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THE POTENTIAL OF GUT MICROBIOME PROFILING FOR PERSONALIZED MANAGEMENT OF PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A REVIEW

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Abstract

Introduction. Chronic obstructive pulmonary disease (COPD) remains a major global health challenge due to the unmet need for early prediction of the disease course and therapy results. Exploring the potential of gut microbiome profiling to develop targeted interventions for COPD could help take a significant step toward implementing precision medicine into clinical practice.
Aim. To analyze recent data regarding gut microbiome profiling for the personalized management of COPD patients based on an assessment of articles from the MEDLINE database.
Materials and methods. The MEDLINE database via PubMed was searched for articles published in 2015-2025 using a combination of predefined keywords. The search resulted in 173 articles, which were initially screened and assessed based on the titles and content of the abstracts. After evaluating relevance, for detailed assessment, we selected 85 articles. Finally, 50 articles were included in the review.
Results. The gut microbiome profiles of various cohorts of COPD patients differed significantly. Several studies report reduced microbial diversity in COPD. Patients with stable COPD had higher levels of Firmicutes and lower levels of Bacteroidetes compared to healthy individuals. COPD exacerbations were accompanied by increased intestinal and pulmonary epithelium permeability, which promoted lung colonization by Enterobacteriaceae. A decreased abundance of the Bacteroidetes genus *Prevotella* was associated with a significantly greater risk of recent severe exacerbation and severe or very severe airflow limitation. COPD patients with predominant emphysema showed increased intestinal endothelial permeability, indicated by higher blood zonulin levels, and reduced commensal bacteria such as *Lactobacilli, Bifidobacteria*, and Bacteroides subspecies. Corticosteroids and antibiotics significantly impact the gut microbial dysbiosis. Supplementation with dietary fibers and gut microbiome-targeted interventions showed some benefits in improving symptoms and slowing the progression of COPD. However, these preliminary findings have not yet been widely used due to the lack of clinical trials.
Conclusions. The gut microbiome is a promising tool for stratifying COPD patients. Future research should clarify the mechanisms and therapeutic effectiveness of restoring the gut microbiome in COPD.

Keywords: **chronic obstructive pulmonary disease, gastrointestinal microbiome, gut microbiome, gut microbiota, precision medicine, personalized medicine.**

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is one of the most common chronic diseases, especially among people over 60 years old, which is characterized by significant heterogeneity of comorbid conditions and the difficulty of predicting the course [1]. According to the World Health Organization, COPD is the third leading cause of death worldwide. Moreover, two-thirds of COPD patients die from non-pulmonary causes, mainly cardiovascular diseases (myocardial infarction, congestive heart failure, stroke) and lung cancer [2]. Aging, reduced physical activity, poor nutrition, smoking, and hypoxia are factors that contribute to the maintenance of oxidative stress, systemic inflammation, and endothelial dysfunction, pathogenetic prerequisites for the coexistence of multimorbidity in COPD. Microbiome changes may play a role in the course of COPD and its comorbidities [3].

Disturbances in the balance between «protective» and «harmful» species of microbial community are associated with the maintenance of systemic inflammation in COPD [4]. The gut microbiome profiling has gained significant attention due to the evolution of sequencing technologies. Investigating the potential of gut microbiome profiling for developing targeted interventions could help reduce systemic inflammation, prevent exacerbations, and improve lung function in patients with COPD. This approach represents a promising avenue for implementing precision medicine in COPD care [5].

AIM

The review aimed to analyze recent data regarding gut microbiome profiling for the personalized management of COPD patients based on an assessment of articles from the MEDLINE database.

MATERIALS AND METHODS

The MEDLINE database via PubMed was searched for articles published in 2015-2025, using the combination of the keywords «(chronic obstructive pulmonary disease OR COPD) AND (Gut Microbiota OR Gut Microbiome OR Gastrointestinal Microbiota OR Gastrointestinal Microbiome OR Intestinal Microbiome OR Intestinal Microbiota)». The search resulted in 173 articles (on March 12, 2025), which were initially screened and assessed based on the titles and content of the abstracts. Of these, 65 were excluded because they did not directly address COPD, and 23 because they did not focus on the gut microbiome in COPD. For detailed assessment, selected 85 articles. Ultimately, the 50 most relevant English-language publications were included in the review.

RESULTS AND DISCUSSION

The human microbiome and COPD

The human microbiome includes a set of microbiota, i.e., microbial communities inhabiting a specific anatomical niche – the gut, oral cavity, respiratory tract, lungs, skin, and vagina, as well as the result of their vital activity – structural elements, metabolites, signaling molecules, and the environmental conditions [6]. Early-life gut microbial colonization is essential in immunomodulatory and metabolic processes [7].

During the first years of life, a child's microbiota composition is dynamic and changes under the influence of various factors [8]. Gradually, the composition of the microbiota stabilizes into a permanent structure with the formation of microbial communities characteristic of a particular person, which are considered healthy microbiota for them. This acquired microbiota is specific and stable for the human throughout adulthood. Therefore, there is no universal healthy microbiota [9].

The gut microbiota is the second-largest genome and the ninth-largest system in the human body, playing a crucial role in maintaining health [10]. With age, there is a decrease in species diversity and evenness in the gut microbiome, and the slower this decrease occurs, the longer a person's lifespan. An increased risk of COPD is accompanied by a decrease in potentially beneficial bacteria and an increase in potentially harmful or pro-inflammatory bacteria [11]. In addition, aging is linked to intestinal barrier dysfunction and hyperpermeability, which indicates a «leaky gut» and contributes to systemic inflammation. This condition is associated with various age-related diseases, including COPD [12].

Thus, a healthy microbiome composition in early life is a protective factor in maintaining immune and metabolic homeostasis. The gut microbiome is not only an indicator of the aging process, but its understanding has the potential to prevent chronic diseases, including COPD [8, 11].

The gut–lung axis

The gut-lung axis is a bidirectional regulatory pathway between the gut and the lungs, which plays an essential role in regulating immune reactions and maintaining pulmonary health [13]. The gut microbiome is the most abundant in the human body. In a healthy person, it contains mainly Firmicutes, Bacteroidetes, Actinobacteria, Proteobacteria, Fusobacteriota, and Verrucomicrobiota microbial phyla. Firmicutes and Bacteroidetes comprise the vast majority, 90% of the gut microbiome [14]. Firmicutes play an important role in metabolic processes, whereas Bacteroides are involved in immunomodulation [15]. Moreover, the ratio between these two phyla is considered a marker of normal intestinal homeostasis, and changes in this ratio are associated with dysbiosis and may contribute to the formation and progression of various pathological conditions, including COPD [16].

The gut microbiota promotes the fermentation of indigestible dietary fibers to short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate, which have protective anti-inflammatory properties, including in the lungs [15, 17]. Unlike healthy individuals, the gut microbiome of COPD patients showed a decrease in Bacteroidetes, an increase in Firmicutes, and a reduction in SCFAs, with the changes becoming more pronounced in cases of severe disease [18, 19]. Fecal transplantation from patients with COPD into mice results in increased lung inflammation, airway remodeling, decreased lung function, and emphysematous changes within a month [19]. A study involving smoking-exposed emphysema mice showed that a high-fiber diet reduced the Firmicutes/Bacteroidetes ratio and enhanced SCFAs metabolism, which reduced airway and systemic inflammation and attenuated emphysema-related pathological changes [20].

Alterations in the gut microbiome, not just the upper and lower respiratory tract microbiome, in COPD underscore the systemic nature of the disease, and their occurrence initiates a vicious cycle of COPD progression [5, 13]. Understanding the bidirectional link between gut dysbiosis and COPD opens its potential as a therapeutic target for treating COPD.

The impact of smoking on the gut microbiome

Research indicates that smokers possess a different composition of gut microbiota compared to nonsmokers [21]. No significant differences in alpha diversity (the species richness, evenness, or diversity within a sample) were observed. Still, notable differences in beta diversity (the dissimilarity in community composition among samples) were found based on smoking status [22, 23]. Compared to never-smokers, current smokers demonstrated a higher relative abundance of the phylum Bacteroidetes while showing a lower relative abundance of the phyla Firmicutes and Proteobacteria. Furthermore, smokers had a reduced Firmicutes/Bacteroidetes ratio [23]. Among never-smokers, the relative abundance of the Firmicutes genus *Lachnospira* was found to be lower, whereas the relative abundance of the Bacteroidetes genus *Prevotella* and the Firmicutes family Veillonellaceae was higher in smokers [22]. Notably, there were no significant differences in gut microbiota composition between never-smokers and former smokers. This suggests that if smokers quit for an extended period, their gut microbiota composition is likely to return to its state before smoking [23].

Alterations in the gut microbiome in stable COPD and across different stages of the disease

Most studies suggested a tendency towards reduced microbial diversity during stable COPD. The composition of the gut microbiome showed no variations between current smokers and non-smokers suffering from COPD, indicating that this is a phenotype related to the disease itself rather than one driven by the effects of cigarette smoke on the gut microbiome [18]. Several studies report that patients with COPD have higher levels of Firmicutes and lower levels of Bacteroidetes compared to healthy individuals, with the changes becoming more pronounced as COPD progresses and lung function declines [16, 24]. An elevated Firmicutes/Bacteroidetes ratio is linked to sustaining inflammation [16].

Research showed that patients with COPD have a lower abundance of multiple members of the Firmicutes family Lachnospiraceae and the Bacteroidetes genus *Prevotella* and a higher abundance of the Firmicutes genus *Streptococcus* (*Streptococcus* species, including *S. parasanguinis_B* and *S. salivarius*) and the Proteobacteria genus *Enterobacter* in the gut microbiota. Furthermore, these microbial imbalances are associated with decreased lung function and may contribute to the progression of COPD [13, 16, 18, 25, 26]. In the early stages of COPD (stages I-II), a higher abundance of the Bacteroides family Prevotellaceae was found compared to healthy individuals. This is likely a protective mechanism, as Prevotellaceae may possess anti-inflammatory properties [19, 26]. With age, there is a depletion of the Actinobacteria genus *Bifidobacterium* and the Firmicutes genus *Lactobacillus*, a common finding in COPD [25].

Another study reveals the gut microbiota of COPD patients and healthy individuals showed a similar dominant population at the phylum level. Although at the genus level, compared to healthy people, the abundance of gut microbiota in patients with stable COPD stage III was changed: the Bacteroidetes genera *Bacteroides* and *Prevotella*, as well as the Firmicutes genera *Faecalibacterium*, *Roseburia*, *Lachnospira*, and *Coprococcus* were lower, while the Bacteroidetes genus Parabacteroides was higher. Furthermore, the relative abundance of *Lachnospira* and *Coprococcus* was negatively correlated to the smoking index and positively correlated with the forced expiratory volume in the first second (FEV_1) and the ratio of the FEV_1 to the forced vital capacity of the lungs (FEV_1/FVC). In contrast, the relative abundance of Parabacteroides was positively related to the smoking index and negatively related to the FEV_1 and FEV_1/FVC [27].

Studies using Mendelian randomization have identified causal relationships between gut microbiome composition and COPD. The Firmicutes genera *Holdemanella* and *Marvinbryantia* are associated with an increased COPD risk. In contrast, the Actinobacteria genus *Collinsella*, the Bacteroidetes genus *Barnesiella*, the Firmicutes genera *Clostridium innocuum* group, Lachnospiraceae UCG004, Lachnospiraceae UCG010, Lachnospiraceae NK4A136 group and family Family XIII were protective factors for COPD [28, 29]. The abundance of the genera *Streptococcus* and *Marvinbryantia* increases under the influence of nicotine [18, 28].

Additionally, COPD has been associated with increased levels of zonulin in the blood, a marker of a «leaky gut». Higher levels of zonulin were found in patients with moderate and severe COPD than those with mild COPD [25, 30]. COPD patients have lower concentrations of SCFAs in the gut microbiome compared to healthy individuals. Moreover, the severity of COPD correlates with reduced levels of SCFAs. The SCFAs possess anti-inflammatory properties, help maintain the intestinal epithelial barrier, balance the gut microbiota, and regulate immunity and inflammation [25]. Among the gut microbiome members known for their role in SCFAs production – particularly butyrate and acetate, which help counteract inflammation through immune regulation – are several genera from the Lachnospiraceae family. These include

Lachnospiraceae UCG004, Lachnospiraceae UCG010, and Lachnospiraceae NK4A136. Representatives of Lachnospiraceae are considered potential prognostic biomarkers for COPD [28].

The gut microbiome in acute exacerbations of COPD

Exacerbations play a critical role in COPD because they are linked to increased mortality, further deterioration of lung function, and a decrease in quality of life. In addition, exacerbations are heterogeneous and may be accompanied by various inflammatory profiles and etiological factors. Dysbiosis is associated with acute exacerbations of COPD (AECOPD), and disease progression may be an indicator of higher mortality risk [5]. The alpha and beta diversities (compared to stable COPD and healthy individuals) of the gut microbiota were lower in AECOPD patients. The relative abundances of Firmicutes and Actinobacteria were decreased, while those of Bacteroidetes and Proteobacteria were increased in AECOPD compared to stable COPD and healthy individuals. The Firmicutes genus *Lachnoclostridium* and the Bacteroidetes genus *Parabacteroides* were predominantly higher in AECOPD [31].

Low serum vitamin D levels, which predispose to AECOPD, may lead to reduced gut microbiota diversity and a high abundance of the Bacteroidetes genera *Bacteroides* and *Prevotella*, as well as the Firmicutes order Clostridiales [32].

Inflammation and immune dysregulation during AECOPD are associated with increased abundance of the Proteobacteria family Enterobacteriaceae. In addition, AECOPD are accompanied by increased intestinal and pulmonary epithelium permeability, which promotes lung colonization by Enterobacteriaceae [25, 33]. A decreased abundance of the Bacteroidetes genus *Prevotella* was associated with a significantly greater risk of recent severe AECOPD and severe or very severe airflow limitation [5, 34].

AECOPD patients exhibit lower concentrations of SCFAs, leading to an increased pro-inflammatory response. This may be due to a reduction in the abundance of SCFA-producing species, which could potentially help predict AECOPD [35].

Thus, the gut microbiome significantly influences immune regulation and systemic inflammation in AECOPD.

Prognostically significant endotypic and phenotypic features in COPD and their relationship with the gut microbiome

COPD patients with predominant emphysema showed increased intestinal endothelial permeability, indicated by higher blood zonulin levels and reduced commensal bacteria such as *Lactobacilli*, *Bifidobacteria*, and *Bacteroides* spp. Additionally, there was an increase in pathogens like Proteobacteria and fungi such as *Saccharomyces* spp., suggesting severe gut dysbiosis [36]. COPD patients with sarcopenia demonstrated elevated levels of blood zonulin and a gut dysbiosis-induced decreased production of SCFAs. In the gut microbiota of these patients, there is an increased abundance of phylum Proteobacteria and pathogenic genera *Escherichia* and *Shigella*. Conversely, there is a decreased abundance of phylum Firmicutes and genus *Faecalibacterium*, Prevotella 9, *Blautia* [37, 38]. A higher Charlson comorbidity index and increased gastrointestinal symptom scores are linked to a greater risk of gut dysbiosis in stable COPD patients. Additionally, a body mass index greater than 23.25 kg/m² and a serum albumin level above 32.5 g/L may serve as protective factors, as they reduce the risk of gut dysbiosis in these patients [39].

High blood eosinophil levels are known to be linked with an increased risk of AECOPD, mortality, decreased FEV_1, and response to corticosteroids. A lower abundance of *Bacteroides* spp. in stable COPD patients was associated with elevated blood eosinophils [40]. Neutrophilic inflammation was linked to the dominance of Proteobacteria, which correlated the activation of pro-inflammatory signaling pathways [41].

There is evidence that SCFAs suppress eosinophilic inflammation and enhance neutrophilic inflammation. The study supports these data about eosinophil-dominant inflammation and reports that lower SCFAs levels and higher mucus plug scores accompanied it. Furthermore, it was found that a lower relative abundance of the Fusobacteriota genus *Fusobacterium* was related to more significant mucus plugging in the airways on chest computed tomography in COPD patients with predominant eosinophilic inflammation [42].

The relationship between comorbidities in COPD and the gut microbiome

Gut dysbiosis in COPD patients is associated with increased systemic inflammation, which contributes to the development of cardiovascular diseases, cancer, and other comorbidities. Direct studies of the impact of the gut microbiome on COPD comorbidities are currently lacking. However, some studies emphasize the role of gut dysbiosis in forming pathogenetic links of comorbidities – systemic inflammation, metabolic disorders, oxidative stress, endothelial dysfunction, etc., and their progression.

The trimethylamine-N-oxide (TMAO) metabolite, which depends on gut microbiota and is influenced by dietary L-carnitine and phosphatidylcholine intake, has been identified as a biomarker that can predict increased cardiovascular risk. Independent association of TMAO with long-term all-cause mortality was found in patients with congestive heart failure, chronic kidney disease, and community-acquired pneumonia. The study in AECOPD patients also reported that increased circulating TMAO levels were linked with long-term all-cause mortality, regardless of the type of exacerbation [43].

The gut microbiota metabolite phenylacetylglutamine was found to be a promising biomarker for COPD. It may aid in identifying individuals at high risk for COPD and provide valuable insights for early prevention and treatment strategies. Previous research has also indicated that phenylacetylglutamine serves as a prognostic marker for heart failure risk and could potentially be a biomarker for lung cancer, suppressing the growth of lung tumors. Furthermore, elevated levels of phenylacetylglutamine may be linked to acute lung injury [44].

The impact of COPD therapy on the gut microbiome

Corticosteroids (both inhaled and systemic) and antibiotics significantly impact microbial dysbiosis, including the gut microbiome. Although most studies have focused on their effects on the lung microbiome, there is evidence of a decrease in microbial diversity and enrichment of Proteobacteria with long-term corticosteroid therapy [5, 15]. Adding probiotics with *Bifidobacterium Lactobacillus* triple live bacteria to COPD treatment using budesonide and ipratropium bromide has been shown to reduce inflammation, inhibit airway remodeling, regulate gut microbiota, and promote lung function recovery [45]. Long-term treatment with macrolides reduced the abundance of the gut microbiota by reducing the number of pathogenic bacteria in COPD patients but did not affect microbial diversity [5, 46]. There is some evidence that azithromycin may reduce the emphysematous changes caused by smoke exposure [47].

Changes in the gut microbiome induced by diet and pulmonary rehabilitation in patients with COPD

Long-term fiber intake reduces the risk of COPD by 30%. The production of SCFAs provides this beneficial effect through the fermentation of dietary fiber. This is facilitated by an increased abundance of SCFA-producing bacteria, consequently reducing the Firmicutes/Bacteroides ratio. Increased dietary fiber intake enhances intestinal barrier function, has anti-inflammatory and immunomodulatory potential, and reduces the progression of COPD and emphysema [25, 29, 48].

A Western diet (high intake of red and processed meat, fried food, refined grains, saturated fats, baked goods, sweets, and low consumption of fruits and vegetables) is associated with an increased risk of developing and progressing COPD. Furthermore, each increase of 50 grams per week in processed red meat intake is linked to an 8% rise in COPD risk. This diet is rich in choline, which, influenced by gut microbiota, leads to elevated levels of circulating TMAO. High levels of TMAO are associated with an increased risk of all-cause mortality [25]. On the other hand, the Mediterranean diet (high consumption of vegetables, fruits, and whole grains) helps maintain lung function [25, 32].

Higher intake of omega-3 polyunsaturated fatty acids is associated with reduced incidence of COPD and contributes to maintaining intestinal immunity and gut microbiota homeostasis [25]. A diet with adequate vitamin D levels is beneficial for patients with COPD. Vitamin D helps regulate intestinal balance by preventing pathogen invasion, reducing inflammation, and maintaining barrier function [25, 32]. Pulmonary rehabilitation helped reduce intestinal permeability (increased zonulin levels) caused by intestinal dysbiosis [36].

Gut microbiome-targeted interventions in COPD

Probiotics may potentially improve symptoms and slow the progression of COPD. However, most studies to date have been conducted in cigarette smoking-induced COPD mouse models. Therefore, these preliminary findings must be validated in large-scale randomized trials before probiotics can be widely used in COPD treatment. Supplementation with *Bifidobacterium longum subsp. longum* attenuated cigarette smoke-induced inflammation and also restored cigarette smoke-induced butyrate depletion [49]. *Lactobacillus rhamnosus* supplementation for 3 months significantly delayed the next moderate-to-severe exacerbation in moderate-to-very severe stable COPD patients. However, the delay in subsequent exacerbation was limited by the duration of probiotic use, so further long-term studies are needed [50]. *Parabacteroides goldsteinii* supplementation also attenuated cigarette smoke-induced inflammation, significantly restoring the smoke-induced body weight loss and improving lung function [48]. Prebiotics are dietary fibers in the form of nutritional supplements that positively influence the gut microbiome's composition. Synbiotics is a combination of probiotics and prebiotics.

Fecal microbiota transplantation (FMT) is a promising therapeutic strategy to regulate COPD symptoms by modulating the gut microbiota. Given the limited research, more animal experiments and clinical trials are needed to investigate its therapeutic effect. Current evidence suggests that FMT from healthy mice to cigarette smoking-induced COPD mice increased the abundance of Bacteroidetes and Lachnospiraceae. These bacteria can catabolize dietary fiber into SCFAs, which help suppress local and systemic inflammation, thus reducing the development of emphysema [25, 48].

CONCLUSIONS

This review demonstrates that the gut microbiome is a promising tool for stratifying COPD patients. The gut microbiome of these patients differs significantly depending on various factors: stable disease or exacerbation, severity of airway obstruction, phenotypic and endotypic features, comorbidities, medications, patient age, dietary habits, lifestyle, etc. In COPD, gut microbiome profiling is a significant step towards personalized management and developing targeted interventions aimed at restoring a healthy gut microbiome and attenuate COPD progression.

Perspectives for further research. Future research should clarify the mechanisms and therapeutic effectiveness of restoring the gut microbiome in COPD patients. Large-scale clinical studies utilizing a multi-omic approach are necessary to identify the bacteria and metabolites that play a key role in COPD pathogenesis. This comprehensive strategy will be a strong foundation for developing gut microbiome-targeted interventions in COPD.

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COMPLIANCE WITH ETHICAL REQUIREMENTS

This review article is based on an analysis of publicly available scientific data published in peer-reviewed journals, clinical guidelines, and databases. No patient-identifying data were used during the work, and no approval from an ethics committee was required, as the study did not involve new clinical interventions or primary collection of patient information.

The authors adhered to the ethical principles of the World Medical Association Declaration of Helsinki and international standards for publication in medical journals, including the recommendations of the ICMJE (International Committee of Medical Journal Editors).

No element of the work contains plagiarism or fabrication of data. All sources of information are appropriately cited and properly formatted.

AUTHOR CONTRIBUTIONS

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Резюме

ПОТЕНЦІАЛ ПРОФІЛЮВАННЯ МІКРОБІОМУ КИШЕЧНИКА ДЛЯ ПЕРСОНАЛІЗОВАНОГО ВЕДЕННЯ ПАЦІЄНТІВ З ХРОНІЧНИМ ОБСТРУКТИВНИМ ЗАХВОРЮВАННЯМ ЛЕГЕНЬ: ОГЛЯД

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Вступ. Хронічне обструктивне захворювання легень (ХОЗЛ) залишається серйозною глобальною проблемою охорони здоров'я через незадоволену потребу в ранньому прогнозуванні перебігу захворювання та результатах терапії. Вивчення потенціалу профілювання кишкового мікробіому при ХОЗЛ допоможе зробити значний крок на шляху впровадження прецизійної медицини.
Мета. Проаналізувати останні дані щодо профілювання кишкового мікробіому для персоналізованого лікування пацієнтів з ХОЗЛ на основі оцінки статей із бази даних MEDLINE.
Матеріали та методи. Ми здійснили пошук статей у MEDLINE, опублікованих у 2015-2025 роках, за допомогою комбінації заздалегідь визначених ключових слів. У результаті було знайдено 173 статті, які були оцінені за назвами та змістом тез. Після оцінки релевантності для детального аналізу ми відібрали 85 статей. До огляду увійшло 50 статей.
Результати. Профілі кишкового мікробіому різних контингентів пацієнтів з ХОЗЛ суттєво відрізнялися. Кілька досліджень повідомляють про зменшення мікробної різноманітності при ХОЗЛ. Пацієнти зі стабільним ХОЗЛ мали вищі рівні Firmicutes і нижчі рівні Bacteroidetes порівняно зі здоровими особами. Загострення ХОЗЛ супроводжувалися підвищенням проникності кишкового та легеневого епітелію, що сприяло колонізації легень Enterobacteriaceae. Зменшення чисельності роду *Prevotella* було пов'язане зі значно більшим ризиком нещодавнього тяжкого загострення й тяжкого або дуже тяжкого обмеження повітряного потоку. Пацієнти з ХОЗЛ та емфіземою мали вищі рівні зонуліну в крові, маркера проникності кишкового ендотелію, та зниження кількості *Lactobacilli, Bifidobacteria* та підвидів Bacteroides. Кортикостероїди та антибіотики істотно впливали на дисбактеріоз кишечника. Споживання харчових волокон та мікробіом-орієнтована терапія показали певні переваги в покращенні симптомів й уповільненні прогресування ХОЗЛ. Однак ці попередні висновки ще не отримали широкого застосування через недостатню кількість клінічних випробувань.
Висновки. Мікробіом кишечника є перспективним інструментом для стратифікації пацієнтів з ХОЗЛ. Майбутні дослідження мають прояснити механізми та терапевтичну ефективність відновлення мікробіому кишечника при ХОЗЛ.

Ключові слова: **хронічне обструктивне захворювання легень, мікробіом шлунково-кишкового тракту, мікробіом кишечника, мікробіота кишечника, прецизійна медицина, персоналізована медицина**

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