 © Authors of the article / Authors of the article

ПАТОЛОГИЯ ЖИВОТНЫХ, МОРФОЛОГИЯ, ФИЗИОЛОГИЯ, ФАРМАКОЛОГИЯ И ТОКСИКОЛОГИЯ/ANIMAL PATHOLOGY, MORPHOLOGY, PHYSIOLOGY, PHARMACOLOGY AND TOXICOLOGY

DOI: https://doi.org/10.60797/IRJ.2026.173.63 EDN: XCHSFY

CORRECTION OF THE BILE ACID POOL SHIFT IN LOACHES WITH EXPERIMENTAL HEPATOPATHIES USING SULFOLITHOCHOLIC ACID

Research article

Ponamarev V.S.[1, *]
[1] ORCID : 0000-0002-6852-3110;
[1] St. Petersburg State University of Veterinary Medicine, Saint-Petersburg, Russian Federation

* Corresponding author (psevdopyos[at]mail.ru)

Suggested: 05.10.2026; Accepted: 23.10.2026; Published: 17.11.2026

Abstract

One of the most informative but still insufficiently studied markers of the hepatobiliary system in fish is the bile acid profile. In healthy fish, the bile acid pool is maintained by a balanced ratio of primary and secondary bile acids. Under hepatic pathology, this balance shifts toward more hydrophobic and cytotoxic fractions, which aggravates hepatocyte damage and supports a vicious circle. In the present study, the possibility of correcting the bile acid pool shift in common loach (*Misgurnus fossilis*) with experimental hepatopathies was assessed using exogenous sulfolithocholic acid (SLCA). The study demonstrated that SLCA administration in feed at doses of 200 and 400 mg/kg for 35 days promoted a dose-dependent restoration of the bile acid ratio, reduced the share of hydrophobic deoxycholic acid, and improved classical biochemical liver markers.

Keywords: loach, bile acids, bile acid pool, sulfolithocholic acid, lithocholic acid sulfate, hepatic steatosis, toxic hepatopathy.

КОРРЕКЦИЯ СДВИГА ПУЛА ЖЕЛЧНЫХ КИСЛОТ У ВЬЮНОВ С ЭКСПЕРИМЕНТАЛЬНЫМИ ГЕПАТОПАТИЯМИ СУЛЬФОЛИТОХОЛЕВОЙ КИСЛОТОЙ

Научная статья

Понамарёв В.С.[1, *]
[1] ORCID : 0000-0002-6852-3110;
[1] Санкт-Петербургский государственный университет ветеринарной медицины, Санкт-Петербург, Российская Федерация

* Корреспондирующий автор (psevdopyos[at]mail.ru)

Предложена: 05.10.2026; Принята: 23.10.2026; Опубликована: 17.11.2026

Аннотация

Одним из наиболее информативных, но все еще недостаточно изученных маркеров гепатобилиарной системы рыб является уровень желчных кислот. У здоровых рыб уровень желчных кислот поддерживается сбалансированным соотношением первичных и вторичных желчных кислот. При патологии печени этот баланс смещается в сторону увеличения количества гидрофобных и цитотоксических фракций, что усугубляет повреждение гепатоцитов и поддерживает порочный круг. В настоящем исследовании оценивалась возможность коррекции изменения пула желчных кислот у вьюна обыкновенного (*Misgurnus fossilis*) с экспериментальными гепатопатиями с использованием экзогенной сульфолитохолевой кислоты (SLCA). Исследование показало, что введение SLCA в корм в дозах 200 и 400 мг/кг в течение 35 дней способствовало дозозависимому восстановлению соотношения желчных кислот, снижению доли гидрофобной дезоксихолевой кислоты и улучшению классических биохимических показателей печени.

Ключевые слова: вьюн, желчные кислоты, пул желчных кислот, сульфолитохолевая кислота, сульфат литохолевой кислоты, стеатоз печени, токсическая гепатопатия.

Introduction

In recent decades, aquaculture has faced a serious challenge: the widespread occurrence of metabolic liver diseases in fish. The transition to high-protein and high-fat feeds, partial replacement of fishmeal with plant components, and deterioration of the ecological state of water bodies create prerequisites for hepatic steatosis and toxic hepatopathies [1], [8], [9]. Loaches, as a valuable object of fishery and aquaculture, possess several physiological features that make them especially susceptible to lipid metabolism disorders. Their liver plays a key role in fat metabolism, and any deviation in feeding or water quality immediately affects its functional state [1], [9].

Bile acids are synthesized exclusively in hepatocytes from cholesterol. They perform not only an emulsifying function in the intestine but also act as signaling molecules regulating lipid and glucose homeostasis through activation of nuclear receptor FXR and membrane receptor TGR5 [1], [3], [5]. In healthy fish, the bile acid pool is characterized by a certain ratio of primary bile acids, such as cholic acid (CA) and chenodeoxycholic acid (CDCA), and secondary bile acids, including deoxycholic acid (DCA) and lithocholic acid (LCA). In liver pathologies, this ratio regularly shifts: the proportion of hydrophobic and cytotoxic fractions increases, which exacerbates hepatocyte damage and creates a vicious circle [3], [5], [8].

 © Authors of the article / Authors of the article

Traditional approaches to the correction of hepatopathies in fish include diet therapy, plant-derived hepatoprotectors, and antioxidants [5], [6], [7]. However, these methods do not always affect the primary cause of disorders, namely the imbalance of the bile acid pool. In clinical practice in warm-blooded animals, bile acid preparations have long been used for cholestatic liver diseases [2], [3], [4], [7]. For fish, this approach remains relatively new, although some studies already demonstrate its potential [4], [8].

Exogenous bile acids administered with feed can not only compensate for the deficiency of endogenous bile acids but also modulate the activity of lipogenesis and β-oxidation enzymes through FXR activation. In addition, they influence the composition of intestinal microbiota, which indirectly affects secondary bile acid metabolism and the general state of the hepatobiliary system [9], [10], [11]. Sulfolithocholic acid is a sulfated derivative of lithocholic acid. Sulfation increases its hydrophilicity, reduces its cytotoxic potential, and promotes its excretion through the enterohepatic route. Therefore, SLCA may be of interest as a pharmacological agent capable of displacing hydrophobic bile acids from the pool and restoring bile acid homeostasis [1], [3], [11].

Loaches are a convenient model object for studying these issues due to their relative hardiness, availability, and similarity of metabolic pathways with other commercial cyprinids. Unlike zebrafish, which are widely used in laboratory studies, loaches have larger sizes, allowing repeated blood sampling without lethal outcome and obtaining sufficient serum volumes for detailed biochemical analysis [8],[10], [11].

The aim of this study was to evaluate the effectiveness of exogenous sulfolithocholic acid for correcting the bile acid pool shift in loaches with experimentally induced hepatobiliary pathologies. The following tasks were implemented: modeling hepatic steatosis and toxic liver damage using a high-fat diet and lead acetate solution; determination of the initial bile acid pool and classical biochemical liver markers; assessment of the dynamics of bile acid pool recovery during SLCA administration at different doses; and comparative analysis of the effectiveness of the studied doses [12], [13], [14], [15].

Research methods and principles

Sexually mature common loaches (*Misgurnus fossilis*) weighing 45–65 g were used as objects of study. The fish were obtained from a fish farm in the Leningrad Region and underwent a 21-day quarantine in a laboratory aquarium complex. The fish were kept in 240 L aquariums at a temperature of 21 ± 1°C, pH 7.0–7.5, hardness 7–9°dH, and aeration providing dissolved oxygen not lower than 6.5 mg/L. A natural photoperiod with a 12-hour light day was maintained using an automatic lighting timer. During quarantine, the fish received a standard commercial feed containing 36% protein and 7% fat twice a day in an amount of 1.8% of body weight [12], [13], [14], [15].

Hepatic steatosis was modeled by transferring fish to a high-fat diet for 28 days. For this purpose, the standard feed was enriched with a mixture of sunflower and linseed oils in a 1:1 ratio to a final fat content of 20%. The control group (n = 18) continued to receive the standard diet. Toxic liver damage was modeled by adding lead acetate to the aquatic environment at a concentration of 0.4 mg/L calculated as metal ion for 96 hours. The choice of lead was due to its wide occurrence in anthropogenically polluted water bodies and proven hepatotoxicity accompanied by oxidative stress [8], [12].

For correction of the identified disorders, chemically pure sodium sulfolithocholate (Sigma-Aldrich, USA) was used. Fish with hepatic steatosis were divided into four groups (n = 14 each): untreated steatosis group (HS), group with SLCA added at 200 mg/kg feed (HS+SLCA-200), and group with SLCA added at 400 mg/kg feed (HS+SLCA-400). A similar division was performed for the groups with toxic damage: toxic hepatopathy without correction (TH), TH+SLCA-200, and TH+SLCA-400. The choice of doses was based on literature data on the tolerability and bioavailability of sulfated bile acids in fish [1], [4], [8]. The pharmacoperiod was 35 days. During this period, the fish received feed with the corresponding additive. The control group (n = 18) received standard feed without additives throughout the experiment [12], [13], [14], [15].

Classical biochemical parameters were determined on a semi-automatic biochemical analyzer SINNOWA BS-3000M (China). Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were determined by a kinetic method using NADH. Alkaline phosphatase (ALP) activity was measured by a colorimetric method with p-nitrophenyl phosphate. Total bilirubin (TBil) was determined by the diazotized sulfanilic acid method. Triglycerides (TG) and total cholesterol (TC) were measured by enzymatic colorimetric methods. Total protein concentration was determined by the biuret method [12], [13], [14], [15].

The concentration of total bile acids (TBA) and individual fractions was determined by an enzymatic method using 3α-hydroxysteroid dehydrogenase. Fraction separation was performed by high-performance liquid chromatography followed by enzymatic detection. The sensitivity of the method was 0.4 μmol/L, and the coefficient of variation was no more than 3.8% [1], [8].

Statistical processing was performed using SPSS 27.0. Normality of distribution was checked by the Shapiro-Wilk test. One-way analysis of variance (ANOVA) with Tukey post hoc test was used to compare several groups. Differences were considered statistically significant at $p < 0.05$. Data are presented as arithmetic mean ± standard deviation (M ± SD) [12], [13], [14], [15].

Main results

Modeling of pathological states was accompanied by changes in both classical biochemical parameters and the bile acid pool. In the group with induced hepatic steatosis, by the 28th day of the high-fat diet, an increase in triglyceride level from 1.4 ± 0.3 to 3.2 ± 0.5 mmol/L ($p < 0.001$) and total cholesterol from 3.1 ± 0.4 to 5.0 ± 0.6 mmol/L ($p < 0.001$) was observed. ALT activity increased 1.7-fold, and AST activity increased 1.4-fold compared with the control. In the group with induced toxic hepatitis, the changes were more pronounced: ALT activity increased 2.1-fold, AST increased 1.9-fold, and total bilirubin increased from 2.4 ± 0.5 to 5.6 ± 0.9 μmol/L ($p < 0.001$), indicating a cholestatic component [1], [5], [8].

The initial bile acid pool in clinically healthy loaches was characterized by predominance of cholic acid over chenodeoxycholic acid, with a CA/CDCA ratio of 1.53 ± 0.14. The total bile acid level in blood serum was 5.1 ± 0.9 μmol/L. In hepatic steatosis, an increase in total bile acids to 13.7 ± 1.8 μmol/L ($p < 0.001$) and a decrease in the CA/CDCA ratio to

 © Authors of the article / Authors of the article

0.76 ± 0.08 were observed. In induced toxic hepatitis, the total bile acid level reached 20.6 ± 2.4 μmol/L, and the CA/CDCA ratio decreased to 0.60 ± 0.07. A tendency toward an increase in deoxycholic acid, a secondary metabolite formed under the action of intestinal microbiota, was also noted. This indicates a violation of enterohepatic circulation of bile acids and enhanced bacterial transformation of primary bile acids [8], [10], [11].

Administration of exogenous sulfolithocholic acid in feed led to pronounced positive dynamics in both experimental groups. Table 1 presents bile acid pool parameters on the 35th day of the experiment.

Table 1 - Bile acid pool of loach blood serum after 35 days of pharmacocorrection

DOI: https://doi.org/10.60797/IRJ.2026.173.63.1

Group	TBA	CA	CDCA	DCA	CA/CDCA
Control	5.1 ± 0.9	2.6 ± 0.4	1.7 ± 0.3	0.5 ± 0.1	1.53
HS (without correction)	13.7 ± 1.8*	4.5 ± 0.7*	5.9 ± 0.9*	2.9 ± 0.6*	0.76*
HS+SLCA-200	9.2 ± 1.2*#	3.6 ± 0.5*#	3.8 ± 0.6*#	1.5 ± 0.4*#	0.95#
HS+SLCA-400	7.4 ± 1.0#	3.3 ± 0.5#	2.9 ± 0.5#	1.0 ± 0.3#	1.14#
TH (without correction)	20.6 ± 2.4*	5.6 ± 0.8*	9.4 ± 1.2*	5.1 ± 0.9*	0.60*
TH+SLCA-200	12.4 ± 1.6*#	4.4 ± 0.6*#	5.1 ± 0.8*#	2.2 ± 0.5*#	0.86#
TH+SLCA-400	9.8 ± 1.3#	4.0 ± 0.6#	3.9 ± 0.6#	1.6 ± 0.4#	1.03#

*Note: μmol/L, M ± SD; * — $p < 0.05$ compared with the control; # — $p < 0.05$ compared with the corresponding untreated group*

Analysis of the data allows the conclusion that SLCA at a dose of 200 mg/kg effectively reduces the level of total bile acids and shifts the CA/CDCA ratio toward physiological values. However, a more pronounced effect was observed with SLCA at 400 mg/kg. In this group, the parameters were closest to the control values, especially in modeling hepatic steatosis. It was noted that in the group with toxic hepatitis, restoration of the bile acid pool was slower; on the 35th day of pharmacocorrection, the CA/CDCA ratio did not completely reach the control values [1], [3], [8].

The dynamics of classical biochemical parameters during pharmacocorrection are presented in Table 2.

Table 2 - Biochemical parameters of loach blood serum on the 35th day of pharmacocorrection

DOI: https://doi.org/10.60797/IRJ.2026.173.63.2

Group	ALT, U/L	AST, U/L	ALP, U/L	TBil, μmol/L	TG, mmol/L	TC, mmol/L
Control	21.5 ± 3.6	35.2 ± 5.1	24.8 ± 3.9	2.4 ± 0.5	1.4 ± 0.3	3.1 ± 0.4
HS (without correction)	36.9 ± 5.4*	49.8 ± 6.2*	31.2 ± 4.7*	3.3 ± 0.6*	3.2 ± 0.5*	5.0 ± 0.6*
HS+SLCA-200	30.4 ± 4.6*#	43.5 ± 5.7*#	28.6 ± 4.3*#	2.9 ± 0.5#	2.5 ± 0.4*#	4.1 ± 0.5*#
HS+SLCA-400	25.7 ± 4.0#	38.9 ± 5.2#	26.1 ± 4.0#	2.6 ± 0.5#	1.9 ± 0.3#	3.4 ± 0.4#
TH (without correction)	44.8 ± 6.1*	68.4 ± 8.0*	40.2 ± 5.9*	5.6 ± 0.9*	2.2 ± 0.4*	4.0 ± 0.5*
TH+SLCA-200	36.5 ± 5.2*#	56.7 ± 7.1*#	34.8 ± 5.2*#	4.2 ± 0.7*#	1.8 ± 0.3#	3.5 ± 0.5#
TH+SLCA-400	29.9 ± 4.5#	46.2 ± 6.0#	29.7 ± 4.5#	3.2 ± 0.6#	1.5 ± 0.3#	3.1 ± 0.4#

*Note: M ± SD; * — $p < 0.05$ compared with the control; # — $p < 0.05$ compared with the corresponding untreated group*

The most indicative dynamics in the context of this study was the activity of transaminases and alkaline phosphatase. In the group with modeled hepatic steatosis receiving SLCA at 400 mg/kg, ALT activity decreased by 30.3% from the initial level (from 36.9 to 25.7 U/L), AST by 21.9%, and ALP by 16.3%. In the group with toxic liver damage, the effect was less pronounced: ALT decreased by 33.3%, AST by 32.5%, and ALP by 26.1%. The triglyceride level in the HS+SLCA-400 group approached the control values (1.9 ± 0.3 mmol/L versus 1.4 ± 0.3 in the control), which confirms the ability of exogenous sulfated bile acid to restore some links of lipid metabolism [1], [3], [5].

It was noted that in all experimental groups with pharmacocorrection, a decrease in the level of deoxycholic acid, a secondary metabolite with pronounced hydrophobic and cytotoxic properties, was observed. The most pronounced decrease in

 © Authors of the article / Authors of the article

DCA (from 2.9 to 1.0 μmol/L in the HS+SLCA-400 group and from 5.1 to 1.6 μmol/L in the TH+SLCA-400 group) indicates restoration of enterohepatic circulation of bile acids and reduction of bacterial transformation of primary bile acids [8], [10], [11].

Comparative analysis of the effectiveness of SLCA doses revealed certain differences. In hepatic steatosis, the 200 mg/kg dose was moderately effective, but SLCA at 400 mg/kg more markedly restored the CA/CDCA ratio and reduced transaminase levels. In toxic liver damage, the difference between doses was more pronounced: SLCA at 400 mg/kg demonstrated a more significant hepatoprotective effect. This may be due to the fact that sulfolithocholic acid, due to its sulfated structure, is more hydrophilic, less cytotoxic, and capable of competing with hydrophobic bile acids for intestinal and hepatic transporters. It promotes displacement of hydrophobic fractions from the pool, reduces their damaging effect on hepatocyte membranes, and modulates FXR-dependent signaling [1], [3], [5], [11].

Correlation analysis revealed a close relationship between restoration of the bile acid ratio and normalization of biochemical parameters. The correlation coefficient between the CA/CDCA ratio and ALT activity was $r = –0.81$ ($p < 0.01$), between CA/CDCA and ALP $r = –0.76$ ($p < 0.01$), and between CA/CDCA and triglyceride level $r = –0.71$ ($p < 0.01$). This confirms that the bile acid pool is not merely a concomitant predictor but also an active link in the pathogenetic process, and its pharmacocorrection naturally leads to improvement of the functional state of the liver [1], [8], [14].

Conclusion

The conducted study confirmed that exogenous sulfolithocholic acid is an effective means of correcting the bile acid pool shift in loaches with experimentally induced hepatobiliary pathologies. Both hepatic steatosis induced by a high-fat diet and toxic hepatitis caused by lead acetate led to changes in the bile acid ratio: an increase in the level of total bile acids, a decrease in the ratio of cholic acid to chenodeoxycholic acid, and accumulation of hydrophobic deoxycholic acid. Administration of sulfolithocholic acid at doses of 200 and 400 mg/kg feed provided a reliable restoration of the bile acid ratio, with the most pronounced effect observed at 400 mg/kg [1], [8].

It was established that correction of the bile acid pool is accompanied by restoration of classical biochemical predictors of liver damage: a decrease in the activity of transaminases, alkaline phosphatase, bilirubin, triglycerides, and total cholesterol. The closest correlations were found between the CA/CDCA ratio and ALT activity ($r = –0.81$), which allows this parameter to be considered an integral indicator of pharmacotherapy effectiveness. Sulfolithocholic acid can be considered a promising agent for further study in aquaculture as a modulator of bile acid homeostasis and hepatoprotection [1], [8], [14].

Финансирование

Исследование выполнено при поддержке гранта Российского научного фонда № 25-26-00274, https://rscf.ru/project/25-26-00274/.

Конфликт интересов

Не указан.

Рецензия

Все статьи проходят рецензирование. Но рецензент или автор статьи предпочли не публиковать рецензию к этой статье в открытом доступе. Рецензия может быть предоставлена компетентным органам по запросу.

Funding

The study was supported by the Russian Science Foundation grant № 25-26-00274, https://rscf.ru/project/25-26-00274/.

Conflict of Interest

None declared.

Review

All articles are peer-reviewed. But the reviewer or the author of the article chose not to publish a review of this article in the public domain. The review can be provided to the competent authorities upon request.

Список литературы / References

1. Попова О.С. Метаболизм жирных кислот у рыб / О.С. Попова, Л.А. Агафонова // Международный ветеринарный вестник. — 2022. — № 1. — С. 61–65. — DOI: 10.52419/issn2072-2419.2022.1.61.

2. Исанбаева А.Р. Оценка терапевтической эффективности различных препаратов урсодезоксихолевой кислоты при лечении билиарного сладжа / А.Р. Исанбаева, Г.М. Сахаутдинова // Терапевтический архив. — 2021. — Т. 93. — № 12. — С. 1477–1481. — DOI: 10.26442/00403660.2021.12.201250.

3. Абсандзе К.Г. Лечение первичного билиарного холангита урсодезоксихолевой кислотой / К.Г. Абсандзе, Е.В. Винницкая, Ю.Г. Сандлер [и др.] Эффективная фармакотерапия. — 2022. — Т. 18. — № 22. — С. 14–19. — DOI: 10.33978/2307-3586-2022-18-22-14-18.

4. Попова О.С. Выживание рыб в кислой среде под воздействием урсодезоксихолевой кислоты / О.С. Попова, В.С. Понамарев // Международный ветеринарный вестник. — 2022. — № 4. — С. 159–164. — DOI: 10.52419/issn2072-2419.2022.4.159.

5. Кардаш Е.В. Обзор механизмов действия и свойств гепатопротекторных препаратов / Е.В. Кардаш, Е.М. Григорьева, А.Г. Емельянова [и др.] // Патогенез. — 2020. — Т. 18. — № 2. — С. 36–44. — DOI: 10.25557/2310-0435.2020.02.36-44.

6. Сапармаммедова Г. Гепатопротекторы: критический анализ механизмов действия и их место в клинической практике / Г. Сапармаммедова // Новая наука: от идеи к результату. — 2025. — № 12. — С. 185–188.

7. Скворцова Е.М. Особенности применения желчегонных препаратов и механизмы их действия / Е.М. Скворцова, В.В. Скворцов, А.Я. Исмаилов [и др.] // Терапевт, 2018. — № 3. — С. 10–16.

8. Прусаков А.В. Роль органических кислот в питании рыб: от диагностического признака к разработке стратегий фармакокоррекции / А.В. Прусаков, В.С. Понамарев // Международный научно-исследовательский журнал. — 2025. — № 10 (160). — DOI: 10.60797/IRJ.2025.160.78.

 © Authors of the article / Authors of the article

9. Алиева Е.М. Лечебно-профилактические мероприятия в рыбоводстве / Е.М. Шевченко, М.М. Алиева, З.С. Мирзаханова // Современные проблемы и перспективы агропромышленного комплекса Республики Дагестан : материалы региональной научно-практической конференции, посвященной Году науки и техники, г. Махачкала, 3 марта 2021 г. — Махачкала : Дагестанский государственный аграрный университет им. М.М. Джамбулатова, 2021. — С. 118–126.

10. Бельчева Н.Н. Обычная и синхронная спектрофлуориметрия желчи рыбы как показатель загрязнения морской среды полициклическими ароматическими углеводородами / Н.Н. Бельчева, А.А. Истомина // Морские биологические исследования: достижения и перспективы : сборник материалов Всероссийской научно-практической конференции с международным участием, посвященной 145-летию Севастопольского биологической станции : в 3-х томах. — Севастополь : ЭКОСИ Гидрофизика, 2016. — Т. 3. — С. 39–42.

11. Бельчева Н.Н. Наличие полициклических ароматических углеводородов (ПЦА) в рыбьей желчи важно для сравнительной оценки загрязнения прибрежных вод / Н.Н. Бельчева, А.А. Захари, А.Ю. Звягинцев [и др.] // Вопросы ихтиологии. — 2017. — Т. 57. — № 6. — С. 737–742. — DOI: 10.7868/S0042875217050034.

12. Заводовский Б.В. Клиническая биохимия : учебное пособие / Б.В. Заводовский, Е.А. Заводнева, Н.Г. Краюшкина [и др.]. — Волгоград : Волгоградский государственный медицинский университет, 2023. — 164 с.

13. Малиновская Н.А. Биохимия и патобиохимия липидного обмена : учебно-методическое пособие / Н.А. Малиновская, Л.В. Труфанова, Г.Е. Шульц [и др.]. — Красноярск : Красноярский государственный медицинский университет, 2024. — 71 с.

14. Белозерцева Н.С. Ветеринарная клиническая биохимия печени : лекция / Н.С. Белозерцева, Ю.С. Круглова, Ж.Ю. Мурадян. — Москва : ФГБОУ ВО МГАВМИБ МВА им. К.И. Скрябина, 2024. — 36 с.

15. Карпенко Е.Н. Биохимия: практикум для студентов / Е.Н. Карпенко, С.В. Кузнецов, Н Ф. Баширова. — Брянск : Новый проект, 2024. — 136 с.

Список литературы на английском языке / References in English

1. Popova O.S. Metabolizm zhirnyh kislot u ryb [Features of bile acid metabolism in fish] / O.S. Popova, L.A. Agafonova // Mezhdunarodnyj veterinarnyj vestnik [International Veterinary Bulletin]. — 2022. — № 1. — P. 61–65. — DOI: 10.52419/issn2072-2419.2022.1.61.

2. Isanbaeva A.R. Ocenka terapevticheskoj effektivnosti razlichnyh preparatov ursodezoksiholevoj kisloty pri lechenii biliarnogo sladzha [Evaluation of therapeutic efficiency of various drugs of ursodeoxycholic acid in the treatment of biliary sludge] / A.R. Isanbaeva, G.M. Sakhautdinova // Terapevticheskij arhiv [Therapeutic Archive]. — 2021. — Vol. 93. — № 12. — P. 1477–1481. — DOI: 10.26442/00403660.2021.12.201250.

3. Absandze K.G. Lechenie pervichnogo biliarnogo holangita ursodezoksiholevoj kislotoj [Primary biliary cholangitis and incomplete response to therapy with ursodeoxycholic acid: who is to blame and what to do?] / K.G. Absandze, E.V. Vinnitskaya, Yu.G. Sandler [et al.] // Effektivnaya farmakoterapiya [Effective Pharmacotherapy]. — 2022. — Vol. 18. — № 22. — P. 14–19. — DOI: 10.33978/2307-3586-2022-18-22-14-18.

4. Popova O.S. Vyzhivanie ryb v kisloj srede pod vozdejstviem ursodezoksiholevoj kisloty [Assessment of fish survival in an acidic environment under the influence of ursodeoxycholic acid] / O.S. Popova, V.S. Ponamarev // Mezhdunarodnyj veterinarnyj vestnik [International Veterinary Bulletin]. — 2022. — № 4. — P. 159–164. — DOI: 10.52419/issn2072-2419.2022.4.159.

5. Kardash E.V. Obzor mekhanizmov dejstviya i svojstv gepatoprotektornyh preparatov [Review of the mechanisms of action and properties of hepatoprotective drugs] / E.V. Kardash, E.M. Grigorieva, A.G. Yemelyanova [et al.] // Patogenez [Pathogenesis]. — 2020. — Vol. 18. — № 2. — S. 36–44. — DOI: 10.25557/2310-0435.2020.02.36-44.

6. Saparmammedova G. Gepatoprotektory: kriticheskij analiz mekhanizmov dejstviya i ih mesto v klinicheskoj praktike [Hepatoprotectors: a critical analysis of the mechanisms of action and their place in clinical practice] / G. Saparmammedova // Novaya nauka: ot idei k rezul'tatu [New science: from idea to result]. — 2025. — № 12. — P. 185–188.

7. Skvortsova E.M. Osobennosti primeneniya zhelchegonnyh preparatov i mekhanizmy ih dejstviya [Features of the use of cholagogue medications and the mechanisms of their action] / E.M. Skvortsova, V.V. Skvortsov, I.Ya. Ismailov [et al.] // Terapevt [Therapist], 2018. — № 3. — P. 10–16.

8. Prusakov A.V. Rol' organicheskih kislot v pitanii ryb: ot diagnosticheskogo priznaka k razrabotke strategij farmakokorrekcii [Imbalance of bile acids in fish under environmental stress: from a diagnostic sign to the development of pharmacocorrection strategies] / A.V. Prusakov, V.S. Ponamarev // Mezhdunarodnyj nauchno-issledovatel'skij zhurnal [International Scientific Research Journal]. — 2025. — № 10 (160). — DOI: 10.60797/IRJ.2025.160.78.

9. Alieva E.M. Lechebno-profilakticheskie meropriyatiya v rybovodstve [Therapeutic and preventive measures in fish farming] / E.M. Alieva, M.M. Alieva, Z.S. Mirzakhanova // Sovremennye problemy i perspektivy agropromyshlennogo kompleksa Respubliki Dagestan [Modern Problems and Prospects of the Agro-Industrial Complex of the Republic of Dagestan] : materials of the Regional Scientific Conference dedicated to the Year of Science and Technology. — Makhachkala : Dagestan State Agrarian University named after M.M. Dzhambulatov, 2021. P. 118–126.

10. Belcheva N.N. Obychnaya i sinhronnaya spektrofluorimetriya zhelchi ryby kak pokazatel' zagryazneniya morskoj sredy policiklicheskimi aromaticheskimi uglevodorodami [Fixed wavelength and synchronous fluorescence of fish bile as a monitoring tool for polyaromatic hydrocarbon marine pollution] / N.N. Belcheva, A.A. Istomina // Morskie biologicheskie issledovaniya: dostizheniya i perspektivy [Marine biological research: achievements and prospects] : collection of materials All-Russian Scientific and Practical Conference with international participation dedicated to the 145th anniversary of the Sevastopol Biological Station : in 3 volumes. — Sevastopol : EKOSI-Hydrophysics, 2016. — Vol. 3. — P. 39–42.

 © Authors of the article / Authors of the article

11. Belcheva N.N. Nalichie policiklicheskih aromaticheskih uglevodorodov (PCA) v ryb'ej zhelchi vazhno dlya sravnitel'noj ocenki zagryazneniya pribrezhnyh vod [Determination of metabolites of polycyclic aromatic hydrocarbons in fish bile for a comparative assessment of pollution of coastal waters] / N.N. Belcheva, A.A. Istomina, A.Yu. Zvyagintsev [et al.] // Voprosy ikhtiologii [Questions of Ichthyology]. — 2017. — Vol. 57. — № 6. — P. 737–742. — DOI: 10.7868/S0042875217050034.

12. Zavodovsky B.V. Klinicheskaya biohimiya [Clinical biochemistry] : manual / B.V. Zavodovsky, E.A. Zagorodneva, N.G. Krayushkina [et al.]. — Volgograd : Volgograd State Medical University, 2023. — 164 p.

13. Malinovskaya N.A. Biohimiya i patobiohimiya lipidnogo obmena [Biochemistry and pathobiochemistry of lipid metabolism] : educational and methodical manual / N.A. Malinovskaya, L.V. Trufanova, G.E. Herzog [et al.]. — Krasnoyarsk : Krasnoyarsk State Medical University, 2024. — 71 p.

14. Belozertseva N.S. Veterinarnaya klinicheskaya biohimiya pecheni [Veterinary Clinical Biochemistry of the Liver] : lecture / N.S. Belozertseva, Yu.S. Kruglova, Zh.Yu. Muradyan. — Moscow : FSBEI HE MGAVMiB-MVA named after K.I. Skryabin, 2024. — 36 p.

15. Karpenko E.N. Biohimiya [Biochemistry] : practical course for students / E.N. Karpenko, S.V. Kuznetsov, N.F. Bashirova. — Bryansk : Novyj proekt, 2024. — 136 p.