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BLOOD PHOSPHOLIPIDS AND MUSCLE PROTEIN AS SENSITIVE BIOMARKERS OF T-2 MYCOTOXIN TOXICITY IN COMMON CARP (*CYPRINUS CARPIO* L.)

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*T-2 mycotoxin, a highly toxic trichothecene produced by Fusarium species, poses a significant threat to aquaculture due to its stability and prevalence in contaminated feed. This study aimed to evaluate the effects of dietary exposure to T-2 toxin on total lipid and protein levels in the blood and muscle tissues of common carp (*Cyprinus carpio*), focusing particularly on the interrelationships between biochemical parameters and their potential as biomarkers of toxic stress. Fish weighing 250-300 g were exposed to T-2 toxin at a concentration of 0.25 mg/kg of feed (five times the maximum permissible concentration) for 14 days under controlled laboratory conditions. Total lipids, phospholipids and total protein levels were determined using standard biochemical methods. Statistical analyses included Student's *t*-test, Cohen's *d* effect size, Pearson's correlation and linear regression. The results revealed significant metabolic disturbances induced by T-2 toxin. Total lipids, phospholipids and protein levels in the blood were significantly reduced ($p < 0.05$), with particularly strong effects observed for phospholipids (Cohen's $d = 4.95$). In contrast, muscle tissue exhibited a significant increase in total lipid content ($p = 0.033$), accompanied by a marked depletion of protein ($p < 0.001$; Cohen's $d = 6.51$). This indicates a shift towards lipid accumulation and protein catabolism. Correlation analysis revealed coordinated metabolic regulation in the blood, with strong positive correlations between all parameters. In contrast, muscle tissue showed disrupted metabolic integration, including a strong negative correlation between lipids and protein ($r = -0.72$). Regression analysis confirmed that lipid levels significantly predicted protein depletion in muscle tissue ($R^2 = 0.52$), while phospholipids were strong predictors of protein concentration in blood ($R^2 = 0.66$). Thus, T-2 toxin exposure resulted in pronounced and tissue-specific metabolic alterations, characterised by systemic depletion of circulating biomolecules and local metabolic imbalance in muscle tissue. Blood phospholipids and muscle protein emerged as highly sensitive biomarkers of T-2 toxicity. These findings provide new insights into the mechanisms of mycotoxin-induced metabolic disruption in fish and highlight the importance of integrated biochemical and statistical approaches for early detection of sublethal toxic effects in aquaculture.*

Keywords: T-2 toxin; *Cyprinus carpio*; mycotoxins; lipids; proteins; phospholipids; aquaculture; biomarkers; correlation analysis; regression analysis



ФОСФОЛІПІДИ КРОВІ ТА БІЛОК У М'ЯЗОВІЙ ТКАНИНІ ЯК ЧУТЛИВІ БІОМАРКЕРИ ТОКСИЧНОСТІ МІКОТОКСИНУ Т-2 У КОРОПА ЗВИЧАЙНОГО (*CYPRINUS CARPIO* L.)

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Мікотоксин Т-2, високотоксичний трихотецен, що продукується видами *Fusarium*, становить значну загрозу для аквакультури через свою стабільність і поширеність у забруднених кормах. Метою цього дослідження було оцінити вплив дієтичного надходження токсину Т-2 на рівні загальних ліпідів і білків у крові та м'язовій тканині коропа звичайного (*Cyprinus carpio*), з особливим акцентом на їх потенціал як біомаркерів токсичного стресу. Рибу масою 250-300 г піддавали дії токсину Т-2 у концентрації 0,25 мг/кг корму (у п'ять разів вище за максимально допустиму концентрацію) протягом 14 діб у контрольованих лабораторних умовах. Загальні ліпіди, фосфоліпіди та загальний білок визначали стандартними біохімічними методами. Статистичний аналіз включав *t*-критерій Стьюдента, розмір ефекту Коена (*d*), кореляційний аналіз Пірсона та лінійну регресію. Результати виявили значні метаболічні порушення, індуковані токсином Т-2. Рівні загальних ліпідів, фосфоліпідів і білка в крові були достовірно знижені ($p < 0,05$), при цьому найсильніший ефект спостерігався для фосфоліпідів (d Коена = 4,95). Натомість у м'язовій тканині відзначалося значне підвищення вмісту загальних ліпідів ($p = 0,033$) у поєднанні з вираженим зниженням білка ($p < 0,001$; d Коена = 6,51). Це свідчить про зсув у бік накопичення ліпідів і катаболізму білків. Кореляційний аналіз показав координовану метаболічну регуляцію в крові з сильними позитивними кореляціями між усіма параметрами. Натомість у м'язовій тканині виявлено порушення метаболічної інтеграції, зокрема сильну негативну кореляцію між вмістом ліпідів та білків ($r = -0,72$). Регресійний аналіз підтвердив, що рівні ліпідів є значущими предикторами зниження білка в м'язовій тканині ($R^2 = 0,52$), тоді як фосфоліпіди були сильними предикторами концентрації білка в крові ($R^2 = 0,66$). Таким чином, вплив токсину Т-2 спричинив виражені тканинно-специфічні метаболічні зміни, що характеризуються системним виснаженням циркулюючих біомолекул і локальним метаболічним дисбалансом у м'язовій тканині. Фосфоліпіди крові та м'язовий білок виявилися високочутливими біомаркерами токсичності Т-2. Отримані результати надають нові уявлення про механізми метаболічних порушень, індукованих мікотоксинами у риб, та підкреслюють важливість інтегрованих біохімічних і статистичних підходів для раннього виявлення сублетальних токсичних ефектів в аквакультурі.

Ключові слова: токсин Т-2; *Cyprinus carpio*; мікотоксини; ліпіди; білки; фосфоліпіди; аквакультура; біомаркери; кореляційний аналіз; регресійний аналіз



Introduction. Mycotoxins are toxic secondary metabolites produced by filamentous fungi that pose a significant threat to terrestrial and aquatic organisms alike (Bittner M. et al., 2026). T-2 toxin, a type A trichothecene primarily produced by *Fusarium* species, is one of the most potent inhibitors of protein synthesis and cellular metabolism (Janik E. et al., 2021). Due to its stability and persistence, T-2 toxin often contaminates feed ingredients, including those used in aquaculture, posing a serious risk to fish health and productivity (Vulić A. et al., 2025). Contamination can occur at any stage of feed production or storage, which makes eliminating mycotoxins from aquafeeds extremely challenging (Gruber-Dorninger C. et al., 2025).

Fish are particularly vulnerable to dietary mycotoxins as these compounds can disrupt key physiological and biochemical processes including energy metabolism, immune responses and tissue integrity. Furthermore, unlike terrestrial livestock, fish are continuously exposed to the aquatic environment, which may facilitate additional routes of toxin exposure and bioaccumulation (Oliveira M. and Vasconcelos V., 2020; Rose M., 2026). Lipids and proteins are essential macromolecules involved in structural, energetic and regulatory functions, so alterations in their metabolism can be a sensitive indicator of toxic stress (Rose M., 2026). Circulating lipids and phospholipids, in particular, reflect systemic metabolic status, whereas muscle tissue provides insight into long-term energy storage and protein turnover (Bargui R. et al., 2021). Consequently, analysing blood and muscle tissue simultaneously provides a more comprehensive assessment of an organism's response to toxic insults.

Previous studies have demonstrated that T-2 toxin induces oxidative stress, inhibits ribosomal function and disrupts membrane integrity, ultimately resulting in impaired cellular homeostasis (Yang J. et al., 2020; Chen X. et al., 2021). However, data on the combined effects of T-2 toxin on lipid–protein interactions and their tissue-specific regulation in fish are limited. Furthermore, most existing studies concentrate on individual biochemical endpoints, which may not adequately capture the complexity of metabolic disturbances caused by mycotoxins (Yin H. et al., 2020; Yu X. et al., 2023). Understanding these relationships is essential for identifying reliable biochemical biomarkers of toxicity and elucidating the mechanisms underlying toxin-induced metabolic disturbances.

The present study therefore aimed to investigate the effects of T-2 mycotoxin exposure on lipid and protein metabolism in the blood and muscle tissues of common carp *Cyprinus carpio*, with a particular focus on the interrelationships between biochemical parameters, their statistical significance and their potential as indicators of metabolic disruption. This approach enables the evaluation of both individual parameter changes and coordinated metabolic shifts, which may serve as early warning signals of toxic exposure in aquaculture species. This work was supported by CRASP and Elsevier, who awarded scholarships that greatly enhanced the research capabilities of Ukrainian PhD candidates and academic staff. Without this support, it would not have been possible to conduct the present study at the Institute of Biology at the Pomeranian University in Słupsk.

Materials and methods.

Experimental animals and husbandry. The study examined the common carp (*Cyprinus carpio*). The fish were obtained from a natural water reservoir (the wintering pond of the "Chernihivrybhos" aquaculture facility, Chernihiv, Ukraine). During the rearing period, the fish were fed a standard commercial diet in accordance with the recommended seasonal feeding regimes. Prior to the experiment, the fish were captured and transferred to laboratory aquaria, where they were acclimatised to the controlled conditions over a period of three days. During this period, the fish were not fed (winter



fasting conditions), and only healthy individuals without visible signs of disease were selected for further experimentation. An ichthyopathological examination confirmed the absence of ectoparasites and helminths. All procedures were conducted in accordance with the Local Ethical Committee for Animal Experiments' guidelines (T.H. Shevchenko National University "Chernihiv Colehium", Chernihiv, Ukraine), ensuring minimal stress and humane treatment of the fish throughout the experiment.

Experimental design. The experiment was conducted in 200-litre glass aquaria containing dechlorinated tap water. The fish were stocked at a density of one per 40 dm³ of water. The water temperature was kept close to natural seasonal conditions throughout the experiment. The hydrochemical parameters of the water were continuously monitored and kept within the following optimal ranges: dissolved oxygen, 9.6-12.5 mg/dm³; pH, 7.4-8.4; and ammonia concentration, 0.014 mg/dm³. These conditions ensured that the experimental animals did not experience hypoxia, hypercapnia or thermal stress. The fish weighed between 250 and 300 g, and the experimental period lasted 14 days.

T-2 mycotoxin exposure. The experimental group of fish were exposed to the T-2 mycotoxin via their feed. The toxin was incorporated into the commercial diet at a concentration five times higher than the maximum permissible level (5 MPC; 0.25 mg/kg of feed). The fish were fed once daily throughout the 14-day experimental period. The control group received the same diet, but without T-2 toxin supplementation.

Sample preparation and biochemical analyses. At the end of the experimental period, blood samples were collected from the caudal vein of the fish using sterile syringes. After this, the fish were euthanised in accordance with standard ethical procedures and tissue samples (including muscle tissue) were collected for biochemical analysis.

The muscle tissue was then excised *in situ* under aseptic conditions immediately afterwards. The organs were then perfused with an ice-cold isolation buffer solution to remove any remaining blood. This consisted of a Tris-HCl buffer solution at a pH of 7.2. All of the reagents used were of analytical grade. The muscle tissue was homogenised using a motorised pestle in a glass H500 homogeniser and kept in an ice-water bath to prevent protein degradation. This resulted in a 1:9 (weight/volume) homogenate. The homogenates were then centrifuged at 3,000 rpm (approximately 1,000×g) for 15 minutes at 4 °C. After centrifugation, the supernatant was collected and stored at -25 °C until biochemical analysis. All samples were thawed only once before analysis to minimise protein denaturation and artefacts of oxidation.

Total protein content in tissues was determined using the Lowry method, with commercially available biochemical kits (DIALAB, Austria), following the manufacturer's instructions. Total lipids and phospholipids were quantified using standard biochemical assay kits (Filisit, Ukraine).

Statistical analysis. All data are expressed as the mean ± standard deviation (S.D.). Statistical analyses were performed using standard parametric methods. Differences between the control and experimental groups were evaluated using a Student's t-test, with $p < 0.05$ being considered statistically significant. Effect sizes were calculated using Cohen's *d* to assess the magnitude of the differences between the groups. Pearson's correlation coefficient (*r*) was used to analyse relationships between variables, and linear regression analysis was applied to determine predictive relationships between selected biochemical parameters. The interpretation of Cohen's *d* values followed conventional thresholds: 0.2 (small), 0.5 (medium) and ≥ 0.8 (large) (Stanisz A., 2006, 2007). All statistical analyses were carried out using Statistica 13.3 (TIBCO Software



Inc., Palo Alto, USA). Graphs were generated in Microsoft Excel, and significance levels are indicated in the figure and table.

Research results. The effects of exposure to the T-2 mycotoxin on lipid and protein metabolism in carp *Cyprinus carpio* were evaluated in blood and muscle tissues. All data are presented as the mean \pm standard deviation (S.D.). Statistical differences between the control and experimental groups were assessed using a Student's t-test, with $p < 0.05$ being considered significant. Effect sizes were calculated using Cohen's d and relationships between variables were explored further using Pearson's correlation and linear regression analyses.

Figure 1 shows the levels of total lipids, phospholipids and total proteins in the blood and muscle tissue of carp *Cyprinus carpio* exposed to T-2 toxin.

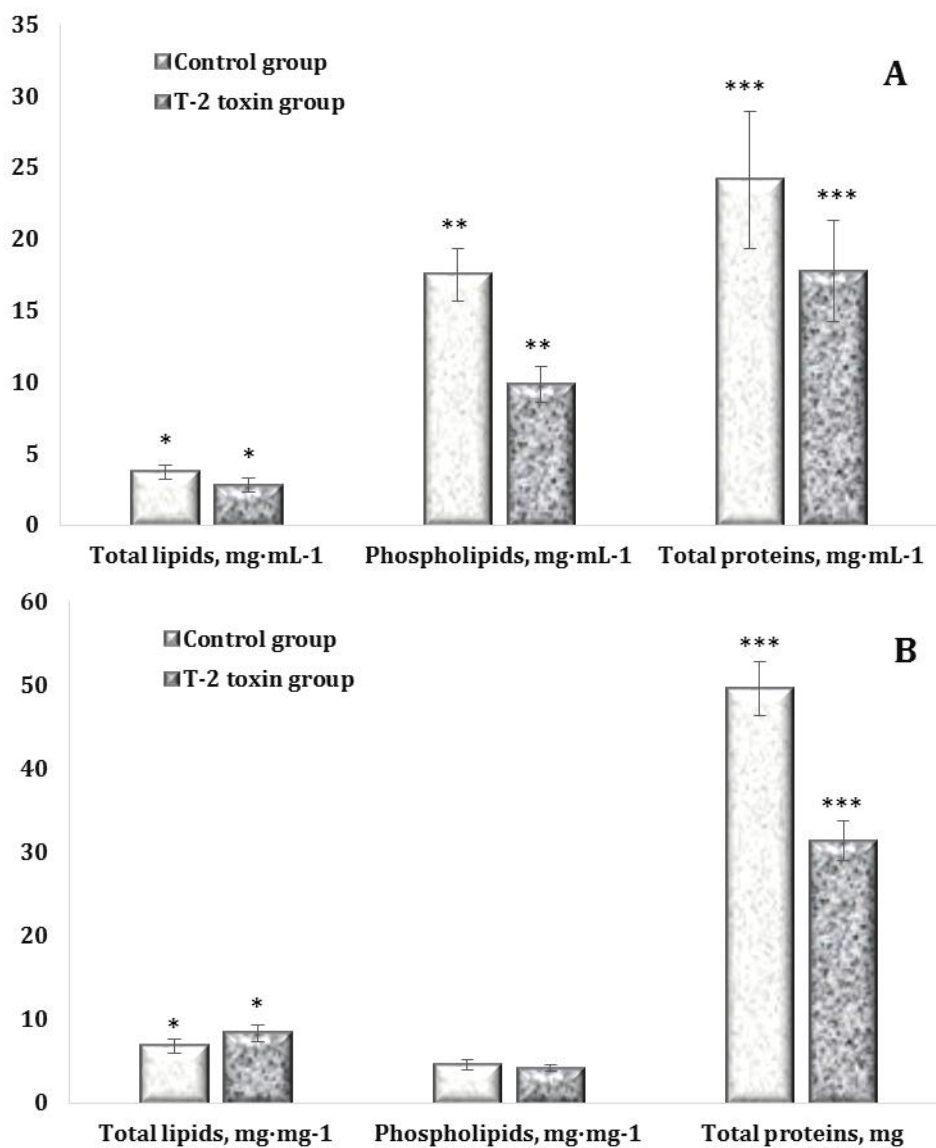


Fig. 1. Levels of total lipids, phospholipids and total proteins in the blood (A) and muscle tissue (B) of carp *Cyprinus carpio* exposed to T-2 toxin.

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

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



Total lipid levels in blood were significantly lower in the T-2-treated group ($2.77 \pm 0.49 \text{ mg}\cdot\text{mL}^{-1}$) than in the control group ($3.69 \pm 0.47 \text{ mg}\cdot\text{mL}^{-1}$; $p = 0.016$), with a large effect size (Cohen's $d = 1.90$). Conversely, muscle tissue exhibited a significant increase in total lipid content following T-2 exposure ($8.31 \pm 0.98 \text{ mg}\cdot\text{mg}^{-1}$ protein), compared to the control group ($6.77 \pm 0.90 \text{ mg}\cdot\text{mg}^{-1}$ protein; $p = 0.033$; Cohen's $d = 1.63$). These results suggest a shift in lipid metabolism, characterised by depletion in circulation and accumulation in muscle tissue (Fig. 1).

A significant decrease in phospholipid levels was observed in the blood of the T-2 group ($9.82 \pm 1.22 \text{ mg}\cdot\text{mL}^{-1}$), compared to the control group ($17.50 \pm 1.82 \text{ mg}\cdot\text{mL}^{-1}$). This difference was highly significant ($p < 0.001$), with an extremely large effect size (Cohen's $d = 4.95$). In muscle tissue, phospholipid levels showed a slight reduction ($4.11 \pm 0.36 \text{ mg}\cdot\text{mg}^{-1}$ protein versus $4.55 \pm 0.58 \text{ mg}\cdot\text{mg}^{-1}$ protein in the control group), which was not statistically significant ($p = 0.187$); however, the effect size remained moderate (Cohen's $d = 0.92$). Therefore, T-2 toxin had a pronounced effect on circulating phospholipids, while structural phospholipids in muscle tissue remained relatively unaffected (Fig. 1).

Total protein concentration in the blood was significantly lower in the T-2 group ($17.73 \pm 3.56 \text{ mg}\cdot\text{mL}^{-1}$) than in the control group ($24.14 \pm 4.82 \text{ mg}\cdot\text{mL}^{-1}$; $p = 0.044$), with a large effect size (Cohen's $d = 1.51$). An even greater effect was seen in muscle tissue, with a marked decrease in protein levels from $49.55 \pm 3.18 \text{ mg/g}$ in the control group to $31.30 \pm 2.37 \text{ mg/g}$ in the T-2-exposed group ($p < 0.001$), corresponding to an extremely large effect size (Cohen's $d = 6.51$).

Pearson's correlation analysis revealed distinct patterns in blood and muscle tissues. In blood, strong positive correlations were observed between all measured parameters, indicating coordinated metabolic regulation: total lipids and phospholipids ($r = 0.74$); total lipids and protein ($r = 0.62$); and phospholipids and protein ($r = 0.81$). In muscle tissue, however, correlations were weaker and more heterogeneous. A weak relationship was found between lipids and phospholipids ($r = 0.28$) and a weak-to-moderate correlation between phospholipids and protein ($r = 0.35$). Interestingly, a strong negative correlation was observed between total lipids and protein ($r = -0.72$), suggesting an inverse relationship between lipid accumulation and protein content.

Linear regression analysis further supported the observed relationships between the studied biochemical parameters. In muscle tissue, a significant inverse association was identified between total lipid content and protein levels, as described by the equation $\text{Protein} = 68.2 - 4.45 \times \text{Lipids}$ ($R^2 = 0.52$, $p = 0.018$). This indicates that approximately 52% of the variability in muscle protein content can be explained by lipid levels. In blood, phospholipids were found to be strong predictors of total protein concentration, as shown by the regression model $\text{Protein} = 4.12 + 1.14 \times \text{Phospholipids}$ ($R^2 = 0.66$, $p = 0.006$). This model explains 66% of the variability in blood protein levels, confirming a strong metabolic link between phospholipids and protein in the bloodstream.

Thus, exposure to the T-2 mycotoxin induced significant and biologically meaningful alterations in the lipid and protein metabolism of *Cyprinus carpio*. These effects manifested as reductions in blood lipids, phospholipids and proteins, alongside a substantial increase in muscle lipid content and severe depletion of muscle proteins. The magnitude of these changes was confirmed by the large effect sizes observed for key parameters, particularly blood phospholipids and muscle protein. The strongest statistical effects were observed for blood phospholipids and muscle protein (both $p < 0.001$; Cohen's $d > 4.0$), indicating that these are the most sensitive biomarkers of T-2 toxicity. Furthermore, correlation and regression analyses revealed coordinated metabolic



regulation in the blood, in contrast to evident metabolic disruption and pronounced lipid–protein imbalance in muscle tissue.

Discussion. This study shows that exposure to the T-2 mycotoxin induces significant and tissue-specific changes to lipid and protein levels in the blood and muscle tissue of common carp *Cyprinus carpio*. These changes reflect a complex metabolic response involving systemic and local regulatory mechanisms. Notably, these effects occurred under sublethal exposure conditions, suggesting that even low levels of T-2 toxin can significantly disrupt metabolic homeostasis in fish. One of the most notable findings was the significant decrease in total blood lipids, accompanied by a simultaneous increase in muscle tissue lipid content. This redistribution suggests a shift in energy metabolism, likely associated with the enhanced mobilisation of circulating lipids and their subsequent deposition in peripheral tissues (Zhang J. et al., 2022; Heikkinen T. et al., 2025). This pattern may be a compensatory response to toxic stress, where energy reserves are reallocated to maintain cellular function in adverse conditions. Alternatively, it may indicate impaired lipid transport and utilisation, which has been linked to the hepatotoxic effects of T-2 toxin reported in previous studies (Wu Q. et al., 2011; An K. et al., 2024, 2025; Wang Y. et al., 2025). Given the liver's central role in lipid metabolism, including lipoprotein synthesis and lipid trafficking, its dysfunction could directly contribute to the altered lipid distribution observed in this study (Janik E. et al., 2021).

Another key observation is the marked reduction in blood phospholipids, which represents a closely related finding. This reduction has an extremely large effect size, indicating high sensitivity to T-2 exposure. Phospholipids are essential components of cellular membranes, playing a critical role in maintaining their fluidity, integrity and signalling. Therefore, their depletion in the blood may indicate membrane damage, increased lipid peroxidation, or impaired phospholipid synthesis (Wang B. & Tontonoz, 2019; Ventura R. et al., 2022). This interpretation is supported by T-2 toxin's well-documented ability to induce oxidative stress and generate reactive oxygen species, which target membrane lipids preferentially (Wu Q. H. et al., 2014; He J. et al., 2024). In contrast, the relatively stable phospholipid levels observed in muscle tissue suggest that structural membrane components are either more resistant to short-term toxic insults or that compensatory mechanisms preserve membrane integrity in this tissue. It is also possible that muscle tissue exhibits lower membrane phospholipid turnover compared to circulating fractions, contributing to their relative stability (Mukund K. & Subramaniam S., 2020).

The effects of T-2 toxin were also evident in protein metabolism, as demonstrated by significant reductions in blood and muscle protein levels. The particularly pronounced decrease in muscle protein, associated with an extremely large effect size, is consistent with the well-documented ability of trichothecenes to inhibit protein synthesis by binding to ribosomal subunits (Janik E. et al., 2021; Gu W. et al., 2023; Vörösházi J. et al., 2024). Additionally, increased proteolysis may contribute to the observed depletion, reflecting a shift towards catabolic processes under toxic stress. This catabolic activation may be mediated by stress-related hormonal responses (e.g. cortisol), which promote protein breakdown in fish under adverse environmental conditions (Canosa L. F. & Bertucci J. I., 2023; Lemos L. S. et al., 2023).

Correlation analysis provides further insight into the relationships between biochemical parameters, helping us to better understand the organisation of these metabolic disturbances. In the blood, strong positive correlations between lipids, phospholipids and proteins suggest coordinated regulation and indicate that these components are closely linked within systemic metabolism. This coherence may reflect homeostatic mechanisms that attempt to stabilise internal conditions despite toxin



exposure. Such coordinated responses are characteristic of integrated metabolic networks, where the disruption of one component is rapidly compensated for by adjustments in others (Jarc E. & Petan T., 2019; Vendruscolo M., 2022).

In contrast, muscle tissue exhibited disrupted relationships, notably a strong negative correlation between lipids and proteins. This suggests a metabolic imbalance in which lipid accumulation occurs at the expense of protein content. This inverse relationship may indicate a shift towards lipid storage or altered energy utilisation pathways, or away from protein synthesis (Adhikari M. et al., 2017). Regression analysis further supports this interpretation by demonstrating that lipid levels significantly predict protein depletion in muscle tissue, explaining over half of its variability. This implies that lipid accumulation may play a mechanistic role in driving protein loss in muscle tissue, rather than being merely a parallel process.

Similarly, the strong predictive relationship between phospholipids and proteins in blood underscores their functional interdependence. Given phospholipids' role in membrane-associated processes and protein transport, their depletion may directly affect protein stability and circulation (Yin, H. et al., 2020; Vörösházi J. et al., 2026). Disruption of lipoprotein complexes and membrane-bound protein systems may therefore be a key mechanism through which lipid and protein alterations occur under T-2 exposure.

Notably, the magnitude of the observed changes, as reflected by large and extremely large effect sizes, underscores the biological significance of these findings. Blood phospholipids and muscle proteins have emerged as particularly sensitive biomarkers of T-2 toxicity, suggesting their potential utility in monitoring sublethal toxic effects in aquaculture systems. Their high sensitivity and clear directional changes make them especially valuable for the early detection of metabolic disturbances, even before overt clinical symptoms appear (Banaee M. et al., 2024; Grădinariu L. et al., 2025).

The metabolic disturbances observed in the current study are consistent with the toxic effects of T-2 mycotoxin previously reported in *Cyprinus carpio*, particularly with regard to oxidative stress, DNA damage and the disruption of biochemical homeostasis. The significant alterations in lipid and protein metabolism identified in our experiment can be explained by trichothecenes' capacity to induce oxidative damage and impair cellular macromolecules. Indeed, it has been demonstrated that T-2 toxin exerts a pronounced genotoxic effect, leading to DNA lesions in common carp during dietary exposure, particularly in the early phases of intoxication, although some adaptive responses may occur over time (Szabó R. T. et al., 2021). This suggests that the biochemical changes observed in our study, particularly the depletion of circulating lipids and proteins, may reflect underlying DNA damage and impaired cellular function.

The redistribution of lipids observed, characterised by decreased blood levels and increased muscle tissue accumulation, may be closely linked to oxidative stress mechanisms. Previous studies have shown that T-2 toxin can modulate lipid peroxidation and the glutathione redox system, although the extent of these changes depends on the duration and dose of exposure (Pelyhe, C. et al., 2016a; Kövesi B. et al., 2020). While short-term exposure can activate antioxidant defence systems, including increased glutathione content and glutathione peroxidase activity, prolonged exposure does not necessarily lead to sustained lipid peroxidation, indicating the presence of compensatory mechanisms (Pelyhe, C. et al., 2016b). Nevertheless, the disruption to lipid metabolism observed in our study is consistent with reports of altered triglyceride levels and increased oxidative stress in carp fed T-2-contaminated diets (Matejova I. et al., 2017).

Thus, the results suggest that the T-2 toxin disrupts metabolic homeostasis at multiple levels, impacting energy storage and structural components and leading to



pronounced imbalances in lipid and protein metabolism. These findings are consistent with known trichothecene toxicity mechanisms, including oxidative stress, mitochondrial dysfunction and protein synthesis inhibition. Due to the presence of a reactive thiol group, the T-2 toxin is a potent inhibitor of protein and DNA synthesis (Hossam E.D.M.O., 2013). It suppresses lymphocyte proliferation, disrupts membrane integrity, impairs antibody production and interferes with dendritic cell development, thereby weakening immune function (Obremski K. et al., 2013; Guilford F. T., & Hope J., 2014). At the cellular level, T-2 toxin induces apoptosis in a wide range of *in vitro* models, including human liver cells and various immune cell lines. Apoptotic changes have also been observed *in vivo* in multiple organs, such as the skin, kidney, brain and bone marrow, in animal models (Afsah-Hejri L. et al., 2013). These effects are largely attributed to oxidative stress driven by reactive oxygen species (ROS), such as hydrogen peroxide, hydroxyl radicals and superoxide anions. Mitochondrial complex I and cytochrome P450 systems play key roles in ROS generation (Mary V. S. et al., 2012).

T-2 toxin primarily inhibits protein synthesis by binding to the 60S ribosomal subunit and blocking peptidyl-transferase activity, thereby preventing polypeptide chain initiation (Henghold W. B., 2004; Afsah-Hejri L. et al., 2013). This effect is particularly pronounced in rapidly proliferating tissues, including the gastrointestinal tract, bone marrow, and erythroid cells (Rai R. B. et al., 2011). The toxin also interferes with multiple enzymes involved in DNA replication, cellular metabolism, and coagulation pathways (Johnsen H. et al., 1988). T-2 toxin has also been shown to modulate host–pathogen interactions by enhancing *Salmonella* uptake in macrophages via the activation of the MAPK/ERK1/2 signalling pathway, which leads to cytoskeletal rearrangements (Antonissen G. et al., 2014).

At the subcellular level, trichothecenes disrupt the structure of mitochondria, the rough endoplasmic reticulum and cellular membranes, while also inhibiting key metabolic enzymes, such as succinate dehydrogenase. This ultimately impairs cellular energetics and mitochondrial protein synthesis (Hossam E.D.M.O., 2013; Afsah-Hejri L. et al., 2013). Their ability to cross the placental barrier and induce apoptosis in foetal tissues further highlights their systemic toxicity (Li Y. et al., 2011). However, some protective effects against T-2-induced oxidative stress have been reported, such as the mitigating role of L-carnitine in hepatocytes (Moosavi M. et al., 2016), and modulation of steroidogenesis via the cAMP-PKA pathway (Wu J. et al., 2015).

The genotoxic and cytotoxic properties of T-2 toxin are closely linked to its ability to inhibit DNA, RNA and protein synthesis, thereby disrupting the cell cycle and promoting apoptosis (Horvatovich K. et al., 2013). Similar to radiation-induced injury, T-2 toxin induces DNA damage, gene mutations, micronuclei formation and chromosomal instability, ultimately impairing intercellular communication and immune competence (Shokri F. et al., 2000).

Apoptosis induced by T-2 toxin is mediated through multiple signalling pathways, particularly stress-activated MAP kinases, including JNK and p38, as well as ERK1/2, which together regulate cell survival and death (Li M. & Pestka J. J., 2008). The toxin can also trigger apoptotic cascades via the upregulation of Fas receptors and the p53 protein, which leads to an altered Bax/Bcl-2 ratio and the activation of caspase-dependent pathways (Bouaziz C. et al., 2008). Furthermore, DNA damage caused by T-2 toxin activates the ATM–Chk2 signalling axis, thereby promoting apoptosis (Rakkestad K. E. et al., 2010). ROS-mediated mechanisms play a central role in these processes by enhancing p53 expression and mitochondrial cytochrome c release, which drives caspase-dependent cell death (Chaudhary M. & Rao P. V., 2010). Furthermore, caspase-



independent pathways involving apoptosis-inducing factor (AIF) contribute to DNA fragmentation and cell death.

Importantly, several studies suggest that T-2-induced apoptosis may be partially reduced. Selenium and its derivatives, including nano-selenium and selenium-chondroitin sulphate complexes, have been shown to reduce apoptosis by modulating the Bax/Bcl-2 ratio and suppressing the activation of the MAPK pathway (Chen J. et al., 2006; Han J. et al., 2013). These findings suggest potential protective strategies against T-2 toxin-induced cellular damage.

Thus, these findings shed new light on the mechanisms of T-2 toxin action in fish and emphasise the importance of combining biochemical and statistical methods in toxicological research. Combining effect size analysis with correlation and regression modelling was particularly valuable for identifying key relationships and sensitive toxicity indicators. These findings also emphasise the importance of monitoring mycotoxin contamination in aquaculture to prevent adverse effects on fish health and productivity. Future studies should focus on long-term exposure scenarios and dose-response relationships involving additional tissues (e.g. the liver) to elucidate the systemic impact of T-2 toxin further and validate the proposed biomarkers under field conditions.

Conclusions. The present study demonstrates that exposure to the T-2 mycotoxin leads to significant and biologically meaningful disturbances in the metabolism of lipids and proteins in *Cyprinus carpio*, with clear tissue-specific patterns. These alterations were consistent across multiple biochemical endpoints, which reinforces the robustness and internal coherence of the observed responses.

Specifically, T-2 toxin induced a decrease in circulating lipids, phospholipids and proteins, alongside an increase in muscle lipid content and a significant reduction in muscle protein. These changes reflect a redistribution of metabolic resources and a shift towards catabolic processes. The strongest effects were observed in blood phospholipids and muscle protein, which exhibited extremely large effect sizes and could therefore serve as sensitive biomarkers of T-2 toxicity. Their responsiveness suggests they could be used to detect subclinical metabolic disturbances in exposed fish populations early on.

Furthermore, correlation and regression analyses revealed coordinated metabolic regulation in the blood, in contrast to the pronounced metabolic disruption observed in muscle tissue, particularly in the form of lipid–protein imbalance. This indicates that systemic homeostasis is partially maintained while local tissue metabolism becomes dysregulated under toxic conditions. This divergence between systemic and tissue-specific responses emphasises the importance of multi-compartment analysis in ecotoxicological assessments. This study provides new insights into integrative metabolic responses to T-2 toxin in fish, highlighting the importance of multi-parameter approaches in ecotoxicological research.

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