TASHKENT STATE MEDICAL UNIVERSITY

ISSN 2181-3485

MEDICAL JOURNAL OF YOUNG SCIENTISTS

YOSH OLIMLAR TIBBIYOT JURNALI

№ 17 (03), 2026

ABOUT JOURNAL
Since 2022, the journal has been included in the current List of peer-reviewed scientific publications.

QUALITY
Is a peer-reviewed publication, registered as a media outlet, has an ISSN

TOSHKENT DAVLAT TIBBIYOT UNIVERSITET
“YOSH OLIMLAR TIBBIYOT JURNALI”

TASHKENT STATE MEDICAL UNIVERSITY
«MEDICAL JOURNAL OF YOUNG SCIENTISTS»

ТАШКЕНТСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
«МЕДИЦИНСКИЙ ЖУРНАЛ МОЛОДЫХ УЧЕНЫХ»

IXTISOSLASHUVI: “TIBBIYOT SOHASI”

ISSN: 2181-3485

Jurnal O‘zbekiston Respublikasi Prezidenti Administratsiyasi huzuridagi Axborot va ommaviy kommunikatsiyalar agentligi tomonidan 2022-yil 18-maydagi 1601-raqami bilan ro‘yxatga olingan.

№ 17 (03), 2026

“Yosh olimlar tibbiyot jurnali” O‘zbekiston Respublikasi Oliy ta’lim, fan va innovatsiyalar vazirligi huzuridagi Oliy attestatsiya komissiyasi Rayosatining 2023-yil 5-maydagi 337/6-son qarori bilan tibbiyot fanlari bo‘yicha dissertatsiyalar asosiy ilmiy natijalarini chop etish tavsiya etilgan milliy ilmiy nashrlar ro‘yxatiga kiritilgan.

Решением Президиума Высшей аттестационной комиссии при Министерстве высшего образования, науки и инноваций Республики Узбекистан от 5 мая 2023 г. № 337/6 «Медицинский журнал молодых ученых» внесен в перечень национальных научных изданий, рекомендованных для публикации основных научных результатов диссертаций по медицинским наукам.

ГЛАВНЫЙ РЕДАКТОР
Боймурадов Шухрат Абдужалилович

ЗАМЕСТИТЕЛЬ ГЛАВНОГО РЕДАКТОРА
Шайхова Гули Исламовна

ОТВЕТСТВЕННЫЙ СЕКРЕТАРЬ
Алимухамедов Дилшод Шавкатович

РЕДАКЦИОННАЯ КОЛЛЕГИЯ:

д.м.н., профессор Азизова Ф.Л.
профессор Азизова Ф.Х.
профессор Аллаева М.Ж.
профессор Даминова Ш.Б.
профессор Каримжонов И.А.
профессор Каримова М.Х.
профессор Набиева Д.А.
профессор Нажмутдинова Д.Қ.
профессор Кирюшин В.А.
профессор Нуриллаева Н.М.
профессор Тешаев Ш.Ж.
профессор Хайдаров Н.К.
профессор Хакимов М.Ш.
профессор Хасанов У.С.
д.м.н. Худойкулова Г.К.
профессор Эрматов Н.Ж.
профессор Маматкулов Б.М.

ЧЛЕНЫ РЕДАКЦИОННОГО СОВЕТА

Агишев И.А.	д.м.н. (Казахстан)
Аглиулин Д.Р.	к.м.н. (Россия)
Алейник В.А.	профессор (Андижан)
Билолов Э.Н.	профессор (Ташкент)
Ганиев А.А.	д.м.н. (Ташкент)
Инаков Ш.А.	Ph.D. (Германия)
Искандарова Г.Т.	профессор (Ташкент)
Исраилов Р.	профессор (Ташкент)
Кайнарбаева М.С.	к.м.н. (Казахстан)
Матназарова Г.С.	профессор (Ташкент)
Мирзоева М.Р.	профессор (Бухара)
Мирмансур	Ph.D. (Индия)
Муртазаев С.С.	д.м.н. (Ташкент)
Орипов Ф.С.	д.м.н. (Самарканд)
Отамурадов Ф.А.	д.м.н. (Термез)
Парпиева Д.А.	д.м.н. (Ташкент)
Рахимова Г.С.	д.м.н. (Ташкент)
Рустамова М.Т.	д.м.н. (Ташкент)
Саломова Ф.И.	профессор (Ташкент)
Сидиков А.А.	д.м.н. (Фергана)
Собиров У.Ю.	профессор (Ташкент)
Тажиева З.Б.	Ph.D. (Ургенч)
Ташкенбаева У.А.	профессор (Ташкент)
Хасанова Д.А.	д.м.н. (Бухара)
Хасанова М.А.	д.м.н. (Ташкент)
Хван О.И.	д.м.н. (Ташкент)
Хожиметов А.А.	профессор (Ташкент)
Холматова Б.Т.	д.м.н. (Ташкент)
Чон Хи Ким	Ph.D. (Южная Корея)
Юлдашев Б.С.	д.м.н. (Ургенч)
Шадманов М.А.	Ph.D. (Андижан)

Адрес редакции:
Ташкентский государственный медицинский университет
100109, г. Ташкент, Узбекистан, Алмазарский район, ул. Фараби 2, тел.: +99878-150-7825, факс: +998 78 1507828, электронная почта: mjys.tma@gmail.com

Dehkanova N.T., Oripov B. / Structural components of the mucosa of the ampula of the papilla of vateri 215

Иброхимов М.Ё., Каримов Х.Я. / Ассоциация полиморфизмов генов COL1A1, MMP9 и TGFB1 с риском развития паховых грыж 218

НАУЧНЫЕ ОБЗОРЫ

Рустамова М.Т., Хайруллаева С.С. / Оилавий шифокорлар амалиётида парҳез овқатланишнинг аҳамияти 222

Boykuziyev X.X. / Alimentar semizlik va metabolik sindromda ochlik orqali davolash usulini qo‘llash 225

Абилов П.М. / Современные представления о патофизиологии коронавирусной инфекции COVID-19: обзор литературы 228

Ahrorov A.A., Xolmatova M.A. / Qandli diabet kasalligi natijasida ko‘zda diabetik retinopatiya hamda morfofunksional o‘zgarishlar rivojlanishi to‘g‘risidagi zamonaviy tasavvurlar (adabiyotlar sharxi) 238

Eshkuvatov K.R. / Rivojlangan mamlakatlarda harbiy-akademik litseylarga nomzodlarning kasallanish ko‘rsatkichlari: dolzarb muammolar, xavf omillari va ularni hal etish yo‘llari (adabiyotlar sharhi) 243

Ibragimova G., Shukurova D.Y. / Arterial gipertoniya va dislipidemiyada dori-darmonsiz davolash: global dalillar asosida milliy model 250

Mamarasulova M.T., Ahrorov A.A., Umarova Z.X. / Ko‘krak bezi saratoni klinik bosqichlari va gistopatologik o‘zgarishlar 256

Mirzaakbarova M.E. / Arterial gipertenziyada fiksirlangan preparatlarning afzalligi 260

Мирхамидова С.М. / Искусственный интеллект и анализ данных в медицине 264

Мирзаева С.С. / Экспериментальные модели тиреотоксикоза и значение их в изучение реактивных изменений в органах и системах организма 268

Okbayev M.B. / Metabolik sindrom haqida tarixiy va epidemiologik ma’lumotlar 274

Zayniyev E.I., Sultonov N.N. / Gemodializ olayotgan bemorlarda yurak struktur o‘zgarishlarini korreksiyalashda kardioprotektorlarning o‘rni 278

Шигакова Л.А. / Влияние хронической внутриутробной экспозиции загрязнителей воздуха на формирование иммунного барьера тонкой кишки в раннем постнатальном онтогенезе 282

Нурмухамедов Х.К. / Электромагнитные излучения как фактор риска для здоровья человека 288

Искандарова Г.Т., Уббиниязова А.М. / Микробиологические риски в маникюрных кабинетах и современные методы их профилактики 291

Джуракулов Б.И. / Гистологические и морфологические маркеры воспалительных процессов 297

Ботиралиева А.А., Абдухаликова Н.Ф. / Этиология, патогенез, клинические проявления, диагностика и лечение СПКЯ (синдрома поликистозных яичников) 303

Шухратова С.С., Абдуллаева М.Т. / Роль электрохимии в медицинских технологиях: применение в сенсорах, имплантах и клинических устройствах 311

Хайдаров Ж.Г., Нуриллаева Н.М. / Популяционно-статистический обзор мутаций гена антитромбина III 318

Отажанова А.Н. / Морфогенез печени у потомства при дефиците йода: данные экспериментальных исследований 322

Parpibaeva D.A., Buvamukhamedova N.T. / Modern methods for diagnosing metabolically associated fatty liver disease 326

Fakhriddinova Sh.F. / Scarlet fever: changes in its course and diagnostic approach 330

Mirzayeva G.P. / Genetic predictors of kidney damage in preeclampsia 333

Хайдаров Ш.М. / Тез-тез касалланувчи болаларни соғломлаштиришга гигиеник ва комплекс ёндашув 338

УДК: 616.36-003.826-07

MODERN METHODS FOR DIAGNOSING METABOLICALLY ASSOCIATED FATTY LIVER DISEASE

Parpibaeva Dinora Ayupovna – D.M.Sc.
Buvamukhamedova Nasiba Tahirovna – Ph.D. student,
Tashkent State Medical University (Tashkent, Uzbekistan)

Abstract: *Non-alcoholic fatty liver disease (NAFLD), now referred to as metabolically associated fatty liver disease (MAFLD), is one of the most common liver diseases worldwide, closely linked to the growing obesity epidemic. Despite the growing prevalence of this disease, there is a notable lack of pharmacological agents specifically designed to treat MAFLD. This gap in therapeutic options can be explained by the multifaceted nature of MAFLD, characterized by an incomplete understanding of its underlying mechanisms, a lack of accurate and accessible imaging tools, and the inadequacy of non-invasive biomarkers for effective diagnosis and monitoring.*

In addition, this review highlights existing methods for diagnosing MAFLD and emphasizes the growing importance of non-coding RNAs as promising diagnostic biomarkers. Today, the urgent need for non-invasive biomarkers combined with accurate and cost-effective diagnostic tools cannot be overstated, as they play a key role in identifying early signs of MAFLD progression.

Keywords: *non-alcoholic fatty liver disease (NAFLD); metabolically associated fatty liver disease (MAFLD); non-alcoholic steatohepatitis (NASH); metabolically associated steatohepatitis (MASH); liver; biomarkers*

СОВРЕМЕННЫЕ МЕТОДЫ ДИАГНОСТИКИ МЕТАБОЛИЧЕСКИ АССОЦИИРОВАННОЙ ЖИРОВОЙ БОЛЕЗНИ ПЕЧЕНИ

Парпибаева Динора Аюповна – д.м.н.
Бувамухамедова Насиба Тахировна – базовой докторант
Ташкентский государственный медицинский университет (Ташкент, Узбекистан)

Аннотация: *Неалкогольная жировая болезнь печени (НАЖБП), которую теперь называют метаболически-ассоциированной жировой болезнью печени (МАЖБП), является одним из наиболее распространенных заболеваний печени во всем мире, тесно переплетаясь с нарастающей эпидемией ожирения. Несмотря на растущую распространенность этого заболевания, существует заметное отсутствие фармакологических препаратов, специально разработанных для лечения МАЖБП. Этот пробел в терапевтических возможностях можно объяснить многогранной природой МАЖБП, характеризующейся неполным пониманием лежащих в ее основе механизмов, отсутствием точных и доступных инструментов визуализации, а также неадекватностью неинвазивных биомаркеров для эффективной диагностики и мониторинга.*

Кроме того, в данном обзоре раскрываются существующие методы диагностики МАЖБП, подчеркивается растущее значение некодирующих РНК как перспективных диагностических биомаркеров. На сегодняшний день невозможно переоценить настоятельную потребность в неинвазивных биомаркерах в сочетании с точными и экономически эффективными диагностическими инструментами, поскольку они играют ключевую роль в выявлении ранних признаков прогрессирования МАЖБП.

Ключевые слова*: неалкогольная жировая болезнь печени (НАЖБП); метаболически-ассоциированная жировая болезнь печени (МАЖБП); неалкогольный стеатогепатит (НАСГ); метаболически-ассоциированный стеатогепатит (МАСГ); печень; биомаркеры*

METABOLIK ASSOTSIRLANGAN JIGAR YOG‘ KASALLIGINI TASHXISLASHDA ZAMONAVIY USULLAR

Parpibayeva Dinora Ayupovna – t.f.d.
Buvamuxamedova Nasiba Taxirovna – tayanch doktoranti
Toshkent davlat tibbiyot universiteti (Toshkent, O‘zbekiston)

Annotatsiya: *Jigarning noalkogol yog‘li kasalligi (JNAYK), hozirda metabolik assotsirlangan jigar yog‘li kasalligi (MAJYK) deb ataladi, butun dunyo bo‘ylab eng keng tarqalgan jigar kasalliklaridan biri bo‘lib, o‘sib borayotgan semizlik epidemiyasi bilan chambarchas bog‘liq. Ushbu kasallikning tarqalishi ortib borayotganiga qaramay, MAJYKni davolash uchun maxsus ishlab chiqilgan farmakologik preparatlarning sezilarli yetishmasligi kuzatilmoqda. Terapevtik imkoniyatlardagi bu bo‘shliqni MAJYKning ko‘p qirrali tabiati bilan izohlash mumkin, bu uning asosidagi mexanizmlarni to‘liq tushunmaslik, aniq va qulay vizualizatsiya vositalarining yo‘qligi, shuningdek, samarali diagnostika va monitoring uchun invaziv bo‘lmagan biomarkerlarning yetarli emasligi bilan tavsiflanadi.*

Bundan tashqari, ushbu sharhda MAJYKni tashxislashning mavjud usullari ochib berilgan, istiqbolli diagnostik biomarkerlar sifatida kodlamaydigan RNKlarning ahamiyati ortib borayotgani ta'kidlangan. Bugungi kunda noinvaziv dori vositalariga bo'lgan kuchli ehtiyojni ortiqcha baholab bo'lmaydi.

Kalit so'zlar: *jigar noalkogol yog' kasalligi (JNAYK); metabolik assotsirlangan jigar yog'li kasalligi (MAJYK); noalkogol steatogepatit (NASG); metabolik steatogepatit (MASG); jigar; biomarkerlar*

Introduction:

Non-alcoholic fatty liver disease (NAFLD) is one of the leading causes of liver disease worldwide, and its prevalence is steadily increasing [1]. Today, it costs more than €35 billion per year in the four largest European countries and more than $100 billion in the United States alone [2]. Since the term "non-alcoholic fatty liver disease" (NAFLD) was introduced into the medical reference book, there has been discussion about changing the name to better reflect the disease process and expand the terminology beyond the superficial histopathological similarity to alcoholic liver disease [2,3]. In early 2020, an international group of experts conducted a consensus process to develop a more appropriate term for this disease. Using a two-stage Delphi consensus method, the term "metabolic dysfunction-associated fatty liver disease," or MAFLD, was proposed [4].

MAFLD includes fatty liver disease, characterized by more than 5% of the liver's mass being fat, with the possibility of progression to non-alcoholic steatohepatitis (NASH), characterized by inflammation, cell damage, and increased severity [4]. In addition, its consequences extend beyond the liver and include cardiovascular complications and links to other metabolic disorders such as obesity and type 2 diabetes mellitus (T2DM) [5,6].

The prevalence of MAFLD has grown alongside the obesity epidemic and is estimated to be around 24% of the general population [6]. The prevalence of this disease is particularly high among people with obesity and T2DM: it affects up to 70% of overweight individuals and more than 90% of those classified as obese [7]. It is alarming that MAFLD can also occur in thin people, and ethnic differences further complicate its prevalence and manifestation [9,12].

Children and adolescents have not been spared from this epidemic, as evidenced by the increase in MAFLD incidence in this demographic group [13]. Despite its growing prevalence, MAFLD still lacks specific pharmacological therapy. This therapeutic gap can be explained by the multifaceted nature of MAFLD, characterized by limited understanding of its pathogenetic mechanisms and the lack of accurate non-invasive biomarkers for diagnosis and monitoring.

The purpose of this review is to clarify the complex mechanisms underlying the pathogenesis of MAFLD.

Pathogenesis of MAFLD

Mechanisms of MAFLD pathogenesis

The prevailing model explaining the development of inflammation and progression of MAFLD is the "multiple hit" model, which involves various stress factors [4,5]. Despite advances in understanding the development of hepatic steatosis, the pathogenesis of non-alcoholic steatohepatitis (NASH) remains incomplete. The progression of NASH is influenced by lipotoxicity, endoplasmic reticulum (ER) stress, mitochondrial dysfunction, oxidative stress, endotoxins from the gut, and changes in the composition of the gut microbiota [12,15]. Lipid overload can provoke lipotoxicity, contributing to the development of inflammation, oxidative stress, and fibrosis. A high-calorie diet and sedentary behavior are key factors in the development of MAFLD. The intake of free fatty acids (FFAs) can disrupt the link between respiration and adenosine triphosphate (ATP) production, which leads to worsening MAFLD [8].

Fatty liver disease occurs due to nutrient overload and a sedentary lifestyle. Many factors contribute to the development of inflammation and MAFLD, which ultimately leads to fibrosis.

The pool of fatty acids (FAs) in the liver is formed from dietary fat, lipolysis of adipose tissue, or de novo lipogenesis (DNL) from carbohydrates or other dietary precursors. In the liver, FAs undergo esterification into triglycerides (TGs) and are assembled into very low-density lipoproteins (VLDL) for release into circulation, oxidation in mitochondria (β-oxidation), or storage in lipid droplets (LDs) (<5% of liver weight). During fasting, LDs undergo lipid hydrolysis (via lipolysis and lipophagy) to provide FFA for β-oxidation. In MAFLD, chronic nutrient overload and insulin resistance lead to an imbalance where the influx of FFA into the liver exceeds their utilization through VLDL secretion or β-oxidation. This lipotoxicity leads to impaired LP lipolysis and increased lipid accumulation in LCs, which accelerates the development of hepatic steatosis (>5% of liver weight).

Fatty liver disease causes endoplasmic reticulum (ER) stress, oxidative stress, and activation of Kupffer cells (KCs) to produce inflammatory cytokines that exacerbate inflammation. In addition, lipotoxicity causes mitochondrial dysfunction and disrupts the electron transport chain (ETC) function, leading to the formation of reactive oxygen species (ROS), which in turn exacerbates mitochondrial damage, perpetuating MAFLD.

Inflammatory cytokines and ROS activate hepatic stellate cells (HSCs) to produce excessive extracellular matrix, leading to progressive fibrosis.

Liver fibrosis, which is reversible in its early stages, is the most powerful predictor of mortality in people with metabolic-associated steatohepatitis (MAS). Therefore, accurate staging of fibrosis and differentiation of MAFLD from early fibrosis are key to identifying patients at risk of disease progression. A range of diagnostic methods are used to diagnose and classify MAFLD, including both traditional and

innovative tools such as imaging and biomarkers, each with its own advantages and limitations.

Blood transaminases: Liver function tests, particularly blood tests for transaminases, are widely used, but their reliability in predicting MAFLD progression remains uncertain. Patients with MAFLD have abnormal and normal levels of liver enzymes, with a decrease in alanine aminotransferase (ALT) levels detected in progressive liver disease. Various biomarker panels are used to assess liver fat, including the hepatic steatosis index (HSI), fatty liver index (FLI), Steatotest, and Liver Fat Score (LFS).

Non-invasive scoring systems: Scoring systems such as Fibrosis-4 (FIB-4), MAFLD Fibrosis Score (NFS), Hepamet Fibrosis Score (HFS), and the automated platelet ratio index (APRI) help determine the risk of MAFLD progression but demonstrate modest sensitivity in diagnosing early stages of NASH and fibrosis. However, there is a noticeable dissonance between these scoring systems when applied to the same patient [15].

Liver biopsy: Despite its invasiveness, cost, sampling errors, and associated risks such as bleeding and, although rare, death, liver biopsy remains the gold standard for diagnosing MAFLD. Histologically, MAFLD manifests as hepatic steatosis, swelling, inflammation, with or without fibrosis [14,15]. Although liver biopsy can distinguish NASH from MAFLD, its drawbacks highlight the need for minimally invasive diagnostic alternatives.

In light of the preventive potential of early detection of MAFLD to prevent the development of fibrosis, efforts are continuing to develop minimally invasive imaging tools and biomarkers to assess MAFLD, the risk of progression, and validate treatment in clinical settings.

Imaging methods:

1. Ultrasound (US): Ultrasound is the primary imaging modality for suspected MAFLD, demonstrating the typical hyperechoic appearance of the liver. However, its effectiveness is limited to the detection of moderate or severe steatosis (>20%) and may be affected by severe fibrosis [14]. New approaches, such as computerized assessment of the liver-to-kidney ratio (H/R) and liver attenuation intensity, offer opportunities for early assessment of steatosis [15].

2. Magnetic resonance spectroscopy (MRS): MRS stands out as the most accurate non-invasive method for quantifying liver fat, based on the separation of the proton signal to differentiate between fat and water fractions. Magnetic resonance imaging that determines the proton density of the fat fraction (MRI-PDFF) is a proven tool for assessing liver fat content, with a relative reduction in liver fat content of 30% associated with an improvement in the histological condition of MAFLD. However, limitations include patient discomfort, cost, and limited availability.

3. Transient elastography (TE): TE using a Fibroscan device with an M sensor assesses liver fibrosis, while the controlled attenuation parameter (CAP) simultaneously assesses steatosis. The XL probe improves accuracy in obese individuals, but limitations remain, particularly in predicting significant liver fibrosis in severely obese individuals [12,13]. Fibrotouch liver elastography is becoming a cost-effective and simple alternative for assessing fibrosis in all patients, regardless of obesity status.

4. Magnetic resonance elastography (MRE): MRE assesses liver stiffness, offering an accurate assessment that is independent of BMI. However, its implementation is hampered by cost, availability, and time constraints on examination.

Early-stage MAFLD biomarker

An important milestone in the field of internal organ pathophysiology was the discovery of the mechanism of the canonical Wnt signaling pathway. This pathway, which relies on β-catenin, regulates adipogenesis in a complex manner and triggers cell apoptosis in various organs and tissues of the body [10,13]. In addition, its involvement in the genesis of insulin resistance has been emphasized [9,10].

The Wnt pathway is initiated by the Frizzled family of transmembrane proteins, as discovered by Vivian S.W. Lee et al. in 2012. Among these proteins, secreted Frizzled-related protein-4 (SFRP4) has become a key player, demonstrating an affinity for liver tissues and confirming its profound importance in the progression of non-alcoholic fatty liver disease (MAFLD) [11,12].

Despite these successes, the diagnostic potential of serum SFRP4 in MAFLD remains largely unexplored. Thus, a thorough investigation of the sensitivity, specificity, and practical utility of serum SFRP4 expression levels as an early-stage MAFLD biomarker is urgently needed and warrants further study.

This discovery not only sheds light on the complex interactions of molecular pathways in the body, but also opens up prospects for the development of diagnostic and therapeutic strategies for the treatment of MAFLD and related metabolic disorders.

Conclusions and prospects

Significant progress has been made in elucidating the pathophysiology of hepatic steatosis and non-alcoholic steatohepatitis (NASH). However, the transition from NASH to fibrosis, which is the most important factor determining mortality in patients with MAFLD, remains poorly understood. This knowledge gap highlights the need for further research aimed at uncovering the mechanisms that determine MAFLD progression.

Identifying accessible non-imaging tools and accurate biomarkers is crucial for improving MAFLD treatment and validating new therapies in clinical trials. Non-invasive and inexpensive methods for accurately determining the stage of MAFLD progression are urgently needed to improve patient care.

Despite recent advances, there is still an unmet need for reliable biomarkers and cost-effective non-invasive tools to accurately determine the stage of MAFLD progression. Addressing these gaps will fa-

cilitate early diagnosis, risk stratification, and treatment monitoring in patients with MAFLD.

The 2018 ASSLD practical guidelines state that weight loss reduces liver steatosis, achieved through a low-calorie diet, increased physical activity, or both. A combination of a low-calorie diet and moderate-intensity exercise is most likely to result in sustained weight loss over time. A 3-5% reduction in body weight improves steatosis, and a 7-10% reduction in body weight is necessary to improve conditions, including fibrosis [13].

Although inflammation plays an important role in disease progression, the strongest predictor of mortality in patients with MAFLD is liver fibrosis. Among patients who lost ~10% of their body weight, 90% showed improvement in MAFLD, and approximately ~45% showed regression of fibrosis. Lifestyle interventions that combine calorie restriction and exercise have a greater effect on reducing liver fat [14]. However, more than 50% of patients included in clinical trials were unable to achieve this level of weight loss. Therefore, despite the fact that lifestyle interventions have a positive effect on the course of MAFLD, it is difficult to achieve sustainable lifestyle changes.

Dietary interventions improve MAFLD progression with or without physical activity; however, the composition of the diet and eating patterns remain controversial [13,14,15]. The picture is somewhat clearer with regard to physical exercise, as most clinical and preclinical studies show that all types and intensities of physical exercise have a positive effect on MAFLD. Physical exercise has been shown to reduce liver steatosis, liver enzyme levels, blood glucose and insulin levels, and improve the lipid profile, both with and without dietary interventions. Even without weight loss, regular physical exercise reduces liver lipid levels.

In conclusion, despite the progress made in understanding the pathophysiology of MAFLD, significant challenges remain in translating this knowledge into effective treatments. Continued research aimed at elucidating the mechanisms of MAFLD progression and developing new diagnostic strategies is essential to improve patient outcomes in the face of this growing epidemic.

REFERENCES

1. Eslam M, Sanyal AJ, George J. MAFLD: A consensus-driven proposed nomenclature for metabolic associated fatty liver disease. *Gastroenterology*. 2020;158(7):1999–2014.

2. Eslam M, Newsome PN, Sarin SK, et al. A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *J Hepatol*. 2020;73(1):202–209.

3. Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *Hepatology*. 2023;78(6):1966–1986.

4. Singh A, Sohal A, Batta A. Recent developments in non-invasive methods for assessing metabolic dysfunction-associated fatty liver disease. *World J Gastroenterol*. 2024;30(39):4324–4338.

5. Hudson D, Afzaal T, Bualbanat H, et al. Modernizing metabolic dysfunction-associated steatotic liver disease diagnostics: the shift from biopsy to non-invasive techniques. *Ther Adv Gastroenterol*. 2024;17:1–14.

6. Abdelhameed F, Kite C, Lagojda L, et al. Non-invasive scores and serum biomarkers for fatty liver in the era of MASLD. *Curr Obes Rep*. 2024;13:510–531.

7. Das S, Agarwal K, Kapoor N, et al. Emerging concepts in the diagnosis and management of MAFLD. *Curr Opin Endocrinol Diabetes Obes*. 2025; 32(6):269–278.

8. Castera L, Friedrich-Rust M, Loomba R. Noninvasive assessment of liver disease in patients with metabolic fatty liver disease. *Gastroenterology*. 2019;156(5):1264–1281.

9. Younossi ZM, Golabi P, de Avila L, et al. Global epidemiology of metabolic associated fatty liver disease. *Hepatology*. 2023;77(4):1335–1347.

10. Wong VW, Adams LA, de Lédinghen V, et al. Noninvasive biomarkers in NAFLD and MAFLD. *Nat Rev Gastroenterol Hepatol*. 2018;15(8):461–478.

11. Tapper EB, Loomba R. Noninvasive imaging biomarker assessment of liver fibrosis by elastography. *Clin Gastroenterol Hepatol*. 2018;16(5): 640–649.

12. Karlas T, Petroff D, Sasso M, et al. Individual patient data meta-analysis of controlled attenuation parameter for diagnosing steatosis. *J Hepatol*. 2017;66(5):1022–1030.

13. Park CC, Nguyen P, Hernandez C, et al. Magnetic resonance elastography vs transient elastography in detection of fibrosis. *Clin Gastroenterol Hepatol*. 2017;15(10):1619–1627.

14. Al-Huneidi LI, Zhao F, Maas R, et al. Liver fat quantification by MRI-PDFF: systematic review and meta-analysis. *J Gastroenterol Hepatol*. 2025;40 (12):2808–2819.

15. Shao C, Ye J, Li X, et al. Discrepancies between NAFLD and MAFLD by steatosis assessment tools. *J Clin Transl Hepatol*. 2022;10(6):1013–1026.

16. Chinese Society of Hepatology. Guideline for the prevention and treatment of metabolic dysfunction-associated fatty liver disease (2024). *Hepatobiliary Surg Nutr*. 2024;13(5):827–846.

17. Алимухамедов Д. Ш. Режим питания при атеросклерозе и других заболеваниях системы кровообращения //Болезни кровообращения. – 2025. – Т. 1. – №. 4. – С. 30-34.

18. Бурибоев Э. М., Азизова Ф. Л. Метабиологическое действие табачных изделий на иммунитет человека //Oriental renaissance: Innovative, educational, natural and social sciences. – 2022. – Т. 2. – №. 12. – С. 213-218.