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Results of the Interdisciplinary International Expert Council on Addressing the Management of Patients with Non-Alcoholic (Metabolic Dysfunction-Associated) Fatty Liver Disease

Marina V. Maevskaya[1]*, Sergey V. Okovityi[2], Igor G. Bakulin[3], Alan S. Bidzhiev[4], Dmitriy V. Glushenkov[1], Olesya Yu. Gurova[5], Alexander Yu. Gorshkov[6], Venera A. Zhakipbekova[7], Oxana Yu. Zolnikova[1], Mirvasit M. Karimov[8], Yulia V. Kotovskaya[9], Igor V. Maev[10], Roman V. Maslennikov[1], Maria Yu. Nadinskaia[1], Marina F. Osipenko[11], Karina L. Raikhelson[12,13], Balnur P. Sultanova[14], Olga N. Tkacheva[9], Ekaterina A. Troshina[15], Zamira Yu. Khalimova[16], Gulnoz A. Khamidullaeva[17], Anna F. Sheptulina[6], Elena N. Shirokova[1], Oksana M. Drapkina[6], Vladimir T. Ivashkin[1]

[1] *I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russian Federation*
[2] *Saint Petersburg State Chemical-Pharmaceutical University, Saint Petersburg, Russian Federation*
[3] *North-Western State Medical University named after I.I. Mechnikov, Saint Petersburg, Russian Federation*
[4] *Center for Hepatopancreatobiliary Surgery, Oncohepatology and Organ Transplantation, National Scientific Oncology Center, Astana, Republic of Kazakhstan*
[5] *Professorial Clinic of Endocrinology and Diabetes, Moscow, Russian Federation*
[6] *National Medical Research Center for Therapy and Preventive Medicine, Moscow, Russian Federation*
[7] *Karaganda Medical University, Karaganda, Republic of Kazakhstan*
[8] *Republican Specialized Scientific and Practical Medical Center for Therapy and Medical Rehabilitation, Tashkent, Republic of Uzbekistan*
[9] *N.I. Pirogov Russian National Research Medical University, Moscow, Russian Federation*
[10] *Russian University of Medicine, Moscow, Russian Federation*
[11] *Novosibirsk State Medical University, Novosibirsk, Russian Federation*
[12] *Saint Petersburg State University, Saint Petersburg, Russian Federation*
[13] *Academician I.P. Pavlov First Saint Petersburg State Medical University, Saint Petersburg, Russian Federation*
[14] *Research Institute of Cardiology and Internal Medicine, Almaty, Republic of Kazakhstan*
[15] *Endocrinology Research Centre, Moscow, Russian Federation*
[16] *Republican Specialized Scientific and Practical Medical Center of Endocrinology named after Academician Y.Kh. Turakulov, Tashkent, Republic of Uzbekistan*
[17] *Republican Specialized Scientific and Practical Medical Center of Cardiology, Tashkent, Republic of Uzbekistan*

Aim: to present the results of an interdisciplinary international expert council on addressing the challenges of managing patients with non-alcoholic (metabolic dysfunction-associated) fatty liver disease in connection with the creation of a new nomenclature, the definition of diagnostic criteria, and the development of approaches to the diagnosis and treatment of fatty liver disease.

Key points. The key role of metabolic dysfunction in non-alcoholic (metabolic dysfunction-associated) fatty liver disease has been proven. Therefore, in the nomenclature, non-alcoholic fatty liver disease (NAFLD) has been replaced by metabolic dysfunction-associated fatty liver disease, and non-alcoholic steatohepatitis has been replaced by metabolic dysfunction-associated steatohepatitis. It is noted that, according to research, the prevalence of fatty liver disease in Russia is high: one-third of patients seeking primary care have this diagnosis. The results of a study of two incretin mimetics, semaglutide and tirzepatide, are presented, along with an opinion on their rational combination with ursodeoxycholic acid. The comorbidity of fatty liver disease with cardiovascular, endocrine, and oncological diseases, chronic kidney disease, and other conditions is discussed.

Conclusion. The expert council included leading specialists: gastroenterologists-hepatologists, cardiologists, internists, and endocrinologists. There were confirmed that treatment for fatty liver disease should be comprehensive and integrated into the overall concept of metabolic health. Algorithms for diagnosis and personalized treatment of fatty liver disease have to be developed for the Russian medical community.

Keywords: non-alcoholic fatty liver disease, metabolically associated fatty liver disease, diagnostics, screening, steatohepatitis, fibrosis, steatosis

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Итоги междисциплинарного международного экспертного совета по решению проблем ведения пациентов с неалкогольной (метаболически ассоциированной) жировой болезнью печени

М.В. Маевская[1]*, С.В. Оковитый[2], И.Г. Бакулин[3], А.С. Биджиев[4], Д.В. Глушенков[1], О.Ю. Гурова[5], А.Ю. Горшков[6], В.А. Жакипбекова[7], О.Ю. Зольникова[1], М.М. Каримов[8], Ю.В. Котовская[9], И.В. Маев[10], Р.В. Масленников[1], М.Ю. Надинская[1], М.Ф. Осипенко[11], К.Л. Райхельсон[12,13], Б.П. Султанова[14], О.Н. Ткачева[9], Е.А. Трошина[15], З.Ю. Халимова[16], Г.А. Хамидуллаева[17], А.Ф. Шептулина[6], Е.Н. Широкова[1], О.М. Драпкина[6], В.Т. Ивашкин[1]

[1] *ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет), Москва, Российская Федерация*

[2] *ФГБОУ ВО «Санкт-Петербургский государственный химико-фармацевтический университет» Министерства здравоохранения Российской Федерации, Санкт-Петербург, Российская Федерация*

[3] *ФГБОУ ВО «Северо-Западный государственный медицинский университет им. И.И. Мечникова» Министерства здравоохранения Российской Федерации, Санкт-Петербург, Российская Федерация*

[4] *Центр гепатопанкреатобилиарной хирургии, онкогепатологии и трансплантации органов, ТОО «Национальный научный онкологический центр», Астана, Республика Казахстан*

[5] *ООО «Профессорская клиника эндокринологии и диабета», Москва, Российская Федерация*

[6] *ФГБУ «Национальный медицинский исследовательский центр терапии и профилактической медицины». Министерства здравоохранения Российской Федерации, Москва, Российская Федерация*

[7] *НАО «Карагандинский медицинский университет», Караганда, Республика Казахстан*

[8] *Республиканский специализированный научно-практический медицинский центр терапии и медицинской реабилитации, Ташкент, Республика Узбекистан*

[9] *ФГАОУ ВО «Российский национальный исследовательский медицинский университет им. Н.И. Пирогова» Министерства здравоохранения Российской Федерации, Москва, Российская Федерация*

[10] *ФГБОУ ВО «Российский университет медицины» Министерства здравоохранения Российской Федерации, Москва, Российская Федерация*

[11] *ФГБОУ ВО «Новосибирский государственный медицинский университет» Министерства здравоохранения Российской Федерации, Новосибирск, Российская Федерация*

[12] *ФГБОУ ВО «Санкт-Петербургский государственный университет», Санкт-Петербург, Российская Федерация*

[13] *ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. академика И.П. Павлова» Министерства здравоохранения Российской Федерации, Санкт-Петербург, Российская Федерация*

[14] *АО «НИИ кардиологии и внутренних болезней», Алматы, Республика Казахстан*

[15] *ФГБУ «НМИЦ эндокринологии им. академика И.И. Дедова» Министерства здравоохранения Российской Федерации, Москва, Российская Федерация*

[16] *Республиканский специализированный научно-практический медицинский центр эндокринологии им. академика Е.Х. Туракулова, Ташкент, Республика Узбекистан*

[17] *Республиканский специализированный научно-практический медицинский центр кардиологии, Ташкент, Республика Узбекистан*

Цель: представить результаты обсуждений в ходе междисциплинарного международного экспертного совета по решению проблем ведения пациентов с неалкогольной (метаболически ассоциированной) жировой болезнью печени в связи с созданием новой номенклатуры, определением диагностических критериев, выработкой подходов к диагностике и лечению жировой болезни печени.

Основные положения. Доказана ключевая роль метаболической дисфункции в неалкогольной (метаболически ассоциированной) жировой болезни печени, поэтому в номенклатуре неалкогольная жировая болезнь печени (НАЖБП) заменена на метаболически ассоциированную жировую болезнь печени, а неалкогольный стеатогепатит — на метаболически ассоциированный стеатогепатит. Отмечено, что, согласно исследованиям, распространенность жировой болезни печени в России высока: треть пациентов, которые обращаются к врачу первичного звена здравоохранения, имеют этот диагноз. Представлены результаты исследования двух инкретиномиметиков — семаглутида и тирзепатида, а также высказано мнение об их рациональной комбинации с урсодезоксихолевой кислотой. Рассмотрены вопросы коморбидности жировой болезни печени с сердечно-сосудистыми, эндокринными, онкологическими заболеваниями, хронической болезнью почек и др.

Заключение. В состав экспертного совета вошли ведущие специалисты: гастроэнтерологи-гепатологи, кардиологи, терапевты, эндокринологи. В ходе дискуссии подтверждено, что лечение жировой болезни печени должно быть комплексным и интегрированным в общую концепцию метаболического здоровья. Необходимо создание алгоритмов диагностики и персонифицированного лечения жировой болезни печени для российского врачебного сообщества.

Ключевые слова: неалкогольная жировая болезнь печени, метаболически ассоциированная жировая болезнь печени, диагностика, скрининг, стеатогепатит, фиброз, стеатоз, аутофагия
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Introduction

In June 2025, an interdisciplinary international expert council was held with the purpose of developing a model of a structured interdisciplinary approach to address current challenges in managing patients with non-alcoholic fatty liver disease (NAFLD), or metabolic dysfunction-associated steatotic liver disease (MASLD), according to the new nomenclature [1]. The expert council was composed of leading specialists from various fields: gastroenterologists-hepatologists, cardiologists, internists, and endocrinologists. The council was chaired by Academician Vladimir T. Ivashkin, Chief Consultant Gastroenterologist of the Russian Ministry of Health, and Academician Oksana M. Drapkina, Chief Consultant Physician of the Russian Ministry of Health.

What formed the basis of the expert council's agenda that required the efforts of collective intelligence? Recently, a new nomenclature of fatty liver disease has been created, diagnostic criteria of NAFLD (MASLD) have been defined, and approaches to diagnosing and treating this disease have been developed. However, not all questions have clear answers, and not all needs of the medical community have been addressed.

The high, increasing prevalence of NAFLD (MASLD) requires the healthcare system to address the issue of screening for this disease. Meanwhile, the question remains: where should the efforts be channelled to: toward the screening a population or high-risk groups for NAFLD (MASLD) and non-alcoholic or metabolic dysfunction-associated steatohepatitis (NASH or MASH) as its progressive form? Which component of NAFLD (MASLD) needs to be screened: fibrosis and/or steatosis? What tools should be used for this? Physicians need to quickly and effectively diagnose the disease, have a clear algorithm for differential diagnosis based on the principle of "from simple to complex and from frequent to rare", stratify the risk using simple and accessible tools, and route the patients. It is necessary to clearly define the role of the primary care physician and the gastroenterologist-hepatologist in managing patients with NAFLD (MASLD), as well as involve endocrinologists and cardiologists in the active detection of NAFLD (MASLD) and risk stratification. In recent years, the NAFLD (MASLD) paradigm has shifted from an organ-centric to a patient-centered one, requiring physicians to understand the pathogenesis of the disease and develop clinical thinking. Possible solutions and approaches to these issues were discussed by the expert council members.

Key points

The work began with the report of the invited expert Professor Arun Sanyal, a world-renowned scientist and a leading expert on NAFLD (MASLD), who noted that non-communicable diseases are responsible for 74 % of all deaths globally, most of which occur in low- or middle-income countries [2].

Professor A. Sanyal focused on the evolution of terminology and understanding of the pathogenesis of NAFLD (MASLD). Historically, the term "non-alcoholic fatty liver disease" (NAFLD) was exclusionary and did not reflect the etiology of the disease. Current data point to the key role of metabolic dysfunction in this disease, which has led to a revision of the nomenclature: the term "non-alcoholic fatty liver disease" has been replaced by a new term, "metabolic dysfunction-associated fatty liver disease". The term "non-alcoholic steatohepatitis" (NASH) has been replaced by "metabolic dysfunction-associated steatohepatitis" (MASH). This transformation is substantiated by the high comorbidity of NAFLD (MASLD) with components of metabolic syndrome: obesity, type 2 diabetes mellitus (T2D), arterial hypertension, and hypertriglyceridemia.

The new nomenclature identifies an umbrella (unifying) term, "steatotic liver disease" (SLD), which includes, in addition to NAFLD (MASLD), alcohol-related liver disease (ALD), fatty liver disease of specific etiology (drug-induced, genetically determined, etc.), as well as new subtypes of SLD, i.e., steatotic liver disease due to moderate alcohol consumption in combination with metabolic dysfunction (Met-ALD) and cryptogenic

SLD. All subtypes of the SLD have their own clear diagnostic criteria [3].

Based on this information, Professor A. Sanyal draws his first conclusion: NAFLD (MASLD) is a common non-communicable disease, and its prevalence is rising worldwide.

Professor A. Sanyal then focused on the systemic nature of NAFLD (MASLD) and its association with cardiometabolic outcomes. A pathogenetic relationship has been established between NAFLD (MASLD) and the development of cardiovascular disease, DM2, chronic kidney disease, and cancers. The presence of liver steatosis independently increases the risk of the outcomes indicated. There is a bidirectional relationship: the severity of metabolic disorders correlates with the progression of liver disease, and vice versa. Ectopic lipid accumulation (in the liver, vascular walls) initiates an inflammatory cascade and fibrogenesis, leading to the dysfunction of end organs.

Professor A. Sanyal draws the second conclusion: NAFLD (MASLD) is closely linked to other non-communicable diseases, necessitating a preventive approach and integrating its treatment into an overall metabolic health management strategy.

The progression of NAFLD (MASLD) follows a classic pathway: steatosis — steatohepatitis — fibrosis — cirrhosis — decompensation/hepatocellular carcinoma — liver transplantation. The two critical concepts in assessing the disease are:

1) activity, which is characterized by three main histological features: steatosis, lobular inflammation and ballooning;

2) stage (fibrosis), which correlates with the risk of developing cirrhosis and death from liver disease.

This leads to the third conclusion: disease activity determines the rate of fibrosis progression, and the fibrosis stage determines the risk of adverse outcomes. This dictates the need for timely diagnosis of the disease and risk stratification.

In primary healthcare settings, non-invasive tests for fibrosis assessment play a key role. For primary fibrosis screening, the FIB-4 index (calculated based on the age, level of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and platelet counts) is used:

- **FIB-4 < 1.3** — low risk of fibrosis progression; re-evaluation in 1–2 years;
- **FIB-4** in the range of **1.3–2.6** — intermediate risk zone;
- **FIB-4 > 2.6** — high risk of clinically significant fibrosis, requiring in-depth investigation.

The second step in assessing fibrosis is liver transient elastography (liver stiffness measurement):

- **liver stiffness < 8 kPa** — low risk of progression to severe fibrosis/cirrhosis;
- **liver stiffness ≥ 8 kPa** — high risk of progression to severe fibrosis/cirrhosis; consultation with a hepatologist is advisable.

Professor A. Sanyal's fourth conclusion: the use of non-invasive tests, in particular FIB-4, allows for effective risk stratification and optimized patient routing.

A. Sanyal turned his attention to the therapeutic strategy for NAFLD (MASLD). Treatment should be comprehensive. It includes lifestyle modification and pharmacotherapy:

- **lifestyle modification** is the foundation; it includes diet, physical activity, and behavioral modification;
- **drug therapy:**

— medications affecting metabolic dysfunction: statins, glucagon-like peptide 1 receptor agonists (GLP-1 RA) and other incretin mimetics, metformin, sodium-glucose cotransporter 2 inhibitors;

— specific therapy for NASH (MASH): for this purpose, hepatotropic drugs approved in each specific region are used. Ursodeoxycholic acid (UDCA) is successfully used for this purpose in a number of countries, including Russia.

In conclusion, Professor A. Sanyal proposed an algorithm of actions in case of suspected NAFLD (MASLD) to assist practicing physicians (Fig. 1).

Professor A. Sanyal once again emphasized that NAFLD (MASLD) is a growing threat to public health worldwide. Addressing this problem requires the involvement of primary care clinicians and physicians, gastroenterologists-hepatologists, cardiologists, endocrinologists, and other specialists, depending on the specific characteristics of the NAFLD (MASLD) features in each patient. A preventive approach aimed at early detection of the disease and intervention in order to optimize its scenario is of key importance.

Of particular importance in this regard are the data cited by Professor Igor G. Bakulin on the association of NAFLD (MASLD) with certain types of cancer: hysterocarcinoma, gallbladder cancer, hepatic cancer, thyroid carcinoma, renal cell carcinoma, bladder cancer, breast cancer, rectal cancer, and colon cancer [4].

Oksana M. Drapkina, Academician of RAS, Chief Consultant Physician of the Russian Ministry of Health, in her presentations, spoke about the epidemiology of NAFLD (MASLD) in Russia based on the results of major epidemiological surveys: DIREG, DIREG2, ESSE-RF2 and ESSE-RF3, and PROMAFLD [5], which she initiated and organized (with the exception of the PROMAFLD survey, which was initiated by the National Medical Research Center for Therapy

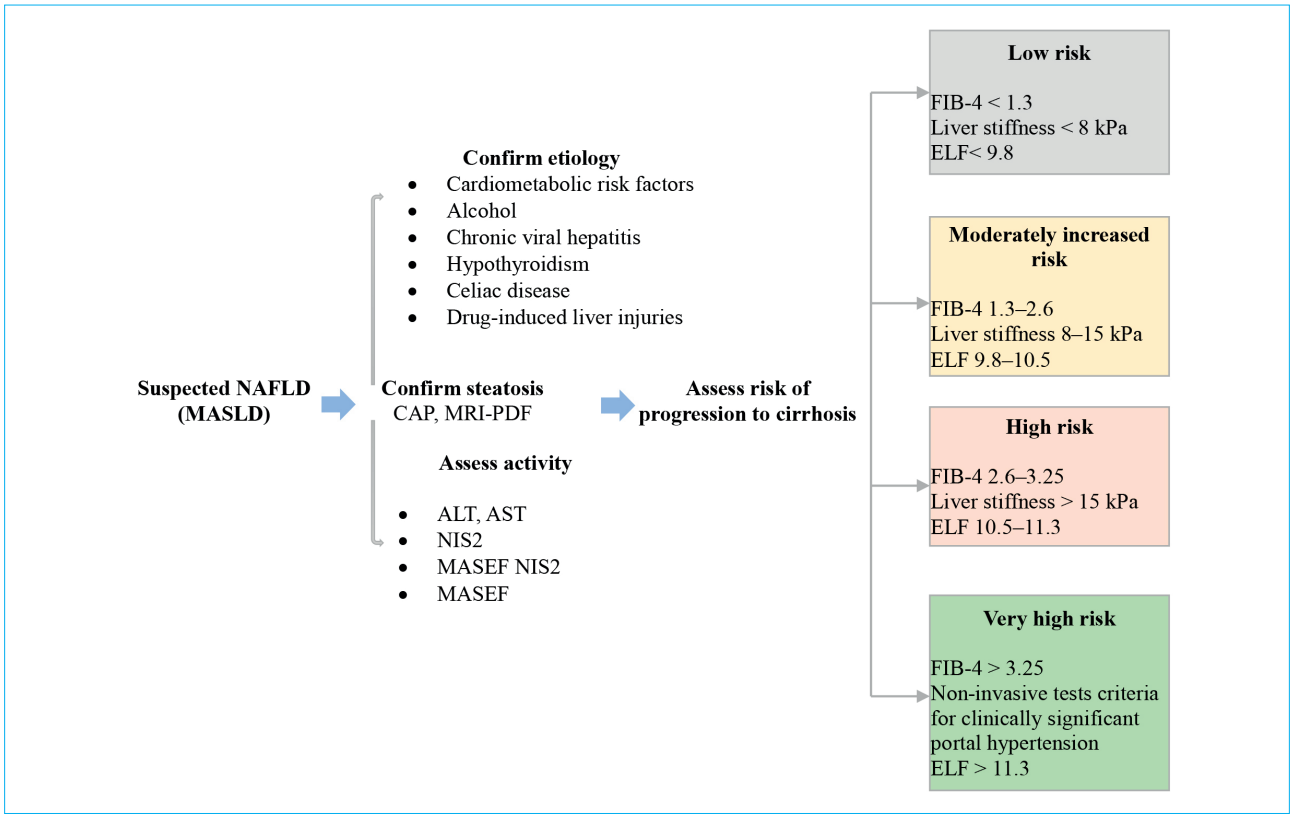

Figure 1. Updated algorithm of physicians' actions for a suspected non-alcoholic (metabolic dysfunction-associated) fatty liver disease: CAP — controlled attenuation parameter; MRI — magnetic resonance imaging; ALT — alanine aminotransferase, AST — aspartate aminotransferase; NIS2 — a two-parameter, sex-corrected blood serum diagnostic technology for assessing the risk of NASH (MASH) in patients with metabolic risk factors; MASEF — a test based on determining twelve lipids in the blood, ALT and AST, as well as body mass index to assess fibrosis (Metabolomics-Advanced Steatohepatitis Fibrosis Score); FIB-4 — Fibrosis-4 Index; ELF (Enhanced Liver Fibrosis) — a three-parameter blood serum test to assess fibrosis

and Preventive Medicine (NMRC TPM) together with the Gilbert Club physicians' community). According to the results of the surveys, the prevalence of NAFLD (MASLD) in our country is high and tends to grow, as it does worldwide. On average, one third of patients seeking primary care are diagnosed with this disease. Academician Igor V. Maev supplemented Academician Drapkina's epidemiological data with the results of his own research: the prevalence of NAFLD (MASLD) among patients of Moscow multidisciplinary hospitals is 47.3 % [6].

An analysis performed on a representative Russian patient sample confirmed the association of NAFLD (MASLD) with arterial hypertension, DM2, dyslipidemia, hyperuricemia, and stroke in men and women, indicating a multifactorial metabolic nature of the disease [5].

It was shown that NAFLD (MASLD) is a new risk factor for the development of arterial hypertension and atherosclerosis. This is due to systemic inflammation, oxidative stress, and vasoconstriction, while genetic predisposition, epigenetic factors, and changes in the composition of the intestinal microbiota also contribute to it. The issues of the NAFLD (MASLD) pathogenesis in association with the risk of cardiovascular disease were also highlighted in a presentation by Professor Yuliya V. Kotovskaya, who cited data from a meta-analysis of 114 studies indicating that NAFLD (MASLD) is associated with endothelial dysfunction and increases the risk of subclinical and overt atherosclerosis [7], including in young people [8]. Older patients meanwhile have an increased risk of complications of cardiovascular disease and atrial fibrillation [9].

Academician Oksana M. Drapkina hypothesized a categorical shift toward increased cardiovascular risk in the presence of NAFLD (MASLD) and the possibility of personalized assessment for individual patients. For this purpose, a modified cardiovascular risk assessment scale taking into account NAFLD (MASLD) as an independent factor is being developed under the guidance of

Academician Oksana M. Drapkina. There is no doubt that current scales of cardiovascular risk stratification (SCORE, SCORE2), widely used in practice, are accurate and precise, and incorporate simple and accessible parameters. However, there remains a residual risk that cannot be explained by traditional and generally accepted factors. To minimize this risk, it is proposed to consider the patient's ethnic origin, family history, renal function, coronary calcium, etc. An approach to improving existing scales with the use of biomarkers of myocardial injury, inflammation, thrombosis, etc. also appears interesting. For instance, according to research results from the NMRC TPM (unpublished data), the presence of inflammation (which occurs in NAFLD (MASLD)) increases the risk of cardiovascular death in men over 75. Russia needs its own risk assessment model, which would improve the effectiveness of screening and preventive examinations of the population.

Academician O.M. Drapkina emphasized the stage of follow-up monitoring for patients with NAFLD (MASLD), identifying the crucial role of a general practitioner in this process, who needs to keep in focus not only the liver condition, but also all metabolic parameters, which determines the frequency of follow-up examinations. For example, individuals with prediabetes and DM2, two or more cardiometabolic risk factors, and signs of clinically significant fibrosis (stage II or higher) should undergo follow-up monitoring once a year. This information is presented in the Clinical Guidelines "Non-Alcoholic Fatty Liver Disease" (2024), which are currently accepted for practical use [10].

Academician O.M. Drapkina concluded that step-by-step adherence to current Russian Clinical Guidelines helps not only to confirm the diagnosis of NAFLD (MASLD), but also to identify extrahepatic manifestations of the disease. This approach allows for a comprehensive assessment of the patient's prognosis and timely implementation of all necessary preventive and therapeutic measures.

There is no doubt that successful treatment of NAFLD (MASLD) is based on non-drug therapy aimed at changing the patient's lifestyle. At the same time, the entire global medical community is following the progress in NAFLD (MASLD) pharmacotherapy. Professors Marina V. Maevskaya and Sergey V. Okovityi shared new data in this area. In addition to the information provided in the Clinical Guidelines "Non-Alcoholic Fatty Liver Disease", the results of a study of two incretin mimetics appeared: semaglutide (a GLP-1 receptor agonist) and tirzepatide (a dual receptor agonist: GLP-1 and glucose-dependent insulinotropic polypeptide).

Semaglutide demonstrated its effectiveness in the treatment of patients with NASH (MASH) and stage II–III fibrosis in the ESSENCE study. Intermediate data were analyzed after 72 weeks of therapy in 800 patients [11] who received semaglutide subcutaneously at a dose of 2.4 mg once a week and were compared with placebo. Resolution of steatohepatitis without worsening fibrosis was observed in 62.9 % of patients compared with 34.3 % in the placebo group; a decrease in fibrosis by ≥ 1 stage without worsening inflammation was observed in 36.8 % of cases in the semaglutide group and in 22.4 % of cases in the placebo group. Combined resolution of steatohepatitis and a reduction in liver fibrosis were observed in 32.7 % of patients in the semaglutide group and in 16.1 % of patients in the placebo group, which is two times less. Based on these data, semaglutide was approved by the FDA for the treatment of NASH (MASH).

The efficacy of tirzepatide was also studied in patients with NASH (MASH) in the SYNERGY-NASH trial, a multicenter, randomized, double-blinded, placebo-controlled phase II trial (tirzepatide dosing) in patients with histologically proven NASH (MASH) and stage II–III fibrosis. Study participants were randomly assigned to receive subcutaneous tirzepatide at doses of 5, 10, or 15 mg or placebo once a week for 52 weeks. The primary efficacy endpoint was resolution of MASH (NASH) without worsening fibrosis; the secondary efficacy endpoint was a reduction in fibrosis by ≥ 1 stage without worsening steatohepatitis after 52 weeks. A total of 190 patients were randomized, and treatment outcomes were assessed in 157 patients.

Resolution of MASH without worsening of fibrosis (the main endpoint) was observed in 10 % of patients in the placebo group, in 44 % of patients in the tirzepatide 5 mg/week group (+34 % to placebo; $p < 0.001$), in 56 % of patients in the tirzepatide 10 mg/week group (+46 %; $p < 0.001$), and in 62 % of patients in the tirzepatide 15 mg/week group (+53 %; $p < 0.001$).

A decrease in fibrosis by ≥ 1 stage without progression of NASH (MASH) (secondary efficacy endpoint) was observed in 30 % of patients in the placebo group, in 55 % of patients in the tirzepatide 5 mg/week group (+25 %), in 51 % of patients in the tirzepatide 10 mg/week group (+22 %), and in 51 % of patients in the tirzepatide 15 mg/week group (+21 %).

Tirzepatide dose-dependently increased the likelihood of regression of NASH (MASH), with the maximum effect at a dose of 15 mg per week, and a decrease in fibrosis was also significant, but without a clear dose dependence [12]. Larger,

longer-term studies are needed to further evaluate the efficacy and safety of tirzepatide in the treatment of NASH (MASH).

Professors Marina V. Maevskaya and Sergey V. Okovityi shared their opinion on the rational combination of incretin mimetics with UDCA, which occupies a central place in the pharmacotherapy of NAFLD (MASLD) in Russian Clinical Guidelines, having a multifactorial action in reduction of steatosis, liver inflammation and prevention of fibrosis progression, as well as a positive effect on lipid and carbohydrate metabolism, reducing the risk of developing cardiovascular disease and its complications. UDCA is used as monotherapy and in various combinations.

The important role of UDCA in NAFLD (MASLD) treatment is to stimulate autophagy via adenosine monophosphate-activated protein kinase, a cellular nutrient sensor. Autophagy is a lysosomal degradation pathway, two types of which, macroautophagy and chaperone-mediated autophagy, regulate a number of critical cellular functions related to the pathophysiological phenomena underlying NAFLD (MASLD).

The intensity of autophagy in the liver decreases in conditions predisposing to the development of NAFLD (MASLD), such as obesity and aging, which suggests that loss of autophagy function may be involved in the pathogenesis of NAFLD (MASLD). Autophagy has important metabolic effects, including increased insulin sensitivity and degradation of intracellular lipids that regulate the development of steatosis. Impaired autophagy in NAFLD (MASLD) and its potentially beneficial effects in preventing steatosis, hepatocyte damage, and inflammation suggest that autophagy-enhancing therapy may be effective for this disease. Professor Sergey V. Okovityi emphasized the key biological effects of UDCA, which underlie its effectiveness shown in clinical trials for NAFLD (MASLD): stimulation of autophagy, interaction with the G protein-coupled bile acid receptor (TGR5/GPBAR1) and insulin receptors, suppression of apoptosis [13–17].

Recent studies have demonstrated new features of UDCA action: it serves as a partial agonist of farnesoid X receptors (FRX) and a modulator of cytochrome P450 -hydroxylase (CYP4A14) activity. It is important that UDCA enhances endogenous secretion of GLP-1 through TGR5/GPBAR1, which is relevant for the treatment of NAFLD as a systemic disease and better control of body weight and carbohydrate metabolism [18]. Professor Sergey V. Okovityi summarized the mechanisms of UDCA action in NAFLD (MASLD) in Figure 2.

The combination of UDCA with incretin mimetics offers several advantages, which relate

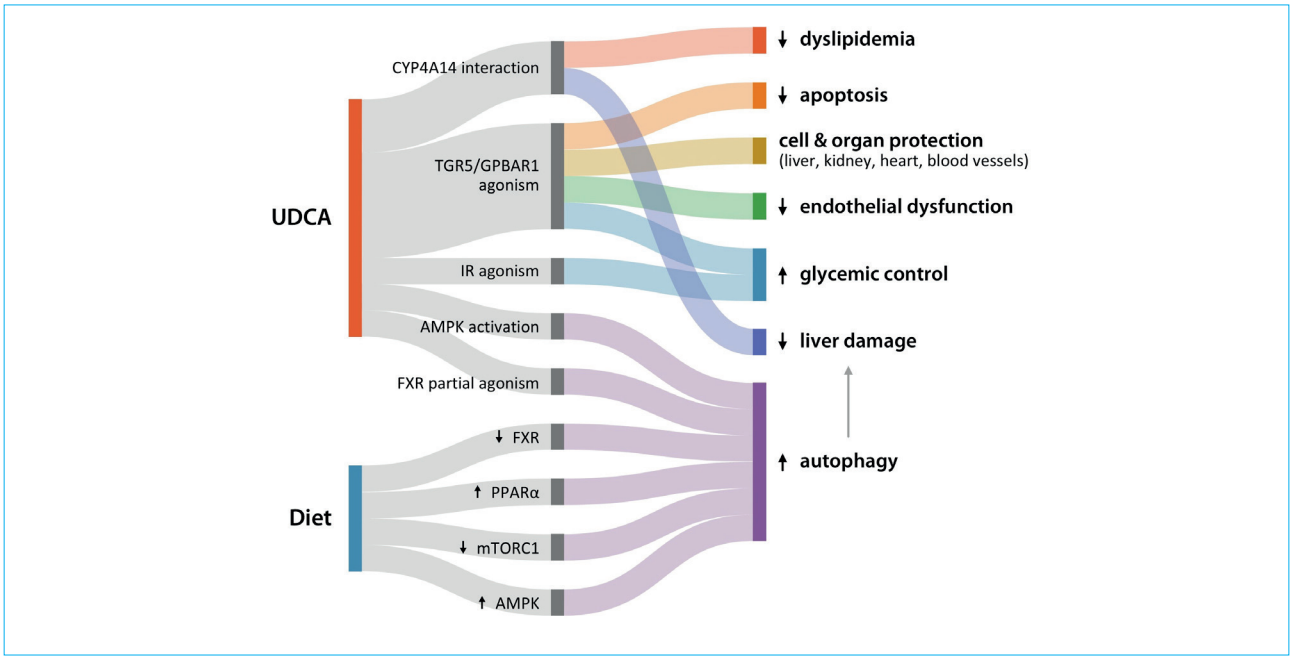

Figure 2. Mechanism of UDCA action in NAFLD (MASLD) (S.V. Okovityi, V.A. Prikhodko, 2025): TGR5/GPBAR1 — G protein-coupled bile acid receptor; IR — insulin receptor; FXR — farnesoid X receptor; PPAR — peroxisome proliferator-activated receptor; mTORC1 — mechanistic target of rapamycin; AMPK — adenosine monophosphate-activated protein kinase; CYP4A14 — monooxygenase catalyzing the omega-hydroxylation of medium-chain fatty acids and arachidonic acid

both to the potentiation of their positive effects and prevention of adverse events, in particular, the formation of sludge and concrements in the gallbladder during rapid weight loss in a patient.

Carbohydrate metabolism disorders in patients with NAFLD (MASLD) require special attention. Ekaterina A. Troshina, Corresponding Member of RAS, discussed this in her presentation, drawing the experts' attention to the common pathogenesis of NAFLD (MASLD), DM2, and obesity. Importantly, the risk of developing NAFLD (MASLD) in young women increases by 50 %, if they had gestational diabetes, and this also applies to their children. These women also have a high risk of developing DM2 [19, 20].

During the work of the expert council, Maria Yu. Nadinskaia, Associate Professor at the Sechenov University, for the first time presented the results of a secondary analysis of the international, multicenter, non-comparative USPEH study.

The results of the initial analysis of the USPEH study were published in 2021 [21]. It included 174 patients with NAFLD (MASLD) without cardiovascular complications. All patients were advised to follow a diet and increase physical activity; UDCA therapy was administered at a dose of 15 mg/kg/day for 6 months.

The primary analysis showed that UDCA use was associated with statistically significant reductions in liver enzymes, total cholesterol and triglyceride levels, as well as a decrease in the hepatic steatosis index (Fatty Liver Index, FLI). No significant effect on liver fibrosis indices (Fibrosis-4 Index, FIB-4 and the NAFLD Fibrosis Score, NFS) was observed. It is important to emphasize that there was no progression of liver fibrosis during treatment.

Furthermore, the primary analysis demonstrated a decrease in carotid intima-media thickness and a reduction in the predicted 10-year risk of atherosclerotic cardiovascular disease in women, as measured by the ASCVD calculator.

Thus, it was shown that UDCA therapy in NAFLD (MASLD) has an effect on liver function parameters and cardiovascular risk factors.

In 2023, a new nomenclature for FLD was proposed, and the diagnostic criteria for MASLD were updated [1]. Analysis of the patient population included in the USPEH study revealed that all patients met the criteria for MASLD: all had liver steatosis detected by ultrasonography and cardiometabolic risk factors, with 75 % of patients having three or more risk factors and 25 % having one or two.

For the purposes of a secondary analysis, patients were stratified using the classical concept of NAFLD (MASLD) progression into subgroups with normal (< 40 U/L) and elevated (≥ 40 U/L) serum ALT levels, conventionally designated as "steatosis" and "steatohepatitis", respectively. In these subgroups, the predicted risk of atherosclerotic cardiovascular disease was assessed using the ASCVD calculator.

Both subgroups were initially found to have an increased predicted 10-year risk of atherosclerotic cardiovascular diseases compared to the calculated values. Furthermore, the risk in the "steatohepatitis" subgroup was statistically significantly higher. Following 6 months of UDCA therapy at a dose of 15 mg/kg/day, a reduction in this predicted risk was observed in both subgroups.

Thus, the results of the secondary analysis complement previously published data and indicate the potential for modification of cardiovascular risk in patients with NAFLD (MASLD), including those with normal ALT levels, who may remain outside the focus of clinical observation. The results shown are of interest from the point of view of comprehensive management of patients with this category of metabolic disorders and require further confirmation in prospective studies.

Therefore, UDCA is a remarkable example of how a deep understanding of molecular and cellular elements has transformed a natural substance into a powerful and multitasking drug with an ever-expanding field of application. Its study continues, and new discoveries may await.

Professor Oxana Yu. Zolnikova, presented that a study conducted by Cao Xinglu, a PhD student at the department, showed that patients with NAFLD have reduced absolute amounts of all SCFAs in the faeces and these changes worsen as the disease progresses into liver cirrhosis, correlating with the main clinical and laboratory characteristics [22, 23]. Teaching Assistant M.S. Reshetova analyzed the metabolomic profile in fatty liver disease of various origins and established characteristic metabolomic signatures [24, 25]. Six distinct metabolomic factors (MF) were identified:

- MF I — acetylcarnitine (C2), oxo-palmitoylcarnitine, oleoylcarnitine and linoleoylcarnitine, uridine, metanephrine, total and asymmetric dimethylarginine;
- MF II — choline, serine, glycine, aspartic acid, phenylalanine, dimethylglycine, and taurine;
- MF III — lauroylcarnitine (C12), dodecenoylcarnitine (C12-1), tetradecenoylcarnitine (C14-1), decanoylcarnitine (C10), caproylcarnitine (C6), myristoylcarnitine (C14);
- MF IV — valerylcarnitine (C5), propionylcarnitine (C3), tiglylcarnitine (C5-1), carnitine (C0), butyrylcarnitine (C4), valine, leucine, lysine, alanine;

• MF V — vitamin B5 (pantothenic acid), cortisol, xanthurenic acid, stearoylcarnitine (C18);
• MF VI — tyrosine, epinephrine, metanephrine.

The specified factors differentiated patients at various stages of the disease and reflected the severity of key metabolic disorders: mitochondrial dysfunction, insulin resistance, anabolic resistance, systemic inflammation, impaired autophagy and microbiota.

Associate Professor Roman V. Maslennikov briefly described changes in the composition of the intestinal microbiota in NAFLD (MASLD) based on the data from a recently published systematic review [26]. This disease is characterized by a decreased intestinal content of beneficial bacteria from the *Ruminococcaceae* family, which produce SCFAs with anti-inflammatory potential, and an increased amount of endotoxin-bearing *Escherichia*. Therefore, the intestinal microbiota in NAFLD (MASLD) has a pronounced pro-inflammatory potential and produces a large amount of endotoxin, which enters the liver tissue with portal vein blood, stimulating the development of NASH (MASH). R.V. Maslennikov also presented an innovative solution of Russian scientists: multiplex PCR kits for the main taxa of the intestinal microbiota, which will allow for the implementation of this analysis in real clinical practice and personalized pro- and prebiotic therapy of intestinal dysbiosis in NAFLD (MASLD) and other diseases.

Conclusion

At the end of the meeting, the experts summarized the results.

• NAFLD (MASLD) refers to a widespread non-communicable disease with a progressively increasing incidence in Russia and worldwide.

• NAFLD (MASLD) is a multisystem disease associated with two main risks: progression of the pathological process in the liver to cirrhosis and hepatocellular carcinoma and the risk of cardiometabolic diseases and their complications. This necessitates a preventive approach to managing patients with NAFLD (MASLD) and integrating the treatment of this disease into the overall strategy of managing the metabolic health of the population.

• The activity of the disease determines the rate of fibrosis progression, and the fibrosis stage determines the risk of adverse outcomes (severe fibrosis, liver cirrhosis, and hepatocellular carcinoma).

• The use of non-invasive tests, in particular FIB-4, allows for effective risk stratification of adverse outcomes of NAFLD (MASLD) and optimized patient routing.

• It is necessary to develop and validate in the Russian Federation a more advanced scale for assessing the risk of developing cardiovascular diseases and their complications, including NAFLD as an independent parameter.

• Treatment of NAFLD should be comprehensive and integrated into the overall concept of metabolic health. This includes two goals: preventing the development of liver cirrhosis and hepatocellular carcinoma, on the one hand, and cardiometabolic complications, on the other.

• Treatment of NAFLD includes lifestyle modification and pharmacotherapy. Preference should be given to drugs with pleiotropic action and their combinations, which have a comprehensive effect on metabolic dysfunction and possess hepatotropic effects. UDCA has shown its effectiveness in the monotherapy for NAFLD, both in reducing steatosis and inflammation and in stabilizing fibrosis. Its ability as a signalling molecule to influence lipid and carbohydrate metabolism is important in the treatment of NAFLD (MASLD) as a systemic disease. The combination of UDCA with incretin mimetics appears very promising in terms of synergy and the prevention of cholelithiasis and requires in-depth study.

• The experts decided to develop algorithms for the diagnosis and personalized treatment for NAFLD (MASLD) for the Russian medical community.

Academician Vladimir T. Ivashkin shared his impressions after the expert council, noting that it had set many urgent and complex tasks. To create simple and well-founded algorithms for physicians, it is necessary to clearly understand the capabilities and limitations of primary care physicians, gastroenterologists-hepatologists, and actively involve endocrinologists in patient treatment. The declared principle of a patient-centered approach requires physicians to have a deep understanding of the pathogenesis of the pathological process in the liver, its relationship with cardiovascular and endocrine diseases, and developed clinical thinking for comprehensive patient management. We can say that a renaissance of the Hippocratic teaching has come: “A doctor treats a patient’s illness”.

The work conducted by the experts will undoubtedly form the basis for updated clinical guidelines, medical practice, and new scientific research.

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Information about the authors

Marina V. Maevskaya* — Dr. Sci. (Med.), Professor, I I.M. Sechenov First Moscow State Medical University (Sechenov University); Vice-President of the Russian Scientific Liver Society.
Contact information: maevskaya_m_v@staff.sechenov.ru;
119991, Moscow, GSP-1, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0000-0001-8913-140X

Sergey V. Okovityi — Dr. Sci. (Med.), Professor, Head of the Department of Pharmacology and Clinical Pharmacology, Saint Petersburg State Chemical and Pharmaceutical University.
Contact information: sergey.okovity@pharminnotech.com;
197022, St. Petersburg, Professora Popova str., 14A.
ORCID: https://orcid.org/0000-0003-4294-5531

Igor G. Bakulin — Dr. Sci. (Med.), Professor, Head of the Department of Propaedeutics of Internal Medicine, Gastroenterology and Dietetics named after S.M. Ryss, North-Western State Medical University named after I.I. Mechnikov.
Contact information: igbakulin@yandex.ru;
191015, St. Petersburg, Kirochnaya str., 41.
ORCID: https://orcid.org/0000-0002-6151-2021

Alan S. Bidzhiev — Gastroenterologist, Center for Hepatopancreatobiliary Surgery, Oncohepatology and Organ Transplantation, National Scientific Oncology Center.
Contact information: alan123145@gmail.com;
010017, Republic of Kazakhstan, Astana, Kerey and Zhanibek Khandar Street, 3/2.

Dmitriy V. Glushenkov — Cand. Sci. (Med), Head of the Medical and Diagnostic Department No. 3 with the Functions of the Admission Department, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: glushenkov_d_v@staff.sechenov.ru;
119991, Moscow, GSP-1, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0009-0001-4663-8275

Сведения об авторах

Маевская Марина Викторовна* — доктор медицинских наук, профессор, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет); вице-президент Российского общества по изучению печени.
Контактная информация: maevskaya_m_v@staff.sechenov.ru;
119991, г. Москва, ГСП-1, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0000-0001-8913-140X

Оковитый Сергей Владимирович — доктор медицинских наук, профессор, заведующий кафедрой фармакологии и клинической фармакологии, ФГБОУ ВО «Санкт-Петербургский государственный химико-фармацевтический университет» Министерства здравоохранения Российской Федерации.
Контактная информация: sergey.okovity@pharminnotech.com;
197022, г. Санкт-Петербург, ул. Профессора Попова, 14а.
ORCID: https://orcid.org/0000-0003-4294-5531

Бакулин Игорь Геннадьевич — доктор медицинских наук, профессор, заведующий кафедрой пропедевтики внутренних болезней, гастроэнтерологии и диетологии им. С.М. Рысса, ФГБОУ ВО «Северо-Западный государственный медицинский университет им. И.И. Мечникова» Министерства здравоохранения Российской Федерации.
Контактная информация: igbakulin@yandex.ru;
191015, г. Санкт-Петербург, ул. Кирочная, 41.
ORCID: https://orcid.org/0000-0002-6151-2021

Биджиев Алан Салихович — врач-гастроэнтеролог, Центр гепатопанкреатобилиарной хирургии, онкогепатологии и трансплантации органов, ТОО «Национальный научный онкологический центр».
Контактная информация: alan123145@gmail.com;
010017, Республика Казахстан, г. Астана, ул. Керей и Жанибек хандар, 3/2.

Глушенков Дмитрий Владимирович — кандидат медицинских наук, заведующий лечебно-диагностическим отделением № 3 с функциями приемного отделения Университетской клинической больницы № 2, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: glushenkov_d_v@staff.sechenov.ru;
119991, г. Москва, ГСП-1, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0009-0001-4663-8275

* Corresponding author / Автор, ответственный за переписку

Olesya Yu. Gurova — Cand. Sci. (Med.), Chief Physician, Professorial Clinic of Endocrinology and Diabetes.
Contact information: gurova79@inbox.ru;
119334, Moscow, Andreevskaya emb., 1, build. 1.
ORCID: https://orcid.org/0000-0003-2576-4421

Alexander Yu. Gorshkov — Cand. Sci. (Med.), Deputy Director for Scientific and Outpatient Clinical Work, National Medical Research Center for Therapy and Preventive Medicine.
Contact information: AGorshkov@gnicpm.ru;
101990, Moscow, Petroverigsky Lane, 10, build. 3.
ORCID: https://orcid.org/0000-0002-1423-214X

Venera A. Zhakipbekova — Cand. Sci. (Med.), Docent, Associate Professor, Karaganda Medical University.
Contact information: Zhakipbekova@qmu.kz;
100000, Republic of Kazakhstan, Karaganda, Gogolya str., 40.
ORCID: https://orcid.org/0000-0003-2721-7907

Oxana Yu. Zolnikova — Dr. Sci. (Med.), Professor at the Department of Internal Disease Propaedeutics, Gastroenterology and Hepatology, Leading Researcher at the Scientific and Technological Institute of Metabolic Health, I.M. Sechenov First Moscow State Medical University (Sechenov University); Chief External Gastroenterologist of the Central Federal District of the Russian Federation.
Contact information: zolnikova_o_yu@staff.sechenov.ru;
119991, Moscow, GSP-1, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0000-0002-6701-789X

Mirvasit M. Karimov — Dr. Sci. (Med.), Professor, Chief Gastroenterologist of Ministry of Health of the Republic of Uzbekistan, Head of the Gastroenterology Department, Republican Specialized Scientific and Practical Medical Center for Therapy and Medical Rehabilitation.
Contact information: mirvasit61@rambler.ru;
100052, Republic of Uzbekistan, Tashkent, Osiyo str., 4.
ORCID: https://orcid.org/0000-0002-1731-0916

Yulia V. Kotovskaya — Dr. Sci. (Med.), Professor, Deputy Director for Scientific Affairs at the Russian Clinical and Research Center of Gerontology, N.I. Pirogov Russian National Research Medical University; Head of the Department for the Implementation of Functions of the National Medical Research Center for Geriatrics.
Contact information: kotovskaya_yv@rgnkc.ru;
129226, Moscow, Pervaya Leonova str., 16.
ORCID: https://orcid.org/0000-0002-1628-5093

Igor V. Maev — Dr. Sci. (Med.), Professor, Academician of the Russian Academy of Science, Head of the Department of Internal Disease Propaedeutics and Gastroenterology, Faculty of Medicine, Russian University of Medicine.
Contact information: igormaev@rambler.ru;
127473, Moscow, Delegatskaya str., 20, build. 1.
ORCID: https://orcid.org/0000-0001-6114-564X

Roman V. Maslennikov — Cand. Sci. (Med.), Associate Professor at the Department of Internal Disease Propaedeutics, Gastroenterology and Hepatology, Leading Researcher at the Scientific and Technological Institute of Metabolic Health,

Гурова Олеся Юрьевна — кандидат медицинских наук, главный врач, ООО «Профессорская клиника эндокринологии и диабета».
Контактная информация: gurova79@inbox.ru;
119334, г. Москва, Андреевская набережная, 1, к. 1.
ORCID: https://orcid.org/0000-0003-2576-4421

Горшков Александр Юрьевич — кандидат медицинских наук, заместитель директора по научной и амбулаторно-поликлинической работе, ФГБУ «Национальный медицинский исследовательский центр терапии и профилактической медицины» Министерства здравоохранения Российской Федерации.
Контактная информация: AGorshkov@gnicpm.ru;
101990, г. Москва, Петроверигский пер., 10, стр. 3.
ORCID: https://orcid.org/0000-0002-1423-214X

Жакипбекова Венера Амантаевна — кандидат медицинских наук, доцент, ассоциированный профессор, НАО «Карагандинский медицинский университет».
Контактная информация: Zhakipbekova@qmu.kz;
100000, Республика Казахстан, г. Караганда, ул. Гоголя, 40.
ORCID: https://orcid.org/0000-0003-2721-7907

Зольникова Оксана Юрьевна — доктор медицинских наук, профессор кафедры пропедевтики внутренних болезней, гастроэнтерологии и гепатологии; ведущий научный сотрудник Научно-технологического института метаболического здоровья, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет); главный внештатный гастроэнтеролог Центрального федерального округа.
Контактная информация: zolnikova_o_yu@staff.sechenov.ru;
119991, г. Москва, ГСП-1, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0000-0002-6701-789X

Каримов Мирвасит Мирвасикович — доктор медицинских наук, профессор, главный гастроэнтеролог Министерства здравоохранения Республики Узбекистан, руководитель отдела гастроэнтерологии, Республиканский специализированный научно-практический медицинский центр терапии и медицинской реабилитации.
Контактная информация: mirvasit61@rambler.ru;
100052, Республика Узбекистан, г. Ташкент, ул. Осие, 4.
ORCID: https://orcid.org/0000-0002-1731-0916

Котовская Юлия Викторовна — доктор медицинских наук, профессор, заместитель директора по научной работе ОСП Российский геронтологический научно-клинический центр, ФГАОУ ВО «Российский национальный исследовательский медицинский университет им. Н.И. Пирогова» Министерства здравоохранения Российской Федерации; начальник управления по реализации функций НМИЦ по гериатрии.
Контактная информация: kotovskaya_yv@rgnkc.ru;
129226, г. Москва, ул. 1-я Леонова, 16.
ORCID: https://orcid.org/0000-0002-1628-5093

Маев Игорь Вениаминович — доктор медицинских наук, профессор, академик РАН, заведующий кафедрой пропедевтики внутренних болезней и гастроэнтерологии лечебного факультета, ФГБОУ ВО «Российский университет медицины» Министерства здравоохранения Российской Федерации.
Контактная информация: igormaev@rambler.ru;
127473, г. Москва, ул. Делегатская, 20, стр. 1.
ORCID: https://orcid.org/0000-0001-6114-564X

Масленников Роман Вячеславович — кандидат медицинских наук, доцент кафедры пропедевтики внутренних болезней, гастроэнтерологии и гепатологии, ведущий научный сотрудник Научно-технологического института метаболи-

I.M. Sechenov First Moscow State Medical University (Sechenov University)
Contact information: maslennikov_r_v@staff.sechenov.ru;
119991, Moscow, GSP-1, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0000-0001-7513-1636

Maria Yu. Nadinskaia — Cand. Sci. (Med.), Associate Professor of the Department of Propaedeutics of Internal Diseases, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: nadinskaya_m_yu@staff.sechenov.ru;
119991, Moscow, GSP-1, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0000-0002-1210-2528

Marina F. Osipenko — Dr. Sci. (Med.), Professor, Head of the Department of Internal Diseases Propedeutics, Novosibirsk State Medical University.
Contact information: ngma@bk.ru;
630091, Novosibirsk, Krasnyi ave., 52.
ORCID: https://orcid.org/0000-0002-5156-2842

Karina L. Raikhelson — Dr. Sci. (Med.), Docent, Professor of Scientific and Educational Center of Gastroenterology and Hepatology of the Medical Institute, Saint Petersburg State University; Professor of Department of General Medical Practice (Family Medicine), Academician I.P. Pavlov First Saint Petersburg State Medical University.
Contact information: kraikhelson@mail.ru;
199106, Saint Petersburg, 21-ya Liniya Vasilyevskogo Ostrova, 8a.
ORCID: https://orcid.org/0000-0002-8821-6142

Balnur P. Sultanova — MD, PhD, 1st Category Endocrinologist, Acting Associate Professor of the Department of Endocrinology, Research Institute of Cardiology and Internal Medicine.
Contact information: balnur4ik@gmail.com;
050000, Republic of Kazakhstan, Almaty, Aiteke Bi Street, 120.
ORCID: https://orcid.org/0000-0003-1974-4248

Olga N. Tkacheva — Dr. Sci. (Med.), Professor, Corresponding Member of the Russian Academy of Sciences, N.I. Pirogov Russian National Research Medical University.
Contact information: tkacheva@rgnkc.ru;
117997, Moscow, Ostrovityanova str., 1.
ORCID: https://orcid.org/0000-0002-4193-688X

Ekaterina A. Troshina — Dr. Sci. (Med.), Professor, Corresponding Member of the Russian Academy of Sciences, Director of the Institute of Clinical Endocrinology, Deputy Director, Endocrinology Research Centre.
Contact information: troshina@inbox.ru;
117292, Moscow, Dmitrya Ulyanovs str., 11.
ORCID: https://orcid.org/0000-0002-8520-8702

Zamira Yu. Khalimova — Dr. Sci. (Med.), Deputy Director for Science, Head of the Department of Neuroendocrinology with Pituitary Neurosurgery, Republican Specialized Scientific and Practical Medical Center of Endocrinology named after Acad. Y.Kh. Turakulov.

ческого здоровья, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: maslennikov_r_v@staff.sechenov.ru;
119991, г. Москва, ГСП-1, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0000-0001-7513-1636

Надинская Мария Юрьевна — кандидат медицинских наук, доцент кафедры пропедевтики внутренних болезней, гастроэнтерологии и гепатологии, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: nadinskaya_m_yu@staff.sechenov.ru;
119991, г. Москва, ГСП-1, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0000-0002-1210-2528

Осипенко Марина Федоровна — доктор медицинских наук, профессор, заведующий кафедрой пропедевтики внутренних болезней, ФГБОУ ВО «Новосибирский государственный медицинский университет» Министерства здравоохранения Российской Федерации.
Контактная информация: ngma@bk.ru;
630091, г. Новосибирск, Красный просп., 52.
ORCID: https://orcid.org/0000-0002-5156-2842

Райхельсон Карина Леонидовна — доктор медицинских наук, доцент, профессор Научно-клинического и образовательного центра гастроэнтерологии и гепатологии медицинского института, ФГБОУ ВО «Санкт-Петербургский государственный университет»; профессор кафедры общей врачебной практики (семейной медицины), ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. академика И.П. Павлова» Министерства здравоохранения Российской Федерации.
Контактная информация: kraikhelson@mail.ru;
199106, г. Санкт-Петербург, 21-я линия Васильевского острова, 8а.
ORCID: https://orcid.org/0000-0002-8821-6142

Султанова Балнур Пердекожаевна — MD, PhD, врач-эндокринолог 1-й категории, и.о. доцента кафедры эндокринологии, АО «НИИ кардиологии и внутренних болезней».
Контактная информация: balnur4ik@gmail.com;
050000, Республика Казахстан, г. Алматы, ул. Айтеке Би, 120.
ORCID: https://orcid.org/0000-0003-1974-4248

Ткачева Ольга Николаевна — доктор медицинских наук, профессор, член-корреспондент РАН, ФГАОУ ВО «Российский национальный исследовательский медицинский университет им. Н.И. Пирогова» Министерства здравоохранения Российской Федерации.
Контактная информация: tkacheva@rgnkc.ru;
117997, г. Москва, ул. Островитянова, 1.
ORCID: https://orcid.org/0000-0002-4193-688X

Трошина Екатерина Анатольевна — доктор медицинских наук, профессор, член-корреспондент РАН, директор Института клинической эндокринологии, заместитель директора, ФГБУ «Национальный медицинский исследовательский центр эндокринологии им. академика И.И. Дедова» Министерства здравоохранения Российской Федерации.
Контактная информация: troshina@inbox.ru;
117292, г. Москва, ул. Дмитрия Ульянова, 11.
ORCID: https://orcid.org/0000-0002-8520-8702

Халимова Замира Юсуповна — доктор медицинских наук, заместитель директора по науке, руководитель отдела нейроэндокринологии с нейрохирургией гипофиза, Республиканский специализированный научно-практический медицинский центр эндокринологии им. академика Е.Х. Туракулова.

Contact information: zam-nar777@mail.ru;
100125, Republic of Uzbekistan, Tashkent, Mirzo Ulugbek Ave., 56.
ORCID: https://orcid.org/0000-0002-3041-0268

Gulnoz A. Khamidullaeva — Dr. Sci. (Med.), Professor, Head of the Department of Arterial Hypertension, Republican Specialized Scientific and Practical Medical Center of Cardiology.
Contact information: gulnoz0566@mail.ru;
100052, Republic of Uzbekistan, Tashkent, Osiyo str., 4.
ORCID: https://orcid.org/0000-0001-9671-9287

Anna F. Sheptulina — Cand. Sci. (Med.), Leading Researcher, Head of the Laboratory of Experimental and Preventive Gastroenterology, National Medical Research Center for Therapy and Preventive Medicine.
Contact information: asheptulina@gnicpm.ru;
101000, Moscow, Petroverginskiy lane, 10, build. 3.
ORCID: https://orcid.org/0000-0001-7230-0780

Elena N. Shirokova — Dr. Sci. (Med.), Professor at the Department of Internal Disease Propaedeutics, Gastroenterology and Hepatology, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: shirokova_e_n@staff.sechenov.ru;
119991, Moscow, GSP-1, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0000-0002-6819-0889

Oksana M. Drapkina — Dr. Sci. (Med.), Professor, Academician of the Russian Academy of Sciences, Director, National Medical Research Center of Preventive Medicine; President of the Russian Society for the Prevention of Non-Communicable Diseases; President of the Russian Scientific Medical Society of Therapists; Chief Specialist in Therapy and General Medical Practice.
Contact information: drapkina@bk.ru;
101000, Moscow, Petroverginskiy lane, 10, build. 3.
ORCID: https://orcid.org/0000-0002-4453-8430

Vladimir T. Ivashkin — Dr. Sci. (Med.), Professor, Academician of the Russian Academy of Sciences, Head of the Department of Internal Disease Propaedeutics, Gastroenterology and Hepatology of the N.V. Sklifosovsky Institute of Clinical Medicine, Head of the Scientific and Technological Institute of Metabolic Health, I.M. Sechenov First Moscow State Medical University (Sechenov University).
Contact information: ivashkin_v_t@staff.sechenov.ru;
119991, Moscow, GSP-1, Trubetskaya str., 8/2.
ORCID: https://orcid.org/0000-0002-6815-6015

Контактная информация: zam-nar777@mail.ru;
100125, Республика Узбекистан, г. Ташкент, просп. Мирзо Улугбека, 56.
ORCID: https://orcid.org/0000-0002-3041-0268

Хамидуллаева Гулноз Абдусаттаровна — доктор медицинских наук, профессор, руководитель отдела артериальной гипертонии, Республиканский специализированный научно-практический медицинский центр кардиологии.
Контактная информация: gulnoz0566@mail.ru;
100052, Республика Узбекистан, г. Ташкент, ул. Осие, 4.
ORCID: https://orcid.org/0000-0001-9671-9287

Шептулина Анна Фароковна — кандидат медицинских наук, ведущий научный сотрудник, руководитель лаборатории экспериментальной и профилактической гастроэнтерологии, ФГБУ «Национальный медицинский исследовательский центр терапии и профилактической медицины» Министерства здравоохранения Российской Федерации.
Контактная информация: asheptulina@gnicpm.ru;
101000, г. Москва, Петроверигский пер., 10, стр. 3.
ORCID: https://orcid.org/0000-0001-7230-0780

Широкова Елена Николаевна — доктор медицинских наук, профессор кафедры пропедевтики внутренних болезней, гастроэнтерологии и гепатологии, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет).
Контактная информация: shirokova_e_n@staff.sechenov.ru;
119991, г. Москва, ГСП-1, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0000-0002-6819-0889

Драпкина Оксана Михайловна — доктор медицинских наук, профессор, академик РАН, директор, ФГБУ «Национальный медицинский исследовательский центр терапии и профилактической медицины» Министерства здравоохранения Российской Федерации; президент Российского общества профилактики неинфекционных заболеваний; президент Российского научного медицинского общества терапевтов; главный внештатный специалист по терапии и общей врачебной практике.
Контактная информация: drapkina@bk.ru;
101000, г. Москва, Петроверигский пер., 10, стр. 3.
ORCID: https://orcid.org/0000-0002-4453-8430

Ивашкин Владимир Трофимович — доктор медицинских наук, профессор, академик РАН, заведующий кафедрой пропедевтики внутренних болезней, гастроэнтерологии и гепатологии Института клинической медицины им. Н.В. Склифосовского, директор Научно-технологического института метаболического здоровья, ФГАОУ ВО «Первый Московский государственный медицинский университет им. И.М. Сеченова» Министерства здравоохранения Российской Федерации (Сеченовский Университет); Главный внештатный специалист гастроэнтеролог Министерства здравоохранения Российской Федерации.
Контактная информация: ivashkin_v_t@staff.sechenov.ru;
119991, г. Москва, ГСП-1, ул. Трубецкая, 8, стр. 2.
ORCID: https://orcid.org/0000-0002-6815-6015

Authors' contributions

All the authors contributed with data collection and analysis, writing of the manuscript, approving final version and its publication.

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