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SMALL INTESTINAL MICROBIAL OVERGROWTH (SIBO, IMO, AND SIFO) IN THE PATHOGENESIS AND MANAGEMENT OF IRRITABLE BOWEL SYNDROME

Julia Danieluk *(Corresponding Author, Email: ji.danieluk@gmail.com)*
Clinical Hospital of Poznań University of Medical Sciences, 49 Przybyszewskiego Street, 60-355 Poznań, Poland
ORCID ID: 0009-0000-1513-6814

Natalia Adamaszek
Clinical Hospital of Poznań University of Medical Sciences, 49 Przybyszewskiego Street, 60-355 Poznań, Poland
ORCID ID: 0009-0007-7839-1581

Michał Gliński
Clinical Hospital of Poznań University of Medical Sciences, 49 Przybyszewskiego Street, 60-355 Poznań, Poland
ORCID ID: 0009-0003-1422-6940

Urszula Kacprzak
Clinical Hospital of Poznań University of Medical Sciences, 49 Przybyszewskiego Street, 60-355 Poznań, Poland
ORCID ID: 0009-0008-2380-3187

Dominika Dołęga-Mostowska
Medical University of Lodz, al. Tadeusza Kościuszki 4, 90-419 Łódź, Poland
ORCID ID: 0009-0003-5448-9956

Julia Sochowska
Clinical Hospital of Poznań University of Medical Sciences, 49 Przybyszewskiego Street, 60-355 Poznań, Poland
ORCID ID: 0009-0001-9484-6634

Agnieszka Buczkowska
Clinical Hospital of Poznań University of Medical Sciences, 49 Przybyszewskiego Street, 60-355 Poznań, Poland
ORCID ID: 0009-0009-3488-2949

Laura Stochaj
Provincial Hospital in Poznań, 7/19 Juraszów Street, 60-479 Poznań, Poland
ORCID ID: 0009-0004-0795-2992

Michalina Flejszman
Poznań University of Medical Sciences, 10 Fredry Street, 61-701 Poznań, Poland
ORCID ID: 0009-0007-9936-6975

Justyna Wójcik
Clinical Hospital of Poznań University of Medical Sciences, 49 Przybyszewskiego Street, 60-355 Poznań, Poland
ORCID ID: 0009-0007-2270-5027

ABSTRACT

Background. Irritable Bowel Syndrome (IBS) is a complex functional disorder characterized by abdominal pain and altered bowel habits. Small Intestinal Bacterial Overgrowth (SIBO) is increasingly recognized as a key pathogenetic factor, shifting the understanding of IBS toward a micro-organic basis. IBS is viewed as a heterogeneous condition with distinct phenotypes: microbiota-driven, post-infectious (PI-IBS), centrally mediated, and genetic variants.
Aim. To evaluate the role of SIBO, Intestinal Methanogen Overgrowth (IMO), and Small Intestinal Fungal Overgrowth (SIFO) in IBS pathogenesis, focusing on prevalence, mechanisms, and targeted therapy.
Material and methods. A literature review was performed using PubMed and Scopus (2000 - 2025). The analysis included 45 sources, including meta-analyses, systematic reviews, and clinical guidelines (Rome IV, Polish Society of Gastroenterology).
Results. Meta-analyses indicate that IBS patients are nearly five times more likely to test positive for SIBO than healthy individuals (OR 4.7). Hydrogen-producing bacteria are linked to diarrhea (IBS-D), while methane delays transit, correlating with constipation (IBS-C). Proposed mechanisms include excessive fermentation gas, bile acid deconjugation, and increased intestinal permeability. SIFO is found in 25% of refractory cases. Targeted eradication particularly with rifaximin achieves symptom resolution, with 48% of treated patients no longer meeting Rome criteria. However, diagnostic inconsistencies in breath tests remain a subject of clinical controversy.
Conclusions. SIBO and IMO represent treatable pathomechanisms in a subset of IBS patients, supporting a transition to personalized, mechanism-based management. Identifying these phenotypes is essential for optimizing targeted antimicrobial and prokinetic therapy. Individualized diagnostics are crucial for managing the micro-organic subtype of this complex syndrome.

KEYWORDS

IBS, SIBO, IMO, Dysbiosis, Microbiome, SIFO

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1. Introduction.

Irritable Bowel Syndrome (IBS) is a common and chronic disorder of gut-brain interaction (DGBI) characterized by recurrent abdominal pain associated with altered bowel habits (Karoń et al, 2024; Park, 2010). It affects approximately 4.1% to 11% of the global population, imposing a significant economic burden and severely reducing quality of life (Karoń et al, 2024; Poon, Law, Major & Andreyev, 2022). The condition can occur across all age groups; however, about half of patients report the onset of symptoms before the age of 35, with IBS being approximately twice as common in women as in men (Jaworowska, Dudzińska, Zagórska & Koch, 2025; Saha, 2014). The diagnosis of IBS has evolved from earlier subjective assessments to standardized symptom-based frameworks, currently culminating in the **Rome IV criteria (2016)** (Lacy & Patel, 2017; Mearin et al, 2016). Unlike previous iterations, Rome IV defines IBS by the presence of recurrent abdominal pain occurring, on average, at least one day per week during the last three months, with symptom onset at least six months prior to diagnosis (Mearin et al, 2016; Schmulson et al, 2025). A major change in Rome IV was the removal of the term “discomfort” from the definition, as it was deemed ambiguous and translated inconsistently across different languages (Lacy, 2017; Mearin et al, 2016). Additionally, the pain must be associated with two or more of the following:

- relation to defecation (either improvement or worsening of pain);
- a change in the frequency of stool;
- a change in the form (appearance) of stool (Lacy, 2017; Schmulson et al, 2025)

While abdominal bloating and distension are not mandatory for a diagnosis, they are now formally recognized as primary and prevalent symptoms (Lu et al, 2020; Shah et al, 2020). To provide targeted treatment, IBS is classified into subtypes based on the predominant bowel habit on days with abnormal bowel movements. The **Bristol Stool Form Scale (BSFS)** is the validated tool used for this classification, categorizing stool into seven types, which is visualised in ***Figure 1.:***

- **Types 1 - 2:** Separate hard lumps or lumpy sausage shapes, indicating constipation;
- **Types 3 - 4:** Sausage-like but with surface cracks or smooth/soft, considered normal;
- **Types 5 - 7:** Soft blobs with clear edges to entirely liquid, indicating diarrhea
- (Sieradzka, Wajda, Mika & Słowik, 2026)

TYPE & ILLUSTRATION	DESCRIPTION	
Type 1	Separate hard lumps (hard to pass)	Severe Constipation
Type 2	Sausage shaped but lumpy	Mild Constipation
Type 3	Like a sausage with cracks	Normal
Type 4	Like a snake, smooth and soft	Normal
Type 5	Soft blobs with clear edges (easily passed)	Lacking Fiber / Loose
Type 6	Fluffy pieces with ragged edges, mush	Mild Diarrhea
Type 7	Watery, entirely liquid, no solid pieces	Severe Diarrhea

Fig. 1. *The Bristol stool form scale (Lacy, 2017).*

The clinical stratification of IBS subtypes is based on the distribution of stool forms according to the Bristol Stool Scale (BSS), as illustrated in ***Figure 2***. This diagnostic framework follows the principle that 25% of BM is the threshold for classification, where the abbreviation BM stands for bowel movement. IBS with predominant diarrhea (IBS-D) is defined by >25% of bowel movements corresponding to Bristol stool types 6 - 7 and <25% corresponding to types 1 - 2. Conversely, IBS with predominant constipation (IBS-C) is characterized by >25% of bowel movements corresponding to types 1 - 2 and <25% corresponding to types 6 - 7. Mixed IBS (IBS-M) involves >25% of bowel movements of both types 6 - 7 and 1 - 2, while cases falling outside these parameters are classified as unclassified IBS (IBS-U) (Lacy, 2017; Mearin et al, 2016).

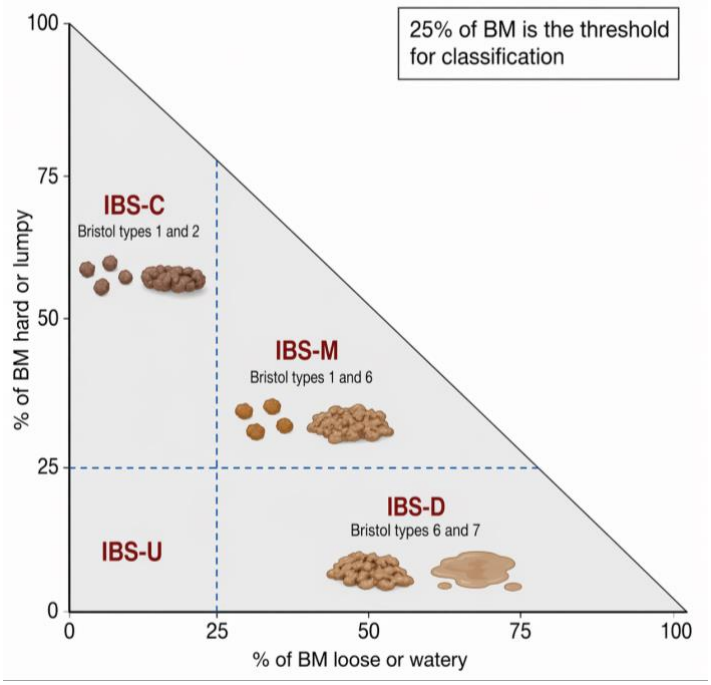

Fig. 2. *Classification of irritable bowel syndrome subtypes based on stool consistency according to the Bristol Stool Form Scale (BSFS) (Lacy, 2017).*

The exact etiology of IBS remains multifactorial and complex, currently understood as a dysregulation of the brain-gut axis (Hillestad et al, 2022; Park, 2010). This bidirectional communication between the central nervous system and the gastrointestinal tract is mediated by neural, hormonal, and immunological pathways, all of which are influenced by genetic predisposition, environmental triggers, and psychosocial factors (Hillestad et al, 2022).

For instance, polymorphisms in genes regulating the serotonergic system and familial clustering suggest a significant hereditary component (Saha, 2014). Physiologically, a hallmark of the condition is visceral hypersensitivity, where patients exhibit an abnormal pain response to normal stimuli (Wu et al, 2019). This is often linked to increased mast cell activation and altered sensory signal processing within the thalamus and prefrontal cortex (Ghoshal, Shukla & Ghoshal, 2017). The psychological dimension of IBS is equally profound, with psychiatric comorbidities such as anxiety, depression, and personality disorders occurring in 70 - 90% of patients (Hillestad et al, 2022; Schmulson et al, 2025). These psychosocial stressors can modulate gut reactivity through the hypothalamic-pituitary-adrenal axis, creating a cycle where emotional distress exacerbates physiological symptoms like pain and altered motility (Hillestad et al, 2022; Park, 2010). Growing evidence increasingly highlights the central role of the gut microbiota as a primary driver of IBS pathophysiology (Banaszak et al, 2023; Hillestad et al, 2022). Intestinal dysbiosis, characterized by a reduction in microbial diversity and shifts in the Firmicutes-to-Bacteroidetes (F/B) ratio, plays a vital role in symptom generation (Banaszak et al, 2023; Su et al, 2018). The F/B ratio serves as a key marker of microbial balance; in many IBS cohorts, an overabundance of *Firmicutes* and a relative depletion of *Bacteroidetes* are observed (Su et al, 2018; Zhuang et al, 2018). This microbial imbalance has direct functional consequences: an increase in *Firmicutes* can lead to enhanced gut barrier permeability, while a lower abundance of *Bacteroidetes* reduces the production of essential anti-inflammatory compounds, such as the short-chain fatty acid butyrate (Su et al, 2018; Yang et al, 2020). These alterations can increase mucosal permeability (leaky gut) and activate the enteric immune system, further contributing to visceral pain and hypersensitivity (Banaszak et al, 2023).

Within this framework of microbial imbalance, **Small Intestinal Bacterial Overgrowth (SIBO)** has emerged as a critical pathogenetic factor. SIBO is a heterogeneous clinical syndrome characterized by an abnormal quantitative and qualitative shift in the small intestinal microbiota, exceeding the physiological threshold of 10^3 - 10^4 CFU/mL (Bures et al, 2010; Wilk et al, 2025). This imbalance often involves the colonization of the upper gastrointestinal tract by colonic species, such as *Streptococcus*, *Escherichia coli*, *Staphylococcus*, and *Klebsiella* (Silva et al, 2025).Beyond these bacterial populations, recent clinical advancements emphasize the significance of other microbial kingdoms within this dysbiotic spectrum, particularly archaea and fungi (Litwiniuk et al, 2023). **Intestinal Methanogen Overgrowth (IMO)** is now recognized as a distinct entity driven predominantly by the archaeon *Methanobrevibacter smithii* (Gandhi et al, 2021). Concurrently, **Small Intestinal Fungal Overgrowth (SIFO**), primarily involving *Candida* species, has been increasingly identified as a contributor to unexplained gastrointestinal symptoms which mimic the symptoms of SIBO (Banaszak et al, 2023; Więckowska-Deroń et al, 2023). Under physiological conditions, the small intestine is protected by several endogenous “gatekeepers” that maintain a relatively sparse microbial environment (Wilk et al, 2025). These include gastric acid secretion, antegrade intestinal motility, specifically the migrating motor complex, an intact ileocecal valve to prevent retrograde translocation, and the bacteriostatic properties of pancreatic and biliary secretions (Silva et al, 2025). A failure in these mechanisms allows bacteria to proliferate, where their metabolites and enzymes induce direct damage to the intestinal (Ghoshal et al, 2017). This leads to the impairment of brush-border disaccharidases and subsequent maldigestion of carbohydrates (Bures et al, 2010). These undigested substrates undergo intraluminal fermentation, resulting in excessive gas production, such as bloating and flatulence, and osmotic diarrhea (Bures et al, 2010; Litwiniuk et al, 2023). Furthermore, bacterial deconjugation of bile acids impairs fat emulsification, potentially manifesting as steatorrhea and deficiencies in fat-soluble vitamins (Bures et al, 2010; Wilk et al, 2025). In severe cases, bacterial sequestering of vitamin B_{12} can lead to megaloblastic anemia and malnutrition (Bures et al, 2010; Silva et al, 2025). The diagnosis of SIBO remains a subject of ongoing clinical debate (Ghoshal & Srivastava, 2014; Wilk et al, 2025). While the microbiological assessment of jejunal aspirates is traditionally considered the gold standard requiring a finding of $\geq 10^3$ to 10^5 CFU/mL its invasive nature, high cost, and technical challenges make it unavailable in routine practice (Bures et al, 2010; Silva et al, 2025). Consequently, the medical community increasingly relies on non-invasive hydrogen (H_2) and methane (CH_4) breath tests (HBT) using glucose or lactulose substrates (Gandhi et al, 2021; Wilk et al, 2025). According to current consensus, a positive result is typically defined by an increase in H_2 of $\geq$ 20 ppm from

baseline within 90 minutes, or a concentration of $CH_4 \geq 10$ ppm at any point during the test (Gandhi et al, 2021; Shah et al, 2020).

The etiology of SIBO is multifactorial, typically involving a synergy of motility-related, structural, and systemic triggers. Impaired intestinal motility remains a cornerstone of its development and is frequently observed in systemic sclerosis, diabetic autonomic neuropathy, and within the context of Irritable Bowel Syndrome (IBS) itself (Bures et al, 2010; Zadrozna et al, 2022). This creates a bidirectional relationship between SIBO and IBS, potentially forming a self-perpetuating cycle of dysbiosis and gastrointestinal dysfunction (Ghoshal et al, 2017; Shah et al, 2020). Structural abnormalities that induce stasis such as intestinal adhesions, strictures common in Crohn's disease, small bowel diverticula, or a history of bariatric surgery and ileocecal resection further facilitate bacterial colonization (Bures et al, 2010; Silva et al, 2025). Additionally, digestive disorders such as exocrine pancreatic insufficiency and cystic fibrosis remove essential bacteriostatic components from the luminal environment (Bures et al, 2010; Wilk et al, 2025). Pharmacological influences also play a significant role; the use of opioids can severely depress motility, while long-term administration of proton pump inhibitors (PPIs) may facilitate overgrowth by removing the gastric acid barrier (Bures et al, 2010; Silva et al, 2025). Finally, systemic conditions such as liver cirrhosis and immunodeficiency states, along with advanced age, are recognized as independent risk factors that increase the prevalence of this condition (Bures et al, 2010; Ghoshal et al, 2014). Understanding the intricate role SIBO plays within this complex web of predisposing factors is essential for evaluating its potential impact on the clinical course and management of patients diagnosed with IBS (Ghoshal et al, 2014; Litwiniuk et al, 2023).

2. Discussion.

2.1 Pathomechanisms.

The clinical and pathophysiological convergence of Irritable Bowel Syndrome (IBS) and Small Intestinal Bacterial Overgrowth (SIBO) represents one of the most transformative paradigm shifts in modern neuro-gastroenterology (Ghoshal et al, 2017; Ghoshal et al, 2014). Historically classified as a purely functional or psychogenic disorder, IBS is now increasingly recognized as having a demonstrable micro-organic basis in a significant subset of the patient population (Ghoshal et al, 2017; Silva et al, 2025). Extensive meta-analyses of case-control studies confirm a robust association between these two conditions, with a pooled odds ratio (OR) ranging from 3.7 to 4.7 for SIBO prevalence in IBS patients compared to healthy controls (Ford, Spiegel, Talley, & Moayyedi, 2009; Shah et al, 2020). While reported prevalence rates vary significantly based on diagnostic methodology ranging from 19% in culture-based studies to as high as 78% in lactulose breath test (LBT) cohorts the pooled prevalence is consistently estimated at approximately 38% (Chen et al, 2018; Shah et al, 2020).

Contrary to traditional definitions of IBS as a non-inflammatory disorder, current research highlights **low-grade mucosal immune activation** as a primary driver of symptom severity (Ghoshal et al, 2017; Litwiniuk et al, 2023). The persistent presence of an abnormal bacterial load in the small intestine acts as a chronic antigenic stimulus, leading to histopathological changes such as increased intraepithelial lymphocyte counts observed in 15.4% of cases and significant mucosal thinning (Bures et al, 2010; Ghoshal et al, 2017). The subsequent compromise of the intestinal barrier acts as a pivotal trigger for the activation of mast cells, which serve as critical immune effector cells (Hillestad et al, 2022; Silva et al, 2025). In IBS cohorts, these cells exhibit increased density and heightened activity in close proximity to the enteric nerve plexuses, particularly the myenteric plexus (Ghoshal et al, 2017; Wu et al, 2019). Upon degranulation, mast cells release a potent array of chemical mediators and neurotransmitters including histamine, serotonin, and substance P thereby, sensitizing afferent sensory fibers (Bures et al, 2010; Ghoshal et al, 2017). Clinical evidence confirms that the physical proximity of these cells to nerve fibers correlates directly with the frequency and intensity of abdominal pain (Ghoshal et al, 2017; Hillestad et al, 2022). This chronic biochemical stimulation results in a significant reduction in the pain threshold, a phenomenon known as visceral hypersensitivity, causing physiological stimuli such as normal luminal gas, digestion, or routine peristaltic contractions to be misinterpreted by the nervous system as acute pain (Hillestad et al, 2022; Park, 2010). Ultimately, this hypersensitivity drives the recurrent abdominal pain required by the Rome IV criteria for a formal IBS diagnosis. This association is further substantiated by the observed elevation in fecal calprotectin, a marker of intestinal inflammation, which was significantly more frequent in SIBO-positive IBS patients ($p < 0.05$) (Chey, Shah, & DuPont, 2020; David, Babin, Picos & Dumitrascu, 2014; Wilk et al, 2025).

The intestinal barrier consists of a monolayer of epithelial cells that must be tightly connected to prevent the uncontrolled translocation of luminal contents into the bloodstream. This integrity is maintained by

specialized tight junction proteins, such as occludin, claudin-1, and zonula occludens-1 (ZO-1). These proteins act as a structural seal within the intercellular spaces, effectively regulating paracellular permeability. However, in IBS the **integrity of the intestinal barrier** is further compromised by a pathological microbiota, often characterized by a high *Firmicutes*-to-*Bacteroidetes* (F/B) ratio (Banaszak et al, 2023; Su et al, 2018). During SIBO, bacteria excessively ferment undigested carbohydrates and catabolize intraluminal proteins, producing toxic by-products such as D-lactate and ammonia (Banaszak et al, 2023; Bures et al, 2010). Structural components released from proliferating bacteria including peptidoglycans, endotoxins like lipopolysaccharide (LPS) and proteins like flagellin activate TLR receptors trigger the production of pro-inflammatory cytokines like IL-6, IL-8 and TNF-α, leading to chronic microscopic mucosal inflammation within the *lamina propria* (Bures et al, 2010; Hillestad et al, 2022). Toll-like receptor 4 (TLR-4) is responsible for recognizing lipopolysaccharides (LPS), which are components of the outer cell membrane of

Gram-negative bacteria (Chey et al, 2020). Conversely, TLR-5 specializes in detecting flagellin, the protein that constitutes bacterial flagella and TLR-2 plays a critical role in recognition of peptidoglycans. Additionally, TLR receptors initiate a signaling cascade leading to the activation of the nuclear factor NF-κB which is essential for TNF-α to induce an increase in intestinal barrier permeability, which creates a vicious cycle mechanism. This process induces direct damage to the intestinal villi and the brush border, impairing disaccharidases (e.g., lactase deficiency) and resulting in carbohydrate maldigestion (Bures et al, 2010). In this scenario, a significantly lowered expression of mRNA of tight junction proteins is observed. Their deficiency leads to the emergence of the phenomenon defined as leaky gut which facilitates the translocation of bacterial products and food antigens, fueling a self-perpetuating cycle of immune activation and motor dysfunction (Banaszak et al, 2023; Hillestad et al, 2022).

Alterations in **gastrointestinal motility** are both a prerequisite for and a consequence of SIBO, determined largely by the symbiotic "cross-feeding" relationship between hydrogen-producing bacteria and methanogenic archaea (IMO) (Gandhi et al, 2021; Ghoshal et al, 2017). Fermentative bacteria consume carbohydrates to produce hydrogen (H_2), which serves as a substrate for *Methanobrevibacter smithii* to generate methane (CH_4) (Gandhi et al, 2021; Wilk et al, 2025). Methane production is exceptionally strong and specific to the constipation phenotype (IBS-C), with an OR of 3.1 (95% CI: 1.7 - 5.6) compared to the diarrhea subtype (IBS-D) (Gandhi et al, 2021). Contemporary research identifies methane as an active neuromuscular transmitter that modulates intestinal physiology by increasing the amplitude of circular muscle contractions in the terminal ileum (Ghoshal et al, 2017; Lu et al, 2020). Rather than facilitating propulsive peristalsis, these contractions lead to luminal segmentation, which drastically retards intestinal transit (Ghoshal et al, 2017; Lu et al, 2020). This inhibitory effect is mediated through cholinergic pathways within the enteric nervous system (ENS) and can be blocked by atropine. The resulting prolonged transit time facilitates excessive water reabsorption in the colon, producing the hard stools characteristic of IBS-C (Gandhi et al, 2021; Ghoshal et al, 2017). In addition to motility changes, the premature colonization of the small intestine leads to the rapid fermentation of FODMAPs, releasing significant volumes of H_2, CH_4, and CO_2 (Ghoshal et al, 2017; Lacy, 2017). Because the small bowel is structurally narrow and acutely sensitive to pressure, this gaseous accumulation triggers luminal distension and visceral hypersensitivity, manifesting as the bloating and pain reported by up to 92% of IBS patients (Ghoshal et al, 2017; Lacy, 2017). Microbiological data (Wu et al, 2019) reveal that in SIBO-positive IBS-D patients, *Prevotella* species often become pathologically dominant, accounting for approximately 32% of the microbiota, while displacing beneficial genera such as *Bacteroides* (Bures et al, 2010; Wu et al, 2019). *Prevotella* possesses aggressive genetic clusters optimized for carbohydrate fermentation, and its abundance correlates positively with higher scores on the IBS Severity Scoring System (IBS-SSS) (Bures et al, 2010; Sieradzka et al, 2026). Furthermore, excessive short-chain fatty acids (SCFAs) generated by these bacteria induce dose-dependent visceral hypersensitivity and stimulate cytokine production (Bures et al, 2010; Wu et al, 2019).

Central to the IBS-D phenotype is the premature **deconjugation of bile salts** by the aberrant microbiota (Bures et al, 2010; Ghoshal et al, 2014). Deconjugated salts fail to form micelles, leading to fat malabsorption, steatorrhea, and subsequent deficiencies in fat-soluble vitamins, particularly vitamins A and D (Bures et al, 2010; Wilk et al, 2025). These liberated bile acids, such as lithocholic acid, exert a potent enterotoxic effect upon reaching the colonic mucosa, stimulating the secretion of water and electrolytes and driving secretory-osmotic diarrhea (Bures et al, 2010; Silva et al, 2025). Finally, proliferating anaerobic bacteria competitively sequester intraluminal vitamin B_{12}, potentially culminating in megaloblastic anemia and neurological sequelae such as polyneuropathy, thus establishing a definitive organic bridge between SIBO and the clinical manifestations of IBS (Bures et al, 2010; Silva et al, 2025).

2.2 Treatment strategies.

The recognition of Small Intestinal Bacterial Overgrowth (SIBO) as a primary pathomechanical driver has shifted therapeutic approaches for Irritable Bowel Syndrome (IBS) from palliative symptom management to mechanism-based microbial eradication (Ghoshal et al, 2017; Litwiniuk et al, 2023). The integration of artificial intelligence and radiomicrobiomics which links neural connectivity patterns from brain imaging with specific microbiota signatures is poised to revolutionize this patient stratification by uncovering complex interactions within the microbiota-gut-brain axis. This shift is substantiated by meta-analyses demonstrating that IBS patients are approximately 4.7 times more likely to harbor SIBO than healthy controls (OR 4.7; 95% CI 3.1 - 7.2) (Bures et al, 2010; Banaszak et al, 2023). This "bacterial hypothesis" is most rigorously supported by the clinical response to non-absorbable antibiotics, with the gastrointestinal-selective agent rifaximin considered the current gold standard (Chey et al, 2020; Wróblewski, Zimna, 2021). Data from dose-finding studies reveal a clear efficacy-dose relationship, where the rate of Glucose Hydrogen Breath Test (GHBT) normalization was significantly higher with a dose of 1600 mg/day (80%) compared to 1200 mg/day (58%; $p < 0.05$) (Bures et al, 2010; Wróblewski et al, 2021). Direct evidence for the SIBO-IBS causal link is further provided by landmark research demonstrating that successful SIBO eradication led to symptom resolution such that 48% of subjects no longer met Rome criteria post-treatment (Park, 2010; Pimentel, Chow, & Lin 2000). Complexity arises in cases of Intestinal Methanogen Overgrowth (IMO), typically associated with the IBS-C subtype, where monotherapy often fails (Gandhi et al, 2021; Ghoshal et al, 2017). In these methanogenic phenotypes, combination therapy with neomycin and rifaximin is superior, improving colonic transit and normalizing breath tests (Gandhi et al, 2021; Wróblewski et al, 2021). Alternative effective agents include ciprofloxacin and metronidazole, which have been shown to significantly decrease viable bacterial counts (Chey et al, 2020; Saha, 2014). Furthermore, it must not be overlooked that SIFO often represents the underlying cause of treatment-resistant symptoms. SIFO management involves a strategic choice between luminal agents, like the non-absorbable nystatin, and systemic triazoles, such as fluconazole (Banaszak et al, 2023; Wilk et al, 2025). Nystatin offers a superior safety profile through localized action, while fluconazole provides broader efficacy for refractory cases, though it requires hepatic monitoring. Integrating these antifungals is vital for managing IBS patients unresponsive to standard antibiotic therapy (Banaszak et al, 2023; Litwiniuk et al, 2023). Dietary interventions serve as essential adjuncts by reducing fermentable substrates available for bacterial metabolism; the Low-FODMAP Diet (LFD) is the most effective model, achieving symptom relief in approximately 70% of patients (Bures et al, 2010; Strużek et al, 2025). However, long-term adherence is cautioned against due to potential reductions in beneficial Bifidobacterium populations (Hillestad et al, 2022; Strużek et al, 2025). In the Polish clinical landscape, the Polish Society of Gastroenterology strongly recommends soluble fiber (10 - 25 g/day) and peppermint oil (180 - 225 mg bid) as first-line non-pharmacological interventions (Gajewski, 2024; Jaworowska et al, 2025). Additionally, the Mediterranean Low-FODMAP Diet is emerging as a sustainable long-term strategy, combining anti-inflammatory benefits with gas restriction (Bures et al, 2010; Siragusa et al, 2021). As these organic models are refined, clinical management must also evolve toward precision nutrition and the implementation of next-generation prebiotics that support epithelial repair by modulating tight junction proteins. Such targeted strategies are designed to provide symptomatic relief without the detrimental loss of beneficial taxa, such as *Bifidobacterium* and *Faecalibacterium prausnitzii*, often observed with long-term restrictive diets. Probiotics, increasingly utilized to restore microbial diversity and strengthen the intestinal barrier, include specific strains like Lactobacillus acidophilus, which modulate intestinal pain by inducing the expression of opioid and cannabinoid receptors (Banaszak et al, 2023). When visceral hypersensitivity dominates the clinical picture, neuromodulators are indicated, with the largest evidence base supporting tricyclic antidepressants (TCAs), such as amitriptyline, and selective serotonin reuptake inhibitors (SSRIs) (Łabuda et al, 2025; Saha, 2014). Notably, clinical trials in IBS-D patients found that low-dose amitriptyline improved stool frequency and reduced the sensation of incomplete evacuation. Despite the success of mechanism-targeted drugs, clinical practice in Poland is restricted by the unavailability of certain FDA-approved agents. While linaclotide is authorized and available in Poland as Constella, other medications such as lubiprostone, alosetron, and eluxadoline are currently not available in the Polish market (Gajewski, 2024; Saha, 2014). This limitation necessitates a rigorous focus on the identification and eradication of SIBO, alongside the optimized use of available antispasmodics, TCAs, and specialized dietary protocols to ensure effective clinical management (Jaworowska et al, 2025; Litwiniuk et al, 2023).

2.3 Limitations of current diagnostic methodologies.

The clinical assessment of Small Intestinal Bacterial Overgrowth (SIBO) within the context of Irritable Bowel Syndrome (IBS) is significantly hindered by the absence of a universally validated diagnostic gold standard, leading to wide variations in reported prevalence rates ranging from 4% to 84% (Chen et al, 2018; Shah et al, 2020). Traditionally, the quantitative culture of proximal jejunal fluid was considered the most reliable method, typically defined by a bacterial count of 10^5 colony-forming units (CFU/mL) (Bures et al, 2010; Wilk et al, 2025). However, this "tarnished" gold standard faces critical challenges, including its invasive nature, high cost, and inability to detect distal overgrowth in the ileum (Silva et al, 2025). Furthermore, culture-based methods are prone to false positives from oropharyngeal contamination and false negatives due to the fact that nearly 70% of intestinal bacteria are difficult to cultivate on conventional media (Bures et al, 2010; Wilk et al, 2025). Recent consensus suggests that a lower threshold of 10^3 CFU/mL may be more clinically relevant for the proximal bowel, a shift that meta-analyses show more than doubles SIBO prevalence in IBS patients from 14% to 33% (Chen et al, 2018; Shah et al, 2020).

Due to these limitations, non-invasive hydrogen (H_2) and methane (CH_4) breath tests (HBT) have emerged as the primary indirect tools for evaluating SIBO (Gandhi et al, 2021; Wilk et al, 2025). The Glucose Hydrogen Breath Test (GHBT), utilizing a substrate rapidly absorbed in the proximal intestine, serves as a highly specific indicator of proximal SIBO, typically defined by a rise of ≥ 20 ppm in hydrogen, though it remains insensitive to distal overgrowth (Litwiniuk et al, 2023; Silva et al, 2025). In contrast, the Lactulose Hydrogen Breath Test (LHBT) uses a non-absorbable disaccharide that reaches the distal bowel, allowing for assessment of the entire intestinal length (Gandhi et al, 2021; Silva et al, 2025). However, LHBT carries a significant risk of false positives, particularly in the IBS-D subtype where rapid intestinal transit may cause premature colonic entry of the substrate, mimicking SIBO (Ghoshal et al, 2017; Ghoshal et al, 2014).

These diagnostic complexities are highlighted in landmark investigations, such as the study by Ghoshal et al., which utilized GHBT to demonstrate that SIBO was significantly more prevalent in patients with chronic non-specific diarrhea (CNSD) at 21.9%, compared to other IBS forms (8.5%) and healthy controls (2%) (Ghoshal, Nehra, Mathur & Rai, 2019; Ghoshal et al, 2010). While these results suggest that clinicians should maintain a higher index of suspicion for SIBO in diarrhea-predominant populations, researchers caution that prevalence figures in the IBS-D group may still be overestimated due to rapid intestinal transit acting as a potential confounder, even when glucose is employed as the substrate (Ghoshal et al, 2017; Ghoshal et al, 2014).

3. Conclusions.

The identification of Small Intestinal Bacterial Overgrowth (SIBO) as a potential driver of Irritable Bowel Syndrome (IBS) represents a transformative shift in clinical gastroenterology, effectively bridging the historical gap between functional and organic disorders (Ghoshal et al, 2017; Ghoshal et al, 2014). Extensive epidemiological data, including meta-analyses reporting a pooled odds ratio of 4.7 (OR 4.7; 95% CI 3.1 - 7.2) for SIBO in IBS patients, confirm that this association is a consistent biological reality across diverse populations (Ford et al, 2009; Shah et al, 2020). Pathophysiological evidence suggests that SIBO does not merely coexist with IBS but directly contributes to symptom generation through shared mechanisms within the gut-immune-microbiota axis (Hillestad et al, 2022; Wu et al, 2019).

A key element of this interaction is the disruption of intestinal barrier integrity, manifested by reduced mRNA expression of tight junction proteins such as occludin and claudin-1 (Banaszak et al, 2023; Hillestad et al, 2022). This leads to the translocation of bacterial antigens, including lipopolysaccharide (LPS) and flagellin, into the intestinal lamina propria (Ghoshal et al, 2017; Giorgio et al, 2013). In this context, it is of groundbreaking importance that treatment with rifaximin extends beyond pure antibiotic action. Studies demonstrate that rifaximin can directly inhibit the activation of Toll-like receptor 4 (TLR-4) induced by LPS (Chey et al, 2020; Wróblewski et al, 2021). This process leads to a significant reduction in the production of pro-inflammatory cytokines (such as IL-6 and TNF-α) and chemokines, which consequently results in the suppression of low-grade inflammation and the normalization of visceral hypersensitivity (Chey et al, 2020; Wróblewski et al, 2021).

This pathophysiology differs depending on the type of overgrowth: while classic SIBO and the presence of *Prevotella* species promote the diarrhea-predominant phenotype (IBS-D), the overgrowth of methanogenic archaea currently classified as Intestinal Methanogen Overgrowth (IMO) is associated with the production of methane, which delays intestinal transit, correlating with the constipation-predominant type (IBS-C) (Banaszak et al, 2023; Gandhi et al, 2021). At the same time, Small Intestinal Fungal Overgrowth (SIFO)

represents an often-overlooked factor contributing to treatment-resistant gastrointestinal symptoms, which underscores the need to broaden diagnostics beyond bacterial populations (Banaszak et al, 2023; Wilk et al, 2025).

The therapeutic implications of recognizing these connections are profound. Clinical trials show that targeted antibiotic therapy with rifaximin allows for SIBO eradication in nearly 80% of cases (Bures et al, 2010; Wróblewski et al, 2021). Most importantly, it has been demonstrated that the successful elimination of overgrowth leads to the resolution of IBS diagnostic criteria in 48% of patients, suggesting that for nearly half of the IBS population, this disorder represents a treatable organic syndrome rather than a primary dysfunction of the central nervous system (Park, 2010; Pimentel et al, 2000).

Ultimately, the medical community must transition from a traditional reliance on a broad functional diagnosis toward a personalized, mechanism-based management paradigm (Jaworowska et al, 2025; Litwiniuk et al, 2023). Embracing a classification system that stratifies patients into distinct pathophysiological phenotypes specifically micro-organic (microbiota-driven), post-infectious (PI-IBS), and gut-barrier-related is essential to optimize targeted therapeutic interventions and reduce the global health burden of this complex syndrome (Barbara et al, 2019; Hillestad et al, 2022). The integration of artificial intelligence and radiomicrobiomics which links neural connectivity patterns from brain imaging with specific microbiota signatures is poised to revolutionize this patient stratification by uncovering complex interactions within the microbiota-gut-brain axis. As these organic models are refined, clinical management must also evolve toward precision nutrition and the implementation of next-generation prebiotics that support epithelial repair by modulating tight junction proteins. Such targeted strategies are designed to provide symptomatic relief without the detrimental loss of beneficial taxa, such as *Bifidobacterium* and *Faecalibacterium prausnitzii*, often observed with long-term restrictive diets. Finally, establishing standardized diagnostic protocols and higher-sensitivity testing is the necessary requirement to fully transition IBS from a diagnosis of exclusion to a manageable, evidence-based micro-organic syndrome (Hillestad et al, 2022; Siragusa et al, 2021).

Author's contribution:
Conceptualization: Julia Danieluk and Natalia Adamaszek;
methodology: Dominika Dołęga-Mostowska; software: Michał Gliński;
check: Julia Sochowska, Agnieszka Buczkowska and Justyna Wójcik;
formal analysis: Michał Gliński; investigation: Julia Danieluk and Urszula Kacprzak;
resources: Michalina Flejszman; data curation: Laura Stochaj;
writing-rough preparation: Julia Danieluk; writing-review and editing: Michalina Flejszman and Dominika Dołęga- Mostowska;
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REFERENCES

1. Banaszak, M., Górna, I., Woźniak, D., Przysławski, J., & Drzymała-Czyż, S. (2023). Association between gut dysbiosis and the occurrence of SIBO, LIBO, SIFO, and IMO. *Microorganisms, 11*(3), 573. https://doi.org/10.3390/microorganisms11030573
2. Barbara, G., Grover, M., Bercik, P., Corso, G., Cremon, C., Ohman, L., & Spiller, R. C. (2019). Rome Foundation working team report on post-infection irritable bowel syndrome. *Gastroenterology, 156*(1), 46–58.e7. https://doi.org/10.1053/j.gastro.2018.07.011
3. Bures, J., Cyrany, J., Kohoutova, D., Förstl, M., Rejchrt, S., Kvetina, J., Vorisek, V., & Kopacova, M. (2010). Small intestinal bacterial overgrowth syndrome. *World Journal of Gastroenterology, 16*(24), 2978–2990. https://doi.org/10.3748/wjg.v16.i24.2978
4. Chen, B., Kim, J., Zhang, Y., Du, L., & Dai, N. (2018). Prevalence and predictors of small intestinal bacterial overgrowth in irritable bowel syndrome: A systematic review and meta-analysis. *Journal of Gastroenterology, 53*, 807–818. https://doi.org/10.1007/s00535-018-1476-9
5. Chey, W. D., Shah, E. D., & DuPont, H. L. (2020). Mechanism of action and therapeutic benefit of rifaximin in patients with irritable bowel syndrome: A narrative review. *Therapeutic Advances in Gastroenterology, 13*, 1756284819897531. https://doi.org/10.1177/1756284819897531
6. David, L., Babin, A., Picos, A., & Dumitrascu, D. L. (2014). Small intestinal bacterial overgrowth is associated with intestinal inflammation in irritable bowel syndrome. *Journal of Gastrointestinal and Liver Diseases, 23*(4), 403–408. https://doi.org/10.3748/wjg.v20.i10.2482
7. Ford, A., Spiegel, B., Talley, N., & Moayyedi, P. (2009). Small intestinal bacterial overgrowth in irritable bowel syndrome: Systematic review and meta-analysis. *Clinical Gastroenterology and Hepatology, 7*(12), 1279–1286. https://doi.org/10.1016/j.cgh.2009.06.031
8. Gajewski, P. (Ed.). (2024). *Interna Szczeklika 2024*. Medycyna Praktyczna.
9. Gandhi, A., Shah, A., Jones, M. P., Koloski, N., Talley, N. J., Morrison, M., & Holtmann, G. (2021). Methane-positive small intestinal bacterial overgrowth in inflammatory bowel disease and irritable bowel syndrome: A systematic review and meta-analysis. *Gut Microbes, 13*(1), 1933313. https://doi.org/10.1080/19490976.2021.1933313
10. Ghoshal, U., Nehra, A., Mathur, A., & Rai, S. (2019). A meta-analysis on small intestinal bacterial overgrowth in patients with different subtypes of irritable bowel syndrome. *Journal of Gastroenterology and Hepatology, 35*, 922–931. https://doi.org/10.1111/jgh.14938
11. Ghoshal, U. C., Kumar, S., Mehrotra, M., Lakshmi, C. P., & Misra, A. (2010). Frequency of small intestinal bacterial overgrowth in patients with irritable bowel syndrome and chronic non-specific diarrhea. *Journal of Neurogastroenterology and Motility, 16*(1), 40–46. https://doi.org/10.5056/jnm.2010.16.1.40
12. Ghoshal, U. C., Shukla, R., & Ghoshal, U. (2017). Small intestinal bacterial overgrowth and irritable bowel syndrome: A bridge between functional organic dichotomy. *Gut and Liver, 11*(2), 196–208. https://doi.org/10.5009/gnl16126
13. Ghoshal, U. C., & Srivastava, D. (2014). Irritable bowel syndrome and small intestinal bacterial overgrowth: Meaningful association or unnecessary hype. *World Journal of Gastroenterology, 20*(10), 2482–2491. https://doi.org/10.3748/wjg.v20.i10.2482
14. Giorgio, V., Scarpellini, E., Roccarina, D., Cammarota, G., Pantanella, A., & Fundarò, C. (2013). Prevalence of small bowel bacterial overgrowth in children with irritable bowel syndrome: A case-control study. *Journal of Pediatric Gastroenterology and Nutrition, 57*(5), 611–615. https://doi.org/10.1016/j.jpeds.2009.03.033
15. Hillestad, E. M. R., van der Meeren, A., Nagaraja, B. H., Bjørsvik, B. R., Haleem, N., Benitez-Paez, A., Sanz, Y., Hausken, T., Lied, G. A., Lundervold, A., & Berentsen, B. (2022). Gut bless you: The microbiota-gut-brain axis in irritable bowel syndrome. *World Journal of Gastroenterology, 28*(4), 412–431. https://doi.org/10.3748/wjg.v28.i4.412
16. Jaworowska, A., Dudzińska, E., Zagórska, J., & Koch, W. (2025). Pathogenesis and treatment of irritable bowel syndrome. *Medical Studies/Studia Medyczne, 41*(3), 179–188. https://doi.org/10.5114/ms.2025.154432
17. Karoń, Ł., Zygmunt, A., Karoń, K., Pedrycz, D., Drapała, G., Grabowski, W., & Pedrycz, E. (2024). Targeting the microbiome: The role of low-FODMAP diet in modulating gut health for IBS and inflammatory conditions. *Journal of Education, Health and Sport, 75*, 56153. https://doi.org/10.12775/JEHS.2024.75.56153
18. Łabuda, M., Bogucka, A., Mentel, O., Sowińska, T., Szema, A., Sikora, J., & Knychalska, K. (2025). Mechanism of action, efficacy, and adverse events of selected drugs used to treat irritable bowel syndrome (IBS): A literature review. *Journal of Education, Health and Sport, 81*, 59982. https://doi.org/10.12775/JEHS.2025.81.59982
19. Lacy, B. E., & Patel, N. K. (2017). Rome criteria and a diagnostic approach to irritable bowel syndrome. *Journal of Clinical Medicine, 6*(11), 99. https://doi.org/10.3390/jcm6110099
20. Litwiniuk, M., Zaniuk, M., Hurkała, K., Antonik, D., Denys, B., Góra, K., & Iberszer, K. (2023). Treatment of small intestinal bacterial overgrowth: Conventional antibiotic therapy and alternative therapy—Probiotics and low FODMAP diet. *Journal of Education, Health and Sport, 41*(1), 57–69. https://doi.org/10.12775/JEHS.2023.41.01.005

21. Lu, S., Chen, Y., Guo, H., Liu, Z., Du, Y., & Duan, L. (2020). Differences in clinical manifestations and the fecal microbiome between irritable bowel syndrome and small intestinal bacterial overgrowth. *BMC Gastroenterology, 20*, 132. https://doi.org/10.1016/j.dld.2024.07.011
22. Mayo Clinic. (2023). *Small intestinal bacterial overgrowth (SIBO): Symptoms and causes*. https://www.mayoclinic.org/diseases-conditions/small-intestinal-bacterial-overgrowth/symptoms-causes/syc-20370168
23. Mearin, F., Lacy, B. E., Chang, L., Chey, W. D., Lembo, A. J., Simren, M., & Spiller, R. (2016). Bowel disorders. *Gastroenterology*. Advance online publication. https://doi.org/10.1053/j.gastro.2016.02.031
24. Park, H. (2010). The role of small intestinal bacterial overgrowth in the pathophysiology of irritable bowel syndrome. *Journal of Neurogastroenterology and Motility, 16*, 3–4. https://doi.org/10.5056/jnm.2010.16.1.3
25. Pawełek, K., Marta, P. K., Huzarski, F. M., Ferfecka, G. M., Rosa-Bończak, M., Ossolińska, A., & Morawiecka, N. (2025). What do gases in the large intestine have to do with health? *Journal of Education, Health and Sport, 78*, 57693. https://doi.org/10.12775/JEHS.2025.78.57693
26. Pimentel, M., Chow, E. J., & Lin, H. C. (2000). Eradication of small intestinal bacterial overgrowth reduces symptoms of irritable bowel syndrome. *American Journal of Gastroenterology, 95*(12), 3503–3506. https://doi.org/10.1111/j.1572-0241.2000.03368.x
27. Poon, D., Law, G., Major, G., & Andreyev, H. (2022). A systematic review and meta-analysis on the prevalence of non-malignant, organic gastrointestinal disorders misdiagnosed as irritable bowel syndrome. *Scientific Reports, 12*. https://doi.org/10.1038/s41598-022-05933-1
28. Saha, L. (2014). Irritable bowel syndrome: Pathogenesis, diagnosis, treatment, and evidence-based medicine. *World Journal of Gastroenterology, 20*(22), 6759–6773. https://doi.org/10.3748/wjg.v20.i22.6759
29. Sánchez-Pellicer, P., Álamo-Marzo, J. M., Martínez-Villaescusa, M., Núñez-Delegido, E., Such-Ronda, J. F., Huertas-López, F., Serrano-López, E. M., Martínez-Moreno, D., & Navarro-López, V. (2021). Comparative analysis of gut microbiota in patients with irritable bowel syndrome and healthy controls. *Biomedicines, 9*(10), 1431. https://doi.org/10.3390/jcm14041198
30. Schmulson, M. J., Cruz-Rico, C. L., Mendoza-Domínguez, G., Zaragoza-Galicia, S. A., & Morales-Guzmán, A. S. (2025). Diagnosis of irritable bowel syndrome. *Clínicas de Gastroenterología de México, 25*(1), 1–9. https://doi.org/10.24875/CGME.M25000006
31. Shah, A., Talley, N. J., Jones, M., Kendall, B. J., Koloski, N., Walker, M. M., Morrison, M., & Holtmann, G. J. (2020). Small intestinal bacterial overgrowth in irritable bowel syndrome: A systematic review and meta-analysis of case-control studies. *The American Journal of Gastroenterology, 115*(2), 165–178. https://doi.org/10.14309/ajg.0000000000000504
32. Sieradzka, I., Wajda, K., Mika, W., & Słowik, J. (2026). Dietary and lifestyle factors associated with small intestinal bacterial overgrowth (SIBO): A review from a health education perspective. *Journal of Education, Health and Sport, 87*, 67772. https://doi.org/10.12775/JEHS.2026.87.67772
33. Silva, B. C. da, Ramos, G. P., Barros, L. L., Ramos, A. F. P., Domingues, G., Chinzon, D., & Passos, M. do C. F. (2025). Diagnosis and treatment of small intestinal bacterial overgrowth: An official position paper from the Brazilian Federation of Gastroenterology. *Arquivos de Gastroenterologia, 62*, e24107. https://doi.org/10.1590/S0004-2803.24612024-107
34. Siragusa, N., Baldassari, G., Ferrario, L., Passera, L., Rota, B., Pavan, F., Santagata, F., Capasso, M., Londoni, C., Manfredi, G., Consalvo, D., Lasagni, G., Pozzi, L., Lombardo, V., Mascaretti, F., Scricciolo, A., Roncoroni, L., Elli, L., Vecchi, M., & Costantino, A. (2021). The ten dietary commandments for patients with irritable bowel syndrome: A narrative review with pragmatic indications. *Nutrients, 13*(10), 3443. https://doi.org/10.3390/nu17152496
35. Spiegel, B. M. R. (2011). Questioning the bacterial overgrowth hypothesis of irritable bowel syndrome: An epidemiologic and evolutionary perspective. *Gastroenterology, 140*(6), 1868–1871. https://doi.org/10.1016/j.cgh.2011.02.030
36. Strużek, K., Karamus, K., Rejmak, R., Biłogras, J., Borowska-Łygan, M., Urban, W., & Tomaszewski, J. (2025). Low-FODMAP diet in the treatment of irritable bowel syndrome—Efficacy, comparison with other diets, mechanisms, and clinical applications. *Journal of Education, Health and Sport, 80*, 59059. https://doi.org/10.12775/JEHS.2025.80.59059
37. Su, T., Liu, R., Lee, A., Long, Y., Du, L., Lai, S., Chen, X., Wang, L., Si, J., Owyang, C., & Chen, S. (2018). Altered intestinal microbiota with increased abundance of *Prevotella* is associated with high risk of diarrhea-predominant irritable bowel syndrome. *Gastroenterology Research and Practice, 2018*, 6961783. https://doi.org/10.1155/2018/6961783
38. Więckowska-Deroń, M., Leszcz, M., Bieńko, K., Białek, J., Ćwik-Błotnicka, D., & Borczyk, J. (2023). SIBO—The present knowledge within the contekst of clinical dependencies and therapy. *Journal of Education, Health and Sport, 23*(1), 22–27. https://doi.org/10.12775/JEHS.2023.23.01.002
39. Wilk, K., Witkowska, E., Ciechański, M., Cholewiński, S., Wilk, B., Stachura, P., & Kasprzyk, A. (2025). Understanding SIBO: A comprehensive review of causes, symptoms, diagnosis, and treatment strategies of small intestinal bacterial overgrowth. *Journal of Education, Health and Sport, 82*, 60508. https://doi.org/10.12775/JEHS.2025.82.60508

40. Wróblewski, H., & Zimna, A. (2021). Use of rifaximin in irritable bowel syndrome treatment. *Journal of Education, Health and Sport, 11*(8), 247–254. https://doi.org/10.12775/JEHS.2021.11.08.025
41. Wu, K.-Q., Sun, W.-J., Li, N., Chen, Y.-Q., Wei, Y.-L., & Chen, D.-F. (2019). Small intestinal bacterial overgrowth is associated with diarrhea-predominant irritable bowel syndrome by increasing mainly *Prevotella* abundance. *Annals of Translational Medicine, 7*(23), 741. https://doi.org/10.1080/00365521.2019.1694067
42. Yamini, D., & Pimentel, M. (2008). Irritable bowel syndrome and small intestinal bacterial overgrowth. *American Journal of Gastroenterology, 103*(12), 3203–3206. https://doi.org/10.1111/j.1572-0241.2008.02258.x
43. Yang, M., Zhang, L., Hong, G., Li, Y., Li, G., Qian, W., Xiong, H., Bai, T., Song, J., & Hou, X. (2020). Duodenal and rectal mucosal microbiota related to small intestinal bacterial overgrowth in diarrhea-predominant irritable bowel syndrome. *Journal of Gastroenterology and Hepatology, 35*(5), 795–803. https://doi.org/10.1111/jgh.14910
44. Zadrozna, K., Wysokińska, O., Małek, A., Iwaniszyn-Zapołoch, K., Wójcik, B., Fabiś, M., & Żyga, J. (2022). Diagnosis and treatment of small intestinal bacterial overgrowth. *Journal of Education, Health and Sport, 12*(12), 42–48. https://doi.org/10.12775/JEHS.2022.12.12.006
45. Zhuang, X., Tian, Z., Li, L., Zeng, Z., Chen, M., & Xiong, L. (2018). Fecal microbiota alterations associated with diarrhea-predominant irritable bowel syndrome. *Frontiers in Microbiology, 9*, 1600. https://doi.org/10.3389/fmicb.2018.01600