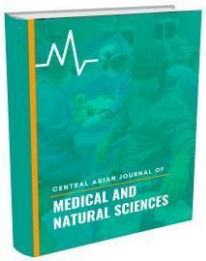

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Article

Gut Microbiota in Children and Adolescents with Obesity

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Abstract: This article analyzes the structural and functional changes of the gut microbiota in children and adolescents with obesity, their impact on metabolic status, and their role in the pathogenesis of obesity. The results of the study show that intestinal dysbiosis, particularly changes in the Firmicutes/Bacteroidetes ratio, a decrease in beneficial bacteria such as Bifidobacterium and Lactobacillus, and an increase in pro-inflammatory microorganisms contribute to the development of obesity, insulin resistance, dyslipidemia, and metabolic syndrome. The article highlights, based on scientific sources, the role of the gut microbiota in energy metabolism, the production of short-chain fatty acids, the maintenance of intestinal barrier integrity, the regulation of immune response, and chronic inflammatory processes. According to the World Health Organization, in 2022, 160 million children and adolescents aged 5–19 years worldwide were affected by obesity, while 390 million were overweight. In Uzbekistan, in 2022, 13,743 cases of obesity were registered among children and adolescents aged 0–17 years, which represents a 23.8% increase compared to 2018. In addition, the article discusses probiotics, prebiotics, synbiotics, diet therapy, and innovative methods of gut microbiota modulation as promising approaches in the комплекс treatment of obesity. According to the study's conclusions, early monitoring and targeted correction of the gut microbiota are of great importance in preventing obesity and its complications in children and adolescents.

Keywords: gut microbiota, obesity, children, adolescents, metabolic syndrome, Firmicutes, Bacteroidetes, short-chain fatty acids, probiotics, dysbiosis.

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1. Introduction

Obesity, defined by the World Health Organization (WHO) as abnormal or excessive fat accumulation that presents a risk to health, is a global issue with potentially serious consequences. Over the past few decades, there has been a rapid increase in the number of children and adolescents who are overweight or obese. According to WHO data, the number of children and adolescents aged 5 to 19 suffering from obesity increased from 11 million in 1975 to 160 million in 2022, while 390 million are overweight. More than

340 million children and adolescents worldwide are overweight, which significantly increases the likelihood of metabolic disorders in adulthood. According to the World Obesity Federation World Obesity Atlas 2023, childhood obesity is projected to double in the coming years, increasing by 100% among boys (up to 208 million) and by 125% among girls (up to 175 million) compared to the 2020 level[1].

The dynamics of these indicators demonstrate a significant increase in the prevalence of this pathology. The prevalence of overweight (including obesity) among children and adolescents aged 5–19 has risen sharply: in 1990, it was only 8%, while by 2022 it had reached 20%. Thus, over three decades, the rate has increased 2.5 times, allowing the situation to be characterized as an epidemic[2].

In the WHO European Region, the problem of childhood obesity remains persistent. According to data from the sixth round of the WHO European Childhood Obesity Surveillance Initiative (COSI), which covered 37 countries and approximately 470,000 children, every fourth child (25%) aged 7–9 years is overweight, and every tenth child (11%) suffers from obesity. Gender differences were also identified: boys are more likely to suffer from obesity (13%) than girls (9%)[3].

In Uzbekistan, in 2022, 13,743 cases of obesity were registered among children and adolescents aged 0–17 years. Compared to 2018, the number of cases increased by 23.8%. The prevalence rate of obesity in this age group was 434.5 per 100,000 children. During the period of 2018–2022, an increase in prevalence by 22.7% was also observed. The highest level was recorded in Tashkent with 1,766 cases, while the lowest was in Jizzakh Region with only 10 cases[4].

2. Materials and Methods

Obesity in children and adolescents is a complex multifactorial condition influenced by genetic, epigenetic, metabolic, and environmental factors. Although in the vast majority of cases childhood obesity is exogenous in nature, in recent years particular attention has been paid to the role of the gut microbiota in the pathogenesis of this disease. Experimental and clinical evidence convincingly demonstrates that changes in the composition and functional activity of the intestinal microbial community can be both a consequence and a cause of the development of overweight and obesity.

The gut microbiota is a collection of microorganisms inhabiting the human gastrointestinal tract. In the large intestine, the number of bacteria reaches peak values of 10^{10}–10^{13} colony-forming units (CFU) per milliliter, which accounts for approximately 5–8% of body mass. Anaerobic bacteria predominate over aerobic bacteria at a ratio of 1000:1. Despite the enormous diversity of microorganisms, five dominant phyla are distinguished: Firmicutes, Bacteroidetes, Actinobacteria, Proteobacteria, and Verrucomicrobia. In a healthy individual, up to 90% of the gut microbiota consists of bacteria belonging to two phyla—Firmicutes and Bacteroidetes.

Colonization of the intestine by bacteria begins immediately after birth, during the passage of the fetus through the mother's birth canal. The composition of the microbiota is influenced by many factors, including the type of feeding (breastfeeding or formula feeding), mode of delivery (vaginal birth or cesarean section), diet, sex, geographical location, ethnicity, and the use of antibiotics. The formation of a relatively stable gut microbial community occurs by the age of 2–3 years.

3. Results and Discussion

The gut microbiota performs numerous functions: it participates in the digestion of dietary fiber with the production of short-chain fatty acids (SCFAs), synthesizes B-group vitamins and vitamin K, maintains the integrity of the intestinal barrier, regulates the immune response, and modulates energy metabolism. It has been proven that the microbiota plays an important role in maintaining metabolic balance and the functioning

of the immune system, and also interacts with the central nervous system through the so-called “gut–brain axis”[5].

Numerous studies have demonstrated that obesity is associated with significant qualitative and quantitative changes in the composition of the gut microbiota. A key observation, confirmed both in experimental animal models and in clinical studies, is the alteration in the ratio of the two main bacterial phyla—Firmicutes and Bacteroidetes. In individuals with obesity, including children and adolescents, there is generally an increase in the relative abundance of Firmicutes bacteria along with a simultaneous decrease in the representation of Bacteroidetes, which leads to an increased Firmicutes/Bacteroidetes (F/B) ratio.

However, the data from various studies are contradictory: some authors point to an increased abundance of Firmicutes in obesity, whereas others, on the contrary, note an increase in Bacteroidetes and a decrease in Firmicutes. Such discrepancies may be due to differences in research methodologies, the characteristics of the studied populations, dietary patterns, and geographical factors.

The study by T. S. Dushina et al. demonstrated that young people with obesity have characteristic features in the relationship between gut microbiota parameters and clinical as well as biochemical indicators. It was found that the degree of intestinal dysbiosis correlates with the severity of metabolic disorders, including insulin resistance and dyslipidemia[6].

When analyzing the results of the use of Colonoflor-16 Premium, a discrepancy was identified between the data obtained from the control group and the reference values of the analysis. In the group of patients with obesity, there was a clear tendency toward a decrease in the levels of Lactobacillus spp. and Bifidobacterium spp.

In addition, compared with the control group (10.3%), the group of patients with obesity showed a significantly higher prevalence of Fusobacterium nucleatum (37.6%) ($p = 0.005$), while the number of Faecalibacterium prausnitzii bacteria significantly decreased ($p = 0.030$) and the number of Prevotella spp. significantly increased ($p = 0.029$).

In young patients with obesity, numerous associations were identified between representatives of the colonic microbiota and the most important anamnestic, anthropometric, and biochemical indicators[7]. In addition to changes in the ratio of the main bacterial phyla, obesity in children is associated with a reduction in overall microbial diversity, which is considered an important indicator of health status. A decrease in gut microbiota diversity is associated with increased intestinal barrier permeability and the development of chronic low-grade inflammation. Children with obesity also show a reduced abundance of representatives of the genus Bifidobacterium and certain butyrate-producing bacteria, which negatively affects the barrier function of the intestine and metabolic homeostasis[8]. The mechanisms through which the gut microbiota participates in the pathogenesis of obesity are diverse and operate at the metabolic, regulatory, and immunological levels.

Intestinal bacteria ferment indigestible carbohydrates (dietary fiber) to produce short-chain fatty acids (SCFAs)—acetate, propionate, and butyrate. SCFAs serve as an additional source of energy for the host organism and provide up to 10% of the daily caloric intake. When the composition of the microbiota shifts toward an increased abundance of bacteria that efficiently ferment polysaccharides, the extraction of energy from food increases, which contributes to the excessive accumulation of adipose tissue[9].

The gut microbiota influences energy balance not only by extracting additional calories from food, but also by modulating the expression of genes involved in energy expenditure and storage. Short-chain fatty acids (SCFAs), produced through microbial fermentation of dietary fiber, activate free fatty acid receptors FFAR2 and FFAR3, which stimulate the secretion of satiety hormones such as peptide YY (PYY) and glucagon-like peptide-1 (GLP-1). These hormones play a crucial role in appetite regulation, glucose homeostasis, and overall metabolic control[10].

Chronic low-grade inflammation is a hallmark of obesity and its associated metabolic disorders. A key contributor to this inflammatory state is bacterial lipopolysaccharide (LPS), a structural component of the outer membrane of Gram-negative bacteria. In conditions of gut dysbiosis, intestinal barrier integrity is often compromised, facilitating the translocation of LPS into the systemic circulation—a phenomenon known as metabolic endotoxemia. LPS activates pattern-recognition receptors, particularly Toll-like receptor 4 (TLR4), which triggers inflammatory signaling pathways through nuclear factor kappa B (NF-κB). This leads to increased production of pro-inflammatory cytokines, exacerbation of systemic inflammation, and the development of insulin resistance[11].

Experimental studies in animal models have demonstrated that obese mice exhibit elevated levels of lipopolysaccharides (LPS) in the bloodstream, which correlate with increased dietary fat intake. Similar findings have been reported in clinical studies: children with obesity show higher levels of systemic inflammatory markers, including C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), which are associated with the severity of gut dysbiosis.

The intestinal microbiota plays a crucial role in the metabolism of bile acids and cholesterol. Gut bacteria are involved in the deconjugation and biotransformation of primary bile acids into secondary bile acids, thereby influencing lipid metabolism, tissue insulin sensitivity, and overall energy homeostasis. Disruption of microbial bile acid metabolism under dysbiotic conditions contributes to the development of dyslipidemia and exacerbates metabolic disturbances in children with obesity.

A healthy gut microbiota maintains the integrity of the intestinal mucosal barrier by stimulating mucin production, enhancing the expression of tight junction proteins such as occludin, claudins, and zonulin, and producing butyrate, which serves as the primary energy substrate for colonocytes. In obesity, reduced butyrate production and disruption of intestinal barrier integrity are commonly observed, creating conditions for increased intestinal permeability and the development of endotoxemia[12].

Age-Related Characteristics of the Gut Microbiota in Obesity

The development of the gut microbiota during childhood is a dynamic process that significantly influences metabolic health in later stages of life. M. Yu. Shcherbakova and colleagues have extensively examined the role of the gut microbiota in the age-related development of obesity, emphasizing that early disturbances in the intestinal microbial community may serve as predictors of metabolic disorders in older age.

Epidemiological studies have demonstrated that the use of antibiotics during the first six months of life may increase the risk of excessive weight gain later in life. Although this factor does not directly affect caloric intake or the host's metabolism, it significantly alters the composition of the gut microbiome. Therefore, early-life influences on the gut microbiota—such as feeding patterns, antibiotic therapy, and dietary characteristics—may have long-term consequences for a child's metabolic health.

In breastfed infants, bifidobacteria predominate and provide protection against the development of obesity. The transition to formula feeding and the introduction of complementary foods are accompanied by diversification of the microbial community and an increased abundance of Firmicutes. During adolescence, the gut microbiota gradually becomes more similar in composition to that of adults; however, in the presence of obesity, more pronounced deviations from the normal microbial profile are observed, which are associated with the progression of metabolic disturbances[13].

Relationship Between Gut Microbiota and Metabolic Status in Childhood Obesity

In a systematic review, X. Yuan and colleagues summarized evidence on the role of the gut microbiota in shaping the metabolic status of children with obesity. The authors emphasized that gut dysbiosis in obese children is associated with the development of insulin resistance, impaired lipid metabolism, elevated levels of pro-inflammatory markers, and an increased risk of metabolic syndrome.

T. S. Dushina and colleagues, in a study conducted at Tyumen State Medical University, identified correlations between specific gut microbiota parameters and clinical and biochemical indicators in young individuals with obesity. The study demonstrated a relationship between the severity of dysbiosis and body mass index (BMI), insulin levels, the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), blood lipid profile parameters, and markers of systemic inflammation.

Obesity that begins in childhood leads to both short-term and long-term adverse consequences for physical and psychosocial health. Children who are overweight have a fivefold higher risk of developing obesity in adulthood compared to children with normal body weight. Overweight and obesity are significant risk factors for the development of metabolic syndrome, Type 2 Diabetes, cardiovascular diseases, and Non-Alcoholic Fatty Liver Disease[14].

Understanding the role of the gut microbiota in the pathogenesis of obesity opens new perspectives for the development of therapeutic strategies aimed at restoring the normal intestinal microbial community. T. V. Tazina and L. V. Evsyukova, in their review, discussed innovative approaches to obesity treatment, including the use of symbiotic preparations for modulating the gut microbiota.

Correction of the gut microbiota can be achieved through several approaches. Probiotics—live microorganisms that exert beneficial effects on the composition and functional activity of the microbiota—have demonstrated the ability to improve metabolic status, reduce systemic inflammation, and decrease insulin resistance. Prebiotics, which are non-digestible food ingredients that stimulate the growth of beneficial bacteria, and symbiotics, a combination of probiotics and prebiotics, have shown a synergistic effect in normalizing the composition of the intestinal microbial community.

The results of both experimental and clinical studies confirm that the комплексное use of symbiotic preparations in combination with lifestyle modifications—such as dietary therapy and increased physical activity—represents a promising strategy for effective obesity management. In addition, the potential use of Ursodeoxycholic Acid preparations for modulating bile acid metabolism and Fecal Microbiota Transplantation as a possible treatment for severe forms of dysbiosis is also being discussed[15].

Diet therapy remains the cornerstone of obesity treatment in children and adolescents, and the composition of the diet has a direct impact on the gut microbiota. A diet rich in dietary fiber, fruits, and vegetables promotes increased microbial diversity and the growth of beneficial bacteria, whereas a diet high in fats and simple carbohydrates leads to depletion of the microbial community and an increased proportion of pro-inflammatory microorganisms.

4. Conclusion

Accumulated evidence convincingly demonstrates a close relationship between the state of the gut microbiota and the development of obesity in children and adolescents. Gut dysbiosis, manifested by an imbalance in the major bacterial phyla, reduced microbial diversity, and altered functional activity of the microbial community, is associated with metabolic disturbances, including insulin resistance, dyslipidemia, and chronic inflammation. The mechanisms through which the gut microbiota contributes to the development of obesity include increased efficiency of energy extraction from food, regulation of appetite and satiety, modulation of intestinal barrier permeability, induction of chronic inflammation through bacterial endotoxemia, and effects on bile acid metabolism.

Particular importance should be given to the fact that the formation of the gut microbial community occurs during the early periods of life, and disturbances arising at this stage may have long-term consequences for metabolic health. This creates opportunities for the early prevention of obesity and its complications through targeted modulation of the gut microbiota. Therapeutic approaches aimed at normalizing the

composition of the gut microbiota—such as the use of probiotics, prebiotics, and symbiotics, as well as dietary therapy—represent a promising direction in the comprehensive treatment of obesity in children and adolescents. However, further large-scale clinical studies involving pediatric cohorts are needed to develop personalized strategies for correcting gut microbiocenosis.

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