*Scientific and technical bulletin of Livestock farming Institute of NAAS of Ukraine, 2026, Is. 137*
***ISSN* 3083-7987 print**

DOI 10.32900/3083-7987-2026-137-194-209
UDC 597.551.2:615.9:577.175.1

# T-2 MYCOTOXIN DISRUPTS TISSUE-SPECIFIC LIPID AND PROTEIN METABOLISM IN COMMON CARP (*CYPRINUS CARPIO*)

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**Received:** May 12, 2026
**Revised:** May 15, 2026
**Published:** May 29, 2026
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*T-2 toxin, a type A trichothecene produced by Fusarium species, is one of the most toxic and environmentally persistent mycotoxins found in aquaculture feed. Despite growing evidence of its immunotoxic and oxidative effects in fish, its impact on tissue-specific lipid and protein metabolism, and the interrelationships between these biochemical pathways, remains poorly understood. This study examined the impact of dietary exposure to T-2 toxin (0.25 mg/kg feed; 5 MPC) on total lipid, phospholipid and protein concentrations in the gills, liver and brain of common carp (Cyprinus carpio). Correlation and regression analyses were also performed to identify coordinated metabolic responses and potential toxicity biomarkers. Exposure to T-2 toxin for 14 days induced pronounced, tissue-specific alterations in biochemical composition. The gills exhibited the most severe metabolic disruption, characterised by significant decreases in total lipids ($p = 0.010$; Cohen's $d = 2.24$) and total proteins ($p < 0.001$; Cohen's $d = 3.32$), indicating severe impairment of energy metabolism and protein homeostasis. In contrast, the brain showed a significant increase in total lipid content ($p = 0.008$; Cohen's $d = 2.22$), suggesting altered lipid distribution and potential neurotoxic effects. Meanwhile, the liver displayed moderate changes, with a significant reduction in protein concentration ($p = 0.031$), but relatively stable lipid profiles. Despite metabolic disturbances, phospholipid levels remained largely unchanged across tissues, indicating the preservation of structural membrane components. Correlation and regression analyses revealed strong lipid–protein coupling in the gills ($r = 0.83$; $R^2 = 0.69$), moderate associations in the brain, and weak or absent relationships in the liver. This highlights differences in the regulation of metabolic networks across tissues. The gills emerged as the most sensitive organ, reflecting tightly coordinated metabolic responses to T-2 exposure. The results demonstrate that the T-2 mycotoxin disrupts systemic energy metabolism and protein homeostasis in C. carpio in a tissue-specific manner. The most pronounced effects were observed in the branchial tissue, alongside distinct lipid accumulation in the neural tissue. These findings identify total lipids and proteins in the gills and brain as sensitive biomarkers of T-2-induced metabolic disturbance, and emphasise the importance of an integrative, multi-tissue approach in the study of*

*mycotoxin toxicity. The study also emphasises that the disruption of coordinated metabolic networks is a key component of T-2 toxicity in fish.*

**Keywords:** T-2 toxin; mycotoxins; *Cyprinus carpio*; lipid metabolism; protein metabolism; oxidative stress; gills; brain; liver; biomarker

# Т-2 МІКОТОКСИН ПОРУШУЄ ТКАНИННО-СПЕЦИФІЧНИЙ МЕТАБОЛІЗМ ЛІПІДІВ І БІЛКІВ У КОРОПА ЗВИЧАЙНОГО (*CYPRINUS CARPIO*)

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**Дата надходження до редакції:** 12.05.2026 р.
**Дата прийняття до друку після рецензування:** 15.05.2026 р.
**Дата публікації:** 29.05.2026 р.
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*Т-2 токсин, мікотоксин типу А з групи трихотеценів, що продукується видами Fusarium, є одним із найтоксичніших і найбільш стійких у довкіллі мікотоксинів, які трапляються у кормах для аквакультури. Незважаючи на зростаючі дані щодо його імунотоксичних та окиснювальних ефектів у риб, його вплив на тканинно-специфічний метаболізм ліпідів і білків, а також взаємозв'язки між цими біохімічними шляхами, залишається недостатньо вивченим. У цьому дослідженні оцінено вплив дієтичного надходження Т-2 токсину (0,25 мг/кг корму; 5 ГДК) на вміст загальних ліпідів, фосфоліпідів і білків у зябрах, печінці та головному мозку коропа звичайного (Cyprinus carpio). Також проведено кореляційний і регресійний аналіз для виявлення скоординованих метаболічних відповідей і потенційних біомаркерів токсичності. Експозиція до Т-2 токсину протягом 14 діб викликала виражені тканинно-специфічні зміни біохімічного складу. Найбільш суттєві порушення метаболізму спостерігалися у зябрах, де зафіксовано значне зниження рівня загальних ліпідів ($p = 0,010$; Cohen's $d = 2,24$) і загальних білків ($p < 0,001$; Cohen's $d = 3,32$), що свідчить про глибоке порушення енергетичного обміну та білкового гомеостазу. Натомість у тканині головного мозку відзначено значне підвищення вмісту загальних ліпідів ($p = 0,008$; Cohen's $d = 2,22$), що може вказувати на змінену розподільну динаміку ліпідів і потенційні нейротоксичні ефекти. Печінка демонструвала помірні зміни: спостерігалося достовірне зниження концентрації білка ($p = 0,031$) при відносно стабільному рівні ліпідів. Попри метаболічні порушення, рівень фосфоліпідів залишався відносно стабільним у всіх тканинах, що свідчить про збереження структурної цілісності клітинних мембран. Кореляційний і регресійний аналіз виявив сильний зв'язок між вмістом ліпідів та білків у зябрах ($r = 0,83$; $R^2 = 0,69$), помірні асоціації у мозку та слабкі або відсутні взаємозв'язки у печінці. Це підкреслює різницю у регуляції метаболічних мереж між тканинами. Зябра виявилися найбільш чутливим органом, демонструючи тісно скоординовані метаболічні реакції на вплив Т-2*

*токсину. Отримані результати свідчать, що Т-2 мікотоксин порушує системний енергетичний метаболізм і білковий гомеостаз у C. carpio у тканинно-специфічний спосіб. Найбільш виражені ефекти спостерігалися в зябровій тканині, а також у вигляді характерного накопичення ліпідів у нервовій тканині. Ці результати ідентифікують загальні ліпіди та білки у зябрах і мозку як чутливі біомаркери метаболічних порушень, індукованих Т-2 токсином, і підкреслюють важливість інтегративного багатотканинного підходу до вивчення токсичності мікотоксинів. Дослідження також показує, що порушення скоординованих метаболічних процесів є ключовим компонентом токсичної дії Т-2 у риб.*

**Ключові слова:** Т-2 токсин; мікотоксини; *Cyprinus carpio*; метаболізм ліпідів; метаболізм білків; окиснювальний стрес; зябра; мозок; печінка; біомаркер

**Introduction.** Mycotoxins are secondary metabolites produced by various species of filamentous fungi that often contaminate agricultural produce and animal feed. T-2 toxin, a type A trichothecene primarily produced by *Fusarium* species, is considered one of the most potent and toxic mycotoxins due to its broad spectrum of biological effects (Adhikari M. et al., 2017). T-2 toxin is highly resistant to environmental degradation and can persist throughout feed processing and storage, posing a significant threat to terrestrial and aquatic organisms alike. In aquaculture systems, the contamination of fish feed with trichothecenes is a growing concern due to the increasing use of plant-derived feed ingredients, which are susceptible to fungal infestation (Janik, E. et al., 2021; Zhang J. et al., 2022).

The toxic effects of T-2 toxin have been extensively documented in mammals, poultry and livestock. Exposure has been associated with growth inhibition, immunosuppression, oxidative stress, reproductive dysfunction and tissue damage (Gu W. et al., 2023; Meng F. et al., 2025). At the cellular level, T-2 toxin inhibits protein synthesis by interacting with the 60S ribosomal subunit, which leads to impaired cellular metabolism, apoptosis and inflammatory responses (Wu Q. et al., 2020; Janik-Karpinska E. et al., 2023). Furthermore, numerous studies have demonstrated that T-2 toxin promotes the generation of reactive oxygen species (ROS), resulting in oxidative damage to lipids, proteins, and nucleic acids (Yin H. et al., 2020; An K. et al., 2024; He J. et al., 2024).

Compared with terrestrial animals, considerably less information is available on the effects of T-2 toxin on fish. Nevertheless, existing studies suggest that fish are particularly susceptible to exposure to mycotoxins due to their constant contact with contaminated feed and aquatic environments (Oliveira M. and Vasconcelos V., 2020). Previous studies have shown that T-2 toxin can cause oxidative stress, histopathological changes, immunotoxicity and metabolic disturbances in different species of fish (Zhang J. et al., 2022; Song W. et al., 2023). However, the mechanisms underlying tissue-specific metabolic responses to T-2 intoxication are not well understood.

Lipids and proteins play a key role in maintaining cellular structure, energy homeostasis and physiological function. Alterations in lipid metabolism may reflect changes in energy utilisation, membrane integrity and oxidative status, whereas disturbances in protein metabolism are closely associated with impaired biosynthesis, increased proteolysis and tissue dysfunction (Zayani Z. et al., 2025; Li Z. et al., 2026). Consequently, measuring total lipids, phospholipids and proteins can provide valuable insights into the metabolic consequences of toxicant exposure (Yoon H. et al., 2021). Furthermore, analysing the relationships between these biochemical parameters may reveal coordinated or disrupted metabolic pathways that cannot be detected by measuring them individually.

**ISSN 3083-7987 print**

The gills, liver and brain are particularly important target organs for toxicological investigations in fish. The gills are directly exposed to the external environment and are the main site of uptake of contaminants (Shah N. et al., 2020). The liver is the central organ of detoxification and metabolic regulation (Topić Popović N. et al., 2023; Wang J. et al., 2025), and the brain is sensitive to oxidative damage due to its high oxygen consumption and lipid-rich composition (Dijkstra P.D. et al., 2024; Ma X. and Wang W.X., 2025). Examining these tissues, therefore, provides a comprehensive assessment of systemic and organ-specific toxic responses.

The common carp (*Cyprinus carpio*) is one of the most economically important freshwater fish species worldwide, and is frequently used as a model organism in ecotoxicological studies (Rahman M.M., 2015; Mihály-Karnai L. et al., 2025). Despite its significance, there is limited information available on the effects of T-2 toxin on lipid and protein metabolism in different carp tissues.

The present study, therefore, aimed to evaluate the effects of T-2 mycotoxin exposure on total lipid, phospholipid and protein concentrations in the gills, liver and brain of *C. carpio*. Additionally, correlation and linear regression analyses were conducted to investigate the relationships between the studied biochemical parameters in different tissues and to identify potential biomarkers of T-2-induced metabolic disturbances. This study was financially supported by the Conference of Rectors of Polish Universities (KRASP) and Elsevier in the form of scholarships, which greatly enhanced the research capabilities of Ukrainian PhD students and academic staff. Without the support of these institutions, the present research at the Institute of Biology, Pomeranian University in Słupsk would not have been possible.

**Materials and methods.**

**Experimental animals and maintenance.** The study was conducted using common carp (*Cyprinus carpio*), which were obtained from the wintering pond of the 'Chernihivrybhosp' aquaculture farm in Chernihiv, Ukraine. During the growing season, the fish were kept in standard aquaculture conditions and fed a commercially available diet according to established seasonal feeding schedules. Prior to the experiment, the fish were transported to the laboratory and acclimatised in experimental aquaria for three days. During this period, the fish were maintained under winter fasting conditions and received no feed. Only clinically healthy individuals showing no external signs of disease or injury were selected for the study. The health status of the fish was verified by ichthyopathological examination, which confirmed the absence of ectoparasitic and helminth infections (Yachna M. et al., 2026). All experimental procedures were carried out in accordance with the recommendations of the Local Ethical Committee for Animal Experiments at T. H. Shevchenko National University 'Chernihiv Collegium' (Chernihiv, Ukraine). Particular attention was paid to minimising handling stress and ensuring animal welfare throughout the experimental period.

**Experimental design.** The exposure experiment was conducted in 200-litre glass aquaria containing dechlorinated tap water. The fish were stocked at a density of one individual per 40 $dm^3$ of water. Environmental conditions were kept close to those naturally occurring during the winter season. Water quality parameters were regularly monitored and kept within the optimal ranges for carp culture: dissolved oxygen concentrations ranged from 9.6 to 12.5 $mg/dm^3$, pH varied from 7.4 to 8.4 and the concentration of ammonia remained at around 0.014 $mg/dm^3$. These conditions prevented the occurrence of hypoxia, hypercapnia or temperature-related stress. The fish used in the experiment weighed between 250 and 300 g, and the exposure period lasted 14 consecutive days.

**T-2 mycotoxin administration.** The experimental fish were exposed to the T-2 mycotoxin through their diet. The toxin was incorporated into a commercial feed mixture at a concentration five times higher than the maximum permissible concentration (5 MPC), equivalent to 0.25 mg of T-2 toxin per kilogram of feed. The exposed group received the contaminated feed once daily throughout the experimental period. The control group of fish were maintained under identical conditions and fed the same diet without T-2 supplementation.

**Tissue collection and biochemical analyses.** At the end of the experiment, the fish were euthanised humanely according to accepted ethical procedures and samples of gill, liver and brain tissue were collected for biochemical analysis. Immediately after dissection, the tissues were rinsed with an ice-cold Tris-HCl buffer solution (pH 7.2) to remove any remaining blood and minimise post-mortem biochemical changes. All chemicals and reagents used in the study were of analytical grade.

The tissue samples were homogenised in an ice-cold buffer solution using a motorised glass homogeniser, maintaining a tissue-to-buffer ratio of 1:9 (w/v). Homogenisation was performed in an ice-water bath to prevent the thermal degradation of proteins and other biomolecules. The resulting homogenates were then centrifuged at 3,000 rpm (approximately 1,000×g) for 15 minutes at 4 °C. Following centrifugation, the resulting supernatant was collected and stored at −25 °C until analysis. To minimise analytical variability, each sample was thawed only once prior to biochemical determinations.

Total protein concentrations were measured using the Lowry method (1951) with commercial diagnostic kits (DIALAB, Austria), following the manufacturer's protocol. Total lipid and phospholipid content was determined using commercially available biochemical assay kits (Filisit, Ukraine).

**Statistical analysis.** The data are presented as mean values ± standard deviation (S.D.). Statistical analyses were performed using parametric methods. Differences between the control group and the T-2-treated group were assessed using a Student's t-test, with values of $p < 0.05$ being considered statistically significant. To evaluate the magnitude of the effects of the treatment, Cohen's d effect sizes were calculated and interpreted according to conventional criteria: values of approximately 0.2, 0.5 and 0.8 or greater indicate small, moderate and large effects, respectively (Stanisz, 2006; Stanisz, 2007).

Relationships among biochemical variables were examined using Pearson's correlation coefficient (r). Additionally, linear regression analyses were performed to evaluate the predictive relationships between selected lipid and protein parameters, and to determine the proportion of variance explained by the individual models. All statistical calculations were conducted using Statistica 13.3 software (TIBCO Software Inc., Palo Alto, CA, USA). Graphical representations of the data were prepared using Microsoft Excel, and statistical significance is indicated in the corresponding figures.

**Results.** The effects of exposure to the T-2 mycotoxin on lipid and protein metabolism were evaluated in the gill, liver and brain tissues of the common carp (*Cyprinus carpio*). All data are presented as the mean ± standard deviation (S.D.). Statistical differences between the control group and the T-2-treated groups were assessed using a Student's t-test, with $p < 0.05$ being considered statistically significant. Effect sizes were calculated using Cohen's d.

Figure 1 shows the concentrations of total lipids, phospholipids, and total proteins in the gill, liver, and brain tissues of carp exposed to the T-2 mycotoxin.

**ISSN 3083-7987 print**

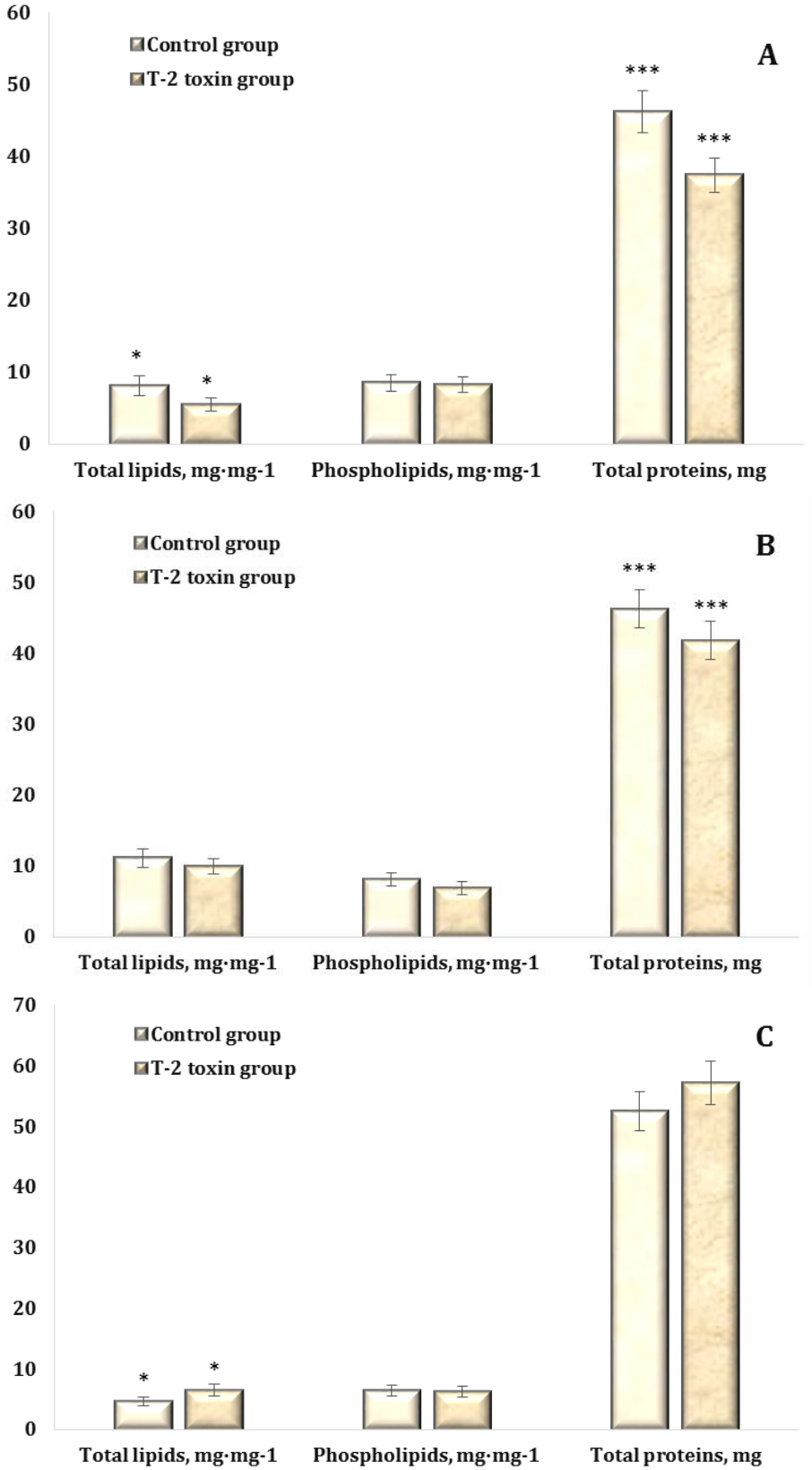


*Fig. 1.* **Levels of total lipids, phospholipids and total proteins in the gill (A), liver (B), and brain tissue (C) of carp *Cyprinus carpio* exposed to T-2 toxin.**

Data are presented as means ± S.D. (n = 6).

*, ** and *** Significant differences ($p < 0.05$) between the untreated control group and the group exposed to T-2 toxin.

Exposure to the T-2 mycotoxin induced marked alterations in lipid and protein metabolism in the gills. Total lipid concentration decreased significantly in the T-2-treated group (5.43 ± 0.94 mg·mg$^{-1}$ protein) compared to the control group (8.10 ± 1.41 mg·mg$^{-1}$ protein; p = 0.010), representing a large effect size (Cohen's d = 2.24). This substantial decrease suggests impaired lipid homeostasis and increased utilisation of lipid reserves within branchial tissues under mycotoxin-induced stress. By contrast, brain tissue exhibited a significant increase in total lipid content, rising from 4.61 ± 0.65 mg·mg$^{-1}$ protein in the control group to 6.46 ± 0.98 mg·mg$^{-1}$ protein in the T-2 group (p = 0.008; Cohen's d = 2.22). No statistically significant changes were detected in liver lipid levels (11.08 ± 1.30 vs. 9.90 ± 5.10 mg·mg$^{-1}$ protein; p = 0.629) and the effect size was small (Cohen's d = 0.32). These findings suggest a redistribution of lipid reserves characterised by reduced circulating lipids and increased lipid accumulation in neural tissue.

Phospholipid levels in the gills remained relatively stable, with no significant difference observed between the control group (8.48 ± 1.19 mg·mg$^{-1}$ protein) and the group exposed to T-2 (8.21 ± 1.12 mg·mg$^{-1}$ protein; p = 0.721). The effect size was negligible to small (Cohen's d = 0.23), suggesting that the composition of phospholipids in the gill membranes was largely preserved despite T-2 intoxication. No significant differences were observed in brain phospholipid concentrations (6.43 ± 0.94 vs. 6.18 ± 0.84 mg·mg$^{-1}$ protein; p = 0.670; Cohen's d = 0.28). In the liver, phospholipid levels decreased following T-2 exposure (8.04 ± 0.90 vs. 6.87 ± 0.87 mg·mg$^{-1}$ protein), though this change was not statistically significant (p = 0.070). Nevertheless, the effect size was large (Cohen's d = 1.32), suggesting a biologically relevant reduction which may become significant in studies with larger sample sizes.

A pronounced effect was observed in terms of total protein concentration. Fish exposed to T-2 toxin had significantly lower levels of protein in their gills (37.40 ± 2.36 mg/g) than the control group (46.20 ± 2.92 mg/g; p < 0.001). This difference was extremely large in magnitude (Cohen's d = 3.32), suggesting severe disruption to protein metabolism and possible impairment to the structure or function of branchial tissue. Of all the biochemical parameters measured in the gills, total protein concentration was the most sensitive to T-2 exposure. A significant decrease in protein concentrations was also observed in the liver, declining from 46.20 ± 2.69 mg/g in the control group to 41.80 ± 2.64 mg/g in the T-2-treated group (p = 0.031). This effect was large in magnitude (Cohen's d = 1.65), indicating impaired hepatic protein metabolism. By contrast, brain protein concentrations tended to increase following exposure to T-2 (57.10 ± 3.60 vs. 52.50 ± 3.28 mg/g protein), though this difference was not statistically significant (p = 0.068). Nevertheless, the corresponding effect size was large (Cohen's d = 1.34), suggesting a substantial biological response.

Correlation analysis revealed tissue-specific relationships among the investigated biochemical parameters. For the gills, Pearson's correlation analysis revealed a pattern of coordinated metabolic regulation between lipid and protein parameters. A strong positive correlation was observed between total lipids and phospholipids (r = 0.70), indicating that changes in overall lipid content were closely associated with variations in membrane lipid fractions. An even stronger positive relationship was found between total lipids and total protein concentration (r = 0.83), suggesting that T-2 mycotoxin-induced lipid depletion was accompanied by a parallel reduction in protein content. In contrast, the correlation between phospholipids and total proteins was relatively weak (r = 0.27), suggesting a less direct association between these two variables. In the liver, all correlations were relatively weak, with coefficients ranging from 0.24 to 0.37, suggesting a less coordinated metabolic response. Brain tissue displayed moderate positive correlations between total

lipids and phospholipids (r = 0.47), and between total lipids and proteins (r = 0.61). However, the association between phospholipids and proteins remained weak (r = 0.18).

Thus, exposure to the T-2 mycotoxin substantially altered the lipid and protein metabolism of *C. carpio*, with the gills showing a strong correlation with total lipid content, which represents a key determinant of metabolic status in this tissue. The particularly high correlation between total lipids and proteins (r = 0.83) supports the hypothesis that disturbances in energy metabolism induced by T-2 are closely linked to protein catabolism and tissue impairment in the gills of *C. carpio*. Hepatic protein levels were also significantly reduced, whereas phospholipid concentrations remained comparatively stable across tissues. The strongest statistical effects were observed for blood protein concentration (Cohen's d = 3.32) and blood and brain lipid levels (Cohen's d > 2.2), indicating that these parameters are particularly sensitive biomarkers of T-2-induced metabolic disturbances. These results suggest that the T-2 mycotoxin disrupts systemic energy metabolism and protein homeostasis, while promoting tissue-specific metabolic adaptations, particularly in neural tissue.

Pearson's correlation analysis was used to construct linear regression models for the strongest identified relationships in the gills, liver and brain tissues of *Cyprinus carpio* exposed to the T-2 mycotoxin. Linear regression analysis showed that total lipid concentration was a strong predictor of total protein levels in gill tissue. The relationship was described by the equation: Protein = 25.39 + 2.43 × Lipids, with a coefficient of determination of $R^2 = 0.69$ ($p = 0.003$). This indicates that approximately 69% of the variability in gill protein concentration can be explained by changes in total lipid levels. This strong positive association supports the close metabolic coupling between lipid reserves and protein metabolism in branchial tissue. A second significant regression was observed between total lipids and phospholipids: Phospholipids = 5.45 + 0.43 × lipids, with $R^2 = 0.50$ ($p = 0.023$). This model shows that almost half of the variation in phospholipid concentration is related to changes in total lipid content. This reflects the coordinated regulation of structural and storage lipid fractions in the gills.

In liver tissue, however, none of the regression models reached statistical significance. The strongest relationship was observed between total lipids and phospholipids: Phospholipids = 6.33 + 0.11 × Lipids, with $R^2 = 0.14$ ($p = 0.294$). Similarly, the associations between lipids and proteins ($R^2 = 0.06$, $p = 0.485$) and between phospholipids and proteins ($R^2 = 0.06$, $p = 0.506$) were weak and non-significant. These results imply that hepatic lipid and protein metabolism remained relatively independent under the experimental conditions and exhibited no strong coordinated response.

Regression analysis of brain tissue revealed a moderate positive association between total lipid and protein concentrations. Protein = 43.82 + 1.98 × Lipids, with $R^2 = 0.38$ ($p = 0.059$). While this relationship did not reach conventional statistical significance, it approached the threshold and suggests that around 38% of the variation in brain protein concentration may be associated with lipid levels. The regression between total lipids and phospholipids was weaker: Phospholipids = 4.55 + 0.32 × lipids, with $R^2 = 0.22$ ($p = 0.175$). Phospholipids were poor predictors of protein concentration ($R^2 = 0.03$, $p = 0.614$).

Linear regression analysis revealed clear, tissue-specific differences in the metabolic effects of the T-2 mycotoxin. The strongest and most statistically significant relationships were observed in the gills, where total lipids accounted for 69% of the variation in protein concentration and 50% of the variation in phospholipid levels. By contrast, liver tissue exhibited weak and non-significant associations among all the biochemical parameters studied, suggesting a less coordinated metabolic response. Brain tissue showed an intermediate pattern, with a moderate lipid–protein relationship

approaching statistical significance. These results suggest that the gills are the tissue that responds most metabolically to T-2-induced alterations, whereas the liver appears relatively resistant to coordinated changes in lipid–protein homeostasis.

**Discussion.** This study shows that exposure to the T-2 mycotoxin causes significant, tissue-specific changes to lipid and protein metabolism in the common carp (*Cyprinus carpio*). While T-2 toxin is primarily recognised for its potent inhibitory effects on protein synthesis and its ability to induce oxidative stress, these findings suggest that it also affects the regulation of lipid homeostasis and energy metabolism. The most significant changes were observed in the gills and brain, while the liver displayed a more moderate biochemical response.

One of the most notable findings was the significant depletion of total lipids in the gills, accompanied by a marked reduction in total protein concentration. The gills are among the most metabolically active tissues in fish, representing the primary interface between the organism and the aquatic environment. Consequently, they are highly susceptible to waterborne toxicants, including mycotoxins. The substantial decrease in lipid content observed in this tissue may reflect the mobilisation of lipid reserves to meet the increased energy demands associated with detoxification processes and the maintenance of cellular homeostasis. Lipids are an important energy source during periods of physiological stress, and their depletion has been reported in fish exposed to various environmental contaminants, including heavy metals, pesticides, and mycotoxins (Wang T. et al., 2021; Gao J. et al., 2025).

The dramatic reduction in branchial protein levels suggests that the T-2 toxin severely disrupts protein metabolism. *Trichothecene* mycotoxins, including T-2, primarily exert their toxicity through the inhibition of ribosomal peptidyl transferase activity, which leads to the suppression of protein synthesis (Adhikari M. et al., 2017; Wang W. et al., 2021). At the same time, oxidative damage caused by reactive oxygen species may speed up protein degradation and activate proteolytic pathways (Di Meo S. et al., 2016). The exceptionally large effect size observed for gill proteins suggests that this parameter could be one of the most sensitive biomarkers of T-2 toxicity in fish. Such alterations may compromise the structure and function of the gills, potentially affecting gas exchange, osmoregulation, and ion transport (Evans D.H., 1987).

Interestingly, phospholipid concentrations in the gills remained relatively stable despite substantial reductions in total lipids and proteins. Phospholipids are essential structural components of biological membranes and are therefore tightly regulated. The maintenance of phospholipid levels may represent a compensatory mechanism aimed at preserving membrane integrity under toxic stress conditions (Shyu P. et al., 2019; Poitelon Y. et al., 2020). A similar preservation of membrane phospholipids, despite the depletion of storage lipids, has been reported in fish exposed to contaminants that induce oxidative stress. This suggests that cellular membrane architecture may be prioritised when adapting to T-2-induced metabolic disturbances.

The strong positive correlations observed between total lipids, phospholipids, and proteins in gill tissue provide further support for the hypothesis of coordinated metabolic regulation. The particularly strong relationship between lipids and proteins ($r = 0.83$) suggests that the depletion of energy reserves and the impairment of protein metabolism are closely interconnected processes. Linear regression analysis confirmed this, showing that lipid levels explained almost 70% of the variability in protein concentrations. These findings suggest that the availability of lipids may play a crucial role in determining protein homeostasis within branchial tissues during exposure to mycotoxins. The significant regression between total lipids and phospholipids also suggests that structural and storage lipid pools respond in a coordinated manner to toxic insults.

In contrast to the gills, the liver exhibited a comparatively moderate response to T-2 intoxication. Although total protein concentrations were significantly reduced, changes in lipid and phospholipid levels failed to reach statistical significance. Nevertheless, the large effect size calculated for phospholipids suggests that biologically meaningful alterations may have occurred despite the limited sample size. As the central organ responsible for xenobiotic metabolism and detoxification, the liver possesses highly developed adaptive and compensatory mechanisms that may mitigate the effects of toxic exposure to some extent (Gu X. and Manautou J.E., 2012; Zhang Y. et al., 2022). Enhanced antioxidant defences, the activation of detoxification enzymes, and metabolic flexibility could explain the relatively weak correlations observed among biochemical parameters in this tissue.

The absence of significant regression models in the liver indicates that lipid and protein metabolism are regulated by multiple, independent pathways rather than by a tightly coordinated metabolic network. This finding contrasts sharply with that observed in the gills and may reflect the liver's greater capacity to maintain metabolic homeostasis in the face of toxicity. Nevertheless, the observed reduction in protein concentration is consistent with the recognised capacity of T-2 toxin to inhibit protein synthesis and cause hepatocellular damage (An K. et al., 2024; Duan L. et al., 2025).

The most intriguing observation was perhaps the significant accumulation of total lipids in brain tissue following T-2 exposure. Unlike in the gills, where lipid depletion predominated, the brain showed a notable increase in lipid content, accompanied by elevated protein levels. Several mechanisms may explain this response. Firstly, disruption to systemic lipid metabolism may promote the redistribution of lipids towards neural tissues. Secondly, T-2-induced oxidative stress may impair β-oxidation and lipid utilisation within neural cells, resulting in intracellular lipid accumulation. Thirdly, neuroinflammatory processes triggered by mycotoxin exposure could alter membrane turnover and stimulate the synthesis of lipid-rich cellular components (Bruce K.D. et al., 2017; Ali O. and Szabó A., 2023).

The brain is particularly vulnerable to oxidative stress due to its high oxygen consumption, the abundance of polyunsaturated fatty acids within it, and its relatively limited antioxidant capacity (Feng J. et al., 2023; Trofin D.-M. et al., 2025). Previous studies have demonstrated that T-2 toxin can cross biological barriers and induce neurotoxicity by causing oxidative damage, mitochondrial dysfunction and apoptosis (Dai C. et al., 2019; Xu C. et al., 2026). Therefore, the increase in total lipids observed in the present study may represent an early manifestation of these neurotoxic effects. The accumulation of lipids within neural tissues has been associated with impaired cellular signalling, mitochondrial dysfunction, and inflammatory responses in numerous toxicological models (Otsuka T. and Matsui H., 2023; Song C. et al., 2024).

Despite the increase in total lipid content, phospholipid concentrations in the brain remained relatively stable. As with the gills, this finding suggests that maintaining membrane phospholipid composition is a priority during toxic stress. Moderate positive correlations were observed between lipids and proteins, and the regression model approached statistical significance. This indicates that lipid accumulation may be linked to broader metabolic changes occurring within neural tissue. However, the weaker relationships observed compared to those in the gills suggest that additional mechanisms contribute to the regulation of brain metabolism during T-2 exposure (Guo P. et al., 2018; Wang Y. et al., 2024).

Thus, the results suggest that the T-2 mycotoxin interferes with both energy metabolism and protein homeostasis in *C. carpio*, although the extent and nature of these effects differ significantly between tissues. The gills were found to be the most responsive

organ, exhibiting severe depletion of lipids and proteins, as well as strong metabolic interrelationships. The liver showed evidence of impaired protein metabolism, yet retained a relatively high degree of metabolic independence and homeostatic control. By contrast, the brain responded with lipid accumulation and moderate alterations in protein metabolism, suggesting the activation of tissue-specific adaptive or pathological mechanisms.

The large effect sizes observed for gill proteins, gill lipids, and brain lipids emphasise the biological relevance of these responses and indicate that these parameters may serve as sensitive biomarkers of T-2 intoxication in fish. Furthermore, correlation and regression analyses reveal that toxic effects are not limited to isolated biochemical changes, but involve the disruption of coordinated metabolic networks, particularly in branchial tissue. These findings expand the current understanding of the mechanisms underlying T-2 toxicity in aquatic organisms, highlighting the importance of examining multiple tissues when assessing the metabolic consequences of mycotoxin exposure.

The present findings are consistent with previous reports demonstrating that T-2 mycotoxin exerts profound hematological, metabolic, and immunotoxic effects in fish species. In common carp (*Cyprinus carpio*), chronic exposure to T-2 toxin has been shown to induce anemia, characterized by reductions in hematocrit, hemoglobin concentration, and erythrocyte counts, alongside leukopenia driven primarily by lymphopenia (Matejova I. et al., 2017). These alterations indicate a marked suppression of hematopoietic processes and immune cell homeostasis, reflecting the systemic toxicity of trichothecene exposure. Similar disturbances in blood physiology have also been observed in rainbow trout (*Oncorhynchus mykiss*), where T-2 toxin exposure resulted in significant modifications of erythrocyte indices, including decreased mean corpuscular hemoglobin and altered erythrocyte morphology (Modra H. et al., 2020).

Importantly, both studies consistently demonstrate that T-2 toxin disrupts innate immune competence in fish. In carp, exposure was associated with altered non-specific immune responses and changes in cytokine profiles in the head kidney, indicating a functional impairment of immune regulation (Matejova I. et al., 2017). In rainbow trout, suppression of humoral immunity was evidenced by a significant reduction in complement-mediated bactericidal activity, alongside dose-dependent changes in leukocyte and lymphocyte populations (Modra H. et al., 2020). These findings collectively suggest that T-2 toxin compromises both cellular and humoral branches of innate immunity, thereby increasing susceptibility to secondary infections.

Another key and consistent finding across studies is the induction of oxidative stress. In common carp, exposure to T-2 significantly increased lipid peroxidation in the liver and caudal kidney. This was accompanied by altered antioxidant enzyme activities, including decreased catalase and glutathione peroxidase activity, as well as increased glutathione S-transferase activity (Matejova I. et al., 2017). These biochemical changes suggest an imbalance between the production of reactive oxygen species and the activity of antioxidant defence systems. Although less extensively characterised in rainbow trout, the observed immunosuppression and haematological disturbances are widely recognised consequences of oxidative damage triggered by trichothecene exposure. This suggests a shared mechanistic pathway of toxicity.

These studies provide strong evidence that the T-2 mycotoxin acts as a potent immunotoxic and pro-oxidative agent in teleost fish, inducing disturbances in haematological parameters, innate immune responses and redox homeostasis in a coordinated manner. The consistency of these effects across species further highlights the ecological and aquaculture relevance of T-2 contamination in feed, and emphasises the vulnerability of fish immune systems to exposure to trichothecenes.

Future studies should investigate the molecular pathways responsible for these alterations. This should include an examination of oxidative stress biomarkers, antioxidant enzyme activities, lipid peroxidation products, and the expression of genes involved in lipid and protein metabolism. This would provide a deeper understanding of the mechanisms linking T-2 exposure to metabolic dysfunction and tissue-specific toxicity in fish.

**Conclusions.** This study shows that exposure to the T-2 mycotoxin causes significant, tissue-specific changes to lipid and protein metabolism in the common carp (*Cyprinus carpio*). Of the organs examined, the gills exhibited the strongest metabolic response, characterised by substantial reductions in total lipid and protein concentrations, as well as strong positive correlations between lipid and protein parameters. These results suggest that the gills are particularly susceptible to metabolic disruption caused by T-2 and could be an early target organ for mycotoxin toxicity. The liver displayed a more moderate response, with significant decreases in protein concentration and relatively weak associations between lipid and protein parameters. This suggests that hepatic compensatory and detoxification mechanisms may mitigate the metabolic consequences of T-2 exposure to some extent. By contrast, the brain primarily responded through significant total lipid accumulation and an increased tendency towards protein content, indicating the activation of distinct neural adaptive or pathological processes.

Correlation and regression analyses revealed clear tissue-specific differences in metabolic regulation. The gills exhibited strong and statistically significant lipid–protein relationships, whereas the liver showed no evidence of coordinated metabolic responses. The brain exhibited intermediate characteristics, displaying moderate associations between lipid and protein metabolism. These findings demonstrate that T-2 toxicity affects both the concentrations of individual biochemical components and the functional relationships among metabolic pathways. The large effect sizes observed for gill proteins, gill lipids, and brain lipids suggest that these parameters are highly sensitive biomarkers of T-2 intoxication in fish. Thus, the results suggest that the T-2 mycotoxin disrupts energy metabolism and protein homeostasis, while promoting tissue-specific metabolic adaptations, particularly in the gills and brain.

Future investigations should focus on the molecular mechanisms underlying these responses, including oxidative stress pathways, lipid peroxidation, antioxidant defence systems, and the expression of genes involved in lipid and protein metabolism. Such studies would contribute to a more comprehensive understanding of T-2-induced toxicity in aquatic organisms and support the development of sensitive biomarkers for environmental and aquaculture risk assessment.

**Acknowledgements.** The authors would like to express their sincere gratitude to the Conference of Rectors of Polish Universities (KRASP) and Elsevier for awarding scholarships that have greatly enhanced the research capabilities of Ukrainian Ph.D. candidates and academic staff. Without this support, it would not have been possible to conduct the present study at the Institute of Biology, Pomeranian University in Słupsk. The authors are very grateful for the opportunity to work in such a dynamic and supportive scientific environment, and for the institutional assistance that was instrumental in the successful completion of this research.

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