УДК 611.36
DOI: https://doi.org/10.35693/SIM630041

Somatotype and microbiome: trends and correlations in liver cirrhosis

Yurii O. Zharikov[1], Aida N. Gadzhiakhmedova[1], Yana V. Kiseleva[2], Roman V. Maslennikov[1], Aliya M. Aliyeva[1], Tatyana S. Zharikova[1], Vladimir N. Nikolenko[1]

[1]I.M. Sechenov First Moscow State Medical University (Sechenov University) (Moscow, Russian Federation)
[2]Pirogov Russian National Research Medical University (Moscow, Russian Federation)

Abstract

Aim – to identify a correlation between the somatotype of a patient with liver cirrhosis and changes in the composition of the intestinal microbiota.

Material and methods. The study included 46 patients diagnosed with liver cirrhosis of various etiologies. The somatotype of the patients was determined using a bioimpedance analyzer of body composition (ABC-01 "Medass"). The intestinal microbiota was analyzed once by 16s rRNA sequencing. The processing of the received data was carried out using the program "Statistica".

Results. Among 46 patients diagnosed with liver cirrhosis, the majority (26 patients) had a mesoendomorphic somatotype, 14 patients were representatives of endomesomorphs. The predominance of representatives of the genera *Streptococcus* (p-value = 0.02), *Campylobacter* (p-value = 0.049) and *Holdemanella* (p-value = 0.048) was statistically significant in the group of endomesomorphs, while bacteria from the genera *Klebsiella* (p-value = 0.01) and *Gammaproteobacteria* (p-value = 0.048) prevailed in the group of mesoendomorphs (p-value = 0.02). Taxa of the intestinal microbiota of *Pyramidobacter* were expressed in patients with the endomorphic somatotype (p-value = 0.016).

Conclusions. Different somatotypes of patients with liver cirrhosis are associated with certain taxa of the intestinal microbiota, which prevail over other families of bacteria.

Keywords: bioimpedance analysis, somatotype, liver cirrhosis, intestinal microbiota.

Conflict of interest: nothing to disclose.

Citation
Zharikov YuO, Gadzhiakhmedova AN, Kiseleva YaV, Maslennikov RV, Aliyeva AM, Zharikova TS, Nikolenko VN. **Somatotype and microbiome: trends and correlations in liver cirrhosis.** *Science and Innovations in Medicine.* 2024;9(3):164-168.
DOI: https://doi.org/10.35693/SIM630041

Information about authors
Yurii O. Zharikov – MD, PhD, Associate professor of the Department of Human Anatomy and Histology, Institute of clinical medicine.
ORCID: https://orcid.org/0000-0001-9636-3807
E-mail: dr_zharikov@mail.ru
Aida N. Gadzhiakhmedova – student of the Institute of clinical medicine.
ORCID: https://orcid.org/0000-0003-2557-5647
E-mail: ai.kidman@mail.ru
Yana V. Kiseleva – resident.
ORCID: https://orcid.org/0000-0002-0009-9245
E-mail: kiselyana17@mail.ru
Roman V. Maslennikov – MD, PhD, Associate professor, Department of Internal Medicine, Gastroenterology and Hepatology of the Institute of clinical medicine.
ORCID: https://orcid.org/0000-0001-7513-1636
E-mail: mmmm00@yandex.ru
Aliya M. Aliyeva – MD, Gastroenterologist, Department of Internal Medicine, Gastroenterology and Hepatology of the Institute of clinical medicine.
ORCID: https://orcid.org/0000-0002-7606-2246
E-mail: aliya1993@mail.ru
Tatyana S. Zharikova – MD, PhD, Associate professor of the Department of Human Anatomy and Histology.
ORCID: https://orcid.org/0000-0001-6842-1520
E-mail: dr_zharikova@mail.ru
Vladimir N. Nikolenko – MD, PhD, Professor, Head of the Department of Human Anatomy and Histology of the Institute of clinical medicine.
ORCID: https://orcid.org/0000-0001-9532-9957
E-mail: vn.nikolenko@yandex.ru

Corresponding Author
Yurii O. Zharikov
Address: Sechenov University, b. 10, 11 Mokhovaya st., Moscow, Russia, 125009. Email: dr_zharikov@mail.ru

Abbreviations
LC – liver cirrhosis; BMI – body mass index; ACM – active cell mass; BFM – body fat mass; BL – body length (height); LBM – lean body mass; BM – body mass; EGDS – esophago-gastroduodenoscopy.

Received: 05.04.2024
Received: 16.06.2024
Published: 06.08.2024

Соматотип и микробиом: тенденции и корреляции при циррозе печени

Ю.О. Жариков[1], А.Н. Гаджиахмедова[1], Я.В. Киселева[2], Р.В. Масленников[1], А.М. Алиева[1], Т.С. Жарикова[1], В.Н. Николенко[1]

[1]ФГАОУ ВО Первый МГМУ имени И.М. Сеченова Минздрава России (Сеченовский Университет) (Москва, Российская Федерация)
[2]ФГАОУ ВО РНИМУ имени Н.И. Пирогова Минздрава России (Москва, Российская Федерация)

Аннотация

Цель – выявление корреляционных связей между соматотипом больных циррозом печени (ЦП) и изменениями в составе их кишечной микробиоты.

Материал и методы. В исследование включены 46 пациентов с диагнозом ЦП различной этиологии. Был определен соматотип пациентов при помощи биоимпедансного анализатора состава тела (АВС-01 «Медасс»). Анализ кишечной микробиоты проводился однократно методом секвенирования гена 16S рРНК.

Результаты. Среди 46 пациентов с диагнозом ЦП большинство (26 пациентов) имели мезоэндоморфный соматотип, 14 пациентов являлись представителями эндомезоморфов. В группе эндомезоморфов статистически значимым оказалось преобладание представителей родов *Streptococcus* (p-value = 0,02), *Campylobacter* (p-value = 0,049) и *Holdemanella* (p-value = 0,048), в то время как в группе мезоэндоморфов преобладали бактерии из родов *Klebsiella* (p-value = 0,01) и *Gammaproteobacteria* (p-value = 0,02). Таксон кишечной микробиоты *Pyramidobacter* были выражены у пациентов с эндоморфным соматотипом (p-value = 0,016).

Выводы. Различные соматотипы пациентов с ЦП ассоциированы с определенными таксонами кишечной микробиоты, которые превалируют над другими семействами бактерий.

Ключевые слова: биоимпедансный анализ, соматотип, цирроз печени, микробиота кишечника.

Конфликт интересов: не заявлен.

Для цитирования:
Жариков Ю.О., Гаджиахмедова А.Н., Киселева Я.В., Масленников Р.В., Алиева А.М., Жарикова Т.С., Николенко В.Н. **Соматотип и микробиом: тенденции и корреляции при циррозе печени.** *Наука и инновации в медицине.* 2024;9(3):164-168.
DOI: https://doi.org/10.35693/SIM630041

Сведения об авторах
Жариков Ю.О. – канд. мед. наук, доцент кафедры анатомии и гистологии человека Института клинической медицины имени Н.В. Склифосовского.
ORCID: https://orcid.org/0000-0001-9636-3807 E-mail: dr_zharikov@mail.ru
Гаджиахмедова А.Н. – студентка Института клинической медицины имени Н.В. Склифосовского.
ORCID: https://orcid.org/0000-0003-2557-5647
E-mail: ai.kidman@mail.ru
Киселева Я.В. – врач-ординатор. ORCID: https://orcid.org/0000-0002-0009-9245
E-mail: kiselyana17@mail.ru
Масленников Р.В. – канд. мед. наук, доцент кафедры пропедевтики внутренних болезней, гастроэнтерологии и гепатологии Института клинической медицины имени Н.В. Склифосовского. ORCID: https://orcid.org/0000-0001-7513-1636
E-mail: mmmm00@yandex.ru
Алиева А.М. – врач-гастроэнтеролог кафедры пропедевтики внутренних болезней, гастроэнтерологии и гепатологии Института клинической медицины имени Н.В. Склифосовского.
ORCID: https://orcid.org/0000-0002-7606-2246
E-mail: aliya1993@mail.ru
Жарикова Т.С. – канд. мед. наук, доцент кафедры анатомии и гистологии человека Института клинической медицины имени Н.В. Склифосовского.
ORCID: https://orcid.org/0000-0001-6842-1520
E-mail: dr_zharikova@mail.ru
Николенко В.Н. – д-р мед. наук, профессор, заведующий кафедрой анатомии и гистологии человека Института клинической медицины имени Н.В. Склифосовского.
ORCID: https://orcid.org/0000-0001-9532-9957
E-mail: vn.nikolenko@yandex.ru

Автор для переписки
Жариков Юрий Олегович
Адрес: Сеченовский Университет, ул. Моховая 11, стр. 10, г. Москва, Россия, 125009.
Email: dr_zharikov@mail.ru

Список сокращений
ЦП – цирроз печени; ИМТ – индекс массы тела; АКМ – активная клеточная масса; ЖМТ – жировая масса тела; ДТ – длина тела (рост); БМТ – безжировая масса тела; МТ – масса тела; ЭГДС – эзофагогастродуоденоскопия.

Получено: 05.04.2024
Одобрено: 16.06.2024
Опубликовано: 06.08.2024

INTRODUCTION

Cirrhosis is the final stage of chronic liver diseases of any etiology. It has a progressive nature, and is accompanied by changes in all organ systems and tissues of the human body (digestive, nervous, endocrine, coagulation, etc.). Liver cirrhosis (LC) has many life-threatening complications (bleeding from esophageal varices, spontaneous bacterial peritonitis, hepatic encephalopathy, etc.); it can be diagnosed at late stages of the disease [1]. The number of patients diagnosed with cirrhosis is steadily increasing, most frequent being the cases of cirrhosis resulting from steatohepatitis, alcoholic hepatitis, and long-term viral hepatitis coming first in etiology.

The only method of radical treatment of cirrhosis is organ transplantation from a deceased or related donor [2]. However, most patients on the transplant waiting list die from complications of LC, while many patients cannot apply for a transplant due to existing contraindications. That is why measures to prevent complications and prolong the compensated course of the disease come to the forefront of LC treatment.

These measures also include correction of the intestinal microbiota, which undergoes significant changes during the course of LC. Some of the characteristic changes are disturbances in the composition and quantity of intestinal microbiota representatives (intestinal dysbiosis) and small intestine bacterial overgrowth (SIBO) [3, 4].

In recent years, many studies were undertaken to prove the benefits of using probiotics in the treatment of non-alcoholic fatty liver disease, but the benefits of probiotics in cirrhosis remain not fully established [5].

One of the possible reasons for the controversial results of the studies is the lack of individual selection of probiotics for each patient with cirrhosis.

AIM

Identify a correlation between the changes of the intestinal microbiota with the somatotype of a patient that might assist implementation of an individual microbiota therapy and improvement of prognosis for patients with liver cirrhosis.

The study included 46 patients with the diagnosis of liver cirrhosis: 18 men and 28 women aged between 43 and 61. To diagnose cirrhosis, general clinical studies were performed (clinical and biochemical blood test, coagulogram), taking of history, physical examination, ultrasound of abdominal organs, esophago-gastroduodenoscopy (EGDS), blood test for viral hepatitis B, C using ELISA and PCR, clarification of alcohol history using the AUDIT questionnaire, blood testing for autoantibodies for individuals suspected of having autoimmune hepatitis. West-Haven criteria were used to diagnose hepatic encephalopathy.

Somatotyping was performed using the Heath-Carter automatic somatotype assessment protocol with bioimpedancemetry (ABC-01 "Medass" analyzer). Bioadhesive electrodes were fixed on the wrist and ankle joints of patients in a supine position. The following formulas are implemented in the software of the ABC-01 "Medass" bioimpedance analyzer to calculate the somatotype components:

$$EN=0,15xBFM/BL;\ ME=0,15xBLM/BL;$$
$$EC=BL/BM^{1/3};\ AC=0,15xACM/BL,$$

where EN, ME and EC – bioimpedance assessments of scores of endo-, meso- and ectomorphy, respectively; AC – analog of the mesomorphy assessment score in terms of active cell mass (ACM); BFM – body fat mass, BL – body length (height), BLM – body lean mass, BM – body mass [6].

To determine changes in intestinal microbiota, a single stool sample was collected from patients into a sterile container, which was immediately frozen to -80°C. Extraction of DNA was performed using the AmpliPrime DNA-sorb-AM reagent kit ("NextBio" LLC), and the obtained material was stored at -20oC. Qualitative and quantitative DNA assessment was performed using NanoDrop 1000 reagents (Thermo Fisher Scientific, Waltham, USA). The amplification was performed on the amplifier Applied Biosystems 2720 Thermal Cycler (Applied Biosystems, CA, United States), using primers: TCG TCGGCAGCGTCAGATGTGTATAAGAGACAGCCTACGG GNGGCWGCAG (forward) and GTCTCGTGGGCTCGGAGA TGTGTATAAGAGACAG-GACTACHVGGGTATCTAATCC (reverse). Amplification program: 95oC in the first 3 minutes; 30 cycles: 95oC for 30 seconds, 55oC for 30 seconds, 72°C for 30 seconds; 72oC for 5 minutes; 4oC. DNA purification was performed using magnetic particles Agencourt AMPure X (Beckman Coulter, Brea, CA, USA). The concentration of the obtained sDNA was measured using the Qubit® 2.0 fluorimeter (Invitrogen, Carlsbad, CA, USA).

The obtained data was processes in the STATISTICA 10 software suite (StatSoft Inc., Tulsa, OK, USA). For quantitative indicators, the distribution pattern was determined using the Shapiro-Wilk test, the median, interquartile range, mean value, and standard deviation. In the case of normal distribution of quantitative characteristics, comparative analysis was performed

using Welch's t-test (when comparing two groups) or ANOVA analysis of variance (when comparing more than two groups). For non-normally distributed quantitative traits, the Mann–Whitney U test (when comparing two groups) or the Kruskal–Wallis test (when comparing more than two groups) were used. Values of p≤0.05 were considered statistically significant.

RESULTS

As far as the cirrhosis etiology is concerned, 32.6% were patients with alcoholic cirrhosis, 10.9% with viral hepatitis C, 10.9% with autoimmune hepatitis. According to the Child-Pugh classification of liver disease, 14 patients had class A, 21 had class B, and 11 had class C. At the time of inclusion in the study, 9 patients had grade 2–3 ascites, and 15 had varying degrees of hepatic encephalopathy. Distribution of cirrhosis by etiology and Child-Pugh class and availability of complications is presented in **Table 1**.

Parameter	Value
Age	55 (43–61)
Men/women	18/28
Cirrhosis etiology:	
Alcoholic cirrhosis	15 (32,6%)
Viral hepatitis C	5 (10,9%)
Primary biliary cholangitis	4 (8,7%)
Primary sclerosing cholangitis	2 (4,3%)
Autoimmune hepatitis	5 (10,9%)
Metabolic-associated liver diseases	4 (8,7%)
Wilson's disease	3 (6,5%)
Liver cirrhosis of mixed and cryptogenic genesis	3 (6,5%)
Child-Pugh cirrhosis classes: A/B/C	14/21/11
Cirrhosis complications:	
Ascites (2-3 degrees), n (%)	9 (19,6%)
Hepatic encephalopathy, n (%)	15 (32,5%)
Somatotypes:	
Endomesomorphic	14 (30,4%)
Mesoendomorphic	26 (56,5%)
Central	3 (6,5%)
Endomorphic	2 (4,3%)
Ectomorphic	1 (2,2%)

Table 1. *General characteristics of the patients included in the study*

Таблица 1. *Общая характеристика пациентов, включенных в исследование*

Microbiome	Somatotype, Me, IQR		p
	endomesomorphic (n = 14)	mesoendomorphic (n = 26)	
Campylobacter	0,002 (0,000-0,019)	0,000 (0,000-0,000)	0,049*
Holdemanella	0,018 (0,000-0,126)	0,000 (0,000-0,013)	0,048*
Streptococcus	0,089 (0,055-0,149)	0,027 (0,002-0,176)	0,020**
Klebsiella	0,000 (0,000-0,000)	0,011 (0,000-0,075)	0,010**
Gammaproteo-bacteria	2,863 (1,131-5,383)	6,026 (3,669-11,9)	0,020**

Note. The table only provides statistically significant results. The asterisk "" marks p<0.05; double asterisk "**", p<0.025.*
Примечания. В таблице представлены только статистически значимые результаты. Знаком «» помечены p < 0,05; «**» – p < 0,025.*

Table 2. *The relative abundance of gut microbiota taxa in patients with endomesomorphic and mesoendomorphic somatotypes*

Таблица 2. *Относительное содержание кишечных бактерий (%) у пациентов эндомезоморфного и мезоэндоморфного соматотипов*

Taxon of intestinal microbiota	Somatotype, Me, IQR			p-value
	Endomeso-morphic (n = 14)	Mesoendo-morphic (n = 26)	Central (n = 3)	
Holdemanella	0,018 (0,00-0,126)	0,000 (0,000-0,013)	0,014 (0,010-0,045)	0,010**
Monoglobus	0,060 (0,012-0,189)	0,032 (0,007-0,123)	0,000 (0,000-0,012)	0,046*
Streptococcus	0,089 (0,055-0,149)	0,027 (0,002-0,176)	0,029 (0,023-0,169)	0,025*
Akkermansia	0,000 (0,000-0,639)	0,021 (0,000-0,867)	0,000 (0,000-0,000)	0,018**
Gamma-proteobacteria	2,863 (1,13-5,138)	6,026 (3,67-11,9)	3,143 (2,691-6,657)	0,025*

Примечания. В таблице представлены только статистически значимые результаты. Знаком «» помечены p < 0,05; «**» – p < 0,025.*

Таблица 3. *Относительное содержание кишечных бактерий различных таксонов у пациентов разных соматотипов*

Table 3. *The relative abundance of gut bacterial taxa in patients with different somatotypes*

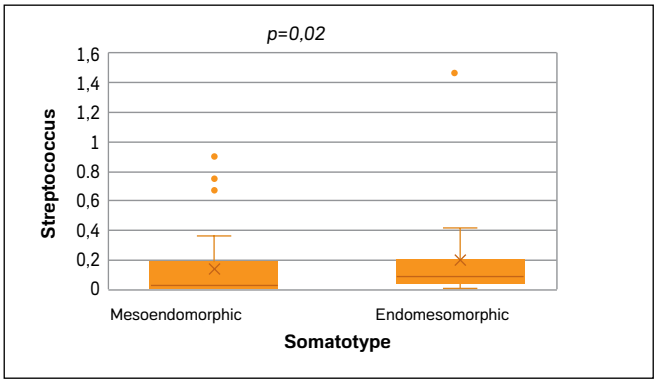

Figure 1. *The relative abundance of Streptococcus in the gut microbiome of patients with mesoendomorphic and endomesomorphic somatotypes.*

Рисунок 1. *Относительное содержание Streptococcus в микробиоме кишечника пациентов мезоэндоморфного и эндомезоморфного соматотипов.*

The majority of patients were endomesomorphs (14/46) and mesoendomorphs (26/46). Three patients had a central somatotype, two had an endomorphic somatotype, and one patient had an ectomorphic somatotype.

Based on the results of sequencing of the intestinal microbiome, statistically significant differences in the prevalence of bacterial genera were revealed among patients with endomesomorphic and mesoendomorphic somatotypes. These differences are shown in **Table 2**.

In the patients of the endomesomorphic somatotype, the following types of bacteria prevailed: *Campylobacter*, *Holdemanella* and *Streptococcus* (**Fig. 1**).

In the patients of the mesoendomorphic somatotype, the following bacteria prevailed: *Klebsiella* (**Fig. 2**), *Gammaproteobacteria* (**Fig. 3**), *Akkermansia* (**Fig. 4**), *Holdemanella* (**Fig. 5**) and *Monoglobus* (**Table 3**).

In the patients with the endomorphic somatotype (2/46), a statistically significant abundance of *Klebsiella* and *Pyramidobacter* was found (**Table 4**).

DISCUSSION

Intestinal dysbiosis is common in liver cirrhosis and is associated with the development of hepatic encephalopathy, decreased serum albumin and cholinesterase levels, systemic inflammation, and worsening of short-term and long-term prognosis. [7].

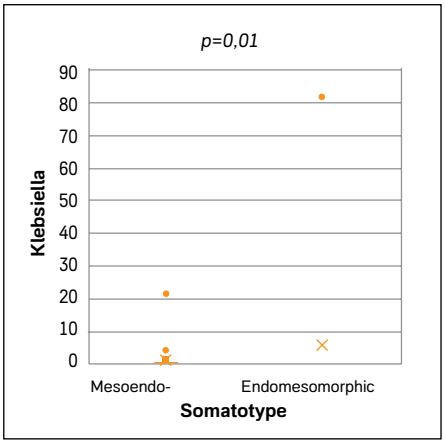

Figure 2. *The relative abundance of Klebsiella in the intestinal microbiome of patients with mesoendo- and endo-mesomorphic somatotypes.*

Рисунок 2. *Относительное содержание Klebsiella в микробиоме кишечника пациентов с мезоэндо- и эндомезоморфного соматотипов.*

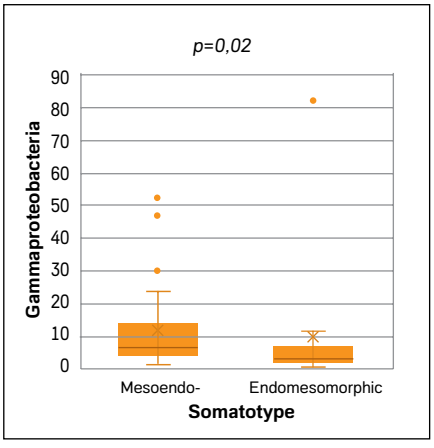

Figure 3. *The relative abundance of Gammaproteobacteria in the intestinal microbiome of patients with mesoendo- and endomesomorphic somatotypes.*

Рисунок 3. *Относительное содержание Gammaproteobacteria в микробиоме кишечника пациентов мезоэндо- и эндомезоморфного соматотипов.*

Cirrhosis causes significant changes in the composition and number of microbiota. It is now known that these changes begin long before the cirrhosis stage and gradually become more marked.

Thus, with cirrhosis, the total content of beneficial bacteria from the Clostridia class (Firmicutes type) decreases, the percentage of opportunistic *Enterococcaceae*, *Staphylococcaceae* and *Enterobacteriaceae* increases, the permeability of the intestinal barrier is disrupted, and bacterial components (lipopolysaccharides, bacterial DNA, etc.) actively enter the bloodstream, as well as living microorganisms. All this combined leads to local intestinal and systemic inflammation. Submucous intestinal edema, infiltration by immune cells, and disorganization of adhesion molecules develop. As a manifestation of cirrhosis, the flow of bile acids into the intestines, which are normally a substrate for "useful" bacteria, decreases, further aggravating the dysbiosis. [10, 11].

Normally, the intestine contains mainly bacteria of the *Firmicutes* and *Bacteroidetes* types, which together make up 90% of the microbiota. In addition to them, a large proportion of bacteria are *Proteobacteria* and *Actinobacteria*. In the state of eubiosis, microorganisms interact with each other through secreted metabolites, maintaining each other's numbers, limiting the growth of opportunistic and pathogenic flora. [8–9]. A comprehensive analysis of the fecal microbiota of healthy Japanese adults by K. Oki *et al*. (2016) identified a novel bacterial lineage associated with a phenotype characterized by high defecation frequency and lean body type. Across the entire population of subjects, stool frequency rates were significantly correlated with the abundance of *Christensenellaceae*, *Mogibacteriaceae*, and *Rikenellaceae* in the fecal microbiota ($p < 0.001$). These three lineages of bacteria were also more prevalent ($p < 0.05$ or 0.01) in lean people (BMI < 25 kg/m^2) than in obese people (BMI > 30 kg/m2). The results showed that the abundance of *Christensenellaceae*, *Mogibacteriaceae* and *Rikenellaceae*, as well as some other bacterial components that together make up the correlation network, were instrumental in the phenotype characterized with high defecation frequency and lean body type [8].

The association of microbiota changes with the presence of complications in cirrhosis was noted in many studies [12]. In the microbiota of patients with decompensation, the *Enterobacteriaceae*, *Staphylococcaceae*, *Enterococcaceae* prevailed, while the number of representatives of *Lachnospiraceae*, *Ruminococcaceae* и *Clostridiales* decreased [13].

Since numerous studies have identified disturbances in the composition of the intestinal microbiome in patients with cirrhosis, it is an urgent task to find factors that influence these disturbances. This article examines the features of the relationship between somatotype and changes in the intestinal microbiome in patients with cirrhosis. Specifically, patients with a predominance of the endomorphic component of the somatotype were found to have a higher content of bacteria of the genera *Campylobacter*, *Holdemanella* and *Streptococcus* than patients with a predominance of the meso- or ectomorphic components. At the same time, patients with a high mesomorphy score showed a predominance of

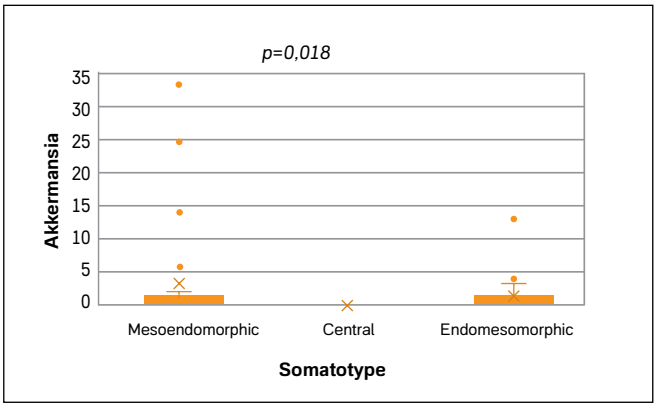

Figure 4. *The relative abundance of Akkermansia in the gut microbiome of patients of different somatotypes.*

Рисунок 4. *Относительное содержание Akkermansia в микробиоме кишечника пациентов разных соматотипов.*

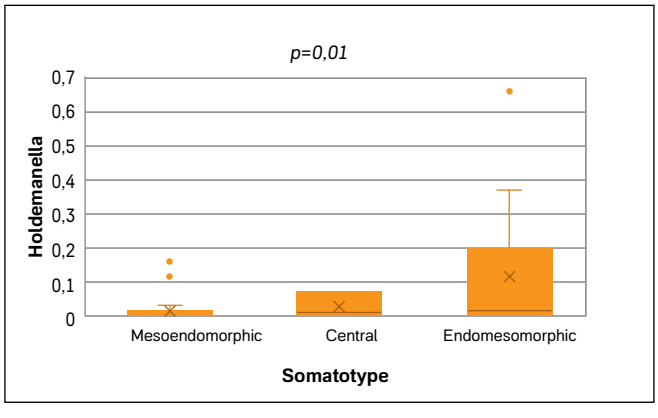

Figure 5. *The relative abundance of Holdemanella in the gut microbiome of patients of different somatotypes.*

Рисунок 5. *Относительное содержание Holdemanella в микробиоме кишечника пациентов разных соматотипов.*

Taxon of intestinal microbiota	Somatotype, Me, IQR					p
	Endomesomorphic (n=14)	Mesoendomorphic (n=26)	Central	эндоморфный (n=2)	эктоморфный (n=1)	
(n=3)	Endomorphic (n=2)	Ectomorphic (n=1)	0,00 (0,00-0,00)	0,11 (0,06-0,17)	0,00 (0,00-0,00)	0,016**
Klebsiella	0,00 (0,00-0,00)	0,01 (0,00-0,08)	0,00 (0,00-0,61)	0,12 (0,06-0,18)	0,02 (0,02-0,02)	0,039*

Note. The table only provides statistically significant results. The asterisk "" marks p<0.05; double asterisk "**", p<0.025.*
Примечания. В таблице представлены только статистически значимые результаты. Знаком «» помечены p < 0,05; «**» – p < 0,025.*

Table 4. *The relative abundance of gut bacterial taxa in patients with different somatotypes*

Таблица 4. *Относительное содержание кишечных бактерий у пациентов разных соматотипов*

bacteria of the genera *Klebsiella*, *Akkermansia*, *Monoglobus* and *Gammaproteobacteria*.

There are no studies on this topic in the scientific literature, which indicates the novelty of our work, but it requires confirmation and supplementation by further large-scale studies.

The results obtained represent the first report on the relationship between gut microbiota and somatotype in liver cirrhosis, as well as the first confirmation that increased Proteobacteria is associated with increased extracellular fluid in patients with liver cirrhosis. In addition, this study is one of the few studies that have examined the relationship between the gut microbiome and sarcopenia in patients with liver cirrhosis. A limitation of our study is the small sample size, but this did not prevent us from obtaining significant results.

CONCLUSIONS

Differences in the composition of the microbiota were revealed among patients with different somatotypes, which may indicate the influence of the somatotype on the characteristic changes in the microbiota. Large multicenter studies with a large number of patients are needed to confirm the correlation between somatotypes and changes in the intestinal microbiota. Identification of this relationship in large studies may facilitate the use of individually selected therapy with pro- and symbiotics for the treatment of patients with cirrhosis.

ADDITIONAL INFORMATION	ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ
Study funding. The study was the authors' initiative without external funding.	***Источник финансирования.*** Работа выполнена по инициативе авторов без привлечения финансирования.
Conflict of Interest. The authors declare that there are no obvious or potential conflicts of interest associated with the content of this article.	***Конфликт интересов.*** Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с содержанием настоящей статьи.
Contribution of individual authors. Yu.O. Zharikov – creating the design of the article, writing the text of the article, searching for literature. A.N. Gadzhiakhmedova – article editing, statistical processing, literature analysis. Ya.V. Kiseleva – writing the text of the article, searching for literature, technical revision of the text. R.V. Maslennikov – literature analysis. A.M. Alieva, T.S. Zharikova – literature search, article editing. V.N. Nikolenko – article editing. All authors gave their final approval of the manuscript for submission, and agreed to be accountable for all aspects of the work, implying proper study and resolution of issues related to the accuracy or integrity of any part of the work.	***Участие авторов.*** Ю.О. Жариков – создание дизайна статьи, написание текста статьи, поиск литературы. А.Н. Гаджиахмедова – правка статьи, статистическая обработка, анализ литературы. Я.В. Киселева – написание текста статьи, поиск литературы, техническая доработка текста. Р.В. Масленников – анализ литературы. А.М. Алиева, Т.С. Жарикова – поиск литературы, правка статьи. В.Н. Николенко – редактирование статьи. Все авторы одобрили финальную версию статьи перед публикацией, выразили согласие нести ответственность за все аспекты работы, подразумевающую надлежащее изучение и решение вопросов, связанных с точностью или добросовестностью любой части работы.

REFERENCES / ЛИТЕРАТУРА

1. Ginès P, Krag A, Abraldes JG, et al. Liver cirrhosis. *Lancet*. 2021;398(10308):1359-1376. DOI: https://doi.org/10.1016/S0140-6736(21)01374-X
2. Yoshiji H, Nagoshi S, Akahane T, et al. Evidence-based clinical practice guidelines for Liver Cirrhosis 2020. *J Gastroenterol*. 2021;56(7):593-619. DOI: https://doi.org/10.1007/s00535-021-01788-x
3. Bajaj JS, Heuman DM, Hylemon PB, et al. Altered profile of human gut microbiome is associated with cirrhosis and its complications. *J Hepatol*. 2014;60(5):940-7. DOI: https://doi.org/10.1016/j.jhep.2013.12.019
4. Maslennikov R, Ivashkin V, Efremova I, et al. Gut dysbiosis is associated with poorer long-term prognosis in cirrhosis. *World J Hepatol*. 2021;13(5):557-570. DOI: https://doi.org/10.4254/wjh.v13.i5.557
5. Maslennikov R, Efremova I, Ivashkin V, et al. Effect of probiotics on hemodynamic changes and complications associated with cirrhosis: A pilot randomized controlled trial. *World J Hepatol*. 2022;14(8):1667-1677. DOI: https://doi.org/10.4254/wjh.v14.i8.1667
6. Kolesnikov VA, Rudnev SG, Nikolaev DV, et al. On a new protocol of the Heath – Carter somatotype assessment using software for body composition bioimpedance analyzer. *Bulletin of Moscow University. Series XXIII. Anthropology*. 2016;4:4-13. [Колесников В.А., Руднев С.Г., Николаев Д.В., и др. О новом протоколе оценки соматотипа по схеме Хит – Картера в программном обеспечении биоимпедансного анализатора состава тела. *Вестник Московского университета. Сер. XXIII. Антропология*. 2016;4:4-13]. URL: file:///C:/Users/Persona/Downloads/o-novom-protokole-otsenki-somatotipa-po-sheme-hit-kartera-v-programmnom-obespechenii-bioimpedansnogo-analizatora-sostava-tela%20(2).pdf
7. Maslennikov R, Ivashkin V, Alieva A, et al. Gut dysbiosis and body composition in cirrhosis. *World J Hepatol*. 2022;14(6):1210-1225. DOI: https://doi.org/10.4254/wjh.v14.i6.1210
8. Oki K, Toyama M, Banno T, et al. Comprehensive analysis of the fecal microbiota of healthy Japanese adults reveals a new bacterial lineage associated with a phenotype characterized by a high frequency of bowel movements and a lean body type. *BMC Microbiol*. 2016;16(1):284. DOI: https://doi.org/10.1186/s12866-016-0898-x
9. Lynch SV, Pedersen O. The human intestinal microbiome in health and disease. *New England journal of medicine*. 2016;375(24):2369-2379. DOI: https://doi.org/10.1056/NEJMra1600266
10. Tilg H, Cani PD, Mayer EA. Gut microbiome and liver diseases. *Gut*. 2016;65(12):2035-2044. DOI: https://doi.org/10.1136/gutjnl-2016-312729
11. Qin N, Yang F, Li A, et al. Alterations of the human gut microbiome in liver cirrhosis. *Nature*. 2014;513(7516):59-64. DOI: https://doi.org/10.1038/nature13568
12. Maslennikov R, Alieva A, Poluektova E, et al. Sarcopenia in cirrhosis: Prospects for therapy targeted to gut microbiota. *World J Gastroenterol*. 2023;29(27):4236-4251. DOI: https://doi.org/10.3748/wjg.v29.i27.4236
13. Garbuzenko DV. Gut microbiota modulation in acute decompensation of liver cirrhosis: theory and therapeutic potential. *Russian Journal of Evidence-Based Gastroenterology*. 2022;11(4):65-76. [Гарбузенко Д.В. Теоретические основы и терапевтический потенциал модуляции микробиоты кишечника при острой декомпенсации цирроза печени. *Доказательная гастроэнтерология*. 2022;11(4):65-76]. DOI: https://doi.org/10.17116/dokgastro20221104165