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REVIEW ARTICLE

Relationship between SIBO and other bowel diseases and a common eating pattern for them. Part III

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Abstract

Introduction and Objective. Conditions resulting from diseases of the brain-gut axis and gum-gut axis show many mutual, often bi-directional interrelationships. The accompanying quantitative and/or qualitative disorders of intestinal microflora may be effectively regulated by implementation of a properly adjusted diet therapy. The aim of the study is to investigate whether there is a relationship between small intestinal bacterial overgrowth (SIBO), and irritable bowel syndrome (IBS), and non-specific inflammatory bowel diseases (IBD), as well as indications for the mode of nutrition.

Review Methods. A literature review was performed using the databases PubMed, Google Scholar and Web of Science. A short synthesis of the collected information was made by a non-systematic literature review.

Brief description of the state of knowledge. From the clinical point of view, SIBO is most often associated with IBS. Both conditions have common symptoms, such as: abdominal pain, flatulence bloating and diarrhea, as well as similar neuropsychological disorders. In turn, IBS have so many characteristics in common with IBD that the term IBS in IBD has even been proposed. Concerning diet therapy, a low FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides and polyols) is the most recommended diet. However, probiotic therapy is recommended to restore microbiological balance.

Summary. At present, an increasing number of studies indicate a close relationship between SIBO and IBD, with SIBO occurring more often in patients suffering from Crohn's disease than those with ulcerative colitis. In order to achieve good outcomes of treatment and prevent the recurrence of these diseases, interdisciplinary and inter-professional cooperation is required in the area of skilful, individualized combination of pharmacotherapy, psychotherapy, probiotic therapy, and diet therapy.

Key words

therapy, irritable bowel syndrome, inflammatory bowel diseases, small intestinal bacterial overgrowth, diet nutritional recommendations

INTRODUCTION

Clinical observations and data from literature indicate the presence of a cause-effect relationship between diseases concerning many systems and organs in the human body. In articles published to-date by the authors of the presented study, the functioning of the gum- gut axis has been confirmed, and the bi-directional character between periodontitis and non-specific inflammatory bowel diseases (IBD), i.e. Crohn's disease (CD), and ulcerative colitis (UC) [1, 2]. *In turn, inflammatory bowel diseases* (IBD) show so many common symptoms with irritable bowel syndrome (IBS), that the term IBS in IBD has even been proposed [3]. Among diseases concerning the gastrointestinal tract, IBS is most often associated with the condition caused by small intestinal bacterial overgrowth (SIBO) [4, 5]. An increasing number of studies also indicate a close relationship between SIBO and IBD [6, 7]. Among the multifactorial etiology of these diseases, the disorder of the brain-gut-microbiome axis seems to be the main element combining them. Quantitative and/or qualitative disorders of microbiota may, to some extent, be regulated by the mode of nutrition adjusted to pathologically changed functions of the gastrointestinal tract.

OBJECTIVE

The aim of the third part of the study is investigation of the relationship between SIBO, and IBS and IBD, as well as assessment of the dietary strategy which may play an important role in the prevention of analyzed diseases and an effective alleviation of their course. For this purpose, a literature review was carried out using the databases PubMed, Google Scholar, and Web of Science. During the literature search the following key words were taken into account, considering terms included in the MeSH thesaurus, i.e. IBS, irritable bowel syndrome, SIBO, intestinal bacterial overgrowth, IBD, inflammatory bowel diseases, CD, Crohn's disease, UC, ulcerative colitis, diet, and nutrition. Abstracts and titles of appropriate articles were analyzed,

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and exclusively full-length articles containing quantitative statistical analyses and meta-analyses were included in the presented review. Studies concerning children and pregnant women, as well as studies on animals, were excluded. w. A short synthesis of the collected information was made by non-systematic literature review.

SIBO – general characteristics. Small intestinal bacterial overgrowth (SIBO) is a clinical disorder of the gastrointestinal tract caused by an excessive number of bacteria in the small intestine [3], with the prevalence of gram-negative and anaerobic species, especially *Streptococcus, Staphylococcus, Bacteroides, Lactobacillus,* and *Enterobacteriaceae–Escherichia, Klebsiella, Proteus)* [8]. In physiological conditions, bacteria inhabiting the large intestine do not colonize the jejunum and proximally located sections of the digestive tract, because it is prevented by endogenous defence mechanisms [8, 9]. In the case of development of SIBO, antibacterial mechanisms are disturbed, such as: decreased acidity of gastric juice, exocrine pancreatic insufficiency, or immunodeficiency syndromes. Anatomical abnormalities play a large role, including small intestine obstruction, diverticula, fistulas, previous ileocecal resections, and primarily small intestine motility disorder [10]. The most frequent symptoms and complaints occurring in SIBO are abdominal pain or discomfort; diarrhea, and sometimes fatty stools may also appear [11]. However, attention should be paid not only to the symptom profile, but any past and concomitant diseases should also be analyzed. SIBO may occur in the course of other diseases, such as diabetic autonomic neuropathy, amyloidosis, hypothyroidism, Hashimoto's disease, or coeliac disease. Moreover, SIBO is a condition that is quite common among patients with irritable bowel syndrome (IBS) [12]. The gold standard in diagnosing SIBO is finding bacteria with more than 10^3 colony-forming units (cfu) per 1 g, or 1 ml in the culture from intestinal contents taken from the proximal part of the small intestine [13]. Nevertheless, considering invasiveness, time-consuming and high costs, this test is not implemented in routine diagnostics.

The diagnosis and identification of SIBO has significantly improved due to the introduction of more sensitive non-invasive methods which involve the measurement in exhaled air of such gases as: hydrogen, hydrogen sulfide and methane, which human cells cannot produce on their own [7]. Hydrogen is produced by the fermentation of sugar by bacteria living in excess in the small intestine. The subsequent gas which may be produced in the small intestine is hydrogen sulfide, which means SIBO-H2S (small intestinal bacteria overgrowth – hydrogen sulfide) [14]. In turn, with archaeal overgrowth (prokaryotic microorganisms), methane is produced, and IMO (*intestinal methanogen overgrowth*) is subsequently diagnosed [15]. SIBO and IMO very frequently occur simultaneously [5, 16]. Small intestinal bacteria overgrowth (SIBO) may be diagnosed using 3 types of respiratory tests [5, 17]:

- hydrogen breath test (HBT), including the measurement of hydrogen and carbon dioxide after previous oral administration of 75 g of glucose, or 10 g of lactose;
- hydrogen-methane breath test (HMBT) – measurement of hydrogen, methane and carbon dioxide;
- TRIO SMART® test (also SBT, sulfur breath test) – measurement of hydrogen, methane, hydrogen sulfide and carbon dioxide.

At present in Poland, the hydrogen breath test and hydrogen-methane breath test are performed, whereas Trio SMARTi has not yet been introduced into general laboratory diagnostics.

The consequence of bacterial overgrowth may lead to various health complications which are associated mainly with depression and anxiety. Patients suffering from SIBO, compared to those who are healthy, show a higher level of neuroticism, lower levels of extroversion and higher levels of anxiety and stress [18]. Apart from psychological disorders, in patients with SIBO/IMO symptoms related to the deficiency of individual nutrients are often observed, including vitamin B12, fat-soluble vitamins and iron. Skin problems also occur, which result from the poor condition of the intestines and their increased permeability.

In addition, patients develop intolerance, most frequently intolerance to histamine, lactose and fructose [19]. Patients are treated mainly with antibiotics, such as neomycin and metronidazole. Recently, the effectiveness of eubiotic rifaximin has also been confirmed. Other biotic agents are also applied, including selected probiotic strains showing a beneficial effect, which is confirmed by the results of examinations using breath tests [20]. The use of herbs showing antibacterial effect is also a common practice [21]. The treatment of bacterial overgrowth requires a holistic approach and an interdisciplinary therapeutic team. In the majority of patients, complete recovery is difficult to achieve, and periods of remission and relapse of the disease may alternate [22].

Relationship between SIBO and irritable bowel syndrome (IBS). Irritable bowel syndrome (IBS) is a functional intestinal disease which belongs to diseases resulting from disorders of gut-brain interaction. Recently these interactions have been extended to the brain -gut-microbiome [23]. According to the Rome IV criteria [24], irritable bowel syndrome (IBS) may be diagnosed when recurrent abdominal pain has occurred ≥1 day per week for the last 3 months, on average, and meets ≥2 out of the following criteria:
1) related with bowel movements;
2) related with a change in the frequency of bowel movements;
3) related with change in the shape (appearance) of the stool.

According to the type of disorder in bowel movements reported by the patient, IBS may be additionally divided into individual sub-types:
– IBS with predominant constipation (IBS-C);
– IBS with predominant diarrhea (IBS-D);
– BS with mixed disorders (IBS-M).

In addition, in patients meeting the criteria of IBS, but cannot be exactly qualified to any of the previously mentioned subtypes, IBS unclassified (IBS-U) is diagnosed [24]. Extra-intestinal symptoms may also occur, concerning mainly disorders of a psychological nature. The results of a meta-analysis conducted by Hu Z. et al. [25] demonstrated that among patients with various subtypes of IBS, the highest level of anxiety and depression was observed in IBS-C, followed by IBS-M. In these patients, psychological examinations and proper psychotherapy are required.

The majority of patients also report painless discomfort in the abdominal cavity, co-occurring with psychological disorders because the pathogenesis of IBS is largely

psychogenic [23]. However, at present it is considered that microbiological factors play a key role [26] which, at the same time, differentiate IBS subtypes. Pathogens which cause inflammatory states of the stomach and intestines, and consequently the development of IBS, produce cytolethal distending toxin B (CdtB). Antibodies produced against CdtB cross-react with cytoskeletal protein vinculin, contributing to impairment of gut motility, which is conducive to bacterial growth. IBS-D then develops which may be diagnosed by positive results of the hydrogen breath test. In turn, IBS-C is related with an elevated level of archaea, in which case positive results of methane test are obtained. Methane gas slows down intestinal contractility, resulting in the constipation characteristic of IBS-C (26). Bacterial dysbiosis and quantitative and qualitative disorders concerning other microorganisms exert an unfavourable systemic effect by reducing the body's immune functions, and disruption of functioning of the brain-gut axis.

Recently, it has been confirmed that SIBO plays an important role in the pathogenesis of symptoms in patients with IBS. It was found that both in patients with SIBO and IBS there occurs an increased number of pathogenic microorganisms, including Enterococcus, Escherichia coli and Klebsiella, as well as archaeon Methanobrevibacter smithii. It was confirmed that SIBO occurs both in patients with irritable bowel syndrome with predominant constipation (IBS-C), and those with IBS with predominant diarrhea (IBS-D), although more often in the case of IBS-D [4].

Studies, as well as clinical observations, indicate that SIBO and IBS have many common symptoms, including abdominal pain, flatulence and diarrhea, as well as similar neuropsychological disorders [5]. The frequency of occurrence of SIBO among patients diagnosed with IBS varies. A systematic review carried out by Shah A. et. al. (25 studies with the participation of 3,192 patients with IBS, and 3,320 persons from the control group), the average frequency of occurrence of SIBO in patients with IBS was significantly higher statistically, compared to the control group (OR = 3.7; 95% CI 2.3 – 6.0) [5]. The differences in the frequency of SIBO in patients with IBS was due to clinical heterogeneity resulting from the lack of uniform criteria for selecting cases and controls, and limited sensitivity and specificity of available diagnostic tests, e. g. SIBO prevalence diagnosed by lactulose breath test was much greater in both patients with IBS (3.6-fold) and controls (7.6-fold), compared with the glucose breath test. A study by Ndong PO et al. showed that the frequency of occurrence of SIBO in IBS was 36.4% (9.7% in hydrogen tests, 26.% in methane tests). The intensity of IBS was not correlated either with the level of hydrogen, or the level of methane in the body. However, it was confirmed that hydrogen production was inversely correlated with the quality of life of the patients [27]. In turn, a study by Ding XW et al. [28] demonstrated that in a group of 50 patients with the diagnosis of IBS, 36 persons (72%) were diagnosed with SIBO (confirmed by breath tests). The study showed that SIBO might prolong the oro-ceacal transit time (OCTT), but did not deteriorate diagnostic parameters determining the diagnosis of IBS. A study by Chuah KH et al. [29] included a multi-ethic group of 186 adult Asians with functional disorders of the digestive tract, and a 58-person control group. SIBO was diagnosed mainly in patients diagnosed with IBS-D, where a dominant problem was diarrhea, and was significantly higher than in the control group (24% vs. 10%).

The above-mentioned studies show that SIBO frequently co-exists with IBS, and SIBO usually does not cause intensification of complaints observed in irritable bowel syndrome.

Relationship between SIBO and IBD. Crohn's disease (CD) and ulcerative colitis (UC) are classified into non-specific inflammatory bowel diseases (IBD), while Crohn's disease is a chronic autoimmune disease in which the inflammatory state may concern any section of the gastrointestinal tract, from the oral cavity to the anus [30]. In the case of UC, inflammatory changes concern the mucous membrane of the rectum, or rectum and colon [31].

While analyzing the relationship between IBD and SIBO it was demonstrated that SIBO occurs more often in patients suffering from Crohn's disease, compared to those with ulcerative colitis [32]. A study conducted by Wanzl et al. [33] showed that in patients with ulcerative colitis the frequency of occurrence of SIBO did not differ from that observed in patients with other diseases of the gastrointestinal tract (e.g. infectious colitis, collagen colitis, or irritable bowel syndrome). This was confirmed by a study by Ghoshal et al. [34], which included 86 patients with IBD (45 with ulcerative colitis and 41 with Crohn's disease). The frequency of occurrence of SIBO was significantly higher in patients with CD (34.1%), compared to those with ulcerative colitis (4.4%).

The co-occurrence of SIBO and CD is justified by specific pathogenesis of CD [35]. In patients with CD, a decreased intestinal motility is observed in which intestinal fistulas and strictures occur, with primarily damage to the ileocecal valve. In turn, SIBO is related with an increased level of endotoxins in serum and the presence of pro-inflammatory cytokines, stimulated by bacterial endotoxins. The above is conducive to the induction of an immune response and increasing inflammation associated with intestinal damage which, consequently, may result in a state of exacerbation of CD [36]. This was reflected in a clinical study by Jinling in a hospital in China. The study covered 73 patients, including 39 with CD alone, and 34 with both CD and SIBO (confirmed by breath test using hydromethane and lactulose). Patients were observed for 18 months and evaluated from the aspect of activity of CD indicator, determining the concentration of calprotectin in stool. It was found that in patients with SIBO the risk of CD recurrence was 2.27 times higher, compared to persons without SIBO. It was also demonstrated that patients with SIBO had a lower level of total protein and serum albumin than those without SIBO (6.2±1.5 g/dl vs. 7.0±0.9 g/dl; and 3.5±0.9 g/dl vs. 4.0±0.6 g/dl, respectively) [37]. In turn, in patients with ulcerative colitis, the inflammatory state lead to changes in gastrointestinal motility and extended the oral-caecal transit time, which predisposed to the development of SIBO [38].

The factors considerably increasing the risk of development of SIBO in IBD was female gender, older age, and intestinal fibrosis and, above all, intestinal surgery (especially resection of the ileocecal valve) [39]. The symptoms of SIBO and IBD are similar, but differentiation between SIBO and exacerbation of the disease in patients with IBD becomes the key for optimizing the treatment of these patients, because the treatment of both diseases is different. It was confirmed that correctly treated SIBO contributes to the reduction of negative symptoms of IBD [40]. In addition, patients with CD, especially those with concomitant intestinal inflammation with stenotic disease, are recommended the diagnostics of

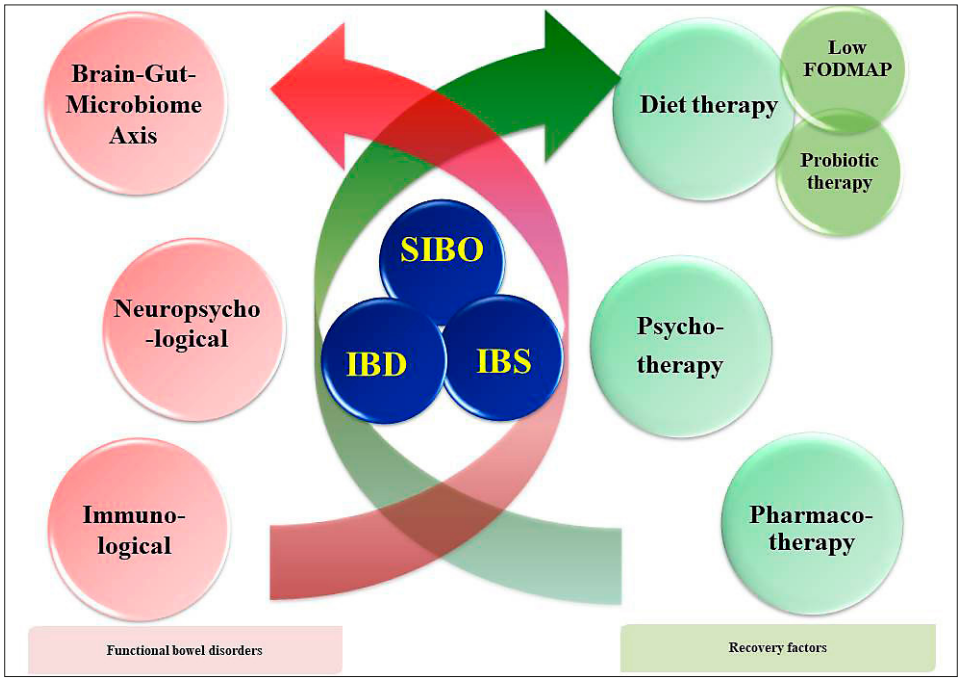

Figure 1. Factors contributing to SIBO, IBS and IBD

SIBO, and subsequently targeted treatment [39, 41]. Currently, both researchers and clinicians consider that the relationship between SIBO and IBD, especially between CD and SIBO, is unquestionable. However, there is no unanimity on whether SIBO is the cause or the effect of CD. On the one hand, patients with CD are more susceptible to developing SIBO than patients without complaints on the part of the intestines [4]. On the other hand, SIBO – through excess bacteria and their metabolites – may intensify intestinal inflammation which, in turn, may exert an unfavourable effect on the course of CD and significantly worsen the prognosis [7]. Therefore, according to some authors, there is a two-way pathology in between CD and SIBO [9], while according to others, SIBO is independently related to clinical symptoms of CD, and primarily with inducing exacerbations of the disease [8].

Common aspects of nutrition in SIBO and IBD and IBS. Improper consumption of nutrients leading to post-prandial hyperglycaemia, and long-term diet inconsistent with the principles of rational nutrition, have a considerable effect on the development and course of systemic inflammatory conditions [42]. The common denominator which improves the quality of life of patients in the event of functional disorders of the digestive tract is the diet. The International Organization for the Study of Inflammatory Bowel Disease (IOIBD) confirms the important role of improper diet in microbiome disorders and pathogenesis of SIBO, as well as IBD and IBS [43]. However, it is worth remembering that dietary recommendations concerning small intestinal bacterial overgrowth (SIBO) are equivocal. At present, it is considered that the most optimum solution is application of a low FODMAP diet (low fermentable oligosaccharides, disaccharides, monosaccharides and polyols). This diet is recommended mainly for adults with irritable bowel syndrome (IBS) [44]. Considering the fact that SIBO very frequently co-occurs with IBS and IBD, the recommendations published in 2020 by the American College of Gastroenterology (ACG) also recommend a low FODMAP diet for patients with SIBO [45]. A study by Prince et al. justified the use of a low FODMAP diet in patients with IBD. A beneficial effect of this diet was confirmed both in patients with CD and those with UC without an active inflammatory state on the reduction of symptoms related with IBD, such as abdominal pain, flatulence and diarrhea [46]. Common aspects of nutrition in SIBO, IBD and IBS, as well as protective and predisposing functional disorders of the digestive tract, is shown in Figure 1.

The safety and effectiveness of a low FODMAP diet in patients with IBD was assessed in a study by Bodini et al. [47]. A low FODMAP diet applied for six weeks was safe for patients with IBD, and was associated with an improvement in inflammation indicators (measured by the concentration of calprotectin), and better quality of life in IBD-Q. In addition, a study by Melgaard et al. [48] showed that after introduction of a low FODMAP diet in patients with UC, there occurred a decrease in abdominal pain and flatulence; however, the symptoms recurred both after placebo challenge and FODMAP. IBS and IBD share many common symptoms, and there are some overlapping mechanisms in these disorders, such as increased gut permeability, altered immune system activation, inflammation, or changes in enteric nervous system and gut microbiota [49]. It was confirmed that a decrease in the intensity of symptoms and the improvement of the quality of life of patients with IBS in IBD was observed after introduction of a low FODMAP diet, whereas a diet rich in FODMAP products exacerbated the symptoms. Products rich in FODMAPs intensify symptoms because their digestion and absorption is very limited; therefore, they enter the intestine in an unaltered form. They show the so-called osmotic effect, i. e. increased water absorption in the small intestine. In addition, in the large intestine they are fermented by intestinal bacteria and produce excessive amounts of gas, causing pain, discomfort and bloating [50]. In turn, a study by McIntosh et al., comparing the effect of low and high content of FODMAP on the intensification of symptoms, metabolomics markers and microbiome in patients with IBS,

showed a slight decrease in the production of hydrogen in persons who consumed a low FODMAP diet [51].

A low FODMAP diet consists of three phases: elimination phase, reintroduction phase and individualization phase. The elimination phase lasts for 4–6 weeks and consists in the exclusion of products containing high amount of FODMAP, or considering these products in a specified amount (size of portions) in particular meals. The time of this restrictive phase should be established carefully under the supervision of a doctor and a dietitian. The re-introduction phase lasts for 6–10 weeks and is aimed at the verification of tolerance to individual FODMAP products. During this phase, the patient should continue the diet applied during the first phase, and gradually introduce the subsequent attempts of testing individual products. The patient should keep a food diary to register the occurrence of particular symptoms and the degree of their intensity at the determined portion of the product. The third phase, i.e. individualization, consists in gradual expansion of the diet and introduction of products containing FODMAP, in amounts, however, well tolerated by the patient. Products which cause mild exacerbation of symptoms may be introduced in smaller amounts, or implemented more rarely. A low FODMAP diet may be difficult to apply in persons suffering from nutrients deficiency, eating disorders, as well as pregnant and breastfeeding women [52]. In such cases, the application is recommended of a gentle-low FODMAP diet consisting in the elimination of only some products characterized by a high content of FODMAP, and/or elimination of several selected FODMAP groups. Restrictions can be extended if necessary. According to this strategy in the first phase, the elimination is recommended of: wheat and rye (and derived products), onions, leek, cauliflower and mushrooms, apple, pear, dried fruit, stone fruit and watermelon, as well as milk and yogurt, and legume seeds [53].

The main source of data concerning the content of individual FODMAP in food products is the database at Monash University in Melbourne, Australia – used worldwide by means of the application Monash University FODMAP Diet®. Currently, researchers from this university created a FODMAP calculator [54]. In Poland, due to its high cost, the calculator is used mainly in scientific and research centres.

It is worth emphasizing that a low FODMAP diet is an elimination and unbalanced diet. Long-term limitation of FODMAP products may increase the risk of undesirable changes in intestinal microbiota. It is not certain whether a diet with a low content of FODMAP is useful and necessary for patients with SIBO, especially when applied for a longer period of time [55]. Resigning from food rich in FODMAP carries the risk of insufficient energy intake. There often occurs a deficiency of nutrients, such as complex carbohydrates, fibre, calcium, iron, zinc, folic acid, vitamins from group B, vitamin D, as well as compounds with antioxidant activity [56]. Ensuring proper consumption of food products that compensate for these deficiencies is very important for balancing the metabolic needs of the body. Before starting a low FODMAP diet it is important to consider both its indications and contraindications before using it.

In the diet therapy of SIBO and IBD, other unconventional diets are also implemented, including the Bi-Phasic diet which consists of two successive phases. The first is the intestinal regenerating phase; the second phase is the period of preventing the growth of microorganisms in the small intestine. The two-phase diet protocol uses a treatment method that limits the side-effects of rapid bacterial and fungal death. Despite generally accepted approaches, the Bi-Phasic Diet should be personalized according to individual tolerance [56]. In turn, a low-sulfur diet is used in patients suffering from hydrogen sulfide overgrowth. The goal of the diet is to limit the supply of foods containing sulfur in order to achieve the lowest possible level, because a high sulfur diet (high in animal protein and fat, and low in fibre) leads to nutrient deprivation for the microbiota [57]. The ketogenic diet is a high-fat diet based on a high supply of fats which may affect both the diversity and quantity of intestinal flora. It is worth mentioning that the side-effects of the diet can be gastrointestinal reactions (e.g. constipation), which can be relieved by adjusting the diet (e.g., supplementing with adequate water, etc.). However, whether the adverse effects of the ketogenic diet are linked to changes in intestinal flora remains to be studied further [58].

The disadvantage of all unconventional diets is the lack of assessment of their long-term effects for the functioning of the body at the organ and cellular levels. Most of the research conducted is limited to a relatively short period, they cover small groups, the correlation with comorbidities is not taken into account, and there is no reassessment of the results obtained. The individualization of diets is also insufficient.

The use of probiotics is a commonly used dietary approach biased towards gastrointestinal microbiome. According to the International Scientific Association for Probiotics and Prebiotics (ISAPP), '*Probiotics are live microorganisms that, when administered in adequate amounts, confer a health benefit on the host'* [59]. Meta-analysis comparing randomized control trials (RCT) of individual strains with trials of many strains, demonstrated that generally in the case of majority of diseases, multi-strain mixtures were no more effective than single-strain probiotics [60]. In recent years, interest has increased in the use of yeast-based probiotics, because not only are they naturally resistant to antibiotics, but also because they can be used in persons undergoing antibiotic therapy. Yeast probiotics are highly resistant to gastrointestinal enzymes, bile salts, pH changes, organic acids and changes in temperature, due to which there is a great probability that they will reach the colon without major changes. Importantly, they are not absorbed from the digestive tract; therefore, the risk of overdose is negligible. As a natural health product, the strain *Saccharomyces cerevisiae* CNCM I-3856 received the following health claim in Canada: '*Helps to reduce abdominal pain and discomfort associated with irritable bowel syndrome (IBS)*'. In addition, *Saccharomyces cerevisiae* CNCM l3856, has been registered in the National Collection of Cultures of Microorganisms. The analgesic effect of this strain is dose dependent, and the maximum effect is 10×10 CFU/daily. In addition, a review of the latest scientific data concerning yeast with probiotic potential carried out in 2021 by Staniszewski A. et al., showed that *Saccharomyces cerevisiae* var. *boulardii* plays a modulating role in the proper functioning of the intestinal microbiome, and is an antagonist against pathogens. It also has an immunomodulatory and trophic effect on the digestive tract. *Saccharomyces cerevisiae* var. *boulardii* colonizes mucosal surfaces and is part of the normal flora of the gastrointestinal tract and respiratory tract. It also mediates pharmacodynamic effects, which has the protective effects of normal, healthy intestinal flora [61].

The consumption of yeast *S. cerevisiae* may exert a beneficial effect on human health due to its ability to colonize the lining of the stomach and intestines. The results of a study conducted

by García-Collinot et al. which included patients with SIBO, demonstrated that supplementation with *Sacharomyces boulardii* (CNCM I 745) in patients with SIBO and systemic sclerosis was associated with considerably higher eradication rates, and a decrease in exhaled hydrogen, compared to metronidazole alone therapy [62]. Analysis performed by Wielgosz-Grochowska JP. et al. showed that monoprobiotics with well-characterized strains may play a beneficial role in the prevention of progression of symptoms of IBS and SIBO. There is a need to focus on common probiotic strains, such as *Sacharomyces boulardii* (CNCM I 745) and *Lactobacillus reuterii* (DSM 17938) which have eradication properties, which may be indispensable in the therapy of in the treatment of inflammatory diseases of the digestive tract [63].

Many studies show the undeniable benefits of using various probiotic strains. However, it is important to note that clinical trials vary in dose, composition, and group of people or materials included the research. The strain should be adapted not only to the disease, but also to its phase. Additional studies, including high-powered RCTs, are needed, to determine which patients may benefit from probiotics and which probiotic strains are the most effective [64].

SUMMARY

In analyzing diseases of the gastrointestinal system: irritable bowel syndrome, inflammatory bowel diseases, and small intestinal bacterial overgrowth, despite the existing differences, have many features in common regarding etiology, course and health consequences. Their frequent co-occurrence and interaction is observed, and the features they have in common include intestinal microbiome and immune disorders. In turn, an increased response to stress results from impaired functioning of the brain-gut axis, and co-occurrence of psychogenic diseases, and often there is a positive feedback. Traumatic experiences, as well as chronic stress, are important factors in the etiology and course of gastrointestinal diseases. In turn, symptoms on the part of the gastrointestinal tract cause psychological discomfort which, in combination with stress, results in exacerbation of the course of the analyzed diseases. Therefore, psychotherapy and psychoeducation which help the patient to learn ways of coping with stress, should be an important element in endeavouring to overcome fear of the symptoms and consequences of the disease.

The diseases require an interdisciplinary and interprofessional approach. It is up to the doctor and dietitian to create a skilful, personalized combination of pharmacotherapy, probiotic therapy and diet therapy. In the field of diet therapy, several diets are promoted; however, the most universal is the low FODMAP diet, i.e. limitation of fermentable oligosaccharides, disaccharides, monosaccharides and polyols.

In turn, the patient must be informed not only about the need to follow the recommended diet, but also about the need to implement other principles for a healthy life style and health promoting personal behaviours concerning the control of body weight, physical activity, sleep hygiene, non-smoking and resigning from stimulants. Only the combination of all these elements may contribute to the achievement of good outcomes. However, most frequently, in the case of chronic diseases the way to recovery is usually not linear and patients must be aware that relapses may occur.

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