World Journal of Pharmaceutical Science and Research
www.wjpsronline.com
Research Article
ISSN: 2583-6579
SJIF Impact Factor: 5.111
Year - 2025
Volume: 4; Issue: 5
Page: 681-696

EFFECT OF ISOQUINOLINE ALKALOIDS – F-5, F-24, AND THEIR DERIVATIVES (KV-6, KV-8) ON THE FUNCTIONAL STATE OF LIVER MITOCHONDRIA IN RATS

Abduvali Abdubokiev[1]*, Shunkor Khushmatov[2], Nurali Ergashev[3], Esohon Komilov[4], Sherzod Zhurakulov[5]

[1]Dept. of Physiology and Anatomy, Namangan State University, Namangan, Uzbekistan.
[2]Dept. for the Development of Scientific and Innovative Activities, Ministry of Higher Education, Science and Innovation of the Republic of Uzbekistan, Tashkent, Uzbekistan.
[3,4]Laboratory of Molecular Biophysics, Institute of Biophysics and Biochemistry under the National University of Uzbekistan, Tashkent, Uzbekistan.
[5]Laboratory of Alkaloid Chemistry, Institute of the Chemistry of Plant Substances named after Academician S.Yu. Yunusov, Academy of Sciences of the Republic of Uzbekistan, Tashkent, Uzbekistan.

Article Received: 12 September 2025 | | ***Article Revised: 02 October 2025*** | | ***Article Accepted: 24 October 2025***

***Corresponding Author: Abduvali Abdubokiev**
Dept. of Physiology and Anatomy, Namangan State University, Namangan, Uzbekistan.
DOI: https://doi.org/10.5281/zenodo.17484637

How to cite this Article: Abduvali Abdubokiev, Shunkor Khushmatov, Nurali Ergashev, Esohon Komilov, Sherzod Zhurakulov (2025). EFFECT OF ISOQUINOLINE ALKALOIDS – F-5, F-24, AND THEIR DERIVATIVES (KV-6, KV-8) ON THE FUNCTIONAL STATE OF LIVER MITOCHONDRIA IN RATS. World Journal of Pharmaceutical Science and Research, 4(5), 681-696. https://doi.org/10.5281/zenodo.17484637

ABSTRACT

In this study, it was found that flavonoid conjugates (KV-6, KV-8) of isoquinoline alkaloids (F-5, F-24) (5-20 μM) in an incubation medium with $[Ca^{2+}]_{out}$=10 mM depending on the concentration (5-20 μM) weaken the process of mitochondrial swelling in the liver of rats. In this case, it was confirmed that these agents at a maximum concentration of 20 μM reduce the swelling process by 47.40±2.32%, 41.86±3.07%, 62.20±3.17% and 56.84±2.24%, respectively, compared to the control. It was noted that this activity is manifested to a greater extent in their conjugates compared to isoquinoline alkaloids (F-5, F-24). The obtained results can be used as a scientific basis for the development of therapeutic drugs for the treatment of liver diseases based on isoquinoline alkaloids and their derivatives.

KEYWORDS: *Isoquinoline alkaloids, каламуш жигар митохондрияси, mitochondrial permeability transition pore.*

INTRODUCTION

Chronic liver diseases (hepatoses, etc.) are a serious medical and social problem worldwide, characterized by a high risk of developing fibrosis, cirrhosis, and hepatocellular carcinoma during pathogenesis.[1,2,3,4]

In liver diseases, morphofunctional changes in mitochondria, disruption of the structure and function of *mito*DNA, and the development of dysfunction of the respiratory complex are observed, which in turn leads to a decrease in ATP biosynthesis and an increase in the formation of ROS.[2,5]

Mitochondria are one of the main dysfunctional structures in the pathogenesis of many diseases and are considered an effective "*target*" in the therapeutic process. [6,7,8,9,10,11,12]

Mitochondria are organelles that play a central role in the cellular energy system of the body, and *mPTP* is the main ion transport system under physiological and pathological conditions.[7,6,13,14,15,16,17]

Mitochondria have a unique structure and functional activity, which have been described in detail by a number of researchers.[18,19,20,21]

The outer membrane of mitochondria contains integral transport proteins that are permeable to inorganic ions and protein molecules (≤10 kDa), forming a matrix consisting of crystals of the inner membrane with high selective permeability. In the mitochondrial membranes there are voltage-dependent anion channels (VDAC), *Bax/Bak, Bcl* - mitochondrial pro-apoptotic proteins, ANT, multienzyme complexes – NADH dehydrogenase (complex I), succinate dehydrogenase (complex II), QH_2 dehydrogenase (complex III), cytochrome oxidase (complex IV), which during respiration form a potential gradient ($\Delta\Psi_{mito.}$) for ATP synthesis and the Krebs cycle (Fig. 1).[22,23,24,25,26,27,28,29,30]

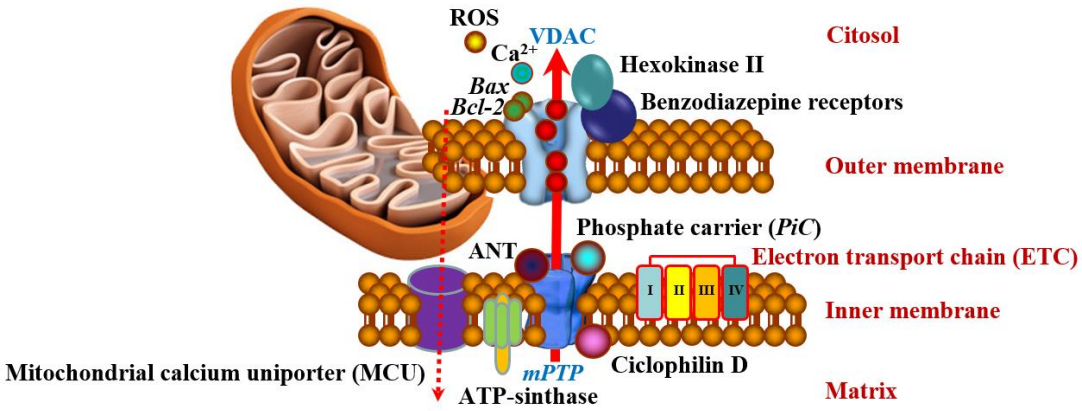

Figure 1: Schematic diagram of the structure of mPTP (*mitochondrial permeability transition pore*).

Bax, Bcl-2 are pro-apoptotic proteins, *ROS* is a reactive oxygen species (free radical), *VDAC* is a voltage-dependent anion channel, *ANT* is an adenine nucleotide translocator.[31,32,33,34,35]

*m*PTP (The mitochondrial permeability transition pore)

Mitochondria, in addition to being the unit of energy supply, are multifunctional organelles involved in a cascade of many physiological and pathological biochemical reactions. In this regard, the Ca^{2+}-dependent megapore (*mPTP*) is a central structural and functional element in such important processes as biosynthesis, ATP synthesis, intracellular signal transduction and oxidative stress. The transition of *mPTP* to the open state, associated with changes in ROS

generation, membrane potential ($\Delta\Psi_{mito.}$), the amount of $[Ca^{2+}]_{mit.}$ (Ca^{2+} homeostasis) and the dynamics of ATP synthesis, indicates the involvement of mitochondria in important physiological and pathophysiological processes.[13,14,36,37,38,39,40,41]

The structural organization, functional units, and regulatory mechanisms of *mPTP* in normal physiological and pathophysiological conditions have been described in detail by several researchers.[6,13,14,35,38,39,42,43,44,45,46,47]

It has been confirmed that *mPTP* plays an important role in maintaining normal physiological metabolism, as well as in pathophysiological conditions in the antioxidant response of hepatocytes (a transition to an open state under the action of free radicals such as ROS and a transition to a closed state under the action of antioxidant agents (ascorbate, etc.) is observed).[32,48,49,50]

mPTP, located in the inner mitochondrial membrane, has a permeability for substances M_r=≤1500 Da, and VDAC, ANT (ADP/ATP antiporter) and cyclophilin D (Cyp-D – *cyclophilin* D) play an important role in its functional regulation.[6,32,48,49,50,51,52,53]

mPTP is a complex protein complex, and an increase in $[Ca^{2+}]_{mit.}$, oxidative stress, generation of ROS (reactive oxygen species) and a change in the value of the internal membrane potential of mitochondria ($\Delta\Psi_{mito}$) transfer mPTP to the open state, while specific blockers (cyclosporin A, etc.) transfer it to the closed state. The experimental mechanism of *mPTP* regulation in liver mitochondria has been described in detail by a number of researchers.[13,18,20,32,34,54,51,55,56]

Initial studies confirmed that when Ca^{2+} (10-50 μM) was added to the incubation medium, the *mPTP* structural-functional complex was formed by ANT, cyclophilin D, and VDAC, which was characterized by a transition of the mitochondrial inner membrane to a state of increased permeability (mitochondrial permeability transition pore). Subsequent studies showed that the phosphate symporter (PiC) and F_1F_0-ATP synthase play an important role in regulating the function of the complex.[34,57,58,59,60]

Mitochondria are the energy-generating units in cells, including hepatocytes, and are involved in the metabolism of proteins, carbohydrates, and lipids, as well as in the e^- transport chain involving CO_2 and H_2O. NADH dehydrogenase (complex I), succinate dehydrogenase (complex II), cytochrome *b* (complex III), and cytochrome c oxidase (complex IV) transport H^+ from the matrix to the intergranular space and form a proton gradient. In this case, in the cascade of oxidation-phosphorylation reactions, ATP-synthaza occurs with the participation of ATP synthase in the reverse H^+ transport (complex V).[7,8,9,61,62,63,64,65]

The functional activity of mitochondria involves the gradient of ions Na^+, Ca^{2+}, K^+, etc. ($[Na^+]_{out}$=145 мМ, $[Na^+]_{in}$=15 мМ, $[Na^+]_{mito.}$=5 мМ, $[Ca^{2+}]_{out}$=2 мМ, $[Ca^{2+}]_{in}$=0,0001-0,02 мМ, $[Ca^{2+}]_{mito.}$=0,5 мМ, $[K^+]_{out}$=4 мМ, $[K^+]_{in}$=150 мМ, $[K^+]_{mito.}$=150 мМ), ion transport systems (the *mito*K_{ATF} channel is supported by a K^+ uniporter, Ca^{2+} uniporter, Na^+/Ca^{2+} exchanger, Na^+/H^+ exchanger, H^+/K^+ exchanger, $H^+/K^+(Na^+)$ exchanger, etc.).[66,67,68,69]

Mitochondria play an important role in Ca^{2+} homeostasis in the cell, and in addition to the mitochondrial Ca^{2+} uniporter, the participation of the Na^+/Ca^{2+} exchanger (NCLX) and the Ca^{2+}/H^+ antiporter (*Letm1*)/K^+/H^+ exchanger has been noted.[18,20,70,71]

It has also been confirmed that ion transport systems (including *mito*BK_{Ca}, *mito*K_{ATP} channel, etc.) play an important role in the functional activity of mitochondria.[72,73,74,75,76,77,78]

Under physiological and pathophysiological conditions, mitochondria play an important role in maintaining the functional activity of the cell, bioenergetic homeostasis and participate in the process of endogenous/exogenous apoptosis. It has been confirmed that under conditions of elevated ROS concentrations, *mPTP*, a non-selective ion channel consisting of a multiprotein structural-functional complex, switches to an open conformational state for a long time, which leads to pathological dysfunction in the cell.[6,7,9,13,14,36,79,80,81,82,83]

Thus, the function of the mPTP multicomplex (VDAC, ATP synthase, Cp-D, ANT, etc.), located in the inner mitochondrial membrane, is regulated by agents such as Ca^{2+}, ADP, and bongkrek acid, and it has been confirmed that its prolonged transition to the open state leads to mitochondria "*swelling*", which in turn leads to the development of various pathologies. In turn, it is of great importance for intracellular signal transmission and Ca^{2+} homeostasis, and is also a central mitochondrial "*target*" for pharmacological agents. [19, 42, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93]

The aim of this study was to analyze the influence of isoquinoline alkaloids – F-5, F-24 and their conjugates (KV-6, KV-8) on the state of *mPTP*.

MATERIALS AND METHODS

The experiments were conducted in 2023-2024 in the Metabolomics laboratory of the Institute of Biophysics and Biochemistry of the National University of Uzbekistan named after Mirzo Ulugbek.

Experimental Animals

The objects of the study were white rat (♀/♂, *m*=170-200 g), which were fed standard food (water) under standard vivarium conditions (room temperature +20±5°C, relative air humidity 75±10%, light regime 12:12 hours).

Ethical Approval

When working with experimental animals in scientific research, the requirements of the rules developed by the International Council for International Organizations of Medical Sciences (1985), the European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes (Strasbourg, 1986), the Declaration developed by the European Union (86/609/EEC), and the Bioethical Statement of the Institute of Biophysics and Biochemistry of the National University of Uzbekistan (No. BRC/IBB; N44/2024/75-1) were observed.

Chemicals and Drugs

Isoquinoline alkaloids (F-5, F-24) and their derivatives with the flavonoid quercetin (KV-6, KV-8), provided by the staff of the S.Yu.Yunusov Institute of Plant Chemistry of the Academy of Sciences of the Republic of Uzbekistan were used as objects of research.

Isolation of liver mitochondria

Rat liver mitochondrial suspension is a convenient experimental object for the analysis of *mPTP* regulatory mechanisms.[32]

In studies on suspensions of rat liver mitochondria, the effect of isoquinoline alkaloids and their conjugates on the state of Ca^{2+}-dependent, cyclosporine A-sensitive *mPTP* in mitochondria (*in vivo*) was analyzed. [20,95]

After the experimental animals were withdrawn from the experiment, the abdominal cavity was surgically opened, the liver was removed, and weighed in a separation medium cooled to 0±0.5°C, mixed with a separation medium (sucrose – 250 mM, Tris-HCl – 10 mM, EDTA – 1 mM, *pH*=7.4) in a ratio of 1:6 g/ml. After homogenization in a glass homogenizer, it was centrifuged in an RS-6MC centrifuge (NV-Lab, Russia) using the standard differential centrifugation method at 1500 rpm for 7-8 minutes (separation of nuclear and cellular fragments), and the mitochondrial supernatant was centrifuged at 6000 rpm for 15-20 minutes (separation of mitochondria) (*t*=0…+2±0.5°C), in EDTA-free separation medium (g/ml ratio 10:1), the test sample was stored in an ice bath in a diluted state. [74, 77, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109]

The mitochondrial protein content was measured by the Lowry method (modified by Peterson), based on spectrophotometric (λ=750 nm) analysis of the intensity of blue color formed during the reaction of protein with the Folin-Ciocalteu reagent, using $CuSO_4 \times 5H_2O$ (0.1% solution) + potassium tartrate (0.2% solution) + Na_2CO_3 (0.2% solution), Folin-Ciocalteu reagent (0.5%). [106,110]

The conductivity of *mPTP* was analyzed spectrophotometrically using a V-5000 spectrophotometer (Shanghai Metash Instruments Co., Ltd.; China) (λ=540 nm) in the following incubation medium: sucrose (250 mM), EDTA (10 mM), KH_2PO_4 – 1 mM, Ca^{2+}-EGTA buffer – 20 mM, succinate (5 mM), rotenone (2 μM), oligomycin (1 μg/ml), TRIS chloride (20 mM), HEPES (20 mM), KH_2PO_4 (1 mM) (pH = 7.4).[77,96,102,105,107,109,111,112,113,114,115,116,117,118,119,120,121,122,123]

Statistical Analysis

Mathematical and statistical processing of the obtained experimental results was carried out by standard methods using special software packages “Microsoft Excel 2007” (Microsoft, USA), OriginPro v. 8.5 SR1 (EULA, USA).

The results are presented as *M*±*m*, where M is the arithmetic mean and m is the standard error of the mean, calculated based on the results of experiments conducted with *n*=3-4 repetitions. Also, the level of statistical significance of the values between the experimental results and the control group was calculated based on Student’s *t*-test and was considered statistically significant at values of $p<0.05$ and $p<0.01$. The level of reliability of the difference between the values of the two experimental groups was calculated using the Student's *t*-test.

RESULTS AND DISCUSSION

In experiments it was established that flavonoid conjugates of isoquinoline alkaloids (KB-6, KB-8) (5-20 μM) do not have a significant effect on the ionic permeability of rat liver mitochondria in an incubation medium with $[Ca^{2+}]_{out}$=0 mM.

Mitochondrial swelling induced by the addition of Ca^{2+} (10 μM) to the incubation medium was used as a control (100%). It was found that cyclosporine A (1 μM) reduced mitochondrial swelling by 47.40±2.32% compared to the control (Fig. 2).

It was established that isoquinoline alkaloids – F-5, F-24 – inhibit mitochondrial swelling depending on the concentration (5–20 μM) under conditions of $[Ca^{2+}]_{out}$=10 mM, and at a maximum concentration of 20 μM, these

alkaloids reduce the swelling process by 47.40±2.32% and 41.86±3.07%, respectively, compared to the control (Fig. 2A, B).

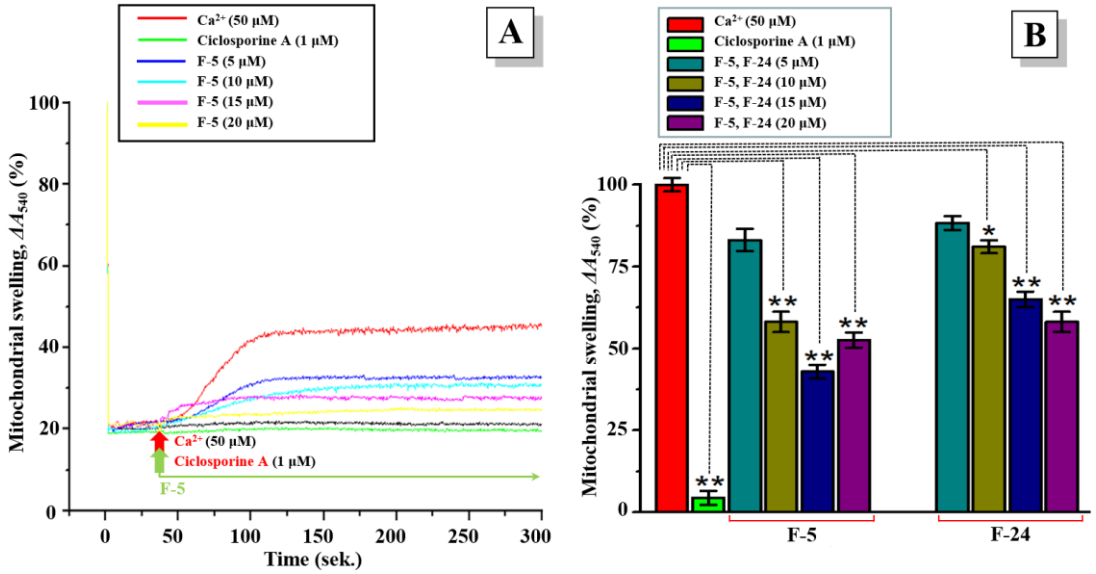

Figure 2: Concentration-dependent (5-20 μM) effects of cyclosporine A (1 μM), isoquinoline alkaloids F-5 (A) and F-24 on mitochondrial swelling (control) under conditions of $[Ca^{2+}]_{out}$=10 mM (B).

A. The ordinate axis shows the mitochondrial swelling process (ΔA_{540}), expressed as a percentage (%) of the maximum, the abscissa axis shows the F-5 concentration (5-20 μM). * – *p<0.05* compared to the control, ** – *p<0.01 (n=3–4).*

It was also established that conjugates of isoquinoline alkaloids (F-5, F-24) with quercetin – KV-6, KV-8 – inhibit the process of mitochondrial swelling depending on the concentration (5–20 μM) under conditions of $[Ca^{2+}]_{out}$=10 mM, and at a maximum concentration of 20 μM, these agents reduce the swelling process by 62.20±3.17% and 56.84±2.24%, respectively, compared to the control (Fig. 3A,B).

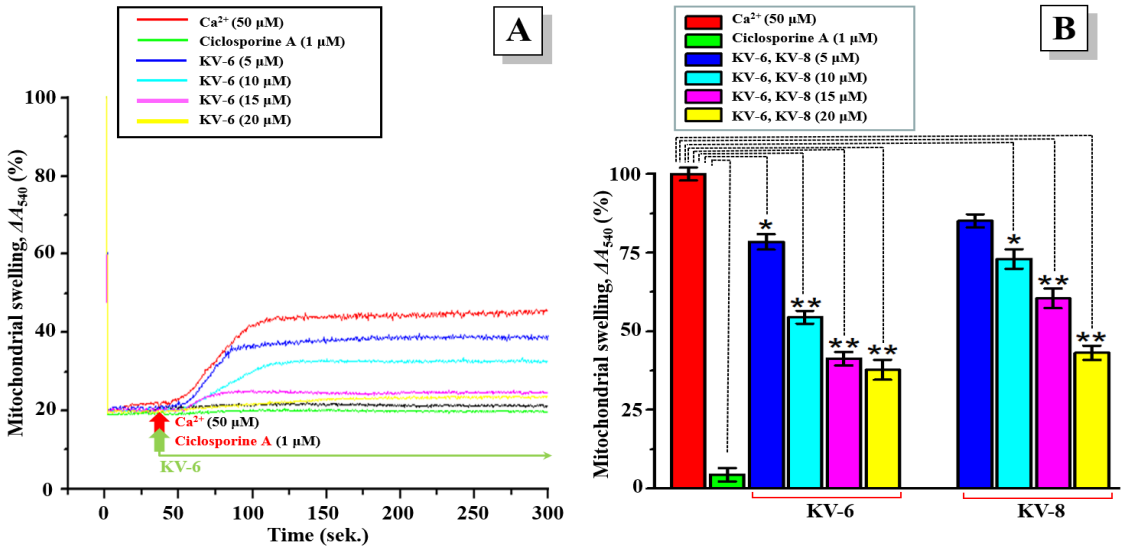

Figure 3. Concentration-dependent effect on mitochondrial swelling of cyclosporine (1 μM) (control) A, conjugates of KV-6 (A), KV-8 isoquinoline alkaloids (F-5, F-24) – (5-20 μM) (B) under conditions of $[Ca^{2+}]_{out}$=10 mM.

A. The ordinate axis shows the mitochondrial swelling process (ΔA_{540}), expressed as a percentage (%) of the maximum, the abscissa axis shows the concentration of KV-6 (5-20 μM) compared to the control, * – $p<0.05$ ** – $p<0.01$ (n=3-4).

Thus, the regulation of Ca^{2+} homeostasis in mitochondria is of great importance for maintaining normal physiological processes in the cell, as well as from a therapeutic point of view, since an increase in the amount of $[Ca^{2+}]_{mito}$ ensures the transition of *mPTP* to an open conformational state. Under the influence of Ca^{2+} as an activator, the process of "*mitochondrial swelling*" occurs under conditions of increased permeability of the mitochondrial membrane *mPTP* ("*permeabilization*").[13, 71, 94, 124, 125, 126, 127, 128, 129]

Under physiological conditions, the transition of *mPTP* to the open state is responsible for the transport of Ca^{2+} ions from the matrix to the external environment (maintaining Ca^{2+} homeostasis), whereas its irreversible transition to the open state is responsible for the occurrence of pathogenesis (induction of apoptosis, etc.). In turn, it is noted that the regulation of the functional activity of *mPTP* is of current importance from a pharmacotherapeutic point of view.[35,38,39,43,42,46,130,129]

Inhibition of mitochondrial swelling by cyclosporin A (1 μM) in the presence of Ca^{2+} (10 μM) in the incubation medium is explained by the blockade of the functional activity of the *mPTP* regulator cyclophilin D (CypD), as well as by blocking the transition of mPTP to the open state (transition to the closed state) as a result of a decrease in the amount of $[Ca^{2+}]_{mito}$ due to buffering.[126,128,131,132]

Studies have confirmed that isoquinoline alkaloids, polyphenolic compounds (flavonoids, etc.) have a significant effect on the functional activity of mitochondria. In particular, it was noted that isoquinoline alkaloids (F-4, F-24) significantly weaken the mitochondrial dysfunction of the rat heart under conditions of oxidative stress caused by $PbCl_2$ and increase the activity of the K_{ATP}-channel.[133]

It has also been confirmed that isoquinoline alkaloid N-14 (30 μM) increases the activity of the K_{ATP}-channel in rat heart mitochondria.[134]

Polyphenolic compounds have been shown to have a significant inhibitory effect on the open state of *mPTP* in rat liver mitochondria.[135]

CONCLUSION

Thus, studies have shown that isoquinoline alkaloids – F-5, F-24 – reliably inhibit the process of mitochondrial swelling depending on the concentration (5-20 μM) under conditions of $[Ca^{2+}]_{out}$=10 mM. It has been confirmed that this activity is manifested to a greater extent in their conjugates than in isoquinoline alkaloids (F-5, F-24).

The obtained results can be used as a scientific basis for the development of therapeutic drugs for the treatment of liver diseases based on isoquinoline alkaloids and their derivatives.

ACKNOWLEDGEMENT

The author(s) declare that financial support was received for the research, authorship, and/or publication of this article. This work was supported by the grant of the Ministry of Higher Education, Science and Innovation of the Republic of

Uzbekistan (F-OT-2021-465 “Development of simulation models of pathological conditions and diseases of humans and animals *in vitro* and *in vivo*”).

Conflict of interest

The authors do not have any conflict of interest.

Permission to reproduce material from other sources

Not applicable.

Data Availability Statement

This statement does not apply to this article.

Ethics statement

This research did not involve human participants, animal subjects, or any material that requires ethical approval.

Informed Consent Statement

This study did not involve human participants, and therefore, informed consent was not required.

Clinical Trial Registration

This research does not involve any clinical trials.

Author Contributions

AA: Investigation, Methodology, Writing-review and editing. **SK**: Conceptualization, Formal Analysis, Funding acquisition, Methodology, Project administration, Supervision, Writing-review and editing. **NE**: Writing-review and editing. **EK**: Data curation, Formal Analysis, Methodology, Writing-original draft. **SZ**: Funding acquisition, Methodology, Project administration. All authors read and approved the final manuscript.

REFERENCES

1. Delgado-Montemayor C., Cordero-Perez P., Salazar-Aranda R., Waksman-Minskya N. Models of hepatoprotective activity assessment // Medicina Universitaria, 2015; 17(69): 222-228.
2. Ferramosca A., Di Giacomo M., Zara V. Antioxidant dietary approach in treatment of fatty liver: New insights and updates // World J. Gastroenterol. 2017; 21: 23(23): 4146-4157.
3. Ko I.-G., Jin J.-J., Hwang L., Kim S.-H., Kim C.-J., Han J.H., Lee S., Kim H.I., Shin H.P., Jeon J.W. Polydeoxyribonucleotide exerts protective effect against CCl_4-induced acute liver injury through inactivation of NF-κB/MAPK signaling pathway in mice // Int. J. Mol. Sci. 2020; 21(7894): 1-16.
4. Hinds T.D., Creeden J.F., Gordon D.M., Stec D.F., Donald M.C., Stec D.E. Bilirubin nanoparticles reduce diet-induced hepatic steatosis, improve fat utilization, and increase plasma β-hydroxybutyrate // Front. Pharmacol. 2020; 11(594574): 1-11.
5. Malhi H., Gores G.J. Cellular and molecular mechanisms of liver injury // Gastroenterology. 2008; 134: 1641-1654.
6. Waseem M., Wang B.-D. Promising Strategy of *mPTP* modulation in cancer therapy: An emerging progress and future insight // Int. J. Mol. Sci. 2023; 24(5564): 1-26.

7. Casanova A., Wevers A., Navarro-Ledesma S., Pruimboom L. Mitochondria: It is all about energy // Front. Physiol. 2023; 14(1114231): 1-37.
8. Zhang L., Miao M., Bai X.X.M., Wu M., Zhang A. From physiology to pathology: The role of mitochondria in acute kidney injuries and chronic kidney diseases // Kidney Dis. 2023; 9: 342-357.
9. Collier J.J., Olahova M., McWilliams T.G., Taylor R.W. Mitochondrial signalling and homeostasis: From cell biology to neurological disease // Trends in Neurosciences. 2023; 46(2): 137-152.
10. Desai V.M., Rajdeep M.Ch., Singhvi C.G. Photodynamic therapy induced mitochondrial targeting strategies for cancer treatment: Emerging trends and insights // Mol. Pharmaceutics. 2024; 21(4): 1591-1608.
11. Deng Y., Zhu H., Xing J., Gao J., Duan J., Liu P., Zhong G., Cai X. The role of natural products in improving lipid metabolism disorder-induced mitochondrial dysfunction of diabetic kidney disease // Front. Physiol. 2025; 16(1624077): 1-8.
12. Li J., Liu W., Zhang J., Sun C. The Role of mitochondrial quality control in liver diseases: Dawn of a therapeutic era // Int. J. Biol. Sci. 2025; 10:21(4): 1767-1783.
13. Bauer T.M., Murphy E. Role of mitochondrial calcium and the permeability transition pore in regulating cell death // Circulation Research. 2020; 126: 280-293.
14. Kim Y.-R., Jun S., Jung S., Lee B., Lee S.-H., Lee J., Hwang J.-S., Thoudam T., Lee H., Sinam I.S., Jeon J.-H., Lee K.-Y., Min S.-J., Kim U.-K. Inhibition of the mitochondrial permeability transition pore as a promising target for protecting auditory function in cisplatin-induced hearing loss // Biomedicine & Pharmacotherapy. 2025; 182(117767): 1-10.
15. Whitehead M., Harvey J.P., Sladen P.E., Becchi G., Singh K., Sun Y.J., Burgoyne T., Duchen M.R., Yu-Wai-Man P., Cheetham M.E. Disruption of mitochondrial homeostasis and permeability transition pore opening in OPA_1 *i*PSC-derived retinal ganglion cells // Acta Neuropathologica Communications. 2025; 13(28): 1-13.
16. Chen T.-H., Lin S.-H., Lee M.-Y., Wang H.-C., Tsai K.-F., Chou C.-K. Mitochondrial alterations and signatures in hepatocellular carcinoma // Cancer and Metastasis Reviews. 2025; 44(34): 1-19.
17. Li X., Chen W., Jia Z., Xiao Y., Shi A., Ma X. Mitochondrial dysfunction as a pathogenesis and therapeutic strategy for metabolic dysfunction-associated steatotic liver disease // Int. J. Mol. Sci. 2025; 26(4256): 1-29.
18. Crompton M., Barksby E., Johnson N., Capano M. Mitochondrial intermembrane junctional complexes and their involvement in cell death // Biochimie. 2002: 84(2-3): 143-152.
19. Akopova O.V. Rol mitoxondrialnoy pory v transmembrannom obmene kalsiya v mitoxondriyax // Ukr. bioxim. jurn. 2008; 80(3): 40-47. (*In Russian*).
20. Levchenkova O.S., Novikov V.Ye., Pojilova Ye.V. Mitoxondrialnaya pora kak mishen farmakologicheskogo vozdeystviya // Vestnik Smolenskoy gosudarstvennoy meditsinskoy akademii. 2014; 13(4): 24-33. (*In Russian*).
21. Mannella C.A. Structure and dynamics of the mitochondrial inner membrane cristae // Biochimica et Biophysica Acta. 2006; 1763: 542-548.
22. Margineantu D.H., Cox W.G., Sundell L., Sherwood S.W., Beechem J.M., Capaldi R.A. Cell cycle dependent morphology changes and associated mitochondrial DNA redistribution in mitochondria of human cell lines // Mitochondrion. 2002; 1(5): 425-435.
23. Tsai C.W., Wu Y., Pao P.C. et al. Proteolytic control of the mitochondrial calcium uniporter complex // Proceedings of the National Academy of Sciences. 2017; 114(17): 4388-4393.
24. Zahedi A., On V., Phandthong R., Chaili A., Remark G., Bhanu B., Talbot P. Deep analysis of mitochondria and

cell health using machine learning // Scientific reports. 2018; 8(1): 1-15.

25. Bulthuis E.P., Adjobo-Hermans M.J., Willems P.H. et al. Mitochondrial morphofunction in mammalian cells //Antioxidants & Redox Signaling. 2019; 30(18): 2066-2109.
26. Seok J., Jun S., Lee J.O., Kim G.J. Mitochondrial dynamics in placenta-derived mesenchymal stem cells regulate the invasion activity of trophoblast // Int. J. Mol. Sci. 2020; 21(8599): 1-15.
27. Xusainova G.X. Proizvodnye neyroaktivnyx aminokislot kak regulyatory funksionalnogo sostoyaniya mitoxondriy vozbudimyx tkaney krys v norme i pri eksperimentalnyx patologiyax // Diss. ... na soisk. nauchn. step. k.farm.n. – Volgograd, 2022. pp.181. (*In Russian*).
28. Huang D., Chen S., Xiong D., Wang H., Zhu L., Wei Y., Li Y., Zou S. Mitochondrial dynamics: Working with the cytoskeleton and intracellular organelles to mediate mechanotransduction // Aging and Disease. 2023; 14(5): 1511-1532.
29. Monzel A.S., Enriquez J.A., Picard M. Multifaceted mitochondria: Moving mitochondrial science beyond function and dysfunction // Nature Metabolism. 2023; 5: 532-546.
30. Naghdi S., Mishra P., Roy S.S., Weaver D., Walter L., Davies E., Antony A.N., Lin X., Moehren G., Feitelson M.A., Reed C.A., Lindsten T., Thompson C.B., Dang H.T., Hoek J.B., Knudsen E.S., Hajnoczky G. $VDAC_2$ and Bak scarcity in livermitochondria enables targeting hepatocarcinoma while sparing hepatocytes // Nature Communications. 2025; 16(2416): 1-20.
31. Desagher S., Martinou J.-C. Mitochondria as the central control point of apoptosis // Trends in Cell Biology. 2000; 10: 369-377.
32. Paul M.K., Rajinder K., Mukhopadhyay A.K. Characterization of rat liver mitochondrial permeability transition pore by using mitochondrial swelling assay // African Journal of Pharmacy and Pharmacology. 2008; 2(2): 014-021.
33. Cao J.L., Adaniya S.M., Cypress M.W., Suzuki Y., Kusakari Y., Jhun B.S., O.-Uchi J. Role of mitochondrial Ca^{2+} homeostasis in cardiac muscles // Arch Biochem Biophys. 2019; 15(663): 276-287.
34. Korotkov S.M., Novozhilov A.V. The joint influence of Tl^{+} and thiol-modifying agents on rat liver mitochondrial parameters *in vitro* // Int. J. Mol. Sci. 2022; 23(8964): 1-26.
35. Endlicher R., Drahota Z., Stefkova K., Cervinkova Z., Kucera O. The mitochondrial permeability transition pore – Current knowledge of its structure, function, and regulation, and optimized methods for evaluating its functional state // Cells. 2023; 27:12(9):1273: 1-17.
36. Duchen M.R. Roles of mitochondria in health and disease free crossmark: Check for updates // Diabetes. 2004; 53(1): S96-S102.
37. De Marchi E., Bonora M., Giorgi C., Pinton P. The mitochondrial permeability transition pore is a dispensable element for mitochondrial calcium efflux // Cell Calcium. 2014; 56(1): 1-13.
38. Halestrap A.P., Richardson A.P. The mitochondrial permeability transition: A current perspective on its identity and role in ischaemia/reperfusion injury // J. Mol. Cell. Cardiol. 2015; 78: 129-141.
39. Harisseh R., Abrial M., Chiari P., Al-Mawla R., Villedieu C., Tessier N., Bidaux G., Ovize M., Gharib A. A modified calcium retention capacity assay clarifies the roles of extra- and intracellular calcium pools in mitochondrial permeability transition pore opening // J. Biol. Chem. 2019; 21:294(42): 15282-15292.
40. Wacquier B., Combettes L., Dupont G. Dual dynamics of mitochondrial permeability transition pore opening // Scientific Reports. 2020 (3924)10: 1-10.

41. Zhou S., Yu Q., Zhang L., Jiang Z. Cyclophilin *D*-mediated mitochondrial permeability transition regulates mitochondrial function // Review Curr. Pharm. Des. 2023; 29(8): 620-629.
42. Halestrap A.P. What is the mitochondrial permeability transition pore? // Review J. Mol. Cell. Cardiol. 2009; 46(6): 821-831.
43. Miura T., Tanno M. The *mPTP* and its regulatory proteins: Final common targets of signalling pathways for protection against necrosis // Cardiovascular Research. 2012; 94(2): 181-189.
44. Javadov S., Jang1 S., Parodi-Rullan R., Khuchua Z., Kuznetsov A.V. Mitochondrial permeability transition in cardiac ischemia-reperfusion: Whether cyclophilin D is a viable target for cardioprotection? // Cell. Mol. Life Sci. 2017; 74: 2795-2813.
45. Shoshan-Barmatz V., Shteinfer-Kuzmine A., Verma A. $VDAC_1$ at the intersection of cell metabolism, apoptosis, and diseases // Biomolecules. 2020; 10(1485): 1-40.
46. Robichaux D.J., Harata M., Murphy E., Karch J. Mitochondrial permeability transition pore-dependent necrosis // Journal of Molecular and Cellular Cardiology. 2023; 174: 47-55.
47. Perez-Serna A.V., Guzman-Llorens D., Dos Santos R.S., Marroqui L. Bcl-2 and *Bcl*-xL in diabetes: Contributions to endocrine pancreas viability and function // Biomedicines. 2025; 13(223): 1-27.
48. Crompton M. The mitochondrial permeability transition pore and its role in cell death // Biochem. J. 1999; 341: 233-249.
49. Yoshida T.N., Takashi S.S. Mitochondrial membrane permeability transition and cell death // Biochim. Biophys. Acta. Bioenerg. J. 2005; 1757: 1297-1300.
50. Vorobeva N.V., Kondratenko I.V., Vaxlyarskaya S.S., Chernyak B.V., Pinegin B.V. Rol mitoxondrialnoy pory v effektornyx funksiyax neytrofilov cheloveka // Immunologiya. 2020; 41(1): 42-45. (*In Russian*).
51. Karch J., Molkentin J.D. Identifying the components of the elusive mitochondrial permeability transition pore // PNAS. 2014; 111(29): 10396-10397.
52. Baburina Yu.L., Sotnikova L.D., Krestinina O.V. Uchastie translokatornogo belka v induksii i regulyasii otkrytiya pory nespetsificheskoy pronitsaemosti v mitoxondriyax kletok gliomy // Sitologiya. 2022; 64(6): 581-590. (*In Russian*).
53. Flierl A., Schriner S.E., Hancock S., Coskun P.E., Wallace D.C.The mitochondrial adenine nucleotide transporters in myogenesis // Review Free Radic. Biol. Med. 2022; 1:188: 312-327.
54. Ray P.D., Huang B.W., Tsuji Y. Reactive oxygen species (ROS) homeostasis and redox regulation in cellular signaling // Cell Signal. 2012; 24(5): 981-990.
55. Chen W., Guo C., Feng H., Chen Y. Mitochondria: Novel mechanisms and therapeutic targets for secondary brain injury after intracerebral hemorrhage // Front. Aging Neurosci. 2021; 12(615451): 1-10.
56. Bai J., Zhang L., He L., Zhou Y. Altered mitochondrial function: A clue therapeutic strategies between metabolic dysfunction-associated steatotic liver disease and chronic kidney disease? // Front. Nutr. 2025; 12(1613640): 1-25.
57. Haworth R.A., Hunter D.R. The Ca^{2+}-induced membrane transition in mitochondria. II. Nature of the Ca^{2+} trigger site // Arch. Biochem. Biophys. 1979; 195: 460-467.
58. Zoratti M., Szabo I. The mitochondrial permeability transition // Biochim. Biophys. Acta. 1995; 1241: 139-176.
59. Halestrap A.P., Brenner C. The adenine nucleotide translocase: A central component of the mitochondrial permeability transition pore and key player in cell death // Curr. Med. Chem. 2003; 10: 1507-1525.

60. Halestrap A.P. The C-ring of the F_1F_o ATP-synthase forms the mitochondrial permeability transition pore: A critical appraisal // Front. Oncol. 2014; 4(234): 1-3.
61. Shiryaeva A.P., Baidyuk E.V., Arkadieva A.V., Okovitiy S.V., Morozov V.I., Sakut G.A. State of liver mitochondrial respiratory chain in rats with experimental toxic hepatitis // Cell and Tissue Biology. 2007; 1(2): 169-177.
62. Johannsen D.L., Ravussin E. The role of mitochondria in health and disease // Curr Opin Pharmacol. 2009; 30:9(6): 780-786.
63. Hoffman D.L. Screening mitochondrial toxicity. New cost- and time-efficient assay kits designed to profile compounds // Genetic Engineering & Biotech News. 2014; 34(7): 28-29.
64. Surugihalli C., Porter T.E., Chan A., Farley L.S., Maguire M., Zhang C., Kattapuram N., Muyyarikkandy M.S., Liu H.-C., Sunny N.E. Hepatic mitochondrial oxidative metabolism and lipogenesis synergistically adapt to mediate healthy embryonic-to-neonatal transition in chicken // Scientific Reports. 2019; 9(20167): 1-14.
65. Middleton P., Vergis N. Mitochondrial dysfunction and liver disease: Role, relevance, and potential for therapeutic modulation // Ther Adv. Gastroenterol. 2021; 14: 1-19.
66. Bernardi P. Mitochondrial transport of cations: channels, exchangers, and permeability transition // Physiol. Rev. 1999; 79(4): 1127-1155.
67. Fuster D., Zhang J., Shi M., Bobulescu A., Andersson S., Moe O. Characterization of the sodium/hydrogen exchanger NHA_2 // J. Am. Soc. Nephrol. 2008; 19(8): 1547-1556.
68. Gunter T., Sheu S. Characteristics and possible functions of mitochondrial Ca^{2+}-transport mechanisms // Biochim Biophys Acta. 2009; 1787(11): 1291-1308.
69. Cardoso A.R., Queliconi B.B., Kowaltowski A.J. Mitochondrial ion transport pathways: Role in metabolic diseases // Biochimica et Biophysica Acta. 2010; 1797(6-7): 832-838.
70. Gunter T.E., Yule D.I., Gunter K.K., Eliseev R., Salter J. Calcium and mitochondria // FEBS Letters. 2004; 567(1): 96-102.
71. Belosludsev K.N., Dubinin M.V., Belosludseva N.V., Mironova G.D. Transport ionov Ca^{2+} mitoxondriyami: Mexanizmy, molekulyarnye struktury i znachenie dlya kletki // Bioximiya. 2019; 84(6): 759-775. (*In Russian*).
72. Garlid K., Paucek P. Mitochondrial potassium transport: The K^+ cycle // Biochimica et Biophysica Acta. 2003; 1606(1-3): 23-41.
73. Vadzyuk O.B., Kosterin S.A. Indutsirovannoe diazoksidom nabuxanie mitoxondriy miometriya krys kak svidetelstvo aktivatsii ATF-chuvstvitelnogo K^+-kanala // Ukr. bioxim. jurn. 2008; 80(5): 45-51. (*In Russian*).
74. Akopova O.V. Vliyanie aktivatora ATR-zavisimogo K^+-kanala diazoksida na siklosporinchuvstvitelnuyu poru v mitoxondriyax pecheni krys // Ukr. bioxim. jurn. 2011; 83(3): 37-47. (*In Russian*).
75. Komilov E.J., Ergashev N.A., Abdullaeva G.T., Gayibov U.G., Asrarov M.I., Komilov B.J. Lyuteolin va pulikarin flavonoidlarining mitoxondriya ATFga bog‘liq kaliy kanaliga tasiri // O‘zMU xabarlari. 2018; 3/1: 125-128. (*In Uzbek*).
76. Shemarova I.V., Korotkov S.M., Nesterov V.P. Ca^{2+}-zavisimye mitoxondrialnye mexanizmy kardioproteksii // Jurnal evolyusionnoy bioximii i fiziologii. 2020; 56(4): 272-284. (*In Russian*).
77. Abdulxakova G.V., Komilov E.Dj., Ergashev N.A., Asrarov M.I., Maxmudov R.R., Kenjaeva M.F. Vliyanie taninov geksagalloil-glyukozy i geptagalloil-glyukozy na ATF-zavisimye kalievye kanaly v usloviyax *in vitro* // “Universum: ximiya i biologiya” (Elektron. nauchn. jurn.). 2025; 5(131): 59-64. (*In Russian*).

78. Szewczyk A., Bednarczyk P., Kulawiak B., Zochowska M., Kalenik B., Lewandowska J., Pytlak K., Gałecka Sh., Wrzosek A., Koprowski P. Mitochondrial potassium channels: New propertiyes and functions // Biochimica et Biophysica Acta – Bioenergetics. 2025; 1866(2): 1-9.
79. Antonenko Yu.N., Avetisyan A.V., Bakeeva L.E., Chernyak B.V., Chertkov V.A., Domnina L.V., Ivanova O.Yu., Izyumov D.S., Xaylova L.S., Klishin S.S., Korshunova G.A., Lyamzaev K.G., Muntyan M.S., Nepryaxina O.K., Pashkovskaya A.A., Pletyushkina O.Yu., Pustovidko A.V., Roginskiy V.A., Rokitskaya T.I., Ruuge E.K., Saprunova V.B., Severina I.I., Simonyan R.A., Skulachev I.V., Skulachev M.V., Sumbatyan N.V., Sviryaeva I.V., Tashlitskiy V.N., Vasilev Yu.M., Vysokix M.Yu., Yagujinskiy L.S., Zamyatnin A.A., Skulachev V.P. Proizvodnoe plastoxinona, adresovannoe v mitoxondrii, kak sredstvo, preryvayushee programmu stareniya 1. Kationnye proizvodnye plastoxinona: Sintez i issledovanie *in vitro* // Bioximiya. 2008; 73(12): 1589-1606. (*In Russian*).
80. Nesci S., Trombetti F., Pagliarani A., Ventrella V., Algieri C., Tioli G., Lenaz G. Molecular and supramolecular structure of the mitochondrial oxidative phosphorylation system: Implications for pathology // Life. 2021; 11(242): 1-53.
81. Ramanathan R., Ali A.H., Ibdah J.A. Mitochondrial dysfunction plays central role in nonalcoholic fatty liver disease // Int. J. Mol. Sci. 2022; 23(7280): 1-20.
82. Kit O.I., Frantsiyants E.M., Shikhlyarova A.I., Neskubina I.V., Ilchenko S.A. Extracellular mitochondria – promising diagnostic agents // Research and Practical Medicine Journal. – 2024. – No. 11(1). – Pp. 40-53.
83. Sadovnikov F.A., Polyakova M.V., Astaxova A.I., Alchinova I.B. Morfologicheskie izmeneniya mitoxondriy stromalnyx kletok kostnogo mozga myshey, podvergnutyx modelirovannoy gipokinezii // Patogenez. 2024; 22(2): 84-88. (*In Russian*).
84. Brustovetsky N., Tropschug M., Heimpel S., Heidkamper D., Klingenberg M. A large Ca^{2+}-dependent channel formed by recombinant ADP/ATP carrier from Neurospora crassa resembles the mitochondrial permeability transition pore // Biochemistry. 2002; 41: 11804-11811.
85. De Pinto V., Messina A., Accardi R., Aiello R., Guarino F., Tomasello M.F., Tommasino M., Tasco G., Casadio R., Benz R., De Giorgi F., Ichas F., Baker M., Lawen A. New functions of an old protein: The eukaryotic porin or voltage dependent anion selective channel (VDAC) // Ital. J. Biochem. 2003; 52(1): 17-24.
86. Galieque S., Tinel N., Casellas P. The peripheral benzodiazepine receptor: A promising therapeutic drug target. // Curr. Med. Chem. 2003; 10(16): 1563-1572.
87. Halestrap A.P. Mitochondrial permeability: Dual role for the ADP/ATP translocator? // Nature. 2004; 430: 983.
88. Tsujimoto Y.N., Takashi S.S. Mitochondrial membrane permeability transition and cell death // Biochim. Biophys. Acta. Bioenerg. J. 2005; 1757: 1297-1300.
89. Murakov S.V., Vospelnikov N.D. Mitoxondrialnye megapory v jizni kletki // Voprosy biol., med. i farmatsevticheskoy ximii. 2006; 2: 44-50. (*In Russian*).
90. Sudakov N.P., Nikiforov S.B., Konstantioov Yu.M., Yakubov L.A., Novikova N.A., Karamysheva A.N. Mexanizmy uchastiya mitoxondriy v razvitii patologicheskix protsessov, soprovojdayushixsya ishemiey i reperfuziey // Byulleten VSNS SO RAMN. 2006; 5(51): 332-336. (*In Russian*).
91. Zorov D.B., Isaev N.K., Plotnikov Ye.Yu., Silachev D.N. Perspektivy mitoxondrialnoy meditsiny // Bioximiya. 2013; 78(9): 1251-1264.
92. Shimanskaya T.V., Strutinskaya N.A., Vavilova G.L. i dr. Siklosporin A – chuvstvitelnaya mitoxondrialnaya pora kak mishen kardioprotektornogo deystviya donora serovodoroda // Ros. Fiziol. J. im. I.M.Sechenova. 2013; 99(2):

261-272. (*In Russian*).

93. Pojilova Ye.V., Levchenkova O.S., Novikov V.Ye. Regulyatornaya rol mitoxondrialnoy pory i vozmojnosti yeyo farmakologicheskoy modulyasii // Obzory po klinicheskoy farmakologii i lekarstvennoy terapii. 2014; 12(3): 13-19. (*In Russian*).
94. Dubinin M.V., Adakeeva S.I., Samartsev V.N. Long-chain α,ω-dioic acids as inducers of cyclosporin A – insensitive nonspecific permeability of the inner membrane of liver mitochondria loaded with calcium or strontium ions // Biochemistry. 2013; 78(4): 412-417.
95. Gornall A.G., Bardawill C.J., David M.M. Determination of serum proteins by means of the biuret reaction // J. Biol. Chem. 1949; 177(2): 751-766.
96. Schneider W.C., Hogeboom G.H. Cytochemical studies of mammalian tissues: The isolation of cell components by differential centrifugation // Cancer Research. 1951; 11(1): 1-22.
97. Anderson N.G. Studies on isolated cell components. VIII. High resolution gradient differential centrifugation // Exp. Cell Res. 1955; 9(3): 446-459.
98. Jonson D., Lardy H. Isolation of liver or kidney mitochondria // In: "Methods in Enzymology". New York ("Acad. Press"): 1969, pp.94-96.
99. Mosolova I.M., Gorskaya I.A., Shols K.F. i dr. Vydelenie intaktnyx mitoxondriy iz pecheni krys // Metody sovremennoy bioximii. Moskva. Izd-vo "Nauka", 1975, pp.45-47. (*In Russian*).
100. Watters C. A one-step biuret assay for protein in the presence of detergent // Anal. Biochem. 1978; 8(2): 695-698.
101. Shiryaeva A.P., Baydyuk ye.V., Arkadev A.V., Okovityy S.V., Morozov V.I., Sakuta G.A. Sostoyanie dyxatelnoy sepi mitoxondriy pecheni krys s eksperimentalnym toksicheskim gepatitom // Sitologiya. 2007; 49(2): 125-132. (*In Russian*).
102. Lanza I.R., Nair S.K. Functional assessment of isolated mitochondria *in vitro* // Methods Enzymol. 2009; 457: 349-372.
103. Michelsen U., Von Hagen J. Isolation of subcellular organelles and structures // Methods Enzymol. 2009; 463: 305-328.
104. Sosimchik I.A., Cherkashina D.V., Somov A.Yu., Petrenko A.Yu. Opredelenie aktivnyx form kisloroda v mitoxondriyax pecheni krys s ispolzovaniem digidrorodamina 123 // Visnik problem biologiï i meditsini. 2009; 4: 36-40.
105. Yegorova M.V., Afanasev S.A. Vydelenie mitoxondriy iz kletok i tkaney jivotnyx i cheloveka: sovremennye metodicheskie priemy // Sibirskiy meditsinskiy jurnal. 2011; 26(1)1: 22-28. (*In Russian*).
106. Gonchar O.A., Mankovska I.N. Moderate intermittent hypoxia/hyperoxia: implication for correction of mitochondrial dysfunction // Cent. Eur. J. Biol. 2012; 7(5): 801-809.
107. Asrarov M.I., Komilov E.Dj., Ergashev N.A., Pozilov M.K., Eshbakova K.A., Toshmatov Z.A., Toshbekova M.X. Mexanizm deystviya flavona lyuteolina na funksii mitoxondriy pecheni krys // Voprosy biologicheskoy, meditsinskoy i farmatsevticheskoy ximii. 2015; 12: 38-43. (*In Russian*).
108. Sinitskiy A.I., Kochkina A.T., Grobovoy S.I. Vliyanie 2-etil-6-metil-3-gidroksipiridina na funksionalnoe sostoyanie mitoxondriy pecheni krysy *in vitro* // Ximiko-farmatsevticheskiy jurnal. 2021; 55(1): 8-12. (*In Russian*).
109. Vakhobjonovna A.G., Komilov E.J., Abdullaev I.Z., Ergashev N.A., Makhmudov R.R., Asrarov M.I. Tannins as modulators in the prevention of mitochondrial dysfunction // Trends in Sciences. 2025; 22(8):10436: 1-12.

110. Peterson G.L. A simplification of the protein assay method of Lowry et al. which is more generally applicable // Analytical Biochemistry. 1977; 83(2): 346-356.
111. Johnson D., Lardy H.A. Isolation of liver or kidney mitochondria // Meth. Enzymol. 1967; 10: 94-101.
112. Petronilli V. et al. Physiological effectors modify voltage sensing by the cyclosporine A-sensitive permeability transition pore of mitochondria // J. Biol. Chem. 1993; 268(29): 939-945.
113. Rybalchenko V.K., Koganov M.M. Struktura i funksii membran // Kiev. – Izd-vo “Vysshaya shkola”, 1998. pp.312. (*In Russian*).
114. He L., Lemasters J. Heat shock suppresses the permeability transition in rat liver mitochondria // J. Biol. Chem. 2003; 278(19): 16755-16760.
115. Rahimova N.M. Eksperimental qandli diabetda jigar mitoxondriyalarining funksional holatiga fitoekdisteroidlar va nerobolning tasiri // T.f.n. ilmiy darajasini olish uchun diss. ... avtoreferati. Toshkent, 2010. pp.3-25. (*In Uzbek*).
116. Alisultanova N.J., Vaxnina N.A., Shadrina V.D., Sidorova L.P., Boyko Ye.R., Chupaxin O.N. Izmenenie aktivnosti suksinatdegidrogenazy mitoxondriy pecheni krys pod vozdeystviem soedineniy klassa 1,3,4-tiadiazina v usloviyax *in vitro* // Izvestiya Samarskogo nauchnogo sentra Rossiyskoy akademii nauk. 2014; 16.5(4): 1205-1208. (*In Russian*).
117. Pozilov M.K., Ergashev N.A., Asrarov M.I., Eshbakova K.A. Salvifolin diterpenoidining jigar mitoxondriyasi nafas olishi va oksidlanishli fosforlanishga tasiri // Infeksiya immunitet i farmakologiya jurnali. 2015; 1: 127-134. (*In Uzbek*).
118. Pozilov M.K. Eksperimental diabetda jigar mitoxondriyasi funksiyasining buzilishlari hamda ularni salvifolin va glikorazmulin bilan korreksiyalash // Biologiya fanlari bo‘yicha falsafa doktori (PhD) ilmiy darajasini olish uchun diss. ... avtoreferati. Toshkent, 2017, pp.5-50. (*In Uzbek*).
119. Pozilov M.K., Asrarov M.I., Abdullajanova N.G. Mavlyanov S.M. Deystvie polifenola gossitana na membrannye protsessy mitoxondriy pecheni krys // O‘zMU Xabarlari. 2018; 3/1: 203-207. (*In Russian*).
120. Xalilov R.A., Xazrieva S.I., Djafarova A.M., Abdullaev V.R. Bioenergeticheskie xarakteristiki mitoxondriy pecheni krys pri nizkix temperaturax tela // Voprosy biologicheskoy, meditsinskoy i farmatsevticheskoy ximii. 2019; 22(5): 35-41. (*In Russian*).
121. Mirzaolimov E., Raximov A., Maxmudova Sh., Nishanbaev S., Abdullaev G. Yurak mitoxondriyasi passiv ion utkazuvchanligiga sofora flavonolonozidning tasiri // O‘zMU xabarlari. 2021; 3/2: 72-75. (*In Uzbek*).
122. Abdullaeva G.T. Polifenol birikmalarning mitoxondriya funksional parametrlariga tasiri hamda ularni antigipoksik faolligi // Biologiya fanlari bo‘yicha fan doktori ilmiy darajasini olish uchun diss. ... Toshkent, 2021; 191. (*In Uzbek*).
123. Sobirova G.K., Pozilov M.K. Effect of a triazole derivative on mitochondrial liver dysfunction in alloxan diabetes // Science and Innovation. International Scientific journal. 2023; 2(5): 238-240.
124. Eisenhofer S., Tookos F., Hense B.A., Schulz S., Filbir F., Zischka H. A mathematical model of mitochondrial swelling // A mathematical model of mitochondrial swelling // Eisenhofer et al. BMC Research Notes. 2010; 3(67): 1-14.
125. Asrarov M.I., Pozilov M.K., Ergashev N.A., Raxmatullaeva M.M. Vliyanie gipoglikemicheskogo sredstva glikorazmulina na funksionalnoe sostoyanie mitoxondriy pri streptozototsin-indutsirovannom diabete // Problemy endokrinologii. 2014; 3: 38-42. (*In Russian*).

126. Mishra J., Davani A.J., Natarajan G.K., Kwok W.-M., Stowe D.F., Camara A.K.S. Cyclosporin A increases mitochondrial buffering of calcium: An additional mechanism in delaying mitochondrial permeability transition pore opening // Cells. 2019; 7:8(9):1052: 1-23.
127. Dubinin M.V., Tenkov K.S., Starinets V.S., Stepanova A.Ye., Belosludsev K.N., Samarsev V.N. Vozrastnaya i tkanevaya osobennosti induksii α,ω,-geksadekandikarbonovoy kislotoy kalsiy-zavisimoy permeabilizatsii mitoxondriy krys // Jurnal evolyusionnoy bioximii i fiziologii. 2019; 55(1): 73-75. (*In Russian*).
128. Bernardi P., Gerle C., Halestrap A.P., Jonas E.A., Karch J., Mnatsakanyan N., Pavlov E., Sheu S.-S., Soukas A.A. Identity, structure, and function of the mitochondrial permeability transition pore: controversies, consensus, recent advances, and future directions // Cell Death & Differentiation. 2023; 30: 1869-1885.
129. Carraro M., Bernardi P. The mitochondrial permeability transition pore in Ca^{2+} homeostasis // Cell Calcium. 2023; 111: 1-8.
130. Kroemer G., Dallaporta B., Resche-Rigon M. The mitochondrial death/life regulator in apoptosis and necrosis // Annu. Rev. Physiol. 1998; 60(1): 619-642.
131. Halestrap A.P., Connem C.P., Griffiths E., Kerr P.M. Cyclosporin A binding to mitochondrial cyclophilin inhibits the permeability transition pore and protects hearts from ischaemia/reperfusion injury // Molecular and Cellular Biochemistry. 1997; 174: 167-172.
132. Lonobile C., Di Nubila A., Simone R., Hushi M., Barbieri S.S. The mitochondrial permeability transition pore in platelets: Mechanisms, physiological roles, and therapeutic perspectives // Antioxidants. 2025; 14(923): 1-21.
133. Muxamedieva I.B., Djumaeva M., Isroiljonov S., Jurakulov Sh.N., Pozilov M. Oksidativ stress sharoitida kalamush yurak mitoxondriyasi yuqori o‘tkazuvchan porasiga ayrim izoxinolin alkaloidlarning tasiri // Universal xalqaro ilmiy jurnal. 2024; 1(4): 6-8. (*In Uzbek*).
134. Pozilov M., Muxamedova I., Raxmatullaeva M., Isroiljonov S., Jurakulov Sh. Yurak mitoxondriyasi ATFga bog‘liq kaliy kanaliga 1-(2-xlor-4,5-metilen-dioksifenil)-2-gidroksietil-6,7-dimetoksi-1,2,3,4-tetragidroksi-izoxinolin alkaloidining tasiri // Farmatsevtika jurnali. 2022; 5: 76-81. (*In Uzbek*).
135. Isamuxamedova D.R., Raimova K.V., Bobojanova F.T. Abdullaeva G.T. 2,3-Digidroksi-glutamat kislotasining mitoxondriyalardagi Sa^{2+}-bog‘liq megaporaga tasiri // Materialy mejdunarodnoy nauchno-prakticheskoy konferensii “TAShNIIVS: Vchera, segodnya i zavtra”. Tashkent, 2021; Farmatsiya, immunitet i vaksina. 2021; 2: 47-48. (*In Uzbek*).