

REVIEW PAPER

IMPORTANCE OF SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO)
IN THE ETIOPATHOGENESIS OF CHRONIC DISEASES

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Summary

Small intestinal bacterial overgrowth (SIBO) is a condition characterized by the abnormal proliferation of commensal or pathogenic microorganisms within the small intestine. There is increasing evidence that SIBO plays a significant role in the development of various chronic diseases, including systemic autoimmune disorders, metabolic syndromes and neurodegenerative conditions. Dysbiosis in the small intestine can disrupt the integrity of the mucosal barrier, induce chronic low-grade inflammation, alter intestinal motility and modulate immune responses. This can contribute to systemic pathologies. Furthermore, SIBO may exacerbate nutrient malabsorption, leading to deficiencies that can accelerate disease progression. Understanding the mechanistic links between SIBO and chronic diseases is essential for developing targeted therapeutic strategies, including antibiotic treatment and dietary interventions. This review critically examines the role of SIBO in the pathophysiological continuum of chronic diseases, emphasizing the need for greater clinical awareness and more robust diagnostic and therapeutic protocols.

Keywords: bacterial overgrowth syndrome, dysbiosis, gut microbiota, chronic diseases, inflammation

Introduction

The human digestive tract is the second largest system in the body after the cardiovascular system [1]. By definition, the gut microbiota is a collection of all

microorganisms found in the human digestive tract [1,2]. These microorganisms (including all bacteria, viruses, fungi, archaea and protozoa) number approximately 10^{14} CFU/ml (CFU/ml – colony-forming units per millilitre). The human gut microbiota is made up of around 90% bacteria from the *Firmicutes* and *Bacteroidetes* classes [1-3]. These microorganisms are associated with the proper functioning of the human body, including maintaining intestinal integrity, protecting against pathogens, forming the intestinal epithelium, producing vitamins and supporting the immune system [1-3]. The lowest number of microorganisms is found in the stomach and duodenum, where concentrations range from 10^3 to 10^4 CFU/ml due to the low pH and a very short transit time of food in these organs. The highest concentrations of bacteria are found in the ileum, where the concentrations are about 10^8 CFU/ml, and in the colon, with approximately 10^{11} CFU/ml [2,4]. The large intestine is home to the richest reservoir of intestinal microorganisms, with a mass of bacteria amounting to 1.5-2 kg living there. Differences in microbiota composition between different individuals are related to human population diversity, including living environment, genotype, age, health condition and diet. Despite growing interest in the human intestinal microbiota, its composition remains poorly understood [2-5]. Changes in the composition or number of microorganisms, i.e. dysbiosis, may be related to abdominal pain, diarrhea, bloating, gas and constipation, and can disrupt the body's homeostasis. These gastrointestinal problems may result in reduced nutrient absorption and deficiencies, as well as hypoproteinemia and anemia [1-4]. An increased permeability of lipopolysaccharides (LPS) can trigger an inflammatory response, which is the main cause of intestinal microbiota overgrowth [2]. Small intestinal bacterial overgrowth (SIBO) is a condition associated with differences in the quality, quantity and location of the small intestinal microbiota [1,4,6]. The functional symptoms of the gastrointestinal tract that accompany SIBO can alter the clinical presentation of chronic diseases and exacerbate their progression [6]. SIBO is characterized

by poor absorption, impaired gastric acid secretion, poorer digestion of food, abnormal intestinal anatomy and impaired gastrointestinal motor function [7]. Changes in the intestinal microbiota affect the metabolism of immune, immunosuppressive and inflammatory cells in diseases related to the body's immunity [8]. The presence of SIBO and many other conditions – including cardiovascular diseases, gastrointestinal disorders, autoimmune diseases, neurological diseases, metabolic diseases, dermatological diseases, genetic diseases, endocrine diseases, mental health conditions, nephrological diseases, developmental disorders and gastrointestinal cancers, are connected to the composition of an individual's intestinal microbiota [1,8].

Aim of the work

The aim of this review paper is to present the relationship between gut microbiota and SIBO and to demonstrate its impact on the development of various chronic diseases, which may be helpful to other researchers in the future.

Methods

A computer search of PubMed and Google Scholar was conducted, tracing back to references from January 2020 to March 2025 in order to identify suitable literature. The search initially yielded 93 records. The database search filters included year of publication, text availability and article type. Included studies were limited to those published in English, involving adult and elderly patients and those available as full text without access restrictions. Exclusion criteria included articles involving pediatric populations, publications requiring paid access and papers not written in English. After applying these criteria, 33 articles were

excluded. The databases were searched using the terms “Small Intestinal Bacterial Overgrowth”, “gut microbiota”, “microbiome”, “SIBO”, “dysbiosis”, “chronic diseases”, “inflammation” in various combinations with the terms “related”, “treatment”, “disease”. Additional publications were retrieved from the citations of the manuscripts identified. Review papers, randomized clinical trials, protocols and guidelines were eligible for inclusion. Reference lists were manually checked, and 10 duplicates were removed. Finally, 50 sources met the inclusion criteria and were included in this narrative review.

Literature review results

Gut microbiota

An American geneticist and microbiologist Joshua Lederberg, who won the 1958 Nobel Prize in Physiology and Medicine for discovering the mechanisms of genetic recombination in bacteria, was the first to use the term “microbiome” in 2001 [1]. The term refers to the genome of the microorganisms that live in the human body [1]. The human gut microbiota consists of over 1,500 species belonging to over 50 types, with bacteria from the *Firmicutes* and *Bacteroidetes* classes predominating. The total number of all microorganisms is estimated to be approx. 10^{14} CFU/ml [2,6,9].

The gut microbiota significantly impacts many bodily functions, particularly those related to the body’s immune system. These include the processing of nutrients, the prevention of pathogen invasion through colonization of the mucosal surface, the synthesis of various metabolic products and maintenance of psychosomatic health, all of which are associated with the composition and quality of the microorganisms inhabiting the gut

[1,2,10]. Maintaining bacterial balance is therefore crucial for immunity and homeostasis throughout the organism [3,10].

The gut microbiota plays a crucial role in ensuring that the body functions properly. It is involved in basic physiological processes, including nutrient absorption, digestion and the production of epithelial cells. It also stimulates the synthesis of mucins, which protect the epithelium from pathogens, and produces vitamins from the B and K groups, as well as short-chain fatty acids. It acts as a key component of the immune system, secreting substances that can inhibit the growth of harmful viruses or bacteria, such as bacteriocins, organic acids, lactoperoxidases and hydrogen peroxide [2,3,11,12]. The microorganisms living in the digestive tract create a diverse ecosystem that changes dynamically throughout a person's life. They also support the absorption of sodium, calcium, magnesium and potassium, which are essential for the body to function properly [1]. The process of food passing through the large intestine is slower and allows for a greater variety of microorganisms to be present. Their concentration ranges from 10^{10} to 10^{14} CFU/g of gastrointestinal content, and they represent around 800 species that are either pathogenic, symbiotic or opportunistic [1,2]. The large intestine is dominated by anaerobic, facultatively anaerobic species belonging to genera such as *Bacillus*, *Fusobacterium*, *Bifidobacterium*, *Clostridium*, *Bacteroides*, *Enterococcus*, *Peptostreptococcus*, *Eubacterium* and *Ruminococcus* [1,4,5,13]. The continuous regulation of the host immune response and body homeostasis is linked to the commensal microbiota, which plays a key role in human health [8]. However, even small changes in the composition of the gut microbiota may be correlated with the development of various diseases and may be both the cause and the effect of dysfunction in individual systems and the whole body [2,6,8]. Proper colonization of the gastrointestinal tract is therefore extremely important. A reduction in the number of *Bifidobacteria*, which stimulates the immune system, leads to malnutrition and general mild inflammation [2,4,6].

The gut microbiota varies from person to person. A more diverse, balanced and abundant gut microbiota has been associated with a longer and healthier life [14,15]. People who are obese or overweight have significantly fewer genes from part of the gut microbiota than people of normal body weight, resulting in a much lower total number of bacteria [2,16]. Several diseases can develop as a result of compromised immunity, which can be caused by reduced numbers of beneficial microorganisms, impaired intestinal barrier function or an increased influx of pathogens [16,17]. Dysbiosis may increase the risk of developing certain chronic diseases and can also lead to bacterial overgrowth in the small intestine [1,14,18].

Small intestinal bacterial overgrowth – SIBO

Maintaining the health and homeostasis of the body depends heavily on the symbiotic relationship between the microorganisms living in the digestive system and the human body as a whole [11,19-21]. SIBO is characterized by quantitative and qualitative changes in the composition of microorganisms [1,2,14]. Migration of bacteria from the upper or lower digestive tract can lead to the development of SIBO [19,21]. Notably, overgrowth originating from the oral microbiota is often associated with bacteria such as *Prevotella* and *Streptococcus gramineus* [2]. SIBO pathogenesis related to the large intestine is frequently caused by bacteria such as *Escherichia coli*, *Clostridoides* spp., *Enterococcus* spp., *Proteus mirabilis* or *Klebsiella pneumoniae* [1,19-21]. The most common non-invasive method of diagnosing SIBO involves measuring the levels of methane/hydrogen in the patient's breath after they have consumed a specific carbohydrate substance [20,22]. Culturing endoscopic fluid aspirated from the lumen of the small intestine enables an accurate determination of bacterial numbers [21,23]. According to the definition of SIBO, a count of more than 10^5 CFU/ml of bacteria in the aspirate collected during endoscopy from the third part of the

duodenum indicates small intestinal bacterial overgrowth [1,19,21]. The clinical presentation of SIBO is often non-specific, resembling common symptoms of many different diseases. The most common symptoms include excessive gas accumulation, leading to a sensation of fullness, abdominal bloating, changes in bowel habits and diffuse abdominal cramps [19,21,23]. More severe cases may present with chronic fatigue, steatorrhea, weight loss, malnutrition and problems with concentration [21,23,24].

SIBO is also associated with impaired absorption of fat-soluble vitamins – A, D, E and B12, as well as iron. This can lead to the development of bone metabolism disorders, micro- or macrocytic anemia and polyneuropathy [19,21-24]. However, patients with SIBO usually do not experience deficiencies in folic acid or vitamin K, because these substances are produced by the intestinal microbiome [21]. An increased permeability to lipopolysaccharides can trigger an inflammatory response, thereby contributing to chronic inflammation, which is associated with SIBO [2,6,19]. Additionally, the development of this disease may also be influenced by: irregularities in the composition and operation of the intestinal wall, increased gastric pH, elevated levels of leptin, ghrelin or trimethylamine N-oxide and proinflammatory cytokines [1,4,6,8,14,21]. In a physiological state, the acidic pH of the stomach, the intestinal immune system, pancreatic enzymes, small intestinal peristalsis and the intestinal barrier all prevent excessive bacterial colonization of the small intestine [1,19,21]. Disruption to these protective mechanisms can potentially lead to SIBO [1,24].

The main causes of SIBO are impaired motility of food through the small intestine, delayed esophageal-gastric transit time and increased gastric pH (e.g. due to prolonged use of proton pump inhibitors or following gastric surgery) [1-4,24]. Hypochlorhydria and abnormally low gastric acid secretion can also lead to the development of SIBO. Reduced gastric acid is unable to effectively limit the growth of ingested bacteria through the digestive tract [4]. Patients diagnosed with *Helicobacter pylori*, elderly people, individuals

experiencing stress, smokers and alcoholics are at high risk of SIBO [1,4,13]. Nutrient deficiencies, such as those involving zinc, iron and vitamin B, as well as long-term use of antacids and drugs for heartburn or ulcers, increase the risk of developing SIBO [1,2,25]. Using new molecular methods based on sequencing of the 16S ribosomal RNA gene may lead to the discovery of new bacterial species and improve our understanding of their role in SIBO development and its correlation with other diseases [1,25]. The rapid development of molecular biology, bioinformatics analysis, genomics and high-throughput sequencing techniques has shown a complex relationship between the gut microbiota and the entire human immune system [7]. Altered gut microbiota has been linked to impaired immune responses, with gut microbiota metabolites significantly influencing genetic and epigenetic regulation and immune cell metabolism [26-28]. The primary objective of SIBO treatment is to reduce or eliminate the excess microorganisms present in the small intestine. This not only alleviates symptoms but also maintains remission, prevents possible relapses and corrects any nutritional and vitamin deficiencies, thereby restoring intestinal microbiota balance [21,24,25]. Antibiotics are the gold standard for eradicating SIBO and provide significant symptom relief [22].

The current treatment for SIBO involves the use of antibiotics such as metronidazole, doxycycline, chloramphenicol, cephalexin, norfloxacin, ciprofloxacin, rifaximin, neomycin and tetracycline, as well as a combination of amoxicillin and clavulanate, combinations like amoxicillin, ciprofloxacin and metronidazole and a combination of trimethoprim and sulfamethoxazole [7,22,25,28]. Rifaximin, in particular, is a selective gastrointestinal antibiotic offering several advantages, including the fact that it is not absorbed systemically, it has a broad spectrum of antimicrobial activity, it boasts an excellent safety profile, and it minimally impacts the human gut microbiome [22,28]. SIBO treatment may also include diet therapy [24].

While SIBO is not a life-threatening condition, it can definitely worsen a patient's health and negatively affect comorbidities [19,25,29]. Despite extensive recent research on SIBO, this disease remains overlooked by many specialists and their patients [19,28,29].

SIBO and the occurrence of other diseases

The majority of symptoms associated with small intestinal bacterial overgrowth are confined to the gastrointestinal tract. There is growing evidence of a link between SIBO and various diseases [30]. SIBO stimulates the immune system, leading to increased secretion of pro-inflammatory cytokines in the intestinal mucosa thus contributing to increased intestinal permeability [2,6]. Intestinal motility can be markedly influenced by the presence of various types of fistulas, strictures or a history of surgical interventions, which may also contribute to gastrointestinal microbiota dysbiosis [2]. A range of complex diseases is associated with chronic low-grade inflammation, which plays a crucial role in disrupting the microbial balance of the gut [2,4]. This inflammatory state, often linked to increased intestinal permeability to LPS and endotoxins – especially in individuals with obesity – can promote excessive energy intake, elevated short-chain fatty acid production and adipocyte hyperplasia. This is due to a decrease in the number of *Bacteroides* bacteria relative to *Firmicutes* bacteria [2,31]. SIBO often develops alongside slower esophageal transit, which reduces the proper removal of bacteria from the small intestine [32]. This situation may be caused by intestinal motor dysfunction associated with intestinal diseases, portal hypertension, autonomic diabetic polyneuropathy and reduced motor stimulation by thyroid hormones [30,32].

The metabolic products of microorganisms in the intestinal microbiota can directly or indirectly affect the cognitive, emotional and immune functions in humans, potentially leading to the development of various diseases [32]. SIBO has a multifactorial impact on

gastrointestinal, neurological, cardiovascular, metabolic, autoimmune, endocrine, dermatological, developmental, psychiatric, genetic, nephrological and bone disorders [2,30].

Neurological diseases

There is a strong link between SIBO and neurological diseases such as Alzheimer's and Parkinson's. SIBO is present in over 45% of patients with these diseases. Alzheimer's, the most common neurodegenerative disease, is characterized by the accumulation of abnormal proteins, such as tau protein and beta-amyloid, in the brain. This leads to neuronal loss and impaired connections [1,33,34]. Alterations in gut microbiota have been implicated in the development of Alzheimer's disease through the increased generation of neurotoxic metabolites capable of crossing the blood-brain barrier and triggering neuroinflammation [1,34,35]. Parkinson's disease, the second most prevalent neurodegenerative disorder among older adults, is marked by the pathological accumulation of α -synuclein and the degeneration of dopaminergic neurons in the brain's substantia nigra [1,33,36]. Beyond the hallmark motor symptoms such as tremors and muscle rigidity, individuals with Parkinson's frequently suffer from gastrointestinal disturbances, including delayed gastric emptying and reduced gastrointestinal motility [36]. SIBO increases intestinal permeability, exposing the mucosa to bacterial exotoxins and LPS. This results in increased α -synuclein amyloid formation and neuronal susceptibility to degeneration [1,36]. Furthermore, SIBO significantly interferes with the absorption of levodopa, the most common drug used to treat Parkinson's disease, which can lead to reduced dopamine levels in the brain [1,36].

Gastrointestinal disorders

Gastrointestinal dysfunction includes hypochlorhydria, which is common in older people and is often associated with long-term use of proton pump inhibitor use and *Helicobacter pylori* colonization, both of which contribute to gastrointestinal dysbiosis [25,37,38]. Additionally, abdominal and pelvic surgeries are also predisposing factors for the overgrowth of intestinal bacteria, which is why SIBO is common in conditions such as non-alcoholic fatty liver disease, Crohn's disease, cirrhosis of the liver, ulcerative colitis, irritable bowel syndrome, coeliac disease or pancreatitis [1,37,38].

Cardiovascular diseases

SIBO is significantly related to the cardiovascular system. Patients with heart disease, deep vein thrombosis, malnutrition and cachexia exhibit a systemic inflammatory and immune response resulting from impaired gut function. This process may result in bacterial translocation, allowing microbes and endotoxins to enter systemic circulation [1,39]. The resulting inflammatory response associated with cardiovascular dysfunction can contribute to left ventricular remodeling and increased apoptosis of cardiomyocytes [1,39]. A decline in cardiac output may cause ischemia in the small intestine, impair intestinal function and enhance gut permeability. Elevated levels of LPS and pro-inflammatory mediators are commonly found in patients with cardiovascular-related edema [1]. Moreover, heightened expression of TLR-4 and inflammatory cytokines can enhance procoagulant activity, increasing the risk of cardiovascular complications. The cytokine storm observed in these conditions disrupts gut microbial balance and has been associated with the onset of SIBO

[1,2]. Metabolites produced by gut bacteria, alongside low levels of vitamin K2, increase the risk of developing atherosclerotic disease [39].

Autoimmune diseases (systemic sclerosis)

Systemic sclerosis is a part of chronic autoimmune connective tissue disease resulting in fibrosis of various organs, including the gastrointestinal tract [1,40]. Around 55% of people with systemic sclerosis experience gastrointestinal symptoms such as abdominal pain, constipation, flatulence and diarrhea [1]. Impaired intestinal motility and inadequate cleansing can lead to SIBO. This disorder is ten times more prevalent in patients with systemic sclerosis than in healthy individuals [1,2,40].

Endocrine disorders (thyroid diseases)

Endocrine disorders often present with gastrointestinal symptoms, such as abdominal discomfort, constipation or diarrhea [1]. Such disturbances can lead to gut dysbiosis, resulting in increased intestinal permeability and systemic inflammation. This inflammatory state may stimulate the immune system to produce elevated levels of antibodies, potentially contributing to the pathogenesis of autoimmune thyroid disorders such as Hashimoto's thyroiditis or Graves' disease [1,2,6,41]. Additional factors implicated in the development of SIBO include reduced intestinal clearance, weakened immune defenses, and the administration of levothyroxine in the treatment of hypothyroidism [37].

Dermatological diseases (rosacea)

The incidence of SIBO in patients with rosacea is approx. 46% [1,32]. Rosacea is a chronic inflammatory skin condition characterized by redness, pustules, papules and transient erythema [42]. While the exact cause of rosacea remains unknown, it is believed to involve changes in the body's immune response linked to the gut-skin axis [1,42]. Studies suggest that individuals with dermatological conditions, including rosacea, are more likely to experience SIBO [1].

Metabolic diseases

In the group of metabolic diseases, SIBO is frequently observed in patients with obesity [1]. A reduced ratio of *Bacteroides* to *Firmicutes* bacteria, excess energy consumption, elevated synthesis of short-chain fatty acids, and expansion in adipose tissue due to adipocyte proliferation contribute to obesity [2,31]. SIBO is prevalent among patients with diabetes, and is associated with worsened glycemic control [1]. The incidence of SIBO is higher in patients with type 1 or type 2 diabetes mellitus than in the general population [43]. Hyperglycemia and autonomic neuropathy cause impaired gastrointestinal peristalsis and promote bacterial overgrowth, resulting in a vicious cycle [2,43].

Mental health disorders

In numerous cases, SIBO is associated with psychological conditions including stress, depression and anxiety. Bacterial overgrowth in the small intestine may interfere with tryptophan metabolism (a precursor of serotonin), thereby contributing to the development of

mood disorders [1,43,44]. Disturbances in the composition of the gut microbiota exacerbate anxiety and depression symptoms, impairing the gastrointestinal tract's proper functioning and contributing to SIBO [45,46]. Patients with SIBO exhibit lower levels of extroversion and higher levels of neuroticism, stress and anxiety [1,46]. Psychological stress can also negatively affect small intestinal transit time and significantly disturb intestinal barrier balance and thus lead to the development of SIBO [1,43-46].

Chronic activation of the hypothalamic-pituitary-adrenal axis can also increase the likelihood of SIBO, as the stress response may disrupt the gut microbiota [40]. Furthermore, people with SIBO often experience mild to moderate anxiety and depression [1,46].

In addition to the mental health issues, SIBO is significantly connected with several medical conditions: which include chronic intestinal pseudo-obstruction, cystic fibrosis, functional abdominal distension, dyspepsia, constipation, diarrhea, chronic renal failure, short bowel syndrome, lactase deficiency, diverticular disease, primary biliary cholangitis, gastroparesis, gallstone disease, acromegaly, multiple sclerosis, spondyloarthropathy, fibromyalgia, asthma, genetic, nephrological and bone diseases [2,32,43-46].

Developmental disorders (autism spectrum disorders)

An inappropriate diet in patients displaying symptoms of autism and SIBO may exacerbate the condition [1,44]. Although the incidence of autism spectrum disorders continues to increase, the mechanisms driving their development are not yet fully understood. In autistic children, the presence of small intestinal bacterial overgrowth has been linked to greater symptom severity [1].

Dysbiosis of the gut microbiota can lead to low-grade systemic inflammation and altered production of neurotransmitters and bacterial metabolites, such as short-chain fatty

acids. These changes exacerbate symptoms in individuals with autism spectrum disorders and have been linked to the gut-brain axis [47-49]. These factors can adversely affect brain function, resulting in increased irritability, difficulty concentrating and stereotyped behavior. Applying therapeutic approaches aimed at eradicating SIBO or enhancing gut microbiota balance through dietary changes may help alleviate common gastrointestinal problems in children with autism and potentially reduce the occurrence of symptoms of autism [48,50].

Diseases associated with SIBO

The presence of SIBO affects the course of diseases in various ways. In a number of cases, a significant correlation has been found between the presence of SIBO and disease severity, but further research is needed in this area to be able to more precisely describe the relationship occurring between SIBO and other conditions (Table 1).

Table 1. Summary of literature review – diseases associated with SIBO

Disease Group	Examples	Mechanisms/Clinical Relevance
Neurological diseases [1,29-32]	Alzheimer's disease, Parkinson's disease	Dysbiosis: ↑ neurotoxic metabolites → neuroinflammation. In PD: ↑ intestinal permeability, α-synuclein aggregation, impaired levodopa absorption → worsening motor symptoms
Gastrointestinal disorders [1,22,33,34]	IBS, Crohn's disease, ulcerative colitis, coeliac disease, NAFLD, liver cirrhosis, pancreatitis	Hypochlorhydria, PPI use, <i>H. pylori</i> , surgeries → dysbiosis → bacterial overgrowth. SIBO frequent in chronic GI diseases
Cardiovascular diseases [1,2,35]	Heart failure, DVT, CAD, subclinical atherosclerosis	SIBO → ↑ gut permeability, LPS, systemic inflammation → bacterial translocation, cardiac remodeling, coagulation, atherosclerosis
Autoimmune diseases [1,2,36]	Systemic sclerosis	Impaired GI motility → SIBO prevalence – 10× higher; symptoms: pain, constipation, bloating,

		diarrhea
Endocrine disorders [1,2,5,37]	Autoimmune thyroid diseases (Hashimoto, Graves'), hypothyroidism	Dysbiosis and permeability → systemic inflammation → ↑ autoantibodies; levothyroxine and impaired clearance contribute to SIBO
Dermatological diseases [1,28,38]	Rosacea	Incidence of SIBO in 46%; gut-skin axis, inflammatory response. Symptoms improve after SIBO treatment
Metabolic diseases [1,2,27,39]	Diabetes (T1DM, T2DM), obesity, hyperlipidemia	Dysbiosis (↓ <i>Bacteroides/Firmicutes</i>), SCFA changes, adipose expansion. Hyperglycemia and neuropathy → impaired motility → SIBO, poor glycemic control
Mental health disorders [1,39-42]	Stress, depression, anxiety	Dysbiosis → altered tryptophan/serotonin metabolism. Stress and HPA axis activation → gut permeability, delayed transit. Mood disorders linked to SIBO prevalence
Developmental disorders [1,40]	Autism spectrum disorders	Dysbiosis → SCFA imbalance, neuroinflammation, neurotransmitter disruption. SIBO linked to ↑ symptom severity; dietary and gut-targeted therapy may reduce symptoms
Other conditions [2,28,39-42]	Chronic intestinal pseudo-obstruction, CF, functional distension, dyspepsia, renal failure, short bowel, lactase deficiency, diverticulosis, PBC, gastroparesis, gallstones, acromegaly, MS, spondyloarthropathy, fibromyalgia, asthma, nephrological and bone diseases	SIBO often exacerbates symptoms and disease severity; exact causal links require further research

Notes: PD – Parkinson's Disease; IBS – Irritable Bowel Syndrome; NAFLD – Non-alcoholic Fatty

Liver Disease; PPI – Proton Pump Inhibitor; GI – Gastrointestinal; DVT – Deep Vein Thrombosis;

CAD – Coronary Artery Disease; LPS – Lipopolysaccharide; T1DM – Type 1 Diabetes Mellitus;

T2DM – Type 2 Diabetes Mellitus; SCFA – Short-chain Fatty Acids; HPA axis – Hypothalamic–

pituitary–adrenal axis; CF – Cystic Fibrosis; MS – Multiple Sclerosis.

Conclusions

Small intestinal bacterial overgrowth is strongly associated with intestinal dysbiosis, which disrupts host homeostasis and promotes chronic inflammation. This imbalance not only generates gastrointestinal symptoms such as bloating, abdominal pain, diarrhea, constipation and malabsorption but also contributes to nutritional deficiencies, anemia, protein loss and an overall decline in quality of life. Beyond local effects, SIBO-related dysbiosis has been linked to a broad spectrum of systemic disorders, including gastrointestinal, cardiovascular, endocrine, autoimmune, neurological, metabolic and psychiatric conditions, as well as certain cancers. These associations highlight the central role of the gut microbiota in maintaining human health and in the pathogenesis of diverse diseases.

Understanding the interactions between microbial composition, immune regulation and host metabolism is essential for more accurate diagnosis and the development of targeted therapeutic strategies. Microbial metabolites are emerging as important modulators of inflammation and disease progression, with potential application as biomarkers for monitoring clinical outcomes.

Future research should focus on large-scale, high-quality studies to clarify causal mechanisms, validate reliable microbiota-based biomarkers and establish effective treatment protocols. Such efforts may improve disease management, guide preventive interventions and ultimately reduce the burden of chronic conditions associated with SIBO.

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