Print ISSN : 0972-8813
e-ISSN : 2582-2780

[Vol. 24(1) January-April 2026]

Pantnagar Journal of Research

(Formerly International Journal of Basic and Applied Agricultural Research ISSN : 2349-8765)

G.B. Pant University of Agriculture & Technology
Pantnagar, U.S. Nagar; Uttarakhand, Website : gbpuat.res.in/PJR

PANTNAGAR JOURNAL OF RESEARCH

Vol. 24(1) January-April, 2026

CONTENTS

https://doi.org/10.67063/pjr.vol.24.issue.01.18

Biochemical changes associated with triclosan exposure in the freshwater fish *Cyprinus carpio*

MADHU SHARMA[1*], YASHPAL[2], RIJUL RANA[2], DEEKSHA[2], DEEPIKA THAKUR[2] and TARANG KUMAR SHAH[1]

[1]Department of Fisheries, DGCN COVAS, CSK Himachal Pradesh Krishi Visvavidyalaya, Palampur, [2] Department of Zoology, SCVB Degree College, Palampur (H.P.)
[*]Corresponding author's email id: madhu.srma@gmail.com

ABSTRACT: The present study evaluated the biochemical responses of the freshwater fish *Cyprinus carpio* post sublethal exposure to triclosan. Fish were exposed to two concentrations of triclosan (0.242 mg/L and 0.484 mg/L) for 7, 14, and 21 days, followed by a 7-day recovery period under laboratory conditions. Biochemical parameters like glucose, cholesterol, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were analyzed to assess metabolic and hepatic stress responses. The results revealed significant, concentration- and time-dependent increases in glucose and cholesterol levels in the exposed groups compared to the control and solvent control groups. Similarly, the activities of hepatic enzymes AST and ALT showed marked elevations during the exposure period, suggesting possible liver dysfunction and cellular damage induced by triclosan. These biochemical alterations indicate disturbances in carbohydrate and lipid metabolism along with impairment of hepatic integrity. However, during the recovery period, most parameters showed a noticeable decline toward control values, indicating partial physiological recovery after removal of the toxicant. Overall, the findings demonstrate that triclosan exposure can induce significant biochemical disturbances in *Cyprinus carpio* and highlight the usefulness of biochemical biomarkers as sensitive indicators of aquatic pollution. This study underscores the potential ecological risks associated with triclosan contamination in freshwater ecosystems.

Key words: Biochemical parameters, *Cyrinus carpio,* Triclosan, Toxicity

Triclosan (TCS) has been widely used since 1968 as an antiseptic, disinfectant, and preservative in clinical and consumer products. It is commonly incorporated into cosmetics, household cleaning agents, and various materials, including medical devices, plastics, textiles, and kitchen utensils (Bedoux *et al*., 2012). Due to its widespread application and persistence, TCS has become a contaminant of global concern, with its occurrence extensively reported in rivers, lakes, and estuarine environments across America, Asia, Africa, Australia, and Europe.

The toxicity of TCS has been widely reported in a diverse array of aquatic species, such as algae, daphnids, and fish (Orvos *et al*., 2002). In aquatic environment, TCS is recognized to trigger oxidative stress by increasing the generation of reactive oxygen species (ROS), leading to mitochondrial dysfunction, as demonstrated in mammalian cell studies (Jin *et al*., 2020; Teplova *et al*., 2017). Furthermore, TCS acts as an endocrine disruptor, adversely affecting reproductive and developmental processes in fish (Wang and Tian, 2015; Iannetta *et al*., 2022; Li *et al*., 2024).

At the molecular level, TCS exposure has been shown to alter gene expression and enzyme activity related to detoxification, metabolism, and neurophysiological functions, along with cytoskeletal organization in *Labeo rohita* (Sharma *et al*., 2022). These molecular alterations are further reflected at the physiological level, where significant alterations in haematological parameters have been reported in *Pangasianodon hypophthalmus* and *Cirrhinus mrigala* (Paul *et al*., 2019; Dar *et al*., 2024). Moreover, TCS has been found to induce cytogenotoxic effects in fish species such as *Danio rerio*, *L. rohita*, and *P. hypophthalmus*, indicating its potential to cause genetic damage (Parenti *et al*., 2019; Sharma *et al*., 2021). Recent studies further emphasize its role as a potent metabolic disruptor, causing alterations in carbohydrate metabolism, amino acid regulation, and nitrogen metabolic processes, together with endocrine and developmental disturbances (Wang *et al*., 2025; Das *et al*., 2025).

In the Indian context, although studies are relatively limited, available reports indicate the widespread occurrence of TCS in aquatic systems. It has been detected in both water and sediment matrices across various regions (Ramaswamy *et al*., 2011; Nag *et al*., 2018; Thilakan *et al*., 2019; Singh and Suthar, 2021; Biswas and Vellanki, 2021; Prasad *et al*., 2024; Kumar *et al*., 2024b). A study conducted in South Indian rivers, including the Cauvery, Vellar, and Tamiraparani rivers, reported comparatively higher environmental risk in the Cauvery River (Ramaswamy *et al*., 2011). Notably, the occurrence of TCS has increased following the COVID-19 pandemic, likely due to the intensified use of TCS-containing personal hygiene products, with several studies reporting elevated levels in rivers and coastal ecosystems (Kumar *et al*., 2021; Gopal *et al*., 2021; Prasad *et al*., 2024).

Despite growing evidence of triclosan toxicity, limited information is available regarding its sublethal effects on economically important freshwater species such as *Cyprinus carpio*, particularly under controlled exposure and recovery conditions. Therefore, the present study aims to evaluate the biochemical responses of *C. carpio* following sublethal exposure to triclosan and to assess the potential for physiological recovery after cessation of exposure. The findings of this study will contribute to a better understanding of triclosan-induced toxicity and highlight the importance of biochemical biomarkers in monitoring aquatic environmental health.

MATERIALS AND METHODS

Fish collection and acclimatization

Sixty specimens of freshwater fish, *Cyprinus carpio* (common carp), with an average length of 18 ± 0.5 cm and body weight of 100 ± 5.5 g, were procured from the fish farm of the Department of Fisheries, Dr. G.C. Negi College of Veterinary and Animal Sciences (DGCN-COVAS), CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur, Himachal Pradesh, India. Prior to experimentation, the fish were acclimatized for 10 days in aerated glass aquaria with a capacity of 100 L. The aquaria were filled with dechlorinated bore-well water, and the temperature was maintained at 22–26°C, with a pH range of 7.6–7.8. Continuous aeration was provided using an air pump to ensure adequate dissolved oxygen levels.

During the acclimatization period, key water quality parameters, including pH, ammonia, total hardness, carbonate hardness, nitrite, and nitrate, were monitored regularly. The fish were fed a commercial pelleted diet once daily. The aquaria were cleaned at regular intervals to maintain optimal water quality and ensure the health and well-being of the fish.

Chemical

Analytical grade triclosan (Sigma Aldrich, USA; CAS-No. 72779-5G-F, purity≥97.0%) was used for the study. The stock solution (10 mg/mL) of TCS was prepared by dissolving the compound in 1% dimethyl sulphoxide (DMSO) solution as solvent. From this stock solution, an appropriate quantity was taken and dissolved in experimental tanks (each containing 100 L water) to get the desired concentration of test solutions. Two Sublethal Concentrations were selected based on the previously reported LC_{50} value 0.968 mg/L for *C. carpio* (Tripathi *et al*., 2024).

Experimental design

Fish were kept in five different groups (*n* = 20/ group)- Control (without TCS); Vehicle control (solvent, 1% DMSO); Exposed to 0.048 mg/L of TCS (1/20th of LC_{50}); Exposed to 0.024 mg/L of TCS (1/40th of LC_{50}). Sampling was done at every 7 days interval and was continued till 28 days (7, 14, 21, and 28 days). At each time interval, five fish were sampled for histopathological and biochemical analysis.

Assessment of Plasma Biochemical Parameters

To evaluate the effects of triclosan exposure on biochemical parameters, blood samples were collected from fish at 7,14, 21 days and 7 days post-exposure. Blood was aseptically withdrawn from the caudal vein using a heparinized syringe to prevent coagulation and ensure sample integrity. Plasma biochemical parameters including Glucose, Cholesterol, AST/SGOT, and ALT/SGPT were analyzed using commercially available diagnostic assay kits designed for serum/plasma samples. For plasma separation, blood samples were centrifuged at 10,000 rpm for 10–15 min using a Tarson Spinning MC-00 centrifuge. The separated plasma was then analyzed using a Vchem Next Biochemistry analyzer according to the manufacturer's instructions.

Statistical analysis

Data were represented as the mean ± standard deviation of the mean (SD). One way analysis of variance (ANOVA) was used to detect a significant difference between exposed and controls, followed by Tukey's post hoc test. The statistical analysis was conducted through Statistical Package for the Social Sciences (SPSS) for Windows (standard version 26) software. Differences were considered significant at level of p <0.05.

RESULTS AND DISCUSSION

Exposure to triclosan caused significant alterations in the biochemical parameters of *Cyprinus carpio* compared to the control and solvent control groups. Blood glucose levels showed a significant dose- and time-dependent increase in the treated groups. At 0.024 mg/L, glucose increased from 7 days to 21 days, while at 0.048 mg/L, it increased from 7 days to 21 days. Similarly, cholesterol levels also increased progressively with exposure duration, reaching 126.82 mg/dl at 0.024 mg/L and 165.82 mg/dl at 0.048 mg/L after 21 days. Hepatic enzyme activities, including AST and ALT, showed a marked elevation in treated fish, indicating possible liver dysfunction. AST levels increased to 136.31 µg/L at 0.024 mg/L and 188.00 µg/L at 0.048 mg/L after 21 days, while ALT levels rose to 39.43 µg/L and 45.86 µg/L, respectively. However, after the 7-day recovery period, most biochemical parameters showed a noticeable decline toward control values, suggesting partial physiological recovery following removal of the toxicant.

The present study demonstrated significant alterations in biochemical parameters of *Cyprinus carpio* following exposure to triclosan, indicating metabolic stress and hepatic dysfunction. Similar responses have been widely reported in fish exposed to triclosan, suggesting common mechanisms of toxicity (Orvos *et al*., 2002; Dann & Hontela, 2011;

Table 1: Changes in selected biochemical parameters (Glucose, Cholesterol, AST/SGOT, and ALT/SGPT) in the blood of common carp, *Cyprinus carpio*, following exposure to sublethal concentrations of Triclosan (0.024 mg/L and 0.048 mg/L) for 7, 14, and 21 days, along with a 7-day recovery period. Values are presented as mean ± standard deviation (SD)

Parameter	C/T	7 Days	14 Days	21 Days	Recovery (7 Days)
Glucose (mg/dl)	Control	55.77 ± 3.21^{c1}	55.44 ± 2.15^{c1}	54.77 ± 3.21^{c1}	55.11 ± 4.31^{a1}
	Solvent control	55.46 ± 1.56^{c1}	54.46 ± 1.56^{c1}	55.80 ± 2.56^{c1}	54.13 ± 2.68^{a1}
	0.024 mg/L	71.56 ± 2.14^{b3}	87.55 ± 1.93^{b2}	106.71 ± 4.55^{b1}	39.71 ± 3.12^{b4}
	0.048 mg/L	95.71 ± 3.36^{a3}	118.19 ± 3.13^{a2}	141.95 ± 4.87^{a1}	40.10 ± 3.92^{b4}
Cholesterol (mg/dl)	Control	60.84 ± 1.96^{c1}	58.17 ± 1.37^{c1}	59.50 ± 1.45^{c1}	57.84 ± 1.61^{a1}
	Solvent control	59.33 ± 2.21^{c1}	59.33 ± 3.37^{c1}	60.66 ± 0.85^{c1}	60.00 ± 2.36^{a1}
	0.024 mg/L	73.79 ± 1.99^{b3}	102.48 ± 2.70^{b2}	126.82 ± 3.39^{b1}	59.59 ± 1.09^{a4}
	0.048 mg/L	84.80 ± 2.46^{a3}	115.62 ± 2.52^{a2}	165.82 ± 7.72^{a1}	60.00 ± 2.36^{a4}
AST / SGOT (µg/L)	Control	41.93 ± 1.46^{c1}	41.63 ± 2.00^{c1}	40.93 ± 1.46^{c1}	42.27 ± 1.99^{a1}
	Solvent control	40.80 ± 2.02^{c1}	43.10 ± 2.72^{c1}	41.33 ± 1.55^{c1}	41.13 ± 2.17^{a1}
	0.024 mg/L	66.27 ± 2.67^{b3}	95.30 ± 3.50^{b2}	136.31 ± 3.86^{b1}	44.53 ± 2.73^{a4}
	0.048 mg/L	75.92 ± 3.73^{a3}	123.93 ± 3.80^{a2}	188.00 ± 4.55^{a1}	43.00 ± 2.17^{a4}
ALT / SGPT (µg/L)	Control	13.43 ± 1.50^{c1}	13.73 ± 0.76^{c1}	13.73 ± 0.76^{c1}	13.73 ± 1.25^{a1}
	Solvent control	13.46 ± 0.97^{c1}	14.20 ± 0.96^{c1}	13.83 ± 1.33^{c1}	13.13 ± 0.72^{a1}
	0.024 mg/L	18.90 ± 0.72^{b3}	29.63 ± 0.95^{b2}	39.43 ± 1.06^{b1}	14.13 ± 1.35^{a4}
	0.048 mg/L	24.80 ± 1.49^{a3}	35.83 ± 1.75^{a2}	45.86 ± 1.62^{a1}	15.03 ± 0.32^{a4}

Values are expressed as Mean ± SD. Different superscript letters (a, b, c) within a column indicate significant differences between treatments, while different numbers (1, 2, 3,4) indicate significant differences among exposure durations at p < 0.05.

Ho *et al*., 2016). Previous studies have shown that triclosan exposure leads to significant disturbances in biochemical indices, including alterations in liver enzyme activities, lipid metabolism, and energy balance, reflecting hepatocellular damage and endocrine disruption in fish (Fang *et al*., 2016; Witorsch *et al*., 2014). These findings support the notion that triclosan exerts its toxic effects through disruption of metabolic and hepatic functions.

The progressive increase in glucose levels observed in the present study reflects a stress-induced hyperglycemic response, likely driven by enhanced glycogenolysis and gluconeogenesis to meet elevated energy demands. Such metabolic responses are regulated by stress hormones like cortisol and catecholamines, which stimulate glucose production and mobilization of energy reserves in fish (Iwama *et al*., 1999; Barton, 2002). Furthermore, cortisol plays a central role in elevating circulating glucose levels by promoting hepatic gluconeogenesis and glycogen breakdown during stress conditions (Vijayan *et al*., 2010; Mommsen *et al*., 1999). Comparable findings have been reported in triclosan-exposed fish, where disruption of energy metabolism and oxidative stress significantly altered carbohydrate balance (Sharma *et al.,* 2021). In some cases, prolonged triclosan exposure has also resulted in fluctuations or depletion of glucose levels during recovery, indicating metabolic exhaustion and impaired homeostasis. The significantly lower glucose levels observed in the recovery group compared to control suggest a post-exposure metabolic adjustment rather than complete physiological restoration. This reduction may be attributed to depletion of energy reserves during triclosan exposure, enhanced glucose utilization for tissue repair and antioxidant defense, and possible impairment of hepatic gluconeogenesis. Similar post-stress metabolic alterations in fish have been associated with toxicant exposure and recovery phases (Kumar *et al*., 2021; Dar *et al*., 2024).

The increase in cholesterol levels suggests disruption of lipid metabolism and possible hepatic impairment. Although limited studies in *C. carpio* directly report cholesterol changes under triclosan exposure, available evidence indicates that triclosan interferes with metabolic and endocrine pathways, leading to altered lipid regulation and physiological imbalance (Bouzidi *et al*., 2023; Liu *et al*., 2024; Sun *et al*., 2020). Recent studies have demonstrated that triclosan exposure induces hepatic oxidative stress, inflammation, and lipid accumulation, ultimately disrupting lipid metabolic pathways in fish (Liu *et al.,* 2024; Adhikari *et al*., 2023; Das *et al*., 2025). Additionally, multi-omics analyses have revealed that triclosan alters genes and metabolites involved in lipid metabolism and energy homeostasis, contributing to metabolic dysfunction and liver damage (Che *et al.,* 2025). Such alterations are consistent with the observed lipid disturbances in the present study.

The significant elevation of AST (SGOT) and ALT (SGPT) activities indicates hepatocellular damage and increased membrane permeability. Similar increases in transaminase activities have been reported in triclosan-exposed fish, where metabolic enzymes such as AST and ALT showed marked elevation in a concentration-dependent manner, reflecting liver injury and tissue damage (Liu *et al*., 2024; Zhang *et al*., 2022; Wang *et al*., 2021). These enzyme alterations are often associated with oxidative stress, as triclosan has been shown to impair antioxidant defense systems, reduce the activities of key enzymes such as superoxide dismutase (SOD) and catalase (CAT), and induce lipid peroxidation in fish tissues (Trivedi *et al*., 2025; Nag *et al*., 2025). Furthermore, recent studies using integrated biochemical and molecular approaches have confirmed that triclosan exposure triggers reactive oxygen species (ROS) generation, mitochondrial dysfunction, and hepatotoxicity in aquatic organisms, thereby reinforcing its role in liver damage (Che *et al*., 2025; Yang *et al*., 2023). Such biochemical disruptions are widely recognized as reliable indicators of hepatic dysfunction and toxicant-induced stress in fish. Similar increases in transaminase activities have been reported in triclosan-exposed fish species, suggesting hepatotoxic effects of this compound. A time- and dose-dependent inhibition of acetylcholinesterase (AChE) activity was observed in the brain tissue and glutathione-S-transferase (GST) activity in the liver tissue showed a significant increase with increasing concentrations of triclosan (Seenivasan *et al*., 2023) Furthermore, triclosan exposure has been reported to cause persistent biochemical disturbances even after the removal of the toxicant, indicating prolonged toxic effects on metabolic pathways (Liu *et al*., 2024; Li *et al*., 2022). Such delayed or incomplete recovery has been attributed to sustained oxidative stress, mitochondrial dysfunction, and slow elimination of triclosan from fish tissues (Sharma *et al*., 2021; Nag *et al*., 2025). Additionally, studies have shown that even after cessation of exposure, alterations in metabolic enzymes and antioxidant systems may persist due to residual tissue accumulation and disruption of endocrine and energy metabolism pathways (Sun *et al*., 2020; Che *et al*., 2025). This is comparable to the partial recovery observed in the present study, where biochemical parameters tended toward normalization but may still reflect underlying physiological stress.

CONCLUSION

Overall, the similarity between the present findings and triclosan-based studies suggests that these toxicants induce biochemical alterations primarily through oxidative stress, metabolic disruption, and hepatocellular damage. The observed dose- and time-dependent changes in glucose, cholesterol, AST, and ALT highlight the sensitivity of these parameters as reliable biomarkers for assessing toxicant-induced stress in fish.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the State Department of Fisheries H.P. (State Adhoc Misc. 2348-46) for providing the financial support necessary to carry out the present research work. The funding assistance rendered by the Department is sincerely appreciated, as it facilitated the successful completion of this study.

REFERENCES

Adhikari, A., Das, B. K., Ganguly, S., Nag, S. K., Sadhukhan, D. and Raut, S. S. (2023). Emerging contaminant triclosan incites endocrine disruption, reproductive impairments and oxidative stress in the commercially important carp, *Catla (Labeo catla)*: an insight through molecular, histopathological and bioinformatic approach. *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*, 268: 109605.

Barton, B. A. (2002). Stress in fishes: a diversity of responses with particular reference to changes in circulating corticosteroids. *Integrative and Comparative Biology*, 42: 517–525.

Bedoux, G., Roig, B., Thomas, O., Virginie Dupont, Barbara Le Bot (2012). Occurrence and toxicity of antimicrobial triclosan and by-products in the environment. *Environmental Science and Pollution Research*, 19: 1044–1065.

Biswas, P. and Vellanki, B. P. (2021). Occurrence of emerging contaminants in highly anthropogenically influenced River Yamuna in India. *Science of the Total Environment*, 782: 146741.

Bouzidi, I., Mougin, K., Beyrem, H., Alghonaim, M. I., Alsalamah, S. A., Qurtam, A. A. *et al*. (2023). Physiological impairment and biochemical modifications induced by triclosan in Mediterranean mussels. *Animals*, 13: 583.

Che, J., Li, Z., Wang, X., Guo, M., Wang, H., Huang, L. *et al*. (2025). Integrated transcriptomics, metabolomics, and histopathology reveal molecular mechanisms of sublethal triclosan exposure in mosquitofish larvae (*Gambusia affinis*). *Aquatic Toxicology*, 286: 107471.

Dann, A. B. and Hontela, A. (2011). Triclosan: environmental exposure, toxicity and mechanisms of action. *Journal of Applied Toxicology*, 31: 285–311.

Dar, O. I., Vinothkanna, A., Aslam, B., Furkh, A., Sharma, S., Kaur, A., Gao, Y.-A. and Jia, A.-Q. (2024). Dynamic alterations in physiological and biochemical indicators of *Cirrhinus mrigala* hatchlings: a sublethal exposure of triclosan. *Science of the Total*

Environment, 924: 171701.

Dar, O. I., Vinothkanna, A., Aslam, B., Furkh, A., Sharma, S., Kaur, A., Gao, Y.-A. and Jia, A.-Q. (2024). Dynamic alterations in physiological and biochemical indicators of *Cirrhinus mrigala* hatchlings: a sublethal exposure of triclosan. *Science of the Total Environment*, 924: 171701.

Das, B. K., Chakraborty, H. J., Ghosh, S., Ganguly, S., Adhikari, A., Kumar, V. and Nag, S. K. (2025). Environmentally relevant concentrations of triclosan modulate the brain proteome profile of *Labeo catla*. *Frontiers in Marine Science*, 12: 1672496.

Fang, J.-L., Vanlandingham, M., da Costa, G. G. and Beland, F. A. (2016). Absorption and metabolism of triclosan after application to the skin of B6C3F1 mice. *Environmental Toxicology*, 31: 609–623.

Gopal, C. M., Bhat, K., Ramaswamy, B. R., Kumar, V., Singhal, R. K., Basu, H. *et al*. (2021). Seasonal occurrence and risk assessment of pharmaceutical and personal care products in Bengaluru rivers and lakes, India. *Journal of Environmental Chemical Engineering*, 9: 105610.

Ho, C. H., Hsiao, C. D., Kawakami, K. and Tse, W. K. F. (2016). Triclosan exposure impairs lipid metabolism in zebrafish embryos. *Aquatic Toxicology*, 173: 29–35.

Iannetta, A., Caioni, G., Di Vito, V., Benedetti, E., Perugini, M. and Merola, C. (2022). Developmental toxicity induced by triclosan exposure in zebrafish embryos. *Birth Defects Research*, 114: 175–183.

Iwama, G. K., Vijayan, M. M., Forsyth, R. B. and Ackerman, P. A. (1999). Heat shock proteins and physiological stress in fish. *American Zoologist*, 39: 901–909.

Jin, J., Chen, N., Pan, H., Xie, W., Xu, H., Lei, S. *et al*. (2020). Triclosan induces ROS-dependent cell death and autophagy in A375 melanoma cells. *Oncology Letters*, 20: 73.

Kumar, G., Kumar, S., Paul, T., Pal, P., Shukla, S. P., Kumar, K. *et al*. (2024). Ecotoxicological risk assessment of triclosan in riverine and estuarine ecosystems: a comparative study. *Marine Pollution Bulletin*, 205: 116667.

Kumar, S., Paul, T., Shukla, S. P., Kumar, K., Karmakar, S., Bera, K. K. and Kumar, C. B. (2021). Biomarkers-based assessment of triclosan toxicity in aquatic environment: a mechanistic review. *Environmental Pollution*, 286: 117569.

Kumar, S., Paul, T., Shukla, S. P., Kumar, K., Karmakar, S., Bera, K. K. and Kumar, C. B. (2021). Biomarkers-based assessment of triclosan toxicity in aquatic environment: a mechanistic review. *Environmental Pollution*, 286: 117569.

Li, Z., Xian, H., Ren, X., Ye, R., Zhong, Y., Huang, Y. *et al*. (2024). Insights into triclosan-induced endocrine disruption: evidence from NHANES and zebrafish models. *Environmental Health*, 23: 45.

Liu, J., Lu, L., Song, H., Liu, S., Liu, G., Lou, B. and Shi, W. (2024). Effects of triclosan on lipid metabolism in *Squalidus argentatus*. *Science of the Total Environment*, 951: 175627.

Mommsen, T. P., Vijayan, M. M. and Moon, T. W. (1999). Cortisol in teleosts: dynamics, mechanisms of action, and metabolic regulation.

Nag, M., Kumar, S., Savaliya, B. D., Varghese, T., Paul, T., Sneha, M. *et al*. (2025). Assessment of integrated biomarker responses for alleviating triclosan toxicity in *Labeo rohita* through *Spirulina platensis* supplementary diet. *Science of the Total Environment*, 1002: 180585.

Nag, S. K., Das Sarkar, S. and Manna, S. K. (2018). Triclosan in water, sediment and fish of River Gomti, India. *International Journal of Environmental Health Research*, 28: 461–470.

Orvos, D. R., Versteeg, D. J., Inauen, J., Capdevielle, M., Rothenstein, A. and Cunningham, V. (2002). Aquatic toxicity of triclosan. *Environmental Toxicology and Chemistry*, 21: 1338–1349.

Parenti, C. C., Ghilardi, A., Della Torre, C., Mandelli, M., Magni, S., Del Giacco, L. and Binelli, A. (2019). Environmental concentrations of triclosan activate cellular defence mechanism and generate cytotoxicity on zebrafish (*Danio rerio*) embryos. *Science of the Total Environment*, 650: 1752–1758.

Paul, T., Shukla, S. P., Kumar, K., Poojary, N. and Kumar, S. (2019). Effect of temperature on triclosan toxicity in *Pangasianodon hypophthalmus*. *Science of the Total Environment*, 668: 104–114.

Prasad, G. S., Rout, S. K., Amin, A. *et al*. (2024). Impact of COVID-19 on the aquatic environment with special emphasis on Indian perspective. *Proceedings of the Indian National Science Academy*, 90: 821–839.

Ramaswamy, B. R., Shanmugam, G., Velu, G., Rengarajan, B. and Larsson, D. G. J. (2011). GC–MS analysis and ecotoxicological risk assessment of triclosan in Indian rivers. *Journal of Hazardous Materials*, 186: 1586–1593.

Seenivasan, D., Pandurengan, P., Arasan, S., Gopalrajan, S. and Paulraj, J. (2023). Short term effects of antimicrobial agent triclosan on *Oreochromis mossambicus*. *Indian Journal of Animal Research*, 57: 788–794.

Sharma, S., Dar, O. I., Singh, K., Kaur, A. and Faggio, C. (2021). Triclosan elicited biochemical and transcriptomic alterations in *Labeo rohita* larvae. *Environmental Toxicology and Pharmacology*, 88: 103748.

Sharma, S., Dar, O. I., Thakur, S., Kesavan, A. K. and Kaur, A. (2022). Environmentally relevant concentrations of triclosan cause transcriptomic and biomolecular alterations in *Labeo rohita* hatchlings. *Environmental Toxicology and Pharmacology*, 96: 104004.

Sharma, S., Iqbal Dar, O., Andotra, M., Sharma, S., Kaur, A. and Faggio, C. (2021). Environmentally relevant concentrations of triclosan induce cyto-genotoxicity and biochemical alterations in *Labeo rohita* hatchlings. *Applied Sciences*, 11: 10478.

Singh, V. and Suthar, S. (2021). Occurrence, seasonal variations and ecological risk of pharmaceuticals and personal care products in River Ganges. *Chemosphere*, 268: 129331.

Sun, L., Ling, Y., Jiang, J., Wang, D., Wang, J., Li, J. *et al*. (2020). Differential mechanisms regarding triclosan induced zebrafish lipid metabolism disorders. *Chemosphere*, 251: 126318.

Teplova, V. V., Belosludtsev, K. N. and Kruglov, A. G. (2017). Mechanism of triclosan toxicity: mitochondrial dysfunction including complex II inhibition, superoxide release and uncoupling of oxidative phosphorylation. *Toxicology Letters*, 275: 108–117.

Thilakan, A. P., Kumar, K., Shukla, S. P., Kumar, S., Saharan, N. and Karmakar, S. (2019). Occurrence of triclosan in Versova Creek of Mumbai, India and its toxicity on selected aquatic organisms. *Journal of Experimental Zoology India*, 22: 737–742.

Trivedi, A., Saxena, V., Banaee, M., Bakhasha, J., Arya, N., Yadav, K. K. *et al*. (2025). Unveiling the crosstalk between unfolded protein response and apoptosis in triclosan induced hepatotoxicity in *Labeo rohita*. *Scientific Reports*, 15: 17089.

Vijayan, M. M. *et al*. (2010). The stress response in fish: molecular and physiological aspects.

Wang, C. and Tian, Y. (2015). Reproductive endocrine-disrupting effects of triclosan: population exposure, present evidence and potential mechanisms. *Environmental Pollution*, 206: 195–201.

Wang, J., Ma, N., Mo, G., Qin, X., Zhang, J., Yao, X. *et al*. (2025). Hazards and health risks of the antibacterial agent triclosan to fish: a review. *Journal of Xenobiotics*, 15: 204.

Wang, Y., Zhao, H., Liu, X., Chen, J. and Li, Z. (2021). Biochemical responses and liver damage in fish exposed to triclosan. *Environmental Toxicology and Chemistry*, 40: 2521–2530.

Witorsch, R. J. (2014). Critical analysis of endocrine disruptive activity of triclosan and its relevance to human exposure. *Critical Reviews in Toxicology*, 44: 535–555.

Yang, F., Xu, C., Li, E., Wang, X., Zhang, Y. and Chen, L. (2023). Mitochondrial dysfunction and ROS-mediated toxicity of triclosan in fish liver. *Journal of Hazardous Materials*, 451: 131156.

Zhang, L., Niu, J. and Wang, Y. (2016). Full life-cycle toxicity assessment on triclosan using rotifer *Brachionus calyciflorus*. *Ecotoxicology and Environmental Safety*, 127: 30–35.

Received: April 02, 2026
Accepted: April 28, 2026