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Small Intestinal Bacterial Overgrowth in Various Specialties of Medical Practice (Literature Review and Expert Council Resolution)

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Aim: to discuss current views on the clinical significance, diagnostic opportunities, and therapeutic approaches in the treatment of small intestinal bacterial overgrowth (SIBO) as an important component in the gut microbiota function assessment, to assess the awareness of physicians and the opportunities in the diagnosis and treatment of this disease in patients in the Federal districts of the Russian Federation, as well as to present the proceedings of the Expert Council held on December 16, 2023 in Moscow.

Key points. SIBO is a common syndrome often associated with irritable bowel syndrome, liver cirrhosis, asthma, and congestive heart failure, being also a predictor of early death in the elderly. Today, in many regions of the Russian Federation, there are limitations for instrumental diagnosis of this disease — lack of awareness among doctors, unavailability of gas analyzers for diagnosing SIBO, lack of information about the need to diagnose SIBO in the standards of compulsory health insurance. Rifaximin is the first-line treatment due to the highest therapeutic efficacy. One of the ways to increase the efficacy of SIBO treatment is to include strain-specific probiotics in the treatment regimen. *Saccharomyces boulardii* CNCM I-745 is thought to be the most studied, promising probiotic. The review also presents statistical data on the issues in the diagnosis and treatment of SIBO in the regions of the Russian Federation.

Conclusion. Optimization of approaches to the diagnosis and treatment of SIBO, the development of domestic gas analyzers, increasing the awareness of physicians in all regions of the Russian Federation, as well as the development and optimization of clinical recommendations appear to be necessary measures to increase the effectiveness of medical care, the duration and quality of life of the Russian population. These goals can be achieved within the framework of Federal programs under the supervision of specialized reference centers of the Ministry of Health of the Russian Federation.

Keywords: small intestinal bacterial overgrowth, gut microbiota, irritable bowel syndrome, liver cirrhosis, chronic heart failure, asthma, aging, malnutrition, probiotics, *Saccharomyces boulardii* CNCM I-745

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Синдром избыточного бактериального роста в практике врачей различных специальностей (Обзор литературы и резолюция Экспертного совета)

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Цель: рассмотреть современные представления о клинической значимости, возможностях диагностики и терапевтических подходах в лечении синдрома избыточного бактериального роста в тонкой кишке (СИБР) как важного компонента оценки функционального состояния микробиоты кишечника, оценить осведомленность врачей и возможности диагностики и лечения данного заболевания у пациентов в федеральных округах РФ, а также представить материалы Экспертного совета, который состоялся 16 декабря 2023 г. в Москве.

Основные положения. СИБР является распространенным синдромом, который часто сочетается с синдромом раздраженного кишечника, циррозом печени, бронхиальной астмой и хронической сердечной недостаточностью, а также служит предиктором ранней смерти у пожилых людей. На сегодня во многих регионах Российской Федерации есть ограничения для инструментальной диагностики данного заболевания: недостаточная осведомленность врачей, недоступность газовых анализаторов для диагностики СИБР, отсутствие информации о необходимости диагностики СИБР в стандартах обязательного медицинского страхования. Терапией первой линии ввиду наибольшей лечебной эффективности служит рифаксимин. Одним из способов повышения эффективности терапии СИБР служит включение в схему лечения штаммоспецифичных пробиотиков. Наиболее изученным и перспективным пробиотиком считается *Saccharomyces boulardii* CNCM I-745. В обзоре также представлены статистические данные о проблемах диагностики и лечения СИБР в регионах РФ.

Вывод. Оптимизация подходов к диагностике и лечению СИБР, разработка отечественных газовых анализаторов, повышение осведомленности врачей во всех регионах РФ, а также разработка и оптимизация клинических рекомендаций представляются необходимыми мерами повышения эффективности оказываемой медицинской помощи, увеличения продолжительности и качества жизни населения РФ. Данные цели могут быть достигнуты в рамках программ федерального значения под контролем специализированных референс-центров Минздрава РФ.

Ключевые слова: синдром избыточного бактериального роста, микробиота кишечника, синдром раздраженного кишечника, цирроз печени, хроническая сердечная недостаточность, бронхиальная астма, старение, мальнутриция, пробиотики, *Saccharomyces boulardii* CNCM I-745

Конфликт интересов: Экспертный совет состоялся при организационной поддержке фармацевтической компании «Биокодекс».

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On December 16, 2023, a meeting of the Expert Council was held in Moscow under the chairmanship of Chief Gastroenterologist of the Ministry of Health of the Russian Federation, Russian Academy of Sciences (RAS) Academician, Professor, V.T. Ivashkin, discussing intestinal bacterial overgrowth in various specialties of medical practice. The Expert Council was also attended by Chief Specialist in General Medical Practice of the Ministry of Health of the Russian Federation, Vice-Rector for Innovation and Clinical Activities of Sechenov University, RAS Corresponding Member, Professor, V.V. Fomin; Chief Geriatrician of the Ministry of Health of the Russian Federation, RAS Corresponding Member, Professor O.N. Tkacheva; Chief Gastroenterologists of the Federal districts of the Russian Federation; opinion leaders and industry representatives. The event was organized by the Sechenov University and the Scientific Community for Human Microbiome Research (CHMR).

In his opening remarks, V.T. Ivashkin, spoke about the changing understanding of the role of gut microbiota in maintaining health and the development of chronic non-infectious human diseases, as well as about modern diagnostic opportunities to study its composition. The accumulated data demonstrate that small intestinal bacterial overgrowth (SIBO) has a negative impact on the course and prognosis of several chronic non-infectious diseases, meaning that timely diagnosis and treatment of SIBO is one of the most important measures to optimize treatment regimens for various diseases and promote the healthy longevity in the population of the Russian Federation.

The Expert Council reports listed below provided insight on the issues and potential opportunities for optimizing the diagnosis and treatment of SIBO.

Academician of RAS, Professor V.T. Ivashkin's report was devoted to the current views on the prevalence, pathogenesis, and clinical manifestations of SIBO.

SIBO is a disease characterized by an increased amount and/or disruption of the microbiota composition in the small intestine, manifested by systemic inflammation, impaired integrity of the intestinal epithelial barrier, and malabsorption [1]. The prevalence of SIBO in the general population is not well known. SIBO is known to occur mainly in women (66 %), and its incidence increases with age [2]. A recent systematic review showed a high prevalence of SIBO among patients with chronic non-communicable diseases (Table 1) [3].

Microbiota density normally varies in different parts of the small intestine, being ≤ 1000 CFU/mL in the duodenum and 10^3 – 10^7 CFU/mL in the jejunum and ileum. The gradient in the quantitative composition of microorganisms is due to several factors. Factors that inhibit the growth of microorganisms, many of which are anaerobes, in the proximal portions include high partial pressure of oxygen; bactericidal activity of primary bile acids, digestive enzymes and hydrochloric acid; propulsive peristalsis; low pH. In the distal parts of the small intestine, the activity of these inhibitory factors is significantly reduced, and microorganisms are cleared from the body due to innate and acquired (adaptive) immune mechanisms interfering with the development of a systemic inflammatory response [4, 5].

Key bacteria responsible for intestinal bacterial overgrowth have not been identified to date; however, most studies indicate obligate anaerobes [6, 7]. An analysis of biopsies of the duodenal, iliac, and sigmoid colon mucosa (16S ribosomal RNA sequencing) showed significant differences

Table 1. Prevalence of SIBO in chronic non-communicable diseases

Таблица 1. Распространенность СИБР при хронических неинфекционных заболеваниях

Organic gastrointestinal diseases Органические заболевания ЖКТ		Liver diseases Заболевания печени	
Short bowel syndrome Синдром короткой кишки	50 %	Liver cirrhosis Цирроз печени	40.8 %
Chronic pseudo-obstruction Хроническая псевдообструкция	23.7–52.6 %	Hepatocellular carcinoma Гепатоцеллюлярная карцинома	71.8 %
Lactase deficiency Лактазная недостаточность	27.6 %	MAFLD МАЖБП	35.0 %
Lactose intolerance in the elderly Непереносимость лактозы у пожилых	90 %	Primary biliary cholangitis Первичный билиарный холангит	32.8 %
Diverticular disease Дивертикулярная болезнь	58.9 %	Endocrine and metabolic diseases Эндокринные и метаболические заболевания	
Celiac disease / Целиакия	18.3 %	Diabetes / Сахарный диабет	29 %
Non-infectious enteritis Неинфекционный энтерит	85.3 %	Hypothyroidism Гипотиреоз	54 %
Crohn's disease / Болезнь Крона	25.4 %	Acromegaly / Акромегалия	43.9 %
Ulcerative colitis Язвенный колит	14.3 %	Hypercholesterolemia Гиперхолестеринемия	78.9 %
Erosive esophagitis Эрозивный эзофагит	65 %	Rheumatoid diseases Ревматоидные заболевания	
<i>Helicobacter pylori</i> infection Инфекция <i>Helicobacter pylori</i>	60.4 %	Systemic scleroderma Системная склеродермия	34 %
Gastroparesis / Гастропарез	60 %	Behçet's disease / Болезнь Бехчета	36 %
Chronic pancreatitis Хронический панкреатит	38.6 %	Spondyloarthropathy Спондилоартропатия	63 %
Acute pancreatitis Острый панкреатит	12.0–17.8 %	Fibromyalgia Фибромиалгия	100 %
Pancreatic cysts Кисты поджелудочной железы	31.6–40.0 %	Condition after surgery Состояние после хирургического вмешательства	
Cholelithiasis Желчнокаменная болезнь	14.8–40.5 %	Bariatric interventions Бариатрические вмешательства	37.0–73.4 %
Stool incontinence Недержание стула	42 %	Gastrectomy Гастрэктомия	61.6–77.6 %
Functional gastrointestinal diseases Функциональные заболевания ЖКТ		Colectomy Колэктомия	62–74 %
Functional dyspepsia Функциональная диспепсия	17.2–53.4 %	Cholecystectomy Холецистэктомия	24.6–46.8 %
Irritable bowel syndrome Синдром раздраженного кишечника	31.0–36.7 %	Malignant neoplasms of the gastrointestinal tract Злокачественные новообразования ЖКТ	
Functional abdominal bloating Функциональное вздутие	43–68 %	Pancreas cancer Рак поджелудочной железы	63.3 %
Functional constipation Функциональный запор	78 %	Cholangiocarcinoma Холангиокарцинома	46.7 %
Functional diarrhea Функциональная диарея	69 %	Other diseases Другие заболевания	
Neurological diseases Неврологические заболевания		Bronchial asthma (allergic) Бронхиальная астма (аллергическая форма)	67 %
Multiple sclerosis Рассеянный склероз	38.1 %	Bronchial asthma (non-allergic) Бронхиальная астма (неаллергическая форма)	43 %
Restless legs syndrome Синдром беспокойных ног	69 %	Chronic heart failure Хроническая сердечная недостаточность	41.7–45.0 %
Alzheimer's disease Болезнь Альцгеймера	49 %	Obstructive sleep apnea syndrome Синдром обструктивного апноэ сна	30.8 %
Autism / Аутизм	31.0 %	Post-COVID-19 / Пост-COVID-19	93.3 %
Spinal cord injuries Травмы спинного мозга	37.5–38.5 %	Deep vein thrombosis Тромбоз глубоких вен	69.8 %
Parkinson's disease Болезнь Паркинсона	46 %	Rosacea Розацеа	10–46 %

in the composition of mucosal microbiota between patients with SIBO and healthy subjects ($p = 0.039$, $p = 0.002$, and $p = 0.007$, respectively). All biopsies of patients with SIBO had increased levels of representatives of the genera *Lactobacillus*, *Prevotella*, *Dialister* and *Ruminococcaceae*, as compared with healthy subjects [8]. There have also been reports that the methanogenic archaea *Methanosphaera stadtmanae* and *Methanobrevibacter smithii* may be the only microorganisms responsible for microbial overload [5]. According to various estimates, 15 to 30 % of patients with SIBO and constipation symptoms are colonized specifically by *Methanobrevibacter* [2, 5].

The pathogenesis of SIBO includes the following stages [2]:

- active bacterial metabolism leads to the accumulation of gases (e.g., H_2 , CH_4 , H_2S , CO_2), which, in combination with the development of visceral hypersensitivity, causes pain;
- the brush border of enterocytes is damaged, leading to malabsorption;
- microbial cleavage of amino acids and peptides in the small intestine increases, and the absorption of fat and fat-soluble vitamins decreases (as a result of excessive deconjugation of bile acid salts), which exacerbates the manifestations of malabsorption;
- competition between the human body and the small intestinal microbiota for vitamins B_1 , B_2 , B_3 , B_5 and B_{12} increases due to the growing number of bacteria that utilize these vitamins;
- the proportion of toxic metabolites of bacterial origin (lipopolysaccharides (LPSs), ammonia, D-lactate, peptidoglycans, and lipoteichoic acid) increases, disrupting the permeability of the mucosal epithelial barrier of the small intestine;
- impaired permeability of the mucosal epithelial barrier is associated with increased bacterial translocation;
- due to the bacterial translocation, the local and systemic immune response (systemic inflammation) intensifies due to an increase in the pool of proinflammatory cytokines (IL-1 α , IL-1 β , IL-6 and tumor necrosis factor alpha (TNF- α)).

Clinical symptoms of SIBO are non-specific, but most often this condition manifests as complete or partial intestinal paresis associated with abdominal bloating and pain, impaired stool frequency (diarrhea or constipation), and symptoms of malabsorption. Signs suggestive of SIBO during a physical examination include a clear tympanic sound on abdominal percussion, “silent” abdomen on auscultation, and periumbilical tenderness on palpation. However, about a third of SIBO patients have no typical complaints, which

complicates the diagnosis. Clinical manifestations of malabsorption in SIBO include steatorrhea, weight loss and weakness, neurological disorders (especially in case of impaired absorption of B vitamins), as well as symptoms associated with deficiencies of the fat-soluble vitamins A, D $_3$, E [2].

In conclusion, V.T. Ivashkin noted that the disorders observed in SIBO affect almost all human organs and systems, therefore, testing for this condition is valuable in patients with various diseases, regardless of the severity of its clinical manifestations.

Professor O.S. Medvedev discussed the current opportunities and prospects for optimizing the diagnosis of SIBO.

Specific SIBO tests are hydrogen breath tests with a carbohydrate (glucose or lactulose) using special gas analyzers. The test is based on the fact that the human body is unable to produce hydrogen (H_2), therefore, its presence in the exhaled air is due solely to microbial fermentation of carbohydrates; as a result, the produced H_2 enters the bloodstream and, after passing through the liver, is eliminated by the lungs [9]. As far back as the last century, clinical studies showed that patients with SIBO have elevated small intestinal H_2 levels due to an excess of anaerobic bacteria that synthesize it. The increase in small intestinal H_2 after the administration of lactulose directly correlated with the increase of its proportion in the exhaled air [10].

This correlation forms the basis of modern respiratory tests that allows evaluating the content of anaerobic bacteria in the small intestine by consecutively analyzing the H_2 level in the exhaled air before and after carbohydrate intake. The main advantages of this method are low cost, noninvasiveness, ease of conducting the test and interpretation of the results.

One of the drawbacks of the breath test is that the exhaled air only indirectly reflects the H_2 level in the small intestine. Presumably most of it is taken up by the liver, which complicates the analysis of microbial activity in the small intestine under excessive bacterial load [11]. There are several approaches to measuring H_2 levels directly in the small intestine. One of these is the use of endoscopic capsules capable of analyzing the H_2 level in various parts of the gastrointestinal tract and remotely transmitting data to an external analyzer. However, this method is not used in clinical practice due to the invasive nature of the procedure and its high cost [11, 12]. Another method is the assessment of the individual hepatic metabolism of H_2 . The patient undergoes a hydrogen breath test while drinking water saturated with a given amount of exogenous H_2 , and its

level in the exhaled air is subsequently analyzed. According to some studies, about 40 % of the original amount of H_2 does not reach the alveoli in the lungs due to metabolism in the liver and other organs [13]. Despite the simplicity and effectiveness of this method, it requires further study.

Another significant limitation for interpretation of levels of exhaled H_2 is its utilization by small intestinal archaea, which metabolize the H_2 produced by bacteria to methane (CH_4) [9, 11]. Such methanogenic archaea of the human body include *Methanobrevibacter smithii*, *Methanosphaera stadtmanae*, *Methanomassiliicoccus strains*, as well as *Methanobrevibacter oralis* (living in the mouth) [14]. These microorganisms demonstrate high utilization of H_2 produced by bacteria and metabolize it to CH_4 [15]. Since both gases are only produced by microorganisms and can be eliminated by the lungs, more correct interpretation of SIBO breath test results would be achieved with simultaneous assessment of the concentrations of H_2 and CH_4 in the exhaled air [16].

In conclusion, O.S. Medvedev stressed that the most significant limitation for the diagnosis of SIBO using a hydrogen breath test is the low availability of gas analyzers in the Russian Federation. To date, only two foreign-made gas analyzers have been authorized in the Russian Federation (from Germany and the United Kingdom), and their use is very limited due to maintenance difficulties. This problem has led the Sechenov University to partner with the Lomonosov Moscow State University and an enterprise (Izhevsk Radio Plant) to develop a Russian hydrogen breath test analyzer, which is expected to be authorized in 2024.

A.I. Ulyanin's report was dedicated to current approaches to SIBO therapy, as well as potential optimization of treatment outcomes.

In modern clinical practice, approaches to the eradication of small intestinal bacterial overgrowth involve direct elimination of microorganisms (using antibiotics) and the creation of conditions that prevent excessive reproduction of bacteria in the small intestine. The preferred treatment strategy for SIBO is antibacterial therapy with rifaximin or systemic antibiotics (norfloxacin, ciprofloxacin, or metronidazole) [2]. Rifaximin is the most studied drug for the treatment of SIBO. A systematic review and meta-analysis of 32 studies of rifaximin in SIBO showed its efficacy in 72.9 % of patients with this condition (95 % CI 65.5–79.8) [17]. Rifaximin is recommended for the treatment of SIBO at a dose of 400 mg 3 times a day for 7–14 days, norfloxacin is used at a dose of 400 mg 2 times a day for 7–10 days, metronidazole at 250 mg 3 times a day for 10 days, while

the dosage of ciprofloxacin is 500 mg 2 times a day for 5–10 days [2]. In the foreign guidelines and the CHMR (Scientific Community for Human Microbiome Research) and RGA (Russian Gastroenterological Association) recommendations for the diagnosis and treatment of SIBO, rifaximin is the first-line drug for the treatment of this disease, while the use of systemic antibiotics should be considered as an alternative treatment [2].

Empiric antibiotic therapy in patients without a confirmed diagnosis of SIBO is not justified, as it exposes them to an unreasonable risk of antibiotic resistance, antibiotic-associated diarrhea, and *C. difficile*-associated disease. The use of probiotics for the entire period of antibiotic therapy is recommended to prevent these complications. The most studied organism, which is resistant to all groups of antibiotics, is *Saccharomyces boulardii* CNCM I-745 given at 250 mg twice daily [18]. A meta-analysis of 21 RCTs showed that *S. boulardii* reduces the risk of AAD in patients treated with antibiotics from 18.7 to 8.5 % (HR = 0.47; 95% CI: 0.38–0.57), as compared with controls [19]. The results of a meta-analysis of 9 RCTs confirmed the positive effect of *Saccharomyces boulardii* in the prevention of the first episode of *C. difficile*-associated disease [20].

S. boulardii CNCM I-745 may be beneficial in the treatment of SIBO not only in terms of preventing complications of antibacterial therapy, but also to improve treatment outcomes. This assumption is based on the results of a recent randomized study involving 40 patients with systemic scleroderma and SIBO. Subjects were randomized into three groups according to the treatment regimen: metronidazole for 7 days and *S. boulardii* for 14 days ($n = 13$), *S. boulardii* only for 7 days ($n = 14$), monotherapy only for 7 days ($n = 13$). Two months after treatment, patients underwent a repeated hydrogen breath test with lactulose, which showed the absence of SIBO in 55 %, 33 % and 25 % of subjects in these groups, respectively [21]. Further studies are needed to evaluate the efficacy of treatment with other antibiotics.

The authors concluded that several issues in the treatment of SIBO remained and required further investigation. These include the need for clear criteria distinguishing between treatment failure and disease recurrence and for diagnostic re-evaluation and SIBO treatment recommendations. Special attention should be paid to studying the potential use of drugs from other pharmacological groups, which may be beneficial due to their effect on the pathogenesis of SIBO (for example, prucalopride and other prokinetics, as well as drugs that

improve the integrity of the intestinal mucosal epithelial barrier).

Professor E.A. Poluektova detailed the effect of SIBO on the pathogenesis and clinical manifestations of irritable bowel syndrome (IBS).

According to various studies, the prevalence of SIBO among IBS patients ranges from 9 to 70 %. In a recently published meta-analysis of 13 clinical trials ($n = 2494$), SIBO was found to be significantly more common in IBS patients than in healthy subjects (30 % vs. 9 %; $p < 0.0001$) [5]. In a study involving 247 patients with IBS, the prevalence of SIBO, according to hydrogen and methane breath tests, was 36.4 % (9.7 % for H_2 and 26.7 % for CH_4). An analysis of the relationship between the disease course and the level of these gases in the exhaled air revealed that patients with IBS diarrhea were more likely to have an increase in H_2 ($p = 0.013$), while patients with constipation typically had an increase in CH_4 ($p = 0.003$). These data highlight the relationship between the methanogenic microorganisms and the inhibition of colonic motility [22].

The main components of the IBS pathogenesis are the developing emotional disorders, impairment of the gastrointestinal mucosal epithelial barrier, and changes in the composition of the colonic microbiota, which are observed in genetically predisposed individuals under the influence of environmental factors. These factors lead to the clinical manifestations of the disease through impaired colonic motility, changes in central and peripheral sensitization, and induction of intestinal wall inflammation as the outcome of bacterial translocation [23].

The effect of SIBO on emotional disorders was demonstrated in a study involving 26 patients with SIBO and 24 healthy individuals. Compared with the control group, patients with SIBO had a higher emotional instability ($p < 0.001$), were less communicative and sociable ($p < 0.001$) and had a higher anxiety level ($p < 0.001$). This group of subjects had significantly higher overall stress levels ($p < 0.001$), as well as an increase in the level of emotional stress ($p < 0.001$) and susceptibility to stress related to external experiences ($p < 0.041$) and internal sensations ($p < 0.001$), as compared with the control group [24].

These emotional disorders in patients with IBS may be caused by impaired tryptophan metabolism. Normally, about 5 % of tryptophan from food is metabolized to serotonin, and the rest is metabolized to kynurenine [25]. Tryptophan is converted to serotonin, mainly in the colon, by the enzyme tryptophan hydroxylase-1, whose activity is influenced by the short-chain fatty acids (SCFA) synthesized by the gut microbiota: acetate,

propionate, and butyrate [26, 27]. Tryptophan is converted to kynurenine in the intestine, liver, and brain by tryptophan dioxygenase (TDO) and indoleamine 2,3-dioxygenase (IDO). TDO activity increases in response to an increase in the level of circulating blood cortisol, while IDO activity is stimulated by proinflammatory cytokines (e.g., interferon-gamma ($IFN-\gamma$), IL-6, TNF- α) [25]. Further metabolism of kynurenine occurs in two ways, with the formation of quinolinic or kynurenic acids, which have opposite effects on the central nervous system. Increased levels of quinolinic acid predispose to the development of neurodegenerative changes in the brain and psychoemotional disorders (anxiety and depression) [28]. Kynurenic acid, on the contrary, has neuroprotective properties and prevents the neurotoxic effects of quinolinic acid [29].

Significantly higher urine levels of kynurenine ($p < 0.05$) and quinolinic acid ($p < 0.001$), as well as an increased ratio of kynurenine to tryptophan ($p < 0.001$) and quinolinic acid to kynurenic acid ($p < 0.001$), were observed in patients with SIBO compared with healthy subjects without SIBO. A significant increase in tryptophan levels ($p < 0.001$) and a decrease in the levels of kynurenine ($p < 0.001$) and quinolinic acid ($p < 0.001$) were observed after radical treatment of SIBO using rifaximin [30]. A separate study demonstrated significantly higher levels of kynurenine in the urine in patients with IBS-C ($p < 0.001$) compared to IBS-D patients and healthy subjects. These data suggest that a change in tryptophan metabolism towards kynurenine in IBS-C patients should be viewed as a predictor of inhibited propulsive colonic motility due to a decrease in the pool of produced serotonin [31].

Cytokine profile changes associated with SIBO disrupt the integrity of the gastrointestinal mucosal epithelial barrier in patients with IBS and predispose to bacterial translocation; the resulting vicious circle of impaired intestinal permeability and inflammation of the intestinal wall leads to local inflammation [32]. Persistent inflammation of the intestinal wall leads to excessive degranulation of mast cells with the release of histamine, which activates visceral sensory neurons; their hyperexcitability results in visceral hypersensitivity [33].

Professor E.A. Poluektova noted that impaired colonic microbiota in IBS was reported in many clinical studies; however, the effect of SIBO on the composition of the colonic microbiota in such patients has not been sufficiently studied. There are also very limited data on the specific changes in the small intestinal microbiota associated with SIBO in patients with IBS and the role of individual microorganisms in the pathogenesis of the disease [5].

Thus, SIBO has a negative impact on key elements of the IBS pathogenesis and complicates the course of the disease (Fig. 1).

The lack of effective treatment for patients with IBS is an urgent healthcare problem, as most disease symptoms persist with treatment without worsening, while the probability of their resolution is only 38 % with observation periods of 12–20 months [34]. The possibility of optimizing the outcomes of IBS treatment by eliminating intestinal bacterial overgrowth was demonstrated in a study involving 127 patients with IBS in combination with SIBO; successful elimination of intestinal bacterial overgrowth leads to significantly greater changes in disease symptoms ($p < 0.001$) [35].

Thus, timely diagnosis and treatment of IBS is an effective measure to increase the quality of care for IBS patients.

M.S. Zharkova presented in detail the impact of SIBO on the prognosis in patients with liver cirrhosis.

Decompensated liver cirrhosis (LC) is characterized by high mortality and appears to be a significant financial burden in outpatient and inpatient settings [36].

SIBO should be considered as a trigger for decompensation of cirrhosis, which causes re-hospitalization of patients. According to a meta-analysis of 21 clinical studies involving 1264 patients with LC and 306 healthy subjects, the prevalence of SIBO in patients with LC is 6.83 times higher than in the control group (95% CI: 4.16–11.21; $p < 0.001$), specifically 40.8 % vs. 10.7 % (95% CI: 34.8–47.1 and 5.7–19.0, respectively). The prevalence of SIBO in decompensated LC was significantly higher than in compensated LC (50.5 % vs. 31.2 %; $p < 0.001$). In addition, SIBO in LC is reliably associated with ascites ($p < 0.001$), minimal hepatic encephalopathy ($p = 0.001$), bacterial translocation ($p = 0.026$), and spontaneous bacterial peritonitis ($p = 0.008$) [37].

SIBO is associated not only with a risk of disease complications, but also with high mortality in patients with CP. A survival analysis over the 12-month follow-up period showed a significantly higher death rate in LC patients with SIBO (51.7 % vs. 15.4 %; $p = 0.042$), with the lowest survival observed in the first 3 months [38]. According to the data of a separate study involving 50 patients with LC (SIBO was detected in 26 (52.0 %) patients: 10 (52.6 %) patients with compensated LC and 16 (51.6 %) with decompensated CP), SIBO (HR = 4.2; 95% CI: 1.2–14.9; $p = 0.028$) and low serum albumin ($p = 0.027$) were significant independent risk factors for death in LC patients. At the same time, SIBO significantly increased the

death rate during the first year of follow-up in patients with decompensated LC and in subsequent years in patients with compensated LC [39].

SIBO increases the permeability of the gastrointestinal mucosal epithelial barrier, which leads to bacterial translocation initiating inflammation of the intestinal wall. The development of intestinal inflammation is associated with the release of vasoactive mediators (e.g., nitric oxide (NO) and prostaglandins), which consistently leads to splanchnic vasodilatation, systemic circulatory dysfunction, hypersecretion of antidiuretic hormone (ADH), stimulation of the renin-angiotensin system (RAAS) and the sympathetic nervous system (SNS). These reactions exacerbate intestinal bacterial translocation and are also predictors of LC decompensation leading to multiple organ failure. At the same time, the chronic systemic inflammation worsens due to an increase of proinflammatory cytokines in response to bacterial LPSs entering the systemic circulation. The proinflammatory immune response leads to a systemic oxidative stress, which also exacerbates bacterial translocation and leads to decompensation of LC with the development of multiple organ dysfunction (Fig. 2) [40, 41].

Patients with LC and SIBO, in contrast to LC patients without SIBO and healthy individuals, are more likely to suffer from circulatory dysfunction due to decreased systolic blood pressure ($p = 0.005$ and $p = 0.011$, respectively) and systemic vascular resistance ($p = 0.001$ and $p = 0.006$, respectively), as well as a higher level of C-reactive protein (CRP) compared with LC patients without SIBO ($p = 0.028$). SIBO is combined with vasodilatation and hyperdynamic circulation in decompensated cirrhosis ($p = 0.002$; $p = 0.012$), but not in compensated LC ($p = 1.000$; $p = 0.474$) [42].

Initial data are now available on the high efficacy of the probiotic *S. boulardii* CNCM I-745 given at 250 mg twice daily for 3 months for the treatment of SIBO in patients with liver cirrhosis, which emphasizes the importance of probiotic support and correction of the composition and functional potential of the colonic microbiota in this disease [43].

Taking into account the above, it seems necessary to further study the relationship between SIBO and colon microbiota in the pathogenesis of cirrhosis to optimize medical tactics and improve the quality of medical care provided to such patients.

Professor O.Y. Zolnikova spoke about the impact of SIBO on the clinical presentation and prognosis in patients with asthma.

For a long time, the contribution of SIBO to asthma remained unclear, so clinical studies

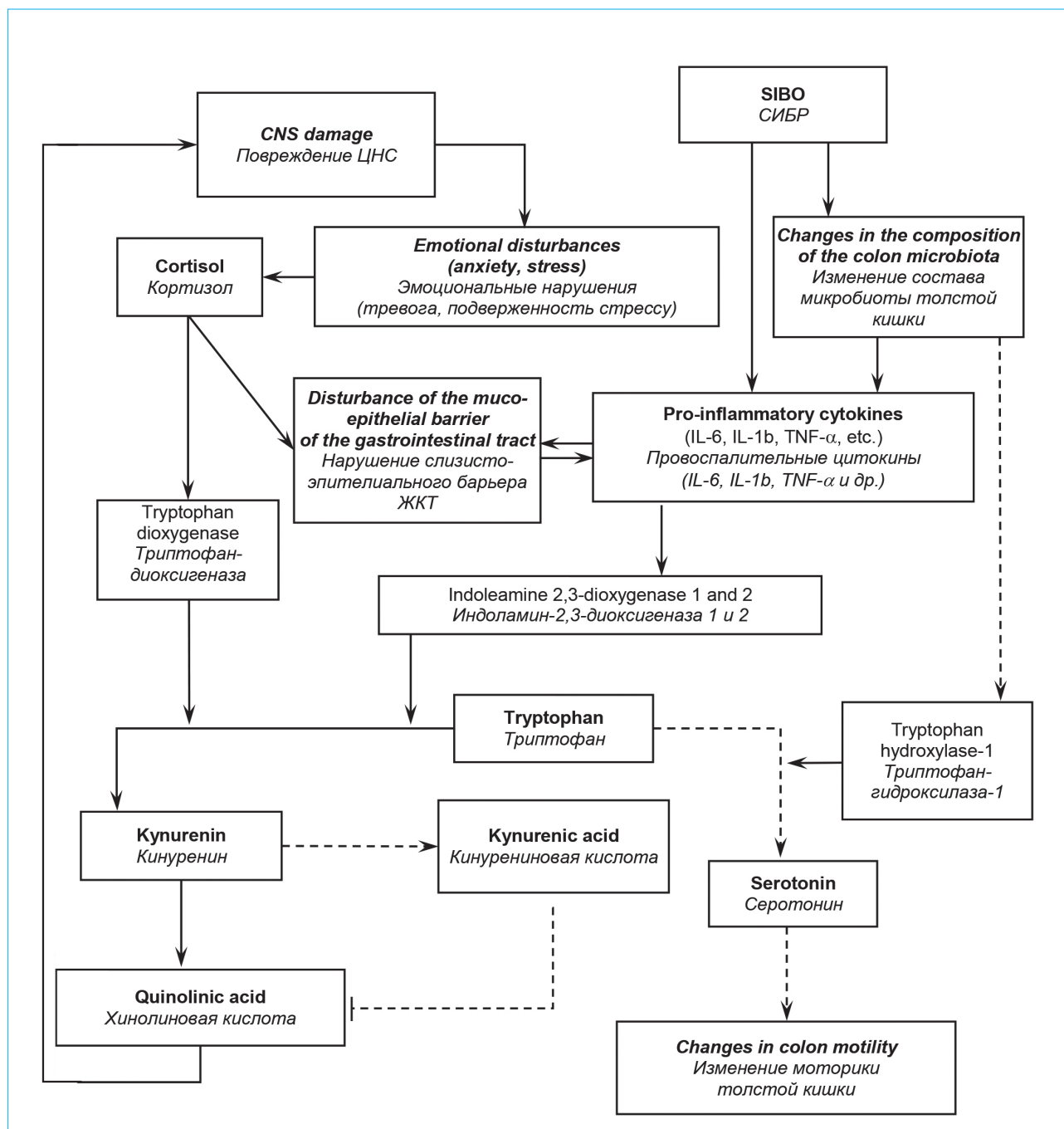


Figure 1. Effect of SIBO on the pathogenesis of irritable bowel syndrome (modified from [23–31]). Continuous arrows represent the stimulating effect of SIBO on physiological pathways, while dotted arrows represent the inhibitory effect. With SIBO, the composition of the intestinal microbiota changes, emotional disorders develop (due to increased levels of kynurenine and quinolinic acid), the muco-epithelial barrier of the gastrointestinal tract is disrupted (due to increased levels of pro-inflammatory cytokines and cortisol), neuroprotective effects in the central nervous system are inhibited (due to decreased levels of kynurenic acid) and changes in colon motility (against the background of decreased serotonin levels)

Рисунок 1. Влияние СИБР на патогенез синдрома раздраженного кишечника (по [23–31] с изменениями). Непрерывные стрелки отображают стимулирующее влияние СИБР на физиологические пути, пунктирные стрелки — угнетающее. При СИБР изменяется состав кишечной микробиоты, развиваются эмоциональные расстройства (ввиду повышения уровня кинуренина и хинолиновой кислоты), нарушается слизисто-эпителиальный барьер ЖКТ (из-за повышения уровня провоспалительных цитокинов и кортизола), угнетаются нейропротекторные эффекты в ЦНС (за счет снижения уровня кинурениновой кислоты) и изменяется моторика толстой кишки (на фоне снижения уровня серотонина)

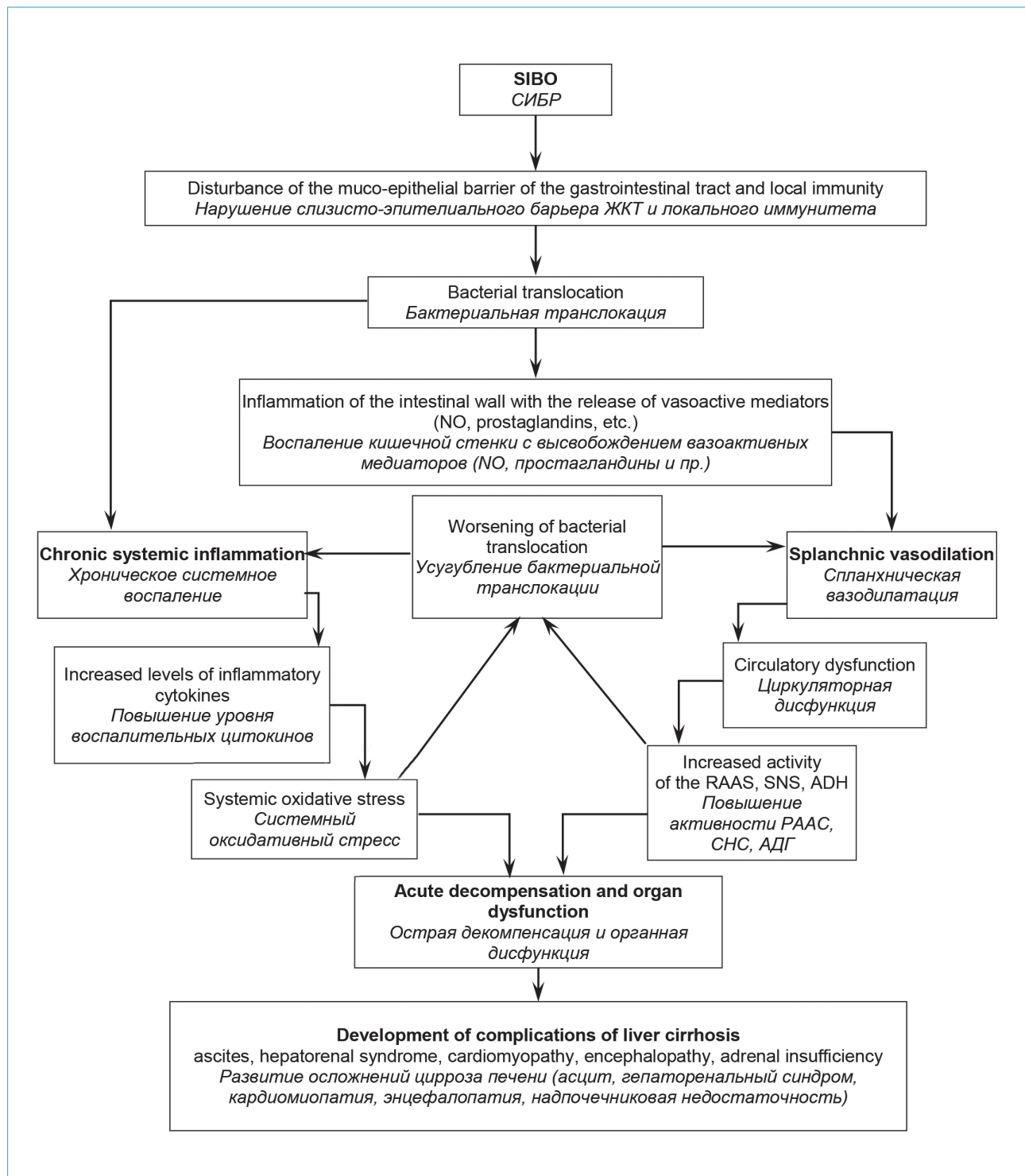


Figure 2. The role of SIBO in the pathogenesis of decompensation of liver cirrhosis and the development of its complications (according to [40, 41] with modifications). SIBO is a predictor of bacterial translocation, which leads to the development of chronic systemic inflammation and splanchnic vasodilation. These processes aggravate bacterial translocation, forming a vicious circle against which decompensation of liver cirrhosis and organ dysfunction develop

Рисунок 2. Роль СИБР в патогенезе декомпенсации цирроза печени и развития его осложнений (по [40, 41] с изменениями). СИБР является предиктором к бактериальной транслокации, которая приводит к развитию хронического системного воспаления и спланхической вазодилатации. Данные процессы усугубляют бактериальную транслокацию, формируя порочный круг, на фоне которого развиваются декомпенсация цирроза печени и органная дисфункция

of this relationship are very limited. Nevertheless, Professor O.Y. Zolnikova's publications clearly demonstrate that SIBO adversely affects the clinical manifestations of asthma and worsens the prognosis in such patients.

SIBO in patients with asthma was characterized by higher blood IgE levels ($p < 0.01$) and sputum eosinophil counts ($p < 0.001$), which was associated with a significantly ($p < 0.01$) more pronounced decrease in the forced expiratory volume in the first second (FEV1), as compared with asthma patients without SIBO. It was noted that the prevalence of SIBO was significantly higher in patients with atopic disease (67 % vs. 43 %; $p = 0.028$), while 40 % of patients had asymptomatic SIBO [44].

It was also noted that SIBO in patients with asthma is associated with specific changes in the composition and functional potential of the gut microbiota. Patients with allergic asthma and SIBO were found to have decreased proportions of bacteria from the classes *Negativicutes* ($p = 0.0008$), *Erysipelotrichia* ($p = 0.01$), *Bacteroidia* ($p = 0.05$) and the families *Erysipelotrichaceae* ($p = 0.01$), *Pseudomonadaceae* ($p = 0.02$), *Rhodospirillaceae* ($p = 0.04$), *Bacillaceae* ($p = 0.02$), as well as increased amounts of organisms from the family *Porphyromonadaceae* ($p = 0.02$) and the genera *Barnesiella*, *Paraprevotella*, *Pyrolobus*, *Bifidobacterium*, *Pseudomonas*, *Coproacter* and *Bacillus* ($p < 0.05$). Patients with non-allergic asthma and SIBO had increased proportions of bacteria from the family Bacteroidaceae ($p = 0.04$) and the genera *Paraprevotella*, *Odoribacter*, *Bacteroides*, *Butyricicoccus* and *Parasutterella* ($p < 0.05$), as compared with such patients without SIBO. Various correlations between changes in the taxonomic profile of bacteria at the phylum level and clinical and laboratory manifestations of asthma have been identified. In allergic asthma, a direct correlation of the level of *Anaerostipes* with the blood immunoglobulin E concentration and FEV1, as well as an inverse correlation with blood and sputum eosinophil counts, has been reported. An inverse correlation of *Faecalibacterium* levels with the blood immunoglobulin E concentration, as well as sputum and blood eosinophil counts, has been demonstrated. *Proteobacteria* levels are directly correlated with FEV1. In patients with non-allergic asthma, the FEV1 value showed an inverse relationship with *Proteobacteria* levels [45]. An SCFA pool analysis of fecal samples revealed a significant decrease in the total amount of fatty acids ($p < 0.001$), acetate, propionate, and butyrate ($p < 0.001$) in all patients with asthma regardless of the disease phenotype, in contrast to healthy individuals [46].

Thus, the negative impact of SIBO on the course of asthma may be due to a decrease in the pool of synthesized SCFAs, an altered composition of the colonic microbiota, and intensified systemic inflammatory immune response. This relationship between the gut microbiota and asthma formed the basis for the concept of the “microbiota – gut – lungs” functional axis, which is considered to play a role in the pathogenesis of a number of bronchopulmonary diseases [47].

In her report, O.Yu. Zolnikova underlined that probiotic support for the eradication of SIBO in patients with asthma allows to improve treatment outcomes. The addition of a multi-strain probiotic to the treatment (rifaximin 800 mg daily for 7 days) results in a significantly greater decrease in blood IgE levels ($p < 0.05$) [48]. Another study demonstrated that the use of probiotics for the eradication of SIBO in such patients increases the duration of remission of asthma and reduces the frequency of hospitalization during the next year ($p < 0.05$) [49]. Finally, the use of probiotics during rifaximin eradication of SIBO in patients with allergic asthma leads to a significantly greater increase in the SCFA pool, as compared with patients receiving only rifaximin ($p < 0.05$) [50]. These data highlight both the importance of diagnosing SIBO in patients with asthma and the need for probiotic support in the treatment of SIBO.

M.V. Fadeeva demonstrated the effect of SIBO on the course of congestive heart failure (CHF).

According to studies, SIBO is a common condition among patients with CHF. According to the author's own data, the prevalence of SIBO among 60 patients with CHF was 42 % ($n = 25$), which was significantly higher ($p = 0.0034$) than its prevalence in a control group of 22 healthy subjects ($n = 2$; 9 %). Interestingly, 60 % of patients with SIBO had no typical complaints of abdominal pain, bloating, impaired frequency, and consistency of stools [51]. In a study involving 102 patients with CHF, SIBO was detected in 38.2 % of subjects by the hydrogen breath test and 47.1 % of patients by the methane breath test [52]. In a later study involving 287 patients with CHF, the hydrogen breath test and methane breath test revealed SIBO in 37 and 28 %, respectively, of patients with preserved ejection fraction (HFpEF) and 25 and 23 %, respectively, of patients with reduced ejection fraction (HFrEF) [53].

A key role in the pathogenesis of CHF is played by neuroendocrine disorders: imbalance of the RAAS, SNS and natriuretic peptides. Current views on the pathogenesis of CHF suggest a role of inflammatory factors as predictors of impairment of these systems [54]. As in liver cirrhosis, hemodynamic disorders in CHF contribute

to edema and hypoxia of the intestinal wall leading to impairment of the gastrointestinal mucosal epithelial barrier, which predisposes to bacterial translocation, maintaining the vicious cycle of systemic inflammation and increased intestinal permeability. Patients with CHF and peripheral edema were found to have a significant increase in plasma concentrations of bacterial LPSs and TNF- α ($p < 0.04$) compared with patients without edema. Interestingly, the LPS level significantly decreased during diuretic therapy ($p = 0.004$), which underscores the role of impaired intestinal permeability and bacterial translocation in the induction of systemic inflammatory response in CHF [55].

It was noted that the severity of systemic inflammation in CHF, as assessed by the plasma levels of IL-1, IL-6, IL-18, TNF- α and CRP, directly correlates with the severity of the disease and mortality. This is due to increased activity of the RAAS and SNS and inhibition of the natriuretic peptide system by proinflammatory cytokines [56]. The natriuretic peptide system exerts an anti-inflammatory effect due to the atrial natriuretic peptide (NT-proBNP), which inhibits the activation of the NF- κ B transcription factor and the release of interleukin-1 β by macrophages. Therefore, inhibition of its activity additionally increases the severity of systemic inflammation in CHF [57]. Another study revealed that an increase in the level of interleukin-6 was significantly ($p < 0.001$) associated with an increase in angiotensin-II levels, which also leads to progression of CHF [58].

Due to the increased severity of systemic inflammation in patients with CHF, ventricular tachycardia is significantly more common in patients with CHF without SIBO (45 % vs. 10.71 %; $p = 0.01557$). The risk of ventricular tachycardia in patients with CHF and SIBO shows a higher correlation with the CRP level ($R = 0.454$; $p = 0.044$) than with CHF severity assessed by the NT-proBNP level ($R = 0.148$; $p = 0.533$) [59].

SIBO in patients with CHF is also associated with higher risks of re-hospitalization for any cause (HR = 1.31; 95% CI: 1.14–1.51; $p < 0.001$), re-hospitalization due to CHF (HR = 1.41; 95% CI: 1.15–1.72; $p = 0.001$), as well as death due to CHF (HR = 1.20; 95% CI: 1.03–1.40; $p = 0.021$) and other causes (HR = 1.21; 95% CI: 1.04–1.40; $p = 0.012$) [52]. A survival analysis in HFpEF and HFrEF patients showed that SIBO increases the risk of re-hospitalization due to CHF in HFpEF patients ($p < 0.001$) and the risk of cardiovascular death in HFrEF patients ($p = 0.011$), being an independent risk factor for these events among all patients with CHF (HR = 2.13; 95% CI: 1.26–3.58; $p = 0.005$) [53].

Data on the effectiveness of a *Saccharomyces boulardii* probiotic are available from a placebo-controlled study involving 20 patients with CHF (NYHA functional class II–III, ejection fraction less than 50 %), whose treatment was combined with *Saccharomyces boulardii* at a dose of 1000 mg per day ($n = 10$) or placebo ($n = 10$) for 3 months. At the end of the follow-up period, the test group demonstrated significant decreases in the left atrial diameter (–0.27 cm vs. +0.22 cm; $p = 0.007$) and CRP (–0.23 pg/mL vs. +0.44 pg/mL; $p = 0.031$), as compared with the placebo group [60].

Concluding, the speaker noted that the timely diagnosis and treatment of SIBO is a necessary measure to improve the quality of medical care for patients with CHF.

RAS Corresponding Member, Professor O.N. Tkacheva spoke about the importance of the diagnosis and treatment of SIBO in the elderly.

In addition to intensified systemic inflammatory response, the negative impact of SIBO on the course of chronic non-infectious diseases and the prognosis in older patients is due to the development of malabsorption. Malabsorption occurring in SIBO is a predictor of malnutrition of the elderly and very old – a disorder due to an imbalance in the intake and consumption of nutrients, which leads to weight loss and a change in body composition. This condition leads to changes in body composition (decrease in lean weight) and body cell mass, impaired physical and mental functioning, and worse overall prognosis for life in the elderly [61].

Timely prevention of malnutrition is an urgent task in geriatrics, since this condition reduces the quality of life of the elderly and increases the duration of inpatient stay, recovery time after acute disease, risks of systemic infections, re-hospitalization, proximal femoral fracture, and death [61]. The main screening tool for malnutrition in the elderly and senile patients is the Mini Nutritional Assessment (MNA) questionnaire scores. The maximum score is 30; a score of 24 or more is typical for normal nutrition and scores in the range of 17–23.5 indicate a risk of malnutrition, while a score of less than 17 means malnutrition [62]. Data obtained in studies of the Russian population showed a high risk of malnutrition among elderly outpatient patients. In a study involving 365 people (mean age – 74.9 ± 6.1 years), malabsorption was detected in 0.3 % of subjects, and a risk of this condition – in 25.8 % of them [63]. Another Russian study assessed the risk of malnutrition using the MNA questionnaire in 611 elderly patients living at home who were aged 65 to 74 years (100 (32.8 %) males and 205 (67.2 %) females)

or 75 years or older (73 (23.9 %) males and 233 (76.1 %) females). The risk of malnutrition was revealed in 17.3 % (106 subjects) of the former group (60.4 % of women and 39.6 % of men), and in 55.7 % of people aged 75 years or older [64].

As O.N. Tkacheva noted, the incidence of SIBO in the population increases with age and is closely associated with chronic non-infectious diseases, which suggests a high prevalence of SIBO among elderly patients with comorbidities. Diagnosis and treatment of SIBO should be regarded as a necessary measure to prevent the development of malnutrition and increase the survival in this group of the Russian population.

During the Expert Council, reports on the prevalence, diagnostic opportunities, and approaches to the treatment of SIBO in Russian regions were presented by the Chief Gastroenterologists of the Federal districts of the Russian Federation: **N.V. Korochanskaya, Dr. Sci. (Med.)** (Southern Federal district); **A.Yu. Baranovsky, Dr. Sci. (Med.)** (North-Western Federal district); **O.P. Alekseeva, Dr. Sci. (Med.)** (Volga Federal district); **I.B. Khlynov, Dr. Sci. (Med.)** (Ural Federal district); **O.Y. Zolnikova, Dr. Sci. (Med.)** (Central Federal district); **V.V. Tsukanov, Dr. Sci. (Med.)** (Siberian Federal district); **S.A. Alekseenko, Dr. Sci. (Med.)** (Far Eastern Federal district); **S.N. Mammaev, Dr. Sci. (Med.)** (North Caucasian Federal district).

The specialists noted that the collection of statistical data on the prevalence, diagnosis, and treatment of SIBO in the regions is associated with significant difficulties due to the absence of obligations to register data in the annual reports of the Chief Gastroenterologists of the Federal districts. However, according to the presented statistics from the Krasnodar Territory for 2022, SIBO was diagnosed in 24.9 % of all registered cases of gastrointestinal diseases. A diagnosis of SIBO is made in most regions based on the clinical manifestations of the disease, due to the absence of gas analyzers for a breath test. Instrumental SIBO tests are carried out only within the framework of voluntary health insurance or paid medical services using gas analyzers of foreign manufacture, which limits the availability of optimal medical care to the population. Another urgent problem in the regions of the Russian Federation is the low awareness of physicians about the approaches to the diagnosis and treatment of SIBO. The Chief Gastroenterologists noted the need for a wide introduction of breath tests for timely diagnosis of the disease and improving the quality of care for patients with gastrointestinal diseases in the Russian regions.

In his final speech, Expert Council Chairperson V.T. Ivashkin noted that the optimization of approaches to the diagnosis and treatment of SIBO in various specialties of medical practice seems to be a highly effective measure to improve the quality of public healthcare in the Russian Federation. However, this is associated with several difficulties requiring organizational measures within the framework of Federal programs under the supervision of specialized reference centers of the Ministry of Health of the Russian Federation.

In 2023, the Sechenov University launched a reference microbiota center of the Ministry of Health of the Russian Federation (headed by Dr. Sci. (Med.), Professor of the Department of Propaedeutics of Internal Diseases, Gastroenterology and Hepatology E.A. Poluektova). The center's objectives include improving microbiota research methods, developing standards and methods for quality control in diagnostics, improving the qualifications of clinicians, as well as implementing innovative approaches in healthcare practice. Thus, the optimization of public healthcare in all regions of the Russian Federation through increased awareness of physicians and the introduction of timely diagnosis and treatment of SIBO into clinical practice can be achieved with the efforts of the Sechenov University reference center with the support of the Ministry of Health of the Russian Federation at the Federal level.

After a discussion of the reports, a resolution was adopted.

Expert Council Resolution

1. Small intestinal bacterial overgrowth (SIBO) is a common condition that complicates chronic non-infectious diseases (including irritable bowel syndrome, liver cirrhosis, asthma, and congestive heart failure), and is also a predictor of worse prognosis and early death in the elderly.

2. Disorders due to SIBO affect almost all human organs and systems; therefore, regardless of the severity of its clinical manifestations, timely diagnosis of SIBO is essential for improving treatment outcomes in a wide range of chronic non-infectious diseases.

3. The hydrogen and methane breath tests with a carbohydrate are a non-invasive, low-cost, and most convenient tool for SIBO diagnosis. Significant limitations associated with the breath tests are the low availability of gas analyzers in the Federal districts of the Russian Federation, the absence of this approach in the Compulsory Health Insurance (CHI) care standards, and low awareness of physicians.

4. Domestic gas analyzers must be developed and subsequently introduced in the regions of the Federal districts of the Russian Federation.

5. The hydrogen breath test with a carbohydrate must be included in the CHI standards for the diagnosis of SIBO in outpatient and inpatient settings.

6. For better awareness of physicians and improved quality of medical care, clinical guidelines for the diagnosis and treatment of SIBO must be developed and information about the diagnosis and treatment of this condition should be added to the current clinical guidelines for the diagnosis and treatment of chronic non-infectious diseases.

7. A Federal program should be initiated to study the impact of timely diagnosis and treatment of SIBO in terms of increasing the quality of medical care in the regions of the Russian Federation.

8. Optimization of the current treatment regimens and development of new SIBO treatment approaches are required to increase the quality and efficacy of SIBO treatment.

9. The therapeutic use of the probiotic strain *Saccharomyces boulardii* CNCM I-745 at a dose of 5×10^9 CFU (250 mg) twice daily for the entire period of antibiotic therapy is an effective measure improving SIBO treatment outcomes.

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