increased gut permeability and inflammation following LFD intervention. Importantly, research shows that alterations in gut microbiota caused by FODMAP restriction can be effectively restored through FODMAP reintroduction and personalization. These findings underscore the importance of considering diet, the colonic environment, and intestinal microbiology together to interpret their interactions and effects on the health of patients with IBS. The health effects of specific gut microbiome features are likely context-dependent, with certain bacterial taxa associated with health in one setting and disease in another. Consequently, it is challenging to identify gut microbiome features following an LFD that are universally beneficial or harmful. Functional redundancy within the microbiome is common, and diversity is likely more important than the increase or decrease in specific taxa following an LFD. In conclusion, this narrative review supports the FODMAP diet as a personalized precision-nutrition therapy that can effectively repair, reinoculate, and rebalance the gut microbiome in patients with IBS.

Authors' Contribution

Review conception, design and manuscript preparation: RV and MV; Both authors reviewed and approved the final version of the manuscript.

Data Availability Statement

Data are available in a public, open access repository.

Ethics Approval and Consent to Participate

Not applicable.

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During the preparation of this work the author(s) used no AI tools to write this paper. The author wrote, reviewed and edited the content as needed manually and take full responsibility for the content of the publication.

Competing Interests

The authors declare that they have no competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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