

IMMUNOREGULATORY ROLE OF INTESTINAL MICROBIOTA IN CHILDREN WITH CHRONIC GASTROINTESTINAL DISEASES

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Annotation: *This research provides comprehensive analysis of the immunoregulatory functions of intestinal microbiota in chronic gastrointestinal diseases. The study presents scientifically grounded explanations of microbiome compositional changes, dysbiosis mechanisms, the microbiota-gut-brain axis, short-chain fatty acid metabolism, effects on immune homeostasis, and probiotic therapy strategies. The findings serve to develop microbiome-based intervention methods in pediatric gastroenterology.*

Keywords: *intestinal microbiota, dysbiosis, short-chain fatty acids, microbiota-gut-brain axis, regulatory T-lymphocytes, toll-like receptors, butyrate, mucosal immunity, probiotics, prebiotics.*

The intestinal microbiota plays a fundamental role in maintaining physiological functions of the digestive system and preserving immune homeostasis. The human intestinal microbiota comprises one hundred trillion microorganisms representing over one thousand species. The prevalence of chronic gastrointestinal diseases in children, including inflammatory bowel diseases, irritable bowel syndrome, chronic gastritis, and gastroduodenitis, has increased by forty to forty-five percent over the past twenty years. In the Republic of Uzbekistan, gastrointestinal diseases constitute eighteen to twenty-two percent of pediatric disease burden. Understanding the complex interplay

between microbiota dysbiosis and immune inflammation, short-chain fatty acid production, mucosal barrier function, and systemic immunity represents a pressing challenge in contemporary pediatric gastroenterology.

In healthy children, the intestinal microbiota composition consists of ninety to ninety-five percent Firmicutes and Bacteroidetes phyla. The Firmicutes to Bacteroidetes ratio ranges from 1.5 to 2.0. In chronic gastrointestinal diseases, this ratio becomes disrupted and dysbiosis develops. In inflammatory bowel diseases, the Firmicutes phylum decreases by thirty to thirty-five percent while Proteobacteria increases two to threefold. Beneficial microorganisms including *Faecalibacterium prausnitzii* and *Akkermansia muciniphila* show concentration reductions of fifty to sixty percent. Dysbiosis severity is assessed using the Dysbiosis Index, which exceeds five points in severe cases.

Short-chain fatty acids, including butyrate, propionate, and acetate, constitute the primary metabolites of intestinal microbiota and possess immunoregulatory functions. Butyrate concentration measures twenty to thirty millimoles per kilogram of feces in healthy children but decreases to eight to twelve millimoles per kilogram in chronic inflammatory conditions. Butyrate serves as the principal energy source for colonocytes, fulfilling seventy to eighty percent of their energy requirements. At the molecular level, butyrate inhibits histone deacetylase enzymes, increases histone acetylation, and stimulates regulatory T-lymphocyte differentiation. Regulatory T-lymphocytes comprise eight to ten percent of lymphocytes in healthy children, whereas this indicator decreases to four to five percent under dysbiotic conditions. The microbiota-gut-brain axis constitutes a bidirectional communication system that establishes connections between the central nervous system and intestinal microbiota through the vagus nerve, immune mediators, and neurometabolites.

The microbiota regulates tryptophan metabolism and participates in producing 5-hydroxytryptophan, the serotonin precursor. Ninety-five percent of serotonin is synthesized in the intestine through microbiota-dependent processes. Under dysbiotic conditions, serotonin synthesis decreases by thirty to forty percent, affecting neuropeptide Y and corticotropin-releasing hormone levels, thereby intensifying stress responses and inflammation.

The mucosal immune system maintains constant interaction with intestinal microbiota. Toll-like receptors recognize microbial pathogen-associated molecular patterns and initiate immune responses. Toll-like receptor 4 recognizes lipopolysaccharide while toll-like receptor 2 identifies peptidoglycan. Under dysbiotic conditions, gram-negative bacterial lipopolysaccharide levels increase two to threefold, activating NF-kappa B signaling through toll-like receptor 4. Consequently, pro-inflammatory cytokine concentrations, including interleukin-1 beta, interleukin-6, and tumor necrosis factor alpha, increase four to fivefold. Clinical trials demonstrate that *Lactobacillus rhamnosus* GG administered at ten to the tenth power colony forming units for eight to twelve weeks increases remission frequency in inflammatory bowel diseases by thirty-five to forty percent. *Bifidobacterium lactis* BB-12 increases butyrate-producing bacterial populations by sixty to seventy percent and stimulates mucosal regeneration.

Intestinal microbiota plays a central role in maintaining immune homeostasis in chronic gastrointestinal diseases. Dysbiosis, short-chain fatty acid deficiency, and disruption of the microbiota-gut-brain axis collectively activate inflammatory processes. Application of microbiome-based therapy including probiotics and prebiotics represents a pathogenetically grounded approach.

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