5. Friedman KG, Tworetzky W. Fetal cardiac interventions: Where do we stand? Arch Cardiovasc Dis. 2020 Feb;113(2):121-128. doi: 10.1016/j.acvd.2019.06.007.
6. Gobergs R, Salputra E, Lubaua I. Hypoplastic left heart syndrome: a review. Acta Med Litu. 2016;23(2):86-98. doi: 10.6001/actamedica.v23i2.3325.
7. Gulgun M, Slack M. Stent Placement in a Neonate with Sano Modification of the Norwood using Semi-Elective Extracorporeal Membrane Oxygenation. Arq Bras Cardiol. 2016 Dec;107(6):600-604. doi: 10.5935/abc.20160080.
8. Ito Y, Yamashita T, Tachibana K. Anesthetic management using high-flow nasal cannula therapy during cardiac catheter examination of a neonate with hypoplastic left heart syndrome. JA Clin Rep. 2022 Oct 12;8(1):83. doi: 10.1186/s40981-022-00572-x.
9. Mayr B, Kido T, Holder S, Wallner M, Vodiskar J, Strbad M, et al. Single-centre outcome of extracorporeal membrane oxygenation after the neonatal Norwood procedure. Eur J Cardiothorac Surg. 2022 Aug 3;62(3):ezac129. doi: 10.1093/ejcts/ezac129.
10. Mohanty SR, Patel A, Kundan S, Radhakrishnan HB, Rao SG. Hypoplastic left heart syndrome: current modalities of treatment and outcomes. Indian J Thorac Cardiovasc Surg. 2021 Jan;37(Suppl 1):26-35. doi: 10.1007/s12055-019-00919-7.
11. Ohye RG, Schranz D, D'Udekem Y. Current Therapy for Hypoplastic Left Heart Syndrome and Related Single Ventricle Lesions. Circulation. 2016 Oct 25;134(17):1265-1279. doi: 10.1161/CIRCULATIONAHA.116.022816.
12. Wilder TJ, Caldarone CA. Apples to oranges: Making sense of hybrid palliation for hypoplastic left heart syndrome. JTCVS Open. 2020 Oct 15;4:47-54. doi: 10.1016/j.xjon.2020.10.002.
13. Yabrodi M, Mastropietro CW. Hypoplastic left heart syndrome: from comfort care to long-term survival. Pediatr Res. 2017 Jan;81(1-2):142-149. doi: 10.1038/pr.2016.194.
14. Yerebakan C, Valeske K, Elmontaser H, Yörüker U, Mueller M, Thul J, et al. Hybrid therapy for hypoplastic left heart syndrome: Myth, alternative, or standard? J Thorac Cardiovasc Surg. 2016 Apr;151(4):1112-21, 1123.e1-5. doi: 10.1016/j.jtcvs.2015.10.066.

Стаття надійшла 24.06.2024 р.

DOI 10.26724/2079-8334-2025-3-93-83-87
UDC 616.34-002-07:577.112.385.4:579.864

A.A. Kerimova, G.M. Gasangulieva, U.A. Aliyeva, A.B. Huseynova, B.A. Namazova
Azerbaijan Medical University, Baku, Azerbaijan

CHANGES OF MICROBIOTA-DERIVED SHORT-CHAIN FATTY ACID LEVELS IN CHILDREN WITH VIRAL DIARRHEA

e-mail: mic_amu@mail.ru

The purpose of the study was to assess the clinical and diagnostic significance of changes in short-chain fatty acids levels in children of the first year of life with viral diarrhea. The study was conducted in 94 children with diarrhea aged 0–1 year. The control group consisted of 30 healthy children. Determination of short-chain fatty acids levels was conducted, using gas-liquid chromatography. According to results, in the mono variant of viral diarrhea, acetic acid decreased by 1.5 times compared to the, in mixed variants – by 1.8 times ($p<0.001$); propionic acid in the mono variant decreased by 1.5 times, in the mixed variant it decreased by 2 times ($p<0.001$); butyric acid in the mono variant decrease of 2.5 times, in the mixed variant it was 3.6 times lower ($p<0.001$); isovaleric acid in the mono variant decreased by 1.2 times ($p<0.05$), while in the mixed course increased (14.1 %).

Key words: short-chain fatty acids, butyric acid, valerianic acid, microbiota, gastroenteritis.

А.А. Керімова, Г.М. Гасангулієва, У.А. Алієва, А.Б. Гусейнова, Б.А. Намазова

ЗМІНИ РІВНЯ КОРОТКОЛАНЦЮГОВИХ ЖИРНИХ КИСЛОТ, СИНТЕЗОВАНИХ МІКРОБІОТОЮ, У ДІТЕЙ З ВІРУСНОЮ ДІАРЕЄЮ

Метою дослідження була оцінка клініко-діагностичної значущості змін рівня коротколанцюгових жирних кислот у дітей першого року життя з вірусною діареєю. Дослідження проведено у 94 дітей з діареєю у віці від 0 до 1 року. Контрольну групу склали 30 здорових дітей. Визначення рівня коротколанцюгових жирних кислот проводилося методом газорідинної хроматографії. Згідно з результатами, при моно-варіанті вірусної діареї оцтова кислота знизилася в 1,5 рази в порівнянні з контролем, при змішаних варіантах – в 1,8 рази ($p<0,001$); пропіонова кислота при моно-варіанті знизилася в 1,5 рази, при змішаному варіанті – в 2 рази ($p<0,001$); масляна кислота при моно-варіанті знизилася в 2,5 рази, при змішаному варіанті була нижчою в 3,6 рази ($p<0,001$). Ізовалеріанова кислота при моно-варіанті знизилася в 1,2 рази ($p<0,05$), а при змішаному перебігу збільшилася (на 14,1 %).

Ключові слова: коротколанцюгові жирні кислоти, масляна кислота, валеріанова кислота, мікробіота, гастроентерит.

The term “microbiota” refers to the diverse ecosystem of bacteria, fungi, archaea, and viruses that inhabit the human gastrointestinal tract. These microbial communities are thought to exert a profound influence on the host’s physiological processes and pathological states [9]. Disruptions in the composition of the gut microbiota have been linked to a wide range of conditions, including gastrointestinal disorders as well as neurological, respiratory, metabolic, hepatic, and cardiovascular diseases [4].

A particularly intriguing focus of recent research concerns the role of microbiota-derived short-chain fatty acids (SCFAs) in shaping pediatric health and disease susceptibility [15]. Evidence indicates that the trillions of microorganisms residing in the gut contribute to the health of infants and children in ways that extend far beyond simple digestive functions. Among SCFAs, key representatives such as acetate, propionate, and butyrate act as important bioactive compounds. These metabolites form critical links between the gastrointestinal system and the immune, metabolic, and nervous systems, thereby influencing overall homeostasis [8].

It is increasingly recognized that the establishment of the human gut microbiota may commence before birth. Maternal environmental exposures during gestation can profoundly influence the maturation and structural composition of the fetal, neonatal, and early-infant intestinal microbiome, thereby shaping long-term health trajectories in the offspring. Multiple studies highlight the decisive role of the mother's gut microbiota – both antenatally and in the postpartum period – in regulating intestinal energy homeostasis and protecting against the onset of metabolic syndrome later in life [6]. Although still under discussion, accumulating findings suggest that microbial colonization in humans could be initiated as early as the first trimester. Moreover, ex vivo experiments demonstrated that a live Micrococcus strain isolated from fetal tissue could suppress interferon-gamma production in human T lymphocytes [2, 12].

The hypothesis of prenatal bacterial colonization corresponds with earlier observations that the fetal intestine may already contain memory T cells and dendritic cells potentially activated by microbial antigens [3]. While the anatomical distribution and taxonomic composition of the gut microbiota in pregnant women differ from those in nonpregnant women, overall species richness and diversity appear relatively stable [5]. In addition, infant birth weight has been shown to correlate strongly with maternal gut microbial composition, with downstream effects on growth and developmental outcomes in early life [6].

Throughout gestation and the early postnatal years, maternal and offspring microbiota remain closely interconnected, particularly regarding the intestinal microbiota of newborns and toddlers [10]. The perinatal period and the first years of life represent a critical window for gut colonization. Factors such as mode of delivery and gestational maturity at birth substantially influence the microbial profile of the neonate. Early-life determinants – including breastfeeding versus formula feeding, maternal nutritional patterns, environmental exposures, and host genetic background – further shape microbial community structure. Given the long-term consequences of this early microbial assembly, its influence on future health outcomes is considerable [11].

The major short-chain fatty acids generated in the human intestine are acetate, propionate, and butyrate, predominantly synthesized by specialized gut microorganisms in the cecum and proximal colon. Non-digestible carbohydrates, including dietary fibers, resistant starches, and non-starch polysaccharides, bypass enzymatic digestion in the small intestine and undergo anaerobic fermentation in the colon, resulting in SCFAs production. In situations of fiber deficiency, amino acids liberated via proteolysis can serve as alternative precursors. Fermentation of branched-chain amino acids – leucine, valine, and isoleucine – yields smaller quantities of less abundant SCFAs such as valerate, formate, and caproate. These metabolites contribute to nutrient absorption, modulation of luminal pH, immune regulation, and maintenance of gastrointestinal integrity [5, 11].

SCFAs uptake by colonocytes occurs through distinct mechanisms depending on ionization state. Protonated SCFAs passively diffuse along a concentration gradient, whereas their non-ionized forms are transported via carrier proteins. Once absorbed, SCFAs enter the portal circulation and are rapidly transported to the liver, where they are metabolized into endogenous molecules such as glucose, fatty acids, and cholesterol, or act as signaling agents. Unmetabolized fractions are eliminated through respiration, urine, or feces. Circulating SCFAs nevertheless exert systemic effects, including influences on cardiometabolic health, which are determined by both receptor engagement and concentration [13].

Beyond serving as an energy source for the colonic mucosa, SCFAs are essential in regulating the differentiation and function of intestinal epithelial cells, adipocytes, and immune cell subsets. Their actions operate through two primary pathways: epigenetic regulation via histone deacetylase inhibition, with butyrate being the most potent inhibitor followed by propionate – both displaying anti-inflammatory and anti-carcinogenic properties and activation of G protein–coupled receptors (GPCRs) expressed on intestinal epithelial cells, adipocytes, neurons, immune cells, and vascular endothelium. Relevant receptors include GPR41 (FFAR3) responsive to butyrate, GPR43 (FFAR2) responsive to acetate, propionate, and butyrate, and GPR109A responsive exclusively to butyrate. These receptor-mediated interactions modulate metabolic, immune, and neural pathways [5].

Taken together, elucidating the biosynthetic routes, absorption mechanisms, and signaling actions of SCFAs offers critical insight into their central role in sustaining host health and preventing disease.

The purpose of the study was to assess the clinical and diagnostic significance of quantitative and qualitative changes in short-chain fatty acids levels in children of the first year of life with viral diarrhea.

Materials and methods. The work was carried out in the period 2022–2023 at the Department of Childhood Diseases I of the Azerbaijan Medical University.

The study was conducted in 94 children with diarrhea aged 0–1 year. Of these, rotavirus gastroenteritis was in 54 patients, adenovirus gastroenteritis - in 8 patients, diarrhea associated with opportunistic microflora – in 32 sick children. The control group consisted of 30 healthy children.

The selection of patients was carried out by the method of continuous sampling as they were admitted to the hospital. For each child observed by us, a specially developed questionnaire was started, which reflected the main anamnestic data characterizing the features of the course of pregnancy, the neonatal period and the first year of the child's life, the nature of feeding, the diseases suffered by the child; the history of the current disease, the dynamics of the development of clinical symptoms, the results of laboratory tests. All children were examined in the acute period (the first two days of hospitalization). The severity of the disease was assessed based on the severity of intoxication symptoms (impaired general health, body temperature, sleep, appetite), as well as the degree of damage to the gastrointestinal tract (the presence and frequency of vomiting, the frequency and nature of stool).

The diagnosis of the disease was identified based on the assessment of anamnestic, clinical, laboratory and epidemiological data.

Laboratory methods included general clinical examinations and a comprehensive study of feces, with microbiological analysis (detection of pathogenic and opportunistic microflora), detection of rotavirus and adenovirus antigens in the coprofiltrate using express agglutination and enzyme immunoassay, as well as determination of SCFA levels (C2 – acetic, C3 – propionic, C4 – butyric, C5 – valerianic, iC4 – isobutyric, iC5 – isovaleric) and their total level, using gas-liquid chromatography.

To determine the rotavirus and adenovirus antigen in feces, the solid-phase enzyme-linked immunosorbent assay (ELISA) method with a ready-made reagent kit “Rotavirus–antigen–ELISA–Best” (VECTOR–BEST), and “Adenovirus-antigen-ELISA-Best” (VECTOR–BEST), were used. The principle of the method lies in the interaction of the rotavirus (adenovirus) antigen with monoclonal antibodies immobilized in the wells of a polystyrene plate. The “antigen–antibody” complex is detected using an enzyme-linked immunosorbent conjugate. After removing the unbound complex and incubating with a TMB (tetramethylbenzidine) solution, the solution in the wells becomes colored. The degree of coloring is directly proportional to the concentration of the rotavirus antigen in the analyzed samples. The reaction is stopped by adding a stop reagent, and the optical density of the solutions in the wells is measured at a wavelength of 450 nm.

The test results were recorded in a specially developed patient examination card, which reflected the clinical presentation of the disease, its diagnostics, and laboratory parameters. The obtained results were processed statistically using the Fisher-Student t-test, the chi-square test (χ^2) and the Wilcoxon U-test. The differences were considered significant when $p<0.05$.

Results of the study and their discussion. As a result of our studies, it was revealed that in children with viral etiology, there is a change in the metabolic activity of the microflora of the large intestine, characterized by various disorders in the spectrum of acetic, propionic, butyric, valerianic acids and their isomers, which has differential diagnostic value. Metabolic disorders in children with viral diarrhea are based on a microbiological imbalance with a deficiency of anaerobes and a decrease in their enzymatic activity. Analysis of the results of biochemical studies on the first day of the disease showed a decrease in the level of both individual volatile fatty acids and their total amount, which is associated with selective inhibition of normal microflora.

Thus, in the mono variant of viral diarrhea, the concentration of acetic acid (C2) decreased by 1.5 times compared to the control group and amounted to 2.642±0.036 mg/ml ($p<0.001$), in mixed variants – by 1.8 times and amounted to 2.302±0.078 mg/ml ($p<0.001$). Moreover, the content of C2 in the mixed course of viral diarrhea was 1.3 times lower than in the mono variant ($p<0.001$). Propionic acid (C3) in the mono variant decreased by 1.5 times compared to the control group and dropped to the level of 0.622±0.016 mg/ml ($p<0.001$), while in the mixed variant it decreased by 2 times and amounted to 0.472±0.024 mg/ml ($p<0.001$). Moreover, in mixed variants, the level of C3 was 1.4 times lower than in the mono variant of viral diarrhea ($p<0.001$).

When analyzing the concentrations of butyric acid (C4) in the mono variant, a decrease of 2.5 times was noted in relation to the control group to a level of 0.587±0.021 mg/ml ($p<0.001$), while in the mixed variant it was 3.6 times lower, which amounted to 0.433±0.031 mg/ml ($p<0.001$). Moreover, in mixed variants, the level of C4 was 1.4 times lower than in the mono variant of viral diarrhea ($p<0.001$).

Comparative analysis of valerianic acid (C5) showed a decrease in its concentration in mono- and mixed variants of viral diarrhea by 1.4 times – to 0.088±0.019 mg/ml ($p<0.001$), and 1.8 times – to 0.073±0.002 mg/ml ($p<0.001$), respectively. Moreover, the content of C5 in mixed variants was 1.2 times lower than in mono variants ($p<0.001$).

Analysis of changes in SCFA isoforms, which are the end product of fermentation of proteolytic intestinal microflora, revealed multidirectional changes in isobutyric and isovaleric acids.

Isobutyric acid (iC4) in mixed variants of viral diarrhea was statistically significantly increased by 1.3 times in relation to its content in mono variants ($p< 0.05$). Also, with mono variants, a decrease in iC4 by 10.3 % was noted in relation to the control group and amounted to 0.064±0.001 mg/ml ($p<0.05$), while with the mixed variant, an increase of 1.2 times to 0.083±0.012 mg/ml was noted.

Isovaleric acid (iC5) in the mono-course of viral diarrhea significantly decreased by 1.2 times in relation to the control group – 0.124±0.019 mg/ml ($p<0.05$), while in the mixed course there was a reliable increase of 14.1 % in relation to the control group – 0.162±0.003 mg/ml ($p<0.05$). The iC5 indicators in mixed variants were significantly higher by 1.3 times than the indicators in the mono-course ($p<0.001$).

Diarrheal diseases represent the most widespread form of acute infectious illnesses across countries with varying socio-political backgrounds. Globally, they rank as the eighth leading cause of mortality across all age groups, and are the fifth most common cause of death in children under the age of five [1]. As we revealed, diarrhea of viral etiology in children of first year of life is characterized with the various changes in the metabolic activity of the microbiota (disturbances in the spectrum of acetic, propionic, butyric, valerianic acids and their isomers).

The data we obtained are consistent with the mechanisms underlying their metabolism and described by other authors. Thus, Sivaprakasam S, et al (2017) indicated that the absorption of short-chain fatty acids in the colon includes various processes. For example, some forms of SCFAs are transported by absorption in the epithelium of the colon through simple diffusion provided by a chemical gradient. It is obvious that disruption of the integrity of the epithelium and its functional state, which usually accompanies viral diarrhea, will lead to disruption of the absorption of a number of SCFAs [13].

Jasim SA, et al (2022) emphasized that many studies indicate that microbiota metabolites act on many cell types to regulate various vital biological processes. In particular, short-chain fatty acids such as butyrate, acetate, and propionate play a key role in maintaining immunity, including host metabolism, intestinal function, and the immune system. This new concept of immune metabolism is critical for disease prevention and treatment. In our opinion, viral diarrhea, by disrupting the balance of SCFAs, creates a vicious cycle in which these pathological changes support each other [7].

It should be noted that our study involved infants under 1 year of age. This is a particularly important period for the development of microbiota, the formation of its functions and, accordingly, the metabolites synthesized by the microbiota, including SCFAs.

Zhang et al. (2022) reported that the composition of the infant's intestinal microbiome can predict the risk of developing and progressing diseases. At the same time, the composition of the infant's intestinal microbiome can be regulated in various ways and used to prevent and treat diseases in infants by changing its composition. The authors point out that differences in the composition of the intestinal microbiota of infants in health and disease, and, indirectly, differences in the levels of metabolites synthesized by them, are of great clinical significance, which allows them to be used for diagnostic purposes [15].

Moreover, some pathogens (sense and use these metabolites for growth and virulence, thereby stimulating inflammation in dysbiosis [9].

The role of SCFAs has been suggested and demonstrated in other gastrointestinal diseases. Several studies have confirmed the findings that butyrate administration alleviated intestinal inflammation and partially restored dysbiosis in mice with necrotizing enterocolitis. Notably, acetate, propionate, and butyrate levels significantly reduced the severity of necrotizing enterocolitis, and subsequent analysis demonstrated their predictive potential before the diagnosis of necrotizing enterocolitis [5].

Xiong et al. (2022) conducted a comparative analysis of the profiles, diversity, and metabolite characteristics of the gut microbiota in infants diagnosed with NEC and food protein-induced allergic coagulopathy (FPAI). The results of the study confirmed significant differences in fecal short-chain fatty acids, including acetate, propionate, butyrate, isovaleric acid, and hexanoic acid, as well as total SCFAs concentrations between the two groups. Therefore, the observed changes in acetate, propionate and butyrate levels may indicate gastrointestinal disturbances associated with NEC and may facilitate early prognosis [14].

Thus, the discussion of differences in SCFAs levels in different health conditions, including formation and absorption processes in normal physiological states and in pathological conditions, is crucial

for the development of potential therapeutic approaches. From this perspective, our research and similar works represent undoubted scientific and practical value.

Conclusions

1. In the mono- variant of viral diarrhea, the concentration of acetic acid (C2) decreased by 1.5 times compared to the ($p<0.001$), in mixed variants – by 1.8 times ($p<0.001$).

2. Propionic acid (C3) in the mono variant decreased by 1.5 times compared to the control group ($p<0.001$), while in the mixed variant it decreased by 2 times ($p<0.001$).

3. Butyric acid (C4) in the mono variant, a decrease of 2.5 times was noted in relation to the control group ($p<0.001$), while in the mixed variant it was 3.6 times lower ($p<0.001$).

4. Isovaleric acid (iC5) in the mono-course of viral diarrhea significantly decreased by 1.2 times in relation to the control group ($p<0.05$), while in the mixed course there was a reliable increase of 14.1 % in relation to the control group ($p<0.05$).

Thus, there were the changes in the metabolic activity of the colon microbiota which has differential diagnostic value in diarrhea of viral etiology.

References

1. Appelkhans OL, Kozishkurt OV, Hrytsenko KS, Boruta NV, Shablii TP, Hruzevskyi OA, et al. Molecular and genetic characteristics of adenovirus as causes of acute diarrhea in the people of southern Ukraine. World of medicine and biology. 2022; 2 (80): 189–193. DOI: 10.26724/2079-8334-2022-2-80-189-193.

2. Blaak EE, Canfora EE, Theis S, Frost G, Groen AK, Mithieux G, et al. Short-chain fatty acids in human gut and metabolic health. Benef Microbes. 2020 Sep 1;11(5):411-455. doi: 10.3920/BM2020.0057.

3. Chun J, Toldi G. The Impact of Short-Chain Fatty Acids on Neonatal Regulatory T Cells. Nutrients. 2022 Sep 6;14(18):3670. doi: 10.3390/nu14183670.

4. Cong J, Zhou P, Zhang R. Intestinal Microbiota-Derived Short Chain Fatty Acids in Host Health and Disease. Nutrients. 2022 May 9;14(9):1977. doi: 10.3390/nu14091977.

5. Hsu CY, Khachatryan LG, Younis NK, Mustafa MA, Ahmad N, Athab ZH, et al. Microbiota-derived short chain fatty acids in pediatric health and diseases: from gut development to neuroprotection. Front Microbiol. 2024 Oct 8;15:1456793. doi: 10.3389/fmicb.2024.1456793.

6. Huang L, Cai M, Li L, Zhang X, Xu Y, Xiao J, et al. Gut microbiota changes in preeclampsia, abnormal placental growth and healthy pregnant women. BMC Microbiol. 2021 Oct 4;21(1):265. doi: 10.1186/s12866-021-02327-7.

7. Jasim SA, Opulencia MJC, Ramírez-Coronel A. A., Abdelbasset W. K., Abed M. H., Markov A. The emerging role of microbiota-derived short-chain fatty acids in immunometabolism. Int. Immunopharmacol. 2022; 110:108983.

8. Mansuy-Aubert V, Ravussin Y. Short chain fatty acids: the messengers from down below. Front Neurosci. 2023 Jul 6;17:1197759. doi: 10.3389/fnins.2023.1197759.

9. Mirzaei R, Dehkhodaie E, Bouzari B, Rahimi M, Gholestani A, Hosseini-Fard SR, et al. Dual role of microbiota-derived short-chain fatty acids on host and pathogen. Biomed Pharmacother. 2022 Jan;145:112352. doi: 10.1016/j.biopha.2021.112352.

10. Pietrzak A, Banasiuk M, Szczepanik M, Borys-Iwanicka A, Pytrus T, Walkowiak J, et al. Sodium Butyrate Effectiveness in Children and Adolescents with Newly Diagnosed Inflammatory Bowel Diseases-Randomized Placebo-Controlled Multicenter Trial. Nutrients. 2022 Aug 11;14(16):3283. doi: 10.3390/nu14163283.

11. Prentice PM, Schoemaker MH, Vervoort J, Hettinga K, Lambers TT, van Tol EAF, et al. Human Milk Short-Chain Fatty Acid Composition is Associated with Adiposity Outcomes in Infants. J Nutr. 2019 May 1;149(5):716-722. doi: 10.1093/jn/nxy320.

12. Rackaityte E, Halkias J, Fukui EM, Mendoza VF, Hayzelden C, Crawford ED, et al. Viable bacterial colonization is highly limited in the human intestine in utero. Nat Med. 2020 Apr;26(4):599-607. doi: 10.1038/s41591-020-0761-3.

13. Sivaprakasam S, Bhutia YD, Yang S, Ganapathy V. Short-Chain Fatty Acid Transporters: Role in Colonic Homeostasis. Compr Physiol. 2017 Dec 12;8(1):299-314. doi: 10.1002/cphy.c170014.

14. Xiong J, Liao XS, Yin T, Liu XC, Bao L, Li LQ. Alterations of the gut microbiota and short chain fatty acids in necrotizing enterocolitis and food protein-induced allergic protocolitis infants: A prospective cohort study. Front Cell Infect Microbiol. 2022 Nov 21;12:1030588. doi: 10.3389/fcimb.2022.1030588.

15. Zhang D, Jian YP, Zhang YN, Li Y, Gu LT, Sun HH, et al. Short-chain fatty acids in diseases. Cell Commun Signal. 2023 Aug 18;21(1):212. doi: 10.1186/s12964-023-01219-9.

Стаття надійшла 7.08.2024 р.