JOURNAL OF MULTIDISCIPLINARY SCIENCES AND INNOVATIONS
VOLUME 5, ISSUE 05
MONTHLY JOURNALS
ISSN NUMBER: 2751-4390
IMPACT FACTOR: 9,08

SPECIFICS OF TREATING LIVER DISEASES AND THE RELEVANCE OF IMPROVING THERAPEUTIC MEASURES

Sokhib Zamon o'g'li Rashidov
Department of the pharmacology of the
Tashkent state medical university
Javokhir Rustamovich Mamashov
Umidjon Khabib o'g'li Abduqakhkhorov
Tashkent state medical university
https://doi.org/10.5281/zenodo.20390053

Abstract. One of the most prevalent pathological issues in the globe is liver disease. Over 1.5 billion people worldwide are impacted by it. Hepatocytes, Kupffer cells, sinusoidal endothelial cells, and hepatic stellate cells (HSCs) are among the several hepatic cell types that have been linked to the development and spread of both acute and chronic liver disorders. Autophagy, fibrogenic factors, cytokines, oxidative stress, and microRNAs are also implicated. Finding novel therapeutic approaches that may be applied in clinical settings is made possible by comprehending the basic pathways behind liver illnesses. Liver illnesses, both acute and chronic, are global issues with complex etiologies. There is still uncertainty regarding the precise pathogenic mechanism behind a number of liver illnesses. Nevertheless, a number of hypothesized mechanisms—such as oxidative stress, inflammation, autophagy, and microRNA—are implicated. The development of novel and successful therapeutic solutions for this bothersome issue is aided by the underlying viewpoint mechanisms. Antioxidant, anti-inflammatory, anti-HSC therapy, gene therapy, cell therapy, gut microbiota, and nanoparticles have recently shown significant promise in the prevention and treatment of liver disorders. The pathophysiology of acute and chronic liver illnesses was examined here, along with recent potential molecular pathways. Additionally, we highlighted recent research on the treatment of liver problems and reviewed recent therapeutic strategies that targeted liver diseases. This review also provides updates on novel diagnostic methods, existing therapies, and possible therapeutic targets that are currently being tested in clinical settings. The development of new therapeutic approaches will benefit greatly from recent developments in our understanding of the pathophysiology of liver diseases.

Keywords. Autophagy, gene therapy, anti-hepatic stellate cells, liver diseases, Cell treatment, effective therapeutic interventions.

Introduction. Chronic liver illnesses are a very common health issue that adds to the growing burden on nations every day. In particular, one of the most well-known causes of morbidity and mortality worldwide is liver cirrhosis, which is a consequence of chronic liver injury. Cheemerla and Balakrishnan estimate that liver cirrhosis killed about 1.32 million people worldwide in 2017. In addition to being the eleventh most common cause of death, liver cirrhosis is now a common reason for living with a disability. Hepatocyte function abruptly declines as a result of acute liver damage. Acute liver failure (ALF), in contrast to liver cirrhosis, usually has no underlying liver issue and deteriorates quickly over the course of days or weeks. The main causes of ALF in terms of etiology are hepatitis B virus infection and drug toxicity, especially from acetaminophen (APAP). However, Wilson's disease, cardiovascular conditions, autoimmune diseases, and other forms of hepatitis are less frequent ALF suspects. Conversely,

there are two types of long-term liver damage: persistent hepatotoxicity and cholestatic disorders that obstruct bile flow [1-5]. Hepatotoxicity can result from a number of conditions, including alcoholism, non-alcoholic steatohepatitis (NASH), hepatitis B, hepatitis D, and hepatitis C viruses. Cholestatic injuries can also result from primary sclerosing cholangitis, biliary cholangitis, and atresia of the bile ducts. Chronic hepatic inflammation, regardless of the source, results in liver fibrosis, which, if left untreated, develops into liver cirrhosis and hepatocellular carcinoma (HCC). Numerous physiological processes have been linked to liver damage, such as the various forms of autophagy, the important involvement of microRNAs (miRNAs), inflammation, the regulation of hepatic cells, and the primary impacts of transcription factors and inflammatory cytokines. When it comes to therapeutic interventions for liver illnesses, certain treatments are essentially based on the disease's underlying etiology. For example, immunosuppressants for autoimmune hepatitis, acetylcysteine for APAP toxicity, and antiviral drugs for hepatitis viruses are taken into consideration. Numerous treatments for liver diseases, including antifibrotic drugs, cell-based treatments, gut microbiota, various nanoparticle systems, gene therapy, and much more, have been covered in recent publications [6-12]. Therefore, our goal is to talk about the most suitable and recent treatment that has been found to be successful for both acute and chronic liver problems, as well as the recently identified pathophysiological causes. A significant worldwide health issue, liver cirrhosis is characterized by the gradual replacement of healthy liver tissue with fibrotic scar tissue, which impairs liver function. This syndrome is linked to high rates of morbidity and mortality worldwide and is caused by a number of chronic liver illnesses. The final stage of chronic liver disease is cirrhosis, which is frequently brought on by alcohol use, viral hepatitis, and non-alcoholic fatty liver disease. A significant reduction in liver function is indicated by the development of cirrhosis, which can occasionally result in potentially fatal consequences. A significant worldwide health burden is caused by liver cirrhosis and associated chronic liver disorders. About 1.47 million fatalities were caused by cirrhosis and other liver-related illnesses, demonstrating their substantial influence on death rates worldwide. The rising incidence of alcohol-related liver disease and non-alcoholic fatty liver disease is the main cause of the rising burden of chronic liver disease [13-21]. It is possible that the fundamental mechanisms causing the start and progression of the illness are not fully known because the elimination of causative factors like ethanol and viruses does not always stop the development of cirrhosis.8 Therefore, the purpose of this review is to present an up-to-date, thorough overview of the characteristics and epidemiology of liver diseases, emphasize the intricate pathogenetic mechanisms involved, and provide an overview of the current clinical treatments and investigational medications undergoing clinical trials that may be useful for future therapeutic management. The goal of this study is to present a thorough summary of current developments in liver cirrhosis and related disorders diagnosis and therapy. We seek to emphasize the changing landscape of cirrhosis care and pinpoint interesting directions for further study and clinical application by looking at the most recent advancements and cutting-edge treatments [22-28].

The main purpose of the presented manuscript is to provide a brief analysis of the specifics of treating liver diseases and the relevance of improving therapeutic measures based on the results of authoritative scientific works.

Hepatic disease epidemiology. Worldwide mortality. One of the main causes of death worldwide is liver disease. According to the Global Burden of Disease 2019 study, 1.26 million people died in 2019 from cirrhosis and other chronic liver illnesses, a 13% rise since 1990. About 830,000 people died from liver cancer in 2020, accounting for 8.3% of all cancer-related fatalities worldwide. Liver cancer is a final consequence of liver illness.Ten Every year, viral hepatitis, particularly HBV and HCV, causes over 1.3 million deaths. Additionally, 5.9% of

fatalities worldwide are attributed to alcohol-associated liver disease (ALD), which affects around 3.3 million people each year. With a predicted 280,000 deaths from MASLD in 2019, the rising death toll is especially significant. Significant regional differences can be seen in liver disease death rates. For instance, Mongolia has the highest liver cancer mortality rate, at 71.0 per 100,000 people, compared to 6.6 in the United States (U.S.) [3-11]. The main causes of this striking disparity include Mongolia's higher rates of HBV and HCV, scarce healthcare resources, and heavy alcohol usage. On the other hand, the United States has much lower death rates due to efficient hepatitis vaccine programs, thorough screening, and cutting-edge treatment choices. Although liver disease mortality is on the rise globally, some high-income nations, like the United States, have seen a 3.2% yearly decline since their peak in 2013. These variations highlight regional and national inequalities in the burden of disease, access to healthcare, and public health initiatives [15-22].

Liver Cirrhosis Pathophysiology. Liver cirrhosis, the last stage of chronic liver injury, is defined by the progressive dysfunction of the liver brought on by the replacement of healthy hepatic tissue with fibrotic scar tissue. Understanding the underlying mechanisms, common etiologies, and associated issues is necessary for effective management. Cirrhosis is caused by a complicated interaction of cellular and molecular processes. Hepatocyte damage and death: Prolonged liver shocks result in apoptosis and hepatocyte destruction. Hepatic stellate cells (HSCs) and Kupffer cells, the local macrophages in the liver, are activated by DAMPs generated by the dying cells. Hepatic stellate cells (HSCs) are activated; in their latent state, HSCs accumulate vitamin A. They become myofibroblast-like cells that create a lot of extracellular matrix components when stimulated by oxidative stress and inflammatory cytokines, which causes fibrosis. Fibrogenesis: The accumulation of fibrotic tissues is facilitated by an imbalance between fibrogenesis, or the formation of scar tissue, and fibrolysis, or the breakdown of scar tissue [7-13]. Tissue inhibitors of metalloproteinases (TIMPs), which are released by activated HSCs, block matrix metalloproteinases (MMPs), the enzymes responsible for matrix degradation.Vascular changes: The hepatic vasculature is altered by progressive fibrosis, which raises blood flow resistance and causes portal hypertension. Intrahepatic vasoconstriction is exacerbated by endothelial dysfunction and inadequate nitric oxide bioavailability. One of the main causes of liver cirrhosis is excessive and prolonged alcohol consumption. The following are included in the pathophysiology of ALD: Alcohol metabolism: Acetaldehyde, a highly reactive and toxic metabolite, is produced in the liver when ethanol is broken down by alcohol dehydrogenase (ADH) and cytochrome P450 2E1 (CYP2E1). Acetaldehyde causes oxidative stress and immunological activation when it forms adducts with cellular proteins. Reactive oxygen species (ROS): DNA damage, lipid peroxidation, and mitochondrial dysfunction are caused by ROS generated during alcohol metabolism [16-22].

Characteristics of liver disorders, both pathological and clinical. clinical characteristics. A wide range of symptoms, from early nonspecific indications to advanced multisystem consequences, are seen in liver illnesses. Conditions include acute viral hepatitis, moderate DILI, early-stage chronic viral hepatitis, MASLD, and initial-phase ALD often start out with mild, nonspecific symptoms. Patients may have mild exhaustion, discomfort in the upper right abdomen, and a diminished appetite. Acute viral hepatitis patients may experience mild jaundice, nausea, and a brief fever. Patients with MASLD frequently exhibit metabolic syndrome symptoms such obesity, dyslipidemia, and hypertension. Abdominal pain and indigestion are possible early signs of ALD. Transaminase levels may slightly increase in mild DILI without causing noticeable symptoms. Interestingly, a large number of people with early-stage liver disease do not exhibit any symptoms and are only found by accident during routine tests or investigations for other purposes. In a variety of disorders, symptoms that were

previously mild or sporadic become increasingly noticeable when liver diseases advance to the intermediate stage [5-16]. Chronic viral hepatitis patients frequently have persistent fatigue, sporadic jaundice, and worsening right upper quadrant pain, which is frequently accompanied by mild hepatomegaly and general malaise. About 70% of individuals with chronic HCV may experience systemic consequences such mixed cryoglobulinemia and cardiovascular problems, highlighting the disease's wide-ranging effects. At the same time, immune-mediated liver disorders show their distinct patterns of progression. 85–95% of individuals with AIH experience nonspecific symptoms like exhaustion, which are usually accompanied by symptoms like jaundice (67–85%) and abdominal pain (50–70%). In conclusion, the development of liver disease is a multistage, intricate process involving several organs and systems. The clinical manifestations at each stage show the degree of liver damage and its systemic influence, ranging from minor early symptoms to potentially fatal consequences in the final stages. Importantly, different kinds of liver disorders might show different clinical signs and proceed at different rates. This knowledge is essential for accurate prognostication, early diagnosis of liver disorders, and the evaluation of disease severity [21-27].

Advances in clinical research. Viral hepatitis is an acute liver illness. Inactivated vaccinations are the main means of preventing acute viral hepatitis caused by viruses like HAV-HEV. Although there isn't yet a specific antiviral for HAV, clinical research indicates that steroids and IFN-β can improve results. Early lamivudine and entecavir therapy for severe acute HBV has been demonstrated to enhance patient outcomes and slow the development of chronic hepatitis. By reducing treatment times, therapeutic approaches like ledipasvir/sofosbuvir have proven successful in treating HIV and HBV co-infections. In acute HCV, however, grazoprevir plus elbasvir showed potential, especially for genotypes 1 or 4. Ribavirin has been successful in lowering the viral load for HEV. There is a dearth of research on acute HDV treatment, which calls for more investigation. Further research indicates that acute viral hepatitis can also be caused by other viruses, including adenovirus, cytomegalovirus (CMV), Epstein-Barr virus (EBV), and TT virus. Due to their low occurrence, rare viral hepatitis is frequently disregarded. These less frequent manifestations highlight the necessity of increased clinical awareness in order to avoid incorrect diagnosis and treatment [4-14]. **Viral hepatitis is a chronic liver condition.** Vebicorvir, a core inhibitor that has demonstrated better efficacy than conventional nucleoside reverse transcriptase inhibitors, is one of the innovations in CHB therapies. When treating multidrug-resistant HBV strains, tenofovir alafenamide improves long-term results. The potential of PD-1 inhibitors and RNA interference treatments, such as ARC-520, to improve immune responses and lower viral loads in HBV patients is being studied. With the advent of DAAs, chronic HCV treatments have changed, allaying worries about VZV reactivation. For HCV patients who did not respond to previous DAA treatments, glecaprevir and pibrentasvir have demonstrated improved results. For HDV patients, bulevirtide plus tenofovir disoproxil fumarate offers fresh hope, while further research is required to validate these results. All things considered, these developments mark a substantial advancement in the treatment of liver illnesses; nonetheless, further study is essential to maximize the long-term safety and effectiveness of these treatments [18-25].

Discussion. Due to its progressive nature and related complications, liver cirrhosis remains a significant worldwide health concern, contributing to high morbidity and mortality. Recent developments in the diagnosis and management of liver cirrhosis and its associated disorders are examined in this review. Liver biopsies are no longer necessary thanks to non-invasive diagnostic techniques including blood biomarkers and elastography, which have greatly enhanced early detection. Advanced imaging methods, such as CT and MRI, improve diagnostic precision even more. Precision medicine is being made possible by molecular and genomic

research, which is simultaneously offering fresh perspectives on the disease's pathogenesis. Pharmacological advancements, like targeted therapy and antifibrotic drugs, have the potential to decrease the progression of the disease. While advances in liver transplantation and artificial liver support systems offer life-saving alternatives, endoscopic procedures such as variceal banding are improving the management of problems. One promising approach to liver repair is regenerative medicine, including stem cell therapy and tissue engineering. In order to treat cirrhosis-related symptoms, such as portal hypertension, ascites, hepatic encephalopathy, and hepatorenal syndrome, new pharmacotherapies and transjugular intrahepatic portosystemic shunt (TIPS) are being used [3-12]. New biomarkers are being added to prognostic scoring systems like the MELD and Child-Pugh to improve risk categorization. In addition to newly developed treatments that are presently being researched, the future of cirrhosis care is probably going to incorporate the integration of artificial intelligence and machine learning for early diagnosis and customized treatments. Widespread adoption is nevertheless hampered by issues including pricing, accessibility, and healthcare inequities despite these developments. In order to improve the outcomes for patients with liver cirrhosis and associated consequences, this study emphasizes the significance of integrating cutting-edge diagnostic and therapeutic approaches into clinical practice. Liver disease aetiology is always changing. MASLD is becoming a global health concern due to the increase in obesity and type 2 diabetes. The prevalence of MASLD, ALD, and DILI is increasing, whereas the incidence of viral hepatitis has dramatically decreased in the Americas and Europe because to vaccination and antiviral drugs [14-22]. Chronic hepatitis B and C are nevertheless common despite improvements in vaccines and antiviral drugs that successfully prevent and treat viral infections, especially in low-income nations with inadequate medical resources. Furthermore, ALD is becoming more common, particularly in younger populations. Liver illnesses account for a substantial portion of the worldwide disease burden notwithstanding increased public health initiatives. In a phase 2b randomized trial, belapectin, a galectin-3 inhibitor, did not demonstrate effectiveness in MASH patients with cirrhosis and portal hypertension, potentially as a result of insufficient therapy duration and dosage. Mice and humans metabolize belapectin differently, according to pharmacokinetic studies. This disparity highlights the necessity of creating more uniform mammalian models, like chimpanzees and pigs, which could close these gaps and enhance translational success [23-28].

Conclusions. Acute and chronic liver disorders are global issues with a complex etiology. There is still uncertainty regarding the precise pathogenic mechanism behind a number of liver illnesses. However, there are other hypothesized mechanisms at play, such as oxidative stress, inflammation, autophagy, and miRNA. More research is needed to fully understand the role of autophagy and miRNA. Additionally, it might offer a fresh approach to developing novel treatments for liver diseases. More research and development are needed for novel treatment approaches such gene therapy, stem cell therapy, gut microbiota, and even nanoparticle formulations. Going forward, our existing knowledge of the etiology has guided ongoing research efforts targeted at treating liver disease and offered insightful information. However, to develop treatment approaches and enhance patient outcomes, a thorough grasp of crucial signaling pathways and their interactions during the evolution of liver disease is necessary.

Liver biopsy is the main method used to diagnose liver disorders, however it is an invasive procedure that is not appropriate for widespread screening. One major obstacle is the lack of trustworthy biomarkers for the accurate diagnosis and staging of particular liver disorders. Therefore, the early diagnosis of silent liver disorders depends on the development of new non-invasive biomarkers and techniques. These developments may make it possible to identify high-risk patients earlier and implement early therapies to stop the progression of the disease. Even while cutting-edge technologies have significantly enhanced our understanding of the

pathophysiology of liver disease, there are still few FDA-approved therapeutic alternatives, and current medical approaches frequently offer negligible long-term survival advantages.

Current mice models are unable to accurately replicate the entire range of human liver illnesses, including ALD and MASLD, due to the complexity of liver disease biology and the significant variation in disease phenotypes. In terms of pathophysiology and treatment outcomes, there are significant differences between human illnesses and mouse models. Several clinical trials have demonstrated that medications that are beneficial in animal models do not provide clinical advantages in humans. Different species have different characteristics of chemical absorption, distribution, metabolism, excretion, and toxicity. As a result, therapeutic doses that are advantageous and non-toxic in mice may be ineffective or cause adverse effects in humans. For instance, galectin-3 did not work well in human patients despite being shown to lessen liver fibrosis and inflammation in mouse MASH models.

References.

1. Aripov A.N., Aripov O.A., Akhundjanova L.L., Nabiev A.U., Nabieva D.A., & Khamroev T.T. (2022). Study the effect of yantacin on some indicators of cellular renewal and on the level of protein expression on rat hepatocytes in chronic heliotrine liver damage. International Journal of Medical Sciences And Clinical Research, 2(05), 06–13. https://doi.org/10.37547/ijmscr/Volume02Issue05-02.
2. Ali FE, Abd El-Aziz MK, Sharab EI, Bakr AG. Therapeutic interventions of acute and chronic liver disorders: A comprehensive review. World J Hepatol. 2023 Jan 27;15(1):19-40. doi: 10.4254/wjh.v15.i1.19.
3. Aripov A. N, Akhunzhanova L. L, Nabiev A. U, Aripov A. O, Khamroev T. T.. Antifibrotic Efficacy of a New Phytocomposition of Essential Phospholipids with Glycyrrhizic Acid, Ecdysterone, Lycopene and Proanthacyanidin in Experimental Severe Chronic Hepatitis Compared with Phosphogliv. Biomed Pharmacol J 2023;16(3).Pages : 1815-1825. DOI : https://dx.doi.org/10.13005/bpj/2761
4. Aripov A.N, Akhunjanova L.L, Khamroev T.T, Aripov Abdumalik Nigmatovich, Akhunjanova Lola Lazizovna, & Khamroev Tolmas Tolibovich. (2022). Differential Analysis of Chronic Toxic Hepatitis Caused by The Introduction of Heliotrin Solution in Various Ways. Texas Journal of Medical Science, 4, 58–62. Retrieved from https://zienjournals.com/index.php/tjms/article/view/670
5. Rashidov S.Z., Rakhimboev S.D., Sanoev Z.I., Abdinazarov I.T., Khamroev T.T., Ismailova D.S., & Elmuradov B.J.. (2022). Study of psychoactive activity potassium salt 5-(o-aminophenyl)-1,3,4- oxadiazole-2-thion (D-361). International Journal of Medical Sciences And Clinical Research, 2(09), 1–5. https://doi.org/10.37547/ijmscr/Volume02Issue09-01
6. Арипов А.Н., Арипов О.А., Ахунджанова Л.Л., Набиев А.Ў., Нишанбаев С.З., Набиева Д.А., Ҳамроев Т.Т. Тажриба шароитида сафорофлавонолозиднинг гепатотроп фаоллигини ўрганиш. Oriental Journal of Medicine and Pharmacology, 2(02), 55–64. https://doi.org/10.37547/supsci-ojmp-02-02-07
7. Zakhidova L.T., Saidkhodjaeva D.M., Sanoev Z.I., Tukhtasheva V.F., Rakhmanova H.A.., Hamroyev T.T. Toxicological Characteristics Of N-Deacetyllappaconitine Under Chronic Administration In White Rats. The American Journal of Applied Sciences, 3(03), 34-41. https://doi.org/10.37547/tajas/Volume03Issue03-06
8. Khamroev T.T., Sanoev Z.I., Rakhimboev S.D., Abdinazarov I.T., Rashidov S.Z. Effect of antiarrhythmic substance N – dezacetyllapoconitin on the central nervous system. ISJ Theoretical &Applied Science, 07 (99), 153-157. http://soi.org/1.1/TAS-07-99-31 Doi:https://dx.doi.org/10.15863

9. Sanoev Z. I, Ismailova D. S, Rakhimboev S. D. O, Khamroev T, T, Elmuradov B. Z, Abdinazarov I. T, Rashidov S. Z. O. Synthesis and Research Anticonvulsant Activity of Annulated Triazolo-Thiadiazine Derivative in Laboratory Animals. Biomed Pharmacol J 2023;16(4). DOI : https://dx.doi.org/10.13005/bpj/2820
10. Sokhib Rashidov Zamon o‘g‘li, Muslimakhon Kamolova Mirzokhidjon qizi, Ikhvoliddin Mirzaev Komiljon o‘g‘li, Nodira Pardaeva Botir qizi, Sevara Rakhmatullaeva Shukhrat qizi/. (2025). The importance of cardiotonic drugs in medical practice, the range of applications and the advantages of their use. International Journal of Cognitive Neuroscience and Psychology, 3(5), 95–100. Retrieved from https://medicaljournals.eu/index.php/IJCNP/article/view/1856
11. Sanoev Zafar Isomiddinovich, Rashidov Sokhib Zamon ugli, Raximboev Sukhrob Davlatyor ugli, Abdinazarov Ibrokhim Tuychievich, Khamroev Tolmas Tolibovich, Ismailova Dilnoza Safaralievna, & Elmuradov Burkhon Juraevich. (2022). Research of Anticonvulsant Activity of Compound 5- (P-Aminophenyl) - 1,3,4-Oxadiazole-2-Thion. Texas Journal of Medical Science, 13, 17–21. Retrieved from https://zienjournals.com/index.php/tjms/article/view/2434
12. Yu. R. Mirzaev, T. T. Khamroev, E. M. Ruzimov, B. N. Khandamov, & Sh. M. Adizov. (2022). Evaluation of the Effect on the Nervous System of Substances with an Alkaloid Structure Having Antitumor Activity. Journal Healthcare Treatment Development(JHTD) ISSN : 2799-1148, 2(06), 6–10. Retrieved from http://journal.hmjournals.com/index.php/JHTD/article/view/1577
13. Aripov A.N., Aripov O.A., Akhunjanova L.L., Nabiev A.O., Nabieva D.A., & Khamroev T.T. (2022). Study the antifibrous efficacy of plant proanthocyanidin in rats with chronic heliotrine liver damage. Frontline Medical Sciences and Pharmaceutical Journal, 2(05), 16–25. https://doi.org/10.37547/medical-fmspj-02-05-03.
14. Sokhib Rashidov Zamon o‘g‘li, Nilufar Ergasheva Ag’zamjon qizi, Elyor Zokirboyev Anvarjon o‘gli, Umidjon Akramov Abdusamad o‘g‘li, & Aziza Egamberdieva Farkhod qizi. (2025). Drugs That Increase the Tone of the Human Body and Pharmacological Characteristics of Immunodeficiency Agents. American Journal of Biomedicine and Pharmacy, 2(5), 300–306. Retrieved from https://biojournals.us/index.php/AJBP/article/view/1065
15. Sokhib Rashidov Zamon o‘g‘li, Murodjon Nabiev Mahammadkarim o‘g‘li, Mo'tabar Yoqubjonova Khusanboy qizi, Shakhzodakhon Bekmurodova Po‘latjon qizi, & Jumanazar To‘ychiev Saidqul o‘g‘li. (2025). Comparative Analysis of Drugs Used for Anemia and Drugs Storing Iron. Research Journal of Trauma and Disability Studies, 4(5), 190–195. Retrieved from https://journals.academiczone.net/index.php/rjtds/article/view/5141
16. Sokhib Rashidov Zamon o‘g‘li, Shakhzoda Abduraimova Abdusattor qizi, Nigora Yusufjonova Mirrakhim qizi, Diyora Turdibekova Erkinjon qizi, & Makhsuma Dovutkho’jayeva Maqsudjonovna. (2025). Classification, Indications for Use, Range of Applications and Disadvantages of Medicines against Nematodes and Leishmania. Research Journal of Trauma and Disability Studies, 4(5), 196–201. Retrieved from https://journals.academiczone.net/index.php/rjtds/article/view/5142
17. Sokhib Rashidov Zamon o‘g‘li, Nigora Yusufjonova Mirrakhim qizi, Diyora Turdibekova Erkinjon qizi, Makhsuma Dovutkho’jaeva Maqsudjonovna, Shakhzoda Abduraimova Abdusattor qizi, Analysis of the effect of medicines used in medical practice for various diseases on the fetus , european journal of modern medicine and practice: Vol. 5 No. 5 (2025) 342-347.

18. Sokhib Rashidov Zamon o‘g‘li, Elyor Zokirboev Anvarjon ògli, Umidjon Akramov Abdusamad o‘g‘li, Aziza Egamberdiyeva Farkhod qizi, Munisa Qo‘shbekova Ro‘zimbek qizi. (2025). Analysis of general and specific pharmacological properties of fat-soluble vitamins. International Journal of Cognitive Neuroscience and Psychology, 3(5), 101–106. Retrieved from https://medicaljournals.eu/index.php/IJCNP/article/view/1857
19. Sah AK, Afzal M, Elshaikh RH, Abbas AM, Shalabi MG, Prabhakar PK, Babker AMA, Khalimova FT, Sabrievna VA, Choudhary RK. Innovative Strategies in the Diagnosis and Treatment of Liver Cirrhosis and Associated Syndromes. Life. 2025; 15(5):779. https://doi.org/10.3390/life15050779
20. Cheemerla S, Balakrishnan M. Global Epidemiology of Chronic Liver Disease. Clin Liver Dis (Hoboken) 2021;17:365–370. doi: 10.1002/cld.1061.
21. Gan, C., Yuan, Y., Shen, H. et al. Liver diseases: epidemiology, causes, trends and predictions. Sig Transduct Target Ther 10, 33 (2025). https://doi.org/10.1038/s41392-024-02072-z
22. Ashenhurst, J. R. et al. Prevalence of alpha-1 antitrypsin deficiency, self-reported behavior change, and health care engagement among direct-to-consumer recipients of a personalized genetic risk report. Chest 161, 373–381 (2022).
23. Hasegawa, K. et al. Clinical practice guidelines for hepatocellular carcinoma: The Japan Society of Hepatology 2021 version (5th JSH-HCC Guidelines). Hepatol. Res. 53, 383–390 (2023).
24. Giannitrapani, L. et al. The changing epidemiology of hepatocellular carcinoma : experience of a single center. Biomed. Res. Int. 2020, 5309307 (2020).
25. Santos, A. A. et al. Spatial metabolomics and its application in the liver. Hepatology 79, 1158–1179 (2024).
26. Han, H. et al. Danger signals in liver injury and restoration of homeostasis. J. Hepatol. 73, 933–951 (2020).
27. Devarbhavi, H. et al. Global burden of liver disease: 2023 update. J. Hepatol. 79, 516–537 (2023).
28. Tapper, E. B. & Parikh, N. D. Diagnosis and management of cirrhosis and its complications: a review. JAMA 329, 1589–1602 (2023).