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THE ROLE OF GUT MICROBIOTA DYSBIOSIS IN GASTROINTESTINAL DISEASES

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Introduction. The gastrointestinal (GI) microbiota comprises trillions of microorganisms, including bacteria, viruses, fungi, and archaea, that inhabit the human gastrointestinal tract and play essential roles in digestion, immune system development, and metabolic regulation [1, 2]. Estimates suggest that the number of bacterial cells in the human body is approximately comparable to the number of human cells [1]. In healthy individuals, the gut microbiota is commonly dominated by members of the phyla Firmicutes and Bacteroidetes, while Actinobacteria, Proteobacteria, and Verrucomicrobia are generally present in lower proportions [7]. Alterations in the composition, diversity, and functional activity of the gut microbiota, collectively referred to as dysbiosis, have been associated with the development and progression of numerous disorders, including inflammatory bowel disease (IBD), irritable bowel syndrome (IBS), Clostridioides difficile infection, and other gastrointestinal pathologies [2, 3]. Consequently, investigating microbiota-related disturbances represents an important area of contemporary clinical gastroenterology.

Keywords: gastrointestinal tract, gut microbiota, dysbiosis, probiotics, inflammatory bowel disease, faecal microbiota transplantation.

Aim of the study. To analyse the role of gut microbiota dysbiosis in gastrointestinal diseases based on contemporary scientific evidence and to evaluate effective strategies for its management.

Main body. The intestinal microbiota performs several essential physiological functions. First, it ferments indigestible dietary fibres, producing short-chain fatty acids (SCFAs), including butyrate, propionate, and acetate. Butyrate serves as a major energy source for colonocytes, supports intestinal epithelial barrier integrity, and exerts anti-inflammatory effects. Second, the microbiota contributes to colonisation resistance by competing with pathogenic microorganisms for nutrients and adhesion sites. Third, it participates in the development and regulation of mucosal immunity and contributes to the synthesis of certain vitamins, including vitamin K and some B group vitamins [2, 7].

Dysbiosis refers to alterations in the composition, diversity, or functional activity of the gut microbiota. Potential contributing factors include inappropriate antibiotic use, an unbalanced diet rich in fats and simple carbohydrates but low in dietary fibre, intestinal infections, chronic stress, prolonged use of proton pump inhibitors, and age-related changes. Dysbiosis may involve a reduction in beneficial microorganisms, particularly butyrate-producing species such as Faecalibacterium prausnitzii, alongside an increase in potentially pathogenic members of the phylum Proteobacteria. These changes may compromise intestinal barrier integrity, increase epithelial permeability, and facilitate the translocation of microbial antigens, thereby contributing to persistent mucosal immune activation and chronic inflammation [2, 3].

Patients with inflammatory bowel diseases, particularly ulcerative colitis and Crohn's disease, frequently exhibit reduced gut microbial diversity and alterations in microbial composition. These changes may include a reduction in certain members of the phylum Firmicutes and an increase in bacteria belonging to the family Enterobacteriaceae. Although the extent to which these microbial

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alterations represent a cause or a consequence of disease remains incompletely understood, substantial evidence suggests that interactions between the microbiota and the intestinal immune system may contribute to the initiation and maintenance of inflammation [3].

Alterations in gut microbial composition have also been associated with irritable bowel syndrome, a disorder characterised by abdominal pain and altered bowel habits. These alterations may interact with disturbances in intestinal motility, visceral sensitivity, and gut-brain signalling. Post-infectious IBS, which develops following an episode of acute gastroenteritis, and small intestinal bacterial overgrowth (SIBO) further illustrate the potential clinical relevance of microbiota-related disturbances. Common symptoms include abdominal pain, bloating, diarrhoea, and constipation [4].

Clostridioides difficile infection represents a prominent example of a microbiota-associated disorder that may develop following antibiotic-induced disruption of the intestinal microbial ecosystem. Antibiotic exposure can impair colonisation resistance, allowing C. difficile to proliferate and produce toxins that cause intestinal inflammation. In patients with recurrent infection, conventional antibiotic treatment may not provide sustained resolution. Clinical trials have demonstrated that faecal microbiota transplantation (FMT), including delivery via the duodenal route, can achieve higher rates of clinical resolution than standard vancomycin therapy in selected patient populations [5].

Several strategies are used to manage microbiota-related disturbances. These include probiotics, defined as live microorganisms that confer a health benefit when administered in adequate amounts; prebiotics, which are substrates selectively utilised by host microorganisms to confer a health benefit; and synbiotics, which combine probiotics and prebiotics. Other approaches include selective antimicrobial treatment when clinically indicated and faecal microbiota transplantation for appropriately selected patients [5, 6]. In addition, a fibre-rich diet incorporating fermented foods, judicious antibiotic prescribing, regular physical activity, and stress management may support a healthy intestinal microbial ecosystem. It is important to recognise that the clinical efficacy of probiotics is strain-specific; therefore, their selection should be guided by evidence from studies evaluating the particular strain, formulation, and clinical indication [6].

Advances in microbiome analysis, including 16S ribosomal RNA gene sequencing and metagenomic sequencing, offer promising opportunities to investigate disease-associated microbial patterns, identify potential biomarkers, and develop personalised therapeutic approaches. These technologies may facilitate the development of microbiota-targeted strategies for the prevention and treatment of gastrointestinal disorders. However, their routine clinical application requires further validation regarding diagnostic accuracy, clinical utility, and reproducibility [2, 3].

The consequences of gut microbiota disturbances may extend beyond the gastrointestinal tract. Through the gut–brain axis, the intestinal microbiota interacts with the nervous, immune, and endocrine systems and may influence gastrointestinal motility, visceral sensitivity, and pain perception. Furthermore, alterations in gut microbial composition and function have been associated with metabolic disorders, including obesity, diabetes mellitus, and metabolic dysfunction-associated steatotic liver disease. These associations highlight the potential importance of the gut microbiota in systemic health, although the underlying causal relationships remain an active area of research [2].

The assessment of suspected dysbiosis should begin with a comprehensive clinical history, including gastrointestinal symptoms, previous antibiotic exposure, dietary habits, and a history of intestinal infections. Depending on the clinical presentation, investigations may include stool microbiological testing, faecal inflammatory markers, hydrogen breath testing, and molecular techniques such as 16S rRNA gene sequencing. Hydrogen breath testing may be useful when SIBO or carbohydrate malabsorption is suspected, while stool testing for specific pathogens is indicated in selected clinical circumstances. Because the composition of a healthy microbiota varies considerably between individuals, microbiome test results should be interpreted in conjunction with the clinical

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presentation and other relevant investigations. At present, there is no universally accepted microbiome profile that independently establishes a diagnosis of generalised intestinal dysbiosis [7].

Conclusion. The gut microbiota is a key component of gastrointestinal health, contributing to digestive processes, intestinal barrier integrity, immune regulation, and metabolic homeostasis. Dysbiosis has been associated with the development and clinical course of several gastrointestinal disorders, including inflammatory bowel disease, irritable bowel syndrome, and Clostridioides difficile infection. Early recognition of clinically relevant microbiota-related disturbances, a balanced diet, rational antibiotic use, and the appropriate application of evidence-based interventions—including selected probiotics, prebiotics, and faecal microbiota transplantation—may improve patient outcomes in specific clinical settings. Further research is required to clarify causal relationships, establish reliable diagnostic criteria, and optimise personalised microbiota-targeted therapies.

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