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## **Dietary and Lifestyle Factors Associated with Small Intestinal Bacterial Overgrowth (SIBO): A Review from a Health Education Perspective**

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### **Abstract**

**Background:** Small intestinal bacterial overgrowth (SIBO) is characterized by bacterial overgrowth in the small intestine, leading to gastrointestinal symptoms and systemic complications. Diet and lifestyle can modulate the gut microbiota and gut motility, influencing the risk of developing SIBO.

**Objective:** The aim of this review is to synthesize the current evidence regarding the influence of dietary and lifestyle factors on the development and progression of SIBO.

**Methods:** A narrative literature review was conducted in PubMed and Scopus, covering publications from 2011 to 2025. Studies examining the association between dietary patterns,

physical activity, and other lifestyle factors with the occurrence or severity of SIBO were included.

Results: A diet high in fermentable carbohydrates (FODMAPs), high in fat, and low in fiber predisposes to SIBO through dysbiosis and impaired gut motility. Low levels of physical activity, irregular meals, snacking, excessive alcohol consumption, long-term use of proton pump inhibitors (PPIs) and smoking are potential risk factor. Interventions including dietary modification, regular meals, physical activity, and the use of probiotics and prebiotics can improve symptoms and modulate the microbiota.

Conclusions: Diet and lifestyle are key factors modulating the pathogenesis and treatment of SIBO. Clinical strategies should combine dietary interventions, lifestyle optimization, and pharmacotherapy. Further prospective and interventional studies are needed to standardize dietary recommendations and the role of lifestyle modification in the prevention and treatment of SIBO.

Keywords: „SIBO”, „diet”, „lifestyle”, „gut microbiota”, „gut motility”

## **Introduction**

Small Intestinal Bacterial Overgrowth (SIBO) is a chronic gastrointestinal disorder characterized by excessive bacterial growth (quantitative and/or qualitative) in the small intestine, leading to digestive and malabsorption dysfunction and nonspecific symptoms such as abdominal pain, excessive intestinal gas, and changes in bowel habits.

The exact incidence of SIBO remains unclear, although it is known that the disorder often coexists with many other conditions. Currently used diagnostic methods, including breath tests and small intestinal aspiration, have their advantages and limitations and should always be interpreted in the context of the overall clinical picture. The therapeutic approach to SIBO has undergone significant changes over the past two decades: remission is typically induced via antibiotic therapy or an elemental diet, while maintenance relies on treatment of the underlying cause and the use of prokinetic medications. It is important to consider lifestyle modifications and plan appropriate nutritional interventions [Sharabi et al., 2024].

## **Epidemiology**

The exact prevalence of SIBO remains difficult to determine unequivocally. This is due to both its nonspecific clinical presentation, which may resemble other gastrointestinal disorders, and the wide variability in prevalence rates across different patient groups.

In studies of individuals with gastrointestinal symptoms, SIBO was diagnosed in over one-third of patients (approximately 33.8%). The presence of this disorder was particularly strongly associated with smoking ( $OR \approx 6.7$ ), as well as with symptoms such as bloating ( $OR \approx 5.4$ ), abdominal pain ( $OR \approx 4.8$ ), and anemia ( $OR \approx 4.1$ ). The risk of SIBO increased gradually with age ( $OR \approx 1.04$  per year), but there was no association with gender or race. Reduced ileocecal pressure, slower intestinal transit, and higher gastric and small intestinal pH were also observed in individuals with SIBO, although parameters of intestinal motility and calprotectin levels did not differ significantly compared with the control group. Lifestyle factors also influenced the incidence; moderate alcohol consumption was associated with higher rates of SIBO (58% vs. 39% in abstainers). In the pediatric population, SIBO was found in the majority of children reporting chronic abdominal pain (approximately 63%) [Efremova et al., 2023]. In patients after surgical procedures modifying the intestinal anatomy – e.g. Roux-en-Y gastric bypass – SIBO was found in approximately 29% of patients within the first three years after surgery, while after more than three years this percentage increased to approximately 53% [Sharabi et al., 2024]. A meta-analysis of 19 studies confirmed that the use of proton pump inhibitors significantly increases the risk of developing SIBO ( $OR 1.71$ ), with its incidence further increasing after one year of continuous therapy. Breath tests also showed that SIBO co-occurs in approximately 68% of patients with functional bowel disorders. An increased incidence of SIBO is also observed in individuals with lactase deficiency compared to the healthy population. Another meta-analysis of 14 studies showed a higher incidence of SIBO in patients with celiac disease – approximately 18.3% of patients met the diagnostic criteria, translating into a five-fold higher risk compared to the control group ( $OR 5.1$ ). An increased incidence of SIBO was also noted in patients after cholecystectomy (46.8% vs. 13.3%;  $OR 5.7$ ). A similar trend was observed in individuals with hyperlipidemia, where SIBO was present in 78.9% of patients, compared with 40% in the control group [Efremova et al., 2023]. This data shows that SIBO appears in a wide variety of disease entities—both functional and organic gastrointestinal disorders, as well as liver disease, endocrine, neurological, and rheumatological disorders, asthma, heart failure, and many other clinical conditions. This broad spectrum of comorbidities makes the epidemiology of SIBO extremely complex and difficult to precisely assess. The diversity of populations affected by this disorder emphasizes the need for a holistic approach to the patient—both at the diagnostic and treatment stages. Considering comorbidities, lifestyle, pharmacotherapy, and individual risk factors is crucial to correctly identify SIBO cases and effectively manage treatment. The introduction of a dedicated ICD-10 code for SIBO (K82.11) by the National Center for Health Statistics in 2024 will enable more precise monitoring and

analysis of the prevalence and incidence of this condition both nationally and globally [Sharabi et al., 2024].

## **Pathophysiology**

The development of SIBO is rarely attributable to a single cause but rather stems from a breakdown in the body's natural defense barriers. The primary mechanisms preventing bacterial overgrowth include efficient intestinal motility, anatomical integrity, and appropriate gastric secretion. Failure in any of these domains predisposes the small intestine to pathological colonization.

### 1. Impaired Intestinal Motility

Growing evidence indicates that intestinal transit time plays a key role in shaping both the composition and activity of the gut microbiome, which has important implications for human health. Population-based studies and smaller analyses have shown that differences in intestinal transit time are a major contributor to the high interindividual variability in the composition of the fecal microbiome. Despite this, measurement of intestinal transit time is rarely considered in microbiome research. This review discusses recent studies demonstrating how and why intestinal transit time varies across the entire gut and within individual segments, and how these variations influence the composition, diversity, and metabolic functions of the microbiota. Furthermore, it describes the mechanisms by which the microbiota may influence intestinal motility, emphasizing the bidirectional nature of the interaction. Considering individual and intraindividual differences in intestinal transit time can improve the interpretation of diet-microbiome interactions and the identification of disease-related microbiome signatures, which are often confounded by variations in intestinal transit time.

Intestinal transit time is influenced by many factors, including age, gender, stress, body mass index, colon anatomy, gut hormones, and diet. Furthermore, the gut microbiota and its metabolites also modulate transit rate, creating a complex network of interdependencies. Differences in transit time have consequences not only for the composition of the microbiota but also for its metabolism, as they alter the availability of substrates in the gastrointestinal tract. Consequently, transit time is a significant factor determining the heterogeneity of the gut microbial community and influences the interdependent metabolism between the host and the microbiota.

Gut microbiota can influence gastrointestinal motility through the production of small molecules that interact with receptors on enterendocrine cells and enteric neurons. Both prolonged and shortened transit times can have significant consequences for host health,

primarily through changes in microbiota composition and metabolism. A key mechanism protecting against SIBO is the Migrating Myoelectric Complex (MMC), a cyclic pattern of electromechanical activity that clears undigested food and bacteria from the small intestine during the interdigestive state. A particularly slow transit time through the small intestine promotes bacterial overgrowth in this section of the intestine, leading to SIBO. Patients with SIBO have high bacterial densities ( $>10^5$  CFU/ml), resulting from impaired peristalsis and insufficient flow of food into the large intestine, which further slows transit time in distal gastrointestinal tracts. Incorporating measurements of intestinal transit time into microbiome studies allows for a better understanding of the relationships between gut microbiota, diet, and disease development. This approach may be important for the prevention, diagnosis, and treatment of various disorders of the gastrointestinal tract and other organs throughout the lifespan [Prochazkova et al., 2023].

## 2. Anatomical Abnormalities and Mucosal Barriers

For many years, altered intestinal anatomy was considered the primary predisposing factor to the development of SIBO. It was believed that the blind loop of the small intestine resulting from bypass surgery favored the retention of food, which in turn led to the overgrowth of intestinal bacteria. Other sequelae of abdominal procedures can also contribute to SIBO development, including postoperative adhesions, anastomotic strictures, and ileocecal valve resection.

In recent years, increasing attention has been paid to intestinal motility disorders as an independent risk factor for SIBO, leading to slowed intestinal transit. Importantly, adhesions can also develop without prior surgery, most often in the course of endometriosis, undiagnosed appendicitis, or ovarian disease. Other motility disorders predisposing to SIBO include autoimmune diseases (systemic sclerosis, celiac disease), inflammatory bowel conditions (Crohn's disease, radiation enteropathy), opioid and anticholinergic use, visceral neuropathies such as gastroparesis, and even autoimmune reactions directed against intestinal structure—such as anti-vinculin antibodies described in post-infectious irritable bowel syndrome [Sharabi et al., 2024]. In patients with inflammatory bowel disease (IBD), SIBO is significantly more common than in the general population—positive tests are found in approximately one-third of patients, and the risk of developing it is more than five times higher compared to healthy individuals. Factors that contribute to increased susceptibility to SIBO include a low body mass index, bloating, excessive gas, prior abdominal surgery, and the presence of strictures or penetrating disease. Dysbiosis, a key element in the pathogenesis of IBD, further contributes to

the disruption of intestinal protective mechanisms. In patients with Crohn's disease, the damaged ileocecal valve, both due to surgical procedures and the inflammation resulting from the disease, does not provide an effective barrier against the receding colonic microflora, facilitating colonization of the small intestine and the development of SIBO [Feng et al., 2025].

### 3. Gut Microbiota Dysbiosis

SIBO is characterized not only by excessive bacterial counts but also by qualitative changes in the small intestinal microbiota. 16S sequencing and shotgun metagenomics studies indicate that *Escherichia coli* and *Klebsiella* strains predominate, and their growth correlates with clinical symptoms and metabolic changes [Leite et al., 2024]. Different SIBO phenotypes include oral-type overgrowth and *Proteobacteria* dominance, which have diagnostic and therapeutic implications [Yersin et al., 2024]. These disorders lead to excessive fermentation of carbohydrates and production of gases ( $H_2$ ,  $H_2S$ ), exacerbating symptoms and possibly inhibiting intestinal motility [Leite et al., 2024; Yersin et al., 2024].

### 4. The Role of Diet and Lifestyle

Diet and lifestyle strongly modulate the gut microbiota and gastrointestinal function. A diet rich in fermentable carbohydrates (FODMAPs), high in fat, or low in fiber promotes bacterial overgrowth and metabolic changes in the small intestine. Certain dietary components, such as fiber and polyphenols, support gut motility and the functioning of the Migrating Motor Complex (MMC), while excess fermentable substrates promote bacterial proliferation. A lifestyle that includes chronic stress, physical inactivity, sleep disorders, and smoking impacts the gut-brain axis, gut motility, and microbiota, increasing the risk of SIBO [Knez et al., 2024; Chomatowski et al., 2025; Foster et al., 2017].

### 5. Multifactorial Pathogenesis

SIBO is the result of the interaction of multiple, overlapping processes, including intestinal motility disorders, dysbiosis, anatomical abnormalities, and the influence of diet and environmental factors. This complex and multifactorial pathophysiological basis is reflected in a broad clinical spectrum – SIBO occurs in patients with gastrointestinal diseases, metabolic, neurological, and endocrine disorders. Therefore, diagnosis and treatment require a holistic approach, considering the interconnected mechanisms underlying the disease. Treatment should include not only interventions aimed at eradicating excess bacteria, such as antibiotic therapy or a balanced diet, but also lifestyle and nutritional modifications that modulate the microbiota and gastrointestinal motility, influencing the long-term effectiveness of the therapy.

## Methods

This narrative review encompasses a wide range of scientific sources, including original clinical trials, systematic reviews, meta-analyses, and current clinical guidelines on the role of diet and lifestyle in the development of SIBO. Publications were selected based on a preliminary analysis of abstracts and full texts, considering their relevance to a multidisciplinary approach to SIBO prevention and treatment. Selection criteria included data validity, methodological quality, and practical clinical relevance. Priority was given to papers with internationally recognized literature and those that addressed medical, psychosocial, and environmental factors influencing SIBO development. As a result, 46 key publications were included in the final analysis and synthesis, allowing for a comprehensive presentation of current strategies related to diet, lifestyle, and the risk of SIBO.

## Dietary Factors in SIBO Pathogenesis

SIBO is associated with numerous nutritional consequences that require medical attention. Bacterial overgrowth can damage the intestinal lining, disrupt metabolism, and lead to restricted food intake, which contributes to malabsorption, weight loss, and micronutrient deficiencies. Therefore, the support of an experienced dietitian who can provide appropriate recommendations is crucial. The following section presents the most important dietary strategies used to manage SIBO.

### 1. Fermentable Oligosaccharides, Disaccharides, Monosaccharides and Polyols (FODMAP)

FODMAPs include fructans, galactans, lactose, fructose, and polyols. The absorption pattern of these nutrients determines their potential impact on the gastrointestinal tract: excess fructose can easily overload GLUT5/GLUT2 transporters, while unabsorbed lactose due to lactase deficiency triggers bacterial fermentation. In SIBO, this fermentation occurs in the small intestine, exacerbating bloating, pain, diarrhea, and leading to false-positive breath tests. Oligosaccharides and polyols, which normally reach the colon and act as prebiotics, are also fermented in the small intestine in SIBO, triggering symptoms.

A low-FODMAP diet involves three stages: elimination, gradual reintroduction, and long-term adjustments based on individual tolerance. While it may reduce symptoms, long-term use can negatively impact the microbiota, as FODMAPs support the growth of beneficial bacteria and the production of short-chain fatty acids. Therefore, caution is advised and long-term restrictions should be avoided. A practical alternative is a low-fermentation diet, which involves limiting the most problematic foods - dairy products, foods high in fructose, highly fermentable vegetables, and sugar alcohols [Vasalsco-Aburto et al., 2025]. On the other hand, long-term adherence to a low-FODMAP diet can lead to a reduction in the number of beneficial gut bacteria, such as *Bifidobacterium*, and reduced production of short-chain fatty acids, which weakens the natural prebiotic effect of fermentable foods. To minimize

these negative effects, short-term elimination periods are recommended, followed by gradual, controlled reintroduction of FODMAP products to determine individual tolerance. Personalizing the diet and monitoring the microbiota by a qualified dietitian allows for maintaining gut microbial balance, reducing symptoms, and limiting the risk of nutritional deficiencies. This approach combines therapeutic efficacy with maintaining intestinal eubiosis and long-term gastrointestinal health [Xu et al., 2024].

## 2. Dietary Fiber and Prebiotic Potential

Research increasingly demonstrates that dietary fiber, particularly its fermentable fractions, influences the gut microbiome by promoting the growth of beneficial microorganisms, including bacteria specialized in fiber degradation. The process of fiber fermentation by these microorganisms results in the production of short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate. These compounds perform important physiological functions, including strengthening the colonic epithelial barrier, modulating immune responses, and regulating host metabolism [Inoue et al., 2025]. Soluble fiber supplementation was associated with beneficial changes in the composition of the gut microbiota. A short-term transition of 16 healthy volunteers from a high-fiber diet to a low-fiber diet (<11 g/1000 kcal) for 7 days resulted in gastrointestinal symptoms in all participants, and two were diagnosed with SIBO. These results underscore the need for further research to develop specific dietary recommendations for this condition [Wielgosz – Grochowska et al., 2022].

However, these data reinforce the concept that fiber is an important component of dietary therapy for small intestine microbial imbalances.

## 3. Impact of High-Fat Diets

In clinical practice, it is often observed that patients with SIBO increase their dietary fat intake in an attempt to reduce carbohydrate-induced symptoms. Notably, different types of fat exert distinct effects on the gut microbiota and pH in the large intestine. Experimental studies have shown that lipids in the duodenum can slow small intestinal motility and impede the elimination of intestinal gas, promoting gas accumulation and bloating. A systematic review of the literature further suggests that fat consumption may significantly influence the severity of functional dyspepsia symptoms [Wielgosz-Grochowska et al., 2024].

A diet high in fat, especially saturated fat, has a complex impact on gastrointestinal function. It alters the composition and activity of the microbiota, modulates the secretion and metabolism of bile acids, influences local immune mechanisms, and can disrupt normal intestinal motility.

These modifications to the intestinal environment promote dysbiosis, weaken the mucosal barrier, and disrupt intestinal transit, which in turn can create conditions conducive to the development of SIBO. Current reviews indicate a high-fat diet as one of the potential factors increasing the risk of small intestinal bacterial overgrowth, but further research is needed [Soliman et al., 2025].

#### 4. Impact of Alcohol and Nicotine

Alcohol may promote SIBO through several mechanisms. First, it compromises the gut barrier because ethanol reduces the expression of tight junction proteins, which increases intestinal permeability and allows bacterial translocation [Sosnowski et al., 2024]. Secondly, alcohol contributes to dysbiosis. Research indicates changes in the composition of bacterial flora in individuals who abuse alcohol, including the growth of potentially pathogenic bacteria. Additionally, in individuals who consume large amounts of alcohol, elevated plasma endotoxin levels may result from several mechanisms: increased endotoxin production in the intestines due to bacterial overgrowth, increased intestinal permeability, allowing it to pass into the blood and delayed endotoxin removal by Kupffer cells in the liver [Chen et al., 2022]. Third, alcohol may disrupt the local gut immune response by disrupting the balance between tolerance and pro-inflammatory responses. This is manifested by reduced numbers of regulatory T cells, increased activity of type 17 T helper cells and TNF- $\alpha$  production, and attenuated antimicrobial mechanisms, which consequently increase inflammation, bacterial translocation, and systemic immune activation [Kreimeyer et al., 2025]. Finally, clinical studies show that SIBO occurs more frequently in patients with severe alcoholic hepatitis than in healthy individuals (25% vs. 3,3%) [Vaithiyam et al., 2025]. Cigarette smoking has been associated with reduced microbiota diversity in the duodenal mucosa. In a study of 102 patients, current and former smokers had significantly lower bacterial diversity and increased relative abundance of strains such as Streptococcus and Veillonella compared with nonsmokers. Such microbial shifts may predispose to dysbiosis and promote bacterial overgrowth in the small intestine [Shanahan et al., 2018]. Additionally, other epidemiological data also indicate that smoking may be a strong predictor of SIBO.

A retrospective study of patients undergoing breath testing demonstrated a significant, independent association between smoking and positive SIBO tests (OR = 6.66) [Liu Chen et al., 2020]. Although available data suggest a significant association between diet and the risk of SIBO, most observations come from studies of limited quality, often cross-sectional or small sample sizes. More rigorous prospective and randomized studies are needed to definitively

confirm the proposed mechanisms. However, preliminary results suggest that the cumulative effects of factors such as a high-FODMAP diet, high fat intake, excessive alcohol consumption, smoking, and low fiber intake may promote bacterial overgrowth in the small intestine.

In clinical practice, when assessing patients with SIBO, it is worth collecting a detailed history of addictions and considering lifestyle interventions (smoking cessation, alcohol restriction) as part of a preventive and therapeutic strategy. In the context of therapeutic management, it is recommended to take these observations into account, especially in patients with active SIBO or a tendency to relapse.

### **Role of Probiotics and Prebiotics**

Probiotics and prebiotics can play an important role in preventing and supporting the treatment of SIBO, although their effectiveness varies and depends on the clinical context. Some research suggests that adding probiotics or prebiotics to antibiotic therapy may improve outcomes—an RCT showed that combined therapy with rifaximin and *Lactobacillus casei* resulted in better symptom improvement than antibiotic monotherapy [Rosania et al., 2013]. More recent systematic analyses also confirm that probiotics can support treatment. A meta-analysis showed that patients taking probiotics had higher rates of decolonization (i.e., removal of excess bacterial flora), reduced hydrogen production, and reduced abdominal pain than those not taking probiotics. However, this study did not demonstrate a positive role for probiotics in preventing SIBO [Zhong et al., 2017].

A review of recent clinical studies also suggests the use of a combination of probiotics and prebiotics, or symbiotics, as a therapeutic strategy for SIBO. In pregnant women with subclinical hypothyroidism, supplementation with synbiotics for 21 days resulted in lower breath test results (hydrogen/methane) and improved gastrointestinal health. Using probiotics in combination with prebiotics may support SIBO treatment through several mechanisms, including regulating intestinal motility—probiotic strains improve the integrity of the intestinal epithelium, strengthen tight junctions between cells, and stabilize intestinal movements, which helps alleviate symptoms such as diarrhea and constipation. They strengthen the intestinal barrier by increasing the number of regulatory T cells, stimulating the production of antibacterial proteins, and promoting the formation of a protective mucosal layer. Additionally, probiotic metabolites, such as lactic acid, short-chain fatty acids, and other organic acids, inhibit pathogen growth, support lysozyme production, and stabilize the microenvironment of the small intestine [Ouyang et al., 2024]. On the other hand, a systematic review of studies in bariatric surgery patients showed limited efficacy of probiotics in treating SIBO in this population, suggesting that the effects may be dependent on the disease context and individual

host characteristics [Wagner et al., 2025]. Therefore, prebiotics and probiotics can provide valuable support in modulating the microbiota and preventing SIBO recurrence, but their selection should be individualized. Prebiotics and probiotics are important as a supplement to, not a replacement for, standard therapy (antibiotics), and their use requires the supervision of a gastroenterologist and dietitian.

### **Impact of Medications (PPIs and Opioids)**

Medications play a significant role in the pathogenesis of SIBO and can be an important element in a holistic approach to bacterial overgrowth. The most documented group are proton pump inhibitors (PPIs). Many studies show that long-term suppression of gastric acid secretion promotes the development of small intestinal bacterial overgrowth. A meta-analysis of 29 studies found that patients taking PPIs had a rate of SIBO of approximately 36.8%, a significantly increased risk compared to those not taking PPIs (OR = 2.14). Furthermore, when analyzing the duration of treatment, the study authors observed a time-dependent effect: each additional month of PPI use increased the risk of SIBO by approximately 4.3%. Approximately 36.8% of patients taking PPIs develop SIBO. Due to the significant consequences for both the gastrointestinal tract and the systemic health of their patients, physicians should exercise extreme caution when using PPIs long-term [Khurmatullina et al., 2025]. Although the literature on opioids and anticholinergics in the context of SIBO is more limited than that on PPIs, it does suggest that these medications may promote small intestinal bacterial overgrowth by slowing intestinal motility. The Asia-Pacific SIBO Consensus Statement lists opioids and anticholinergics as key drug classes that inhibit intestinal transit and predispose to bacterial overgrowth. This study also demonstrated a role for tricyclic antidepressants and antidiarrheal medications in the development of SIBO [Ghoshal et al., 2022]. From a clinical perspective, the issue of the impact of medications on the development of SIBO is important because many of these medications, including proton pump inhibitors, opioids, and anticholinergics, are commonly used in medical practice. Long-term use of these medications may increase the risk of bacterial overgrowth in the small intestine through mechanisms including slowing intestinal motility, disrupting the intestinal barrier, and altering the composition of the microbiota. Awareness of this risk allows physicians to more carefully monitor patients, identify SIBO symptoms early, and modify therapy as needed, which may reduce the incidence of complications and improve patients' quality of life.

### **Physical Activity and Gut Motility**

Exercise acts as a physiological modulator of intestinal motility and may support the activity of the MMC.

A growing body of research indicates that regular physical activity has a beneficial effect on intestinal motility and the health of the microbiota. Literature data shows that exercise modulates intestinal motility, mucosal permeability, and microbiome composition, which may reduce food retention and subsequent bacterial growth [Severo et al., 2025]. Participants who increased their time spent in moderate-to-vigorous physical activity demonstrated significantly faster intestinal transit time. Each additional hour of such activity was associated with a 25.5% reduction in small intestinal transit time and a 16.2% reduction in total intestinal transit time, regardless of age, gender, and body fat percentage [Jensen et al., 2023]. In a study in which 21 participants performed a 20-minute treadmill walk, peristaltic sounds were assessed both at rest and at 15-minute intervals after exercise. These sounds were used as an indirect measure of gut motor activity. Just 1–2 minutes after completing the walk, all peristalsis indices showed a significant increase compared to baseline. This phenomenon may be due to transient changes in the autonomic nervous system and the activation of local intestinal reflexes due to the mechanical vibrations associated with physical activity. The stimulation of gut motor activity may explain the beneficial effect of exercise on constipation and provides clues for the potential use of exercise as a therapeutic approach. Because constipation can also occur with SIBO, these mechanisms may explain why physical activity is important in modulating gastrointestinal function in this group of patients [Katagiri et al., 2025].

Physical activity can affect the digestive tract in several ways, including increasing blood flow to the digestive tract, stimulating the parasympathetic nervous system, and influencing local intestinal reflexes, which together improve peristalsis [Al-Beltagi et al., 2025]. Regular exercise can shape the host gut microbiota in a way that promotes eubiosis - exercise increases bacterial diversity and supports strains that are beneficial for gut function [Li et al., 2024]. From a clinical perspective, low physical activity and motility disorders (such as constipation) may be predisposing factors for SIBO or its recurrence. Promoting moderate, regular physical activity (e.g., walking, yoga, aerobic exercise) can be a simple, noninvasive strategy to prevent SIBO or support its treatment.

### **Meal Frequency and Eating Patterns**

Available data indicate that meal timing may influence gastrointestinal function, but a direct link between frequent snacking and the risk of SIBO has not yet been confirmed in clinical trials. Despite the popularity of recommendations to "lengthen the intervals between meals" in dietary practice, a review of the scientific literature on PubMed shows that there are no peer-reviewed studies that conclusively demonstrate that irregular mealtimes or frequent snacking increase the incidence of SIBO, as assessed by breath testing or small intestinal aspirate.

Hypothesis involving irregular meals in the pathogenesis of bacterial overgrowth is based primarily on mechanistic models of the MMC phases, implying that frequent snacking could inhibit these crucial cleaning waves. In theory, frequent snacking could inhibit MMC, potentially creating conditions conducive to bacterial overgrowth, but this mechanism has not been confirmed in population-based or interventional studies.

### **The Gut - Brain Axis: Stress and Sleep Quality**

Disturbances in the gut-brain axis constitute an important mechanism linking stress and sleep disorders with intestinal motility abnormalities and dysbiosis. Stress activates the hypothalamic-pituitary-adrenal (HPA) axis, increasing cortisol secretion and modulating autonomic tone. Increased sympathetic nervous system activity and decreased parasympathetic nervous system activity translate into inhibition of the MMC and prolonged intestinal transit time. Simultaneously, HPA overload and chronic stress disrupt local mucosal immunity by reducing regulatory T cell function and weakening antimicrobial mechanisms, which promotes increased intestinal permeability and microbial translocation. These changes, including slower transit, a weakened barrier, and impaired local immunoregulation, create an environment predisposing to bacterial overgrowth in the small intestine [Sun et al., 2023; Chaudhry et al., 2023]. Chronic sleep deprivation affects the microbiome through several interrelated mechanisms. Disruption of host circadian rhythms leads to disruptions in the rhythmic expression of gut genes and the secretion of hormones such as melatonin, which alters the microbial metabolic environment. Changes in the microbial metabolome, including bile acid metabolism, observed after periods of sleep deprivation, in turn affect motility, mucosal integration, and local immunoregulation. Experiments in animal models and observational studies in humans have documented reduced microbiome diversity, taxonomic shifts, and altered metabolite profiles following sleep disruption, which may favor ecological conditions that allow bacterial overgrowth in the small intestine [Lin et al., 2024; Sun et al., 2023]. From a clinical perspective, the implications are that assessing and treating sleep disorders and interventions that reduce chronic stress (e.g. cognitive behavioral therapy, relaxation techniques, sleep hygiene optimization) should be considered complementary strategies for preventing and treating SIBO. These interventions may improve gut motility, stabilize the microbiome, and strengthen the intestinal barrier, potentially reducing the risk of relapse after bacterial eradication. Prospective studies investigating the impact of sleep improvement and stress reduction on objective markers of SIBO (breath tests, aspirate/culture, sequencing) are also needed [Cheng et al., 2025; Lin et al., 2024].

## **Impact of Abdominal Surgery and Bariatric Procedures**

Gastrointestinal surgery, such as reconstruction, anastomosis, or resection, is a significant predisposing factor for the development of SIBO. In one study, 43% of patients who underwent bariatric surgery, such as Roux-en-Y or single-anastomosis gastric bypass, had a positive postoperative breath test. This indicates a very high prevalence of this condition. Diagnosis cannot be based solely on clinical symptoms or their severity. SIBO is still frequently underdiagnosed or misinterpreted, which increases the risk of metabolic complications and leads to adverse systemic effects related to changes in the gut microbiota [Novljan et al., 2022].

## **Diagnostic Challenges and Limitations**

SIBO diagnosis is currently based on two main approaches: direct microbiological examination of small intestinal aspirate (jejunal/duodenal aspirate with quantitative culture) and non-invasive breath tests (hydrogen and/or methane breath tests—GBT, LBT). While small bowel aspiration is classically regarded as the “gold standard”, the North American Consensus and European guidelines currently advocate for breath testing as the primary clinical diagnostic tool, citing the invasive nature, high cost, and variable reproducibility of aspirate culture. The quantitative criteria used in practice have changed over time (historically  $\geq 10^5$  CFU/ml, with some studies proposing lower thresholds, e.g.  $\geq 10^3$  CFU/ml), which impacts the comparability of studies and prevalence estimates. Aspirate has the advantage of directly documenting the presence and abundance of bacteria, but it is invasive, expensive, susceptible to contamination by oral or gastric flora, and limited to the sampling site (may not detect focal or distant overgrowth) [Silva et al., 2025; Kashyap et al., 2024]. Breath tests (glucose breath test (GBT) and lactulose breath test (LBT)) have become widely used diagnostic tools due to their noninvasive nature and low cost. However, their diagnostic performance is moderate and variable, with numerous meta-analyses and reviews indicating relatively low to moderate sensitivity and moderate specificity compared to aspirate culture. Comparative publications have emphasized that LBT sensitivity can be low (reported values in the literature often range around 30–42%), while GBT achieves slightly higher sensitivity values (with test-dependent ranges of around 40–55%), while specificity can be moderate (~70% or higher in various analyses). For this reason, positive breath test results must always be interpreted in the clinical context and, if necessary, confirmed with additional testing [Ghoshal, 2011; Rangan et al., 2022]. A significant limitation of breath testing is the influence of oral-cecal transit time (OCTT) and the variability of intestinal transit between individuals and over time within the same individual. In LBT, an early increase in hydrogen may reflect premature passage of substrates

into the colon (false positives), or conversely, a delayed transit may delay gas buildup and produce a false negative result. Hence, the need to correlate breath test results with OCTT measurement or motility assessment. Medications (PPIs, opioids), diet (last meal, FODMAP intake), and physical activity also modify OCTT and the luminal microenvironment, directly affecting breath test results. It should be added that a key criticism of glucose and lactulose breath tests is that early increases in hydrogen levels in exhaled air may be primarily due to increased fermentation in the intestine, rather than the actual presence of bacterial overgrowth in the small intestine. Therefore, studies linking lifestyle (a diet rich in fermentable carbohydrates) to the incidence of SIBO may yield different conclusions depending on the diagnostic method used [Jirapinyo et al., 2019; Lim et al., 2023]. Further limitations concern technical and interpretative standards. In the case of aspirate, heterogeneity of methods (collection site: duodenum vs. jejunum, transport and culture method, aerobic vs. anaerobic culture conditions) and arbitrary diagnostic thresholds are problematic. Breath tests, on the other hand, lack a clear, universally accepted definition of a positive result (different H<sub>2</sub>/CH<sub>4</sub> increase thresholds, different monitoring durations—120 vs. 180 minutes), which impacts the reproducibility and comparability of results between studies. Furthermore, the role of methane (produced by archaea) requires separate interpretation, as methane dominance is often associated with constipation and has a different clinical mechanism (intestinal methanogen overgrowth—IMO) than "classic" SIBO caused by an excess of fermenting bacteria [Pitcher et al., 2022; Tansel et al., 2023].

### **Influence of Diagnostic Methods on Research Outcomes**

The choice of diagnostic method has direct implications for investigating the relationship between SIBO and environmental factors or diet. Breath tests measure functional gas production from fermentation in the gastrointestinal lumen, so they are particularly sensitive to short-term changes in substrate supply (e.g. pre-test FODMAP intake), transit time, and the effects of medications that alter pH or motility (e.g. PPIs, prokinetics, opioids). In practice, this means that a diet rich in fermentable carbohydrates can increase the rate of positive breath tests even in the absence of persistent, quantitative bacterial overgrowth confirmed by aspirate. On the other hand, aspirate—despite its greater specificity in detecting actual bacterial colonization—may not reveal "functional" fermentation activity dependent on current dietary intake and may not capture heterogeneous/point foci of SIBO beyond the sampling site [Lim et al., 2023]. Consequently, epidemiological or interventional studies describing the impact of diet (e.g., FODMAP, low-carbohydrate diet) or lifestyle (smoking, alcohol, physical activity) on the "occurrence of SIBO" may yield contradictory results depending on whether breath tests or

aspirate were used as diagnostic criteria. For example, the association between proton pump inhibitor use and SIBO may be more pronounced in studies based on breath tests, which are sensitive to changes in pH and fermentation, than in studies using aspirate as the standard. Therefore, when assessing dietary or pharmacological effects, it is important to use standardized diagnostic criteria, multimodal diagnostics (breath tests and aspirate/sequencing), and analytical correction for potential confounding factors (transit time, antibiotics, last meal, PPI use) [Tansel et al., 2023; Su et al., 2018].

### **Clinical Implications and Recommendations**

Nutritional and lifestyle interventions can play a significant role in the prevention and treatment of Small Intestinal Bacterial Overgrowth (SIBO). In practice, it's worth considering: establishing regular breaks between meals ( $\geq 3$ –4 hours) and avoiding frequent snacking, as constant snacking throughout the day can exacerbate bloating and related symptoms. It's also recommended to limit excessive intake of fermentable carbohydrates, taking into account individual tolerance. In the context of dietary recommendations for SIBO, it's worth avoiding the typical Western diet, which is rich in refined sugar, animal fats, processed meats, refined grains, excess salt, and other highly processed foods. This type of diet promotes disruption of the gut microbiota, weakens the intestinal barrier, and increases its permeability, which can lead to the entry of unfavorable bacterial metabolites into the bloodstream. Therefore, in the case of SIBO, it's particularly important to limit these foods in favor of vegetables, fruits, whole grains, high-quality protein, fish, nuts, and seeds. Moderate physical activity and treatments that improve gastrointestinal motility can also be helpful, promoting physiological intestinal transit and reducing the risk of bacterial flora stagnation [Knez et al., 2024; Velasco-Aburto et al., 2025]. Due to the possible increased risk of SIBO development with long-term use of proton pump inhibitors, it is worthwhile to implement gradual withdrawal strategies when appropriate. Although appropriate procedures have been developed, they are still rarely implemented in daily practice in many healthcare systems. Broader education and practical implementation of these methods could reduce the risk of SIBO and related problems in individuals taking PPIs long-term [Khurmatullina et al., 2025]. Patient education and preventive measures: informing about the importance of regular mealtimes, a proper diet, physical activity, and the judicious use of medications can reduce the risk of SIBO and its recurrence. These measures should also be promoted in clinical practice and preventative healthcare.

### **Limitations of Current Data and Review**

Significant methodological heterogeneity in existing studies—different diagnostic methods (breath tests, small intestinal aspirate), varying criteria for SIBO diagnosis, and diverse patient

groups—makes it difficult to compare results and draw generalized conclusions. There are insufficient randomized controlled trials (RCTs) confirming the effectiveness of dietary or lifestyle interventions in preventing or treating SIBO. Many available studies are observational reports or reviews, limiting the strength of causal conclusions. Although dietary interventions for SIBO appear to have potential, currently available studies are sparse and have numerous limitations, making it difficult to draw firm clinical conclusions. Therefore, recommendations regarding elimination diets should be approached with caution, especially since severe forms of SIBO may be associated with malabsorption and an increased risk of deficiencies. The most rational approach appears to be a varied, nutritious diet rich in vegetables and whole grains, promoting a balanced microbiota. However, further, better-designed studies are needed to reliably assess the effectiveness and safety of diets in SIBO therapy [Souza et al., 2022]. Another limitation is the focus on publishing positive results while omitting others, which may overestimate the effectiveness of the intervention.

### **Conclusions and Recommendations for Future Research**

Well-designed RCTs should be conducted to assess the impact of specific interventions: dietary (regular meal patterns, low-FODMAP diet compared to a standard diet), lifestyle (physical activity), and pharmacotherapy modifications (reducing PPI and opioid intake) on SIBO recurrence rates, symptom prevalence, quality of life, and microbiological parameters. Prospective cohorts are needed to record risk factors (diet, medications, activity, lifestyle) and monitor SIBO progression using objective methods. Standardization of SIBO diagnosis is necessary, including standardized criteria and the use of multimodal approaches (breath test versus aspirate testing or modern molecular methods) to enable comparison of results between centers and individual patients. Incorporating patient education and conducting population-based and clinical health programs that promote habits conducive to a healthy microbiota and normal gastrointestinal motility is crucial.

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