Int. J. Aquat. Biol. (2025) 13(3): 34-41
ISSN: 2322-5270; P-ISSN: 2383-0956
Journal homepage: www.ij-aquaticbiology.com

Original Article

Assessment of antioxidant defense mechanisms in rainbow trout (*Oncorhynchus mykiss*) following exposure to abamectin: Implications for aquatic toxicology

Ali Sabahi Tajabadi[1], Amir Sattari[*1], Amir Parviz Salati[2]

[1]Department of Food Hygiene and Public Health, Faculty of Veterinary Medicine, Shahid Bahonar University of Kerman, Kerman, Iran.
[2]Department of Fisheries, Faculty of Marine Natural Resources, Khorramshahr University of Marine Sciences and Technologies, Khorramshahr, Iran.

Abstract: The study examines the impact of the pesticide abamectin on Rainbow trout (*Oncorhynchus mykiss*), specifically its effects on antioxidant defense mechanisms. A total of 168 fish were exposed to varying concentrations of abamectin, revealing a 96-hour lethal concentration (LC_{50}) of 15.69 μg/L. Following this, 180 fish were subjected to sub-lethal concentrations (0, 4.5, 5, and 5.5 μg/L) for 14 days. The results indicated that the control group exhibited the highest liver catalase enzyme activity, while lower activity was noted in fish exposed to 5 and 5.5 μg/L of abamectin. Additionally, the activity of superoxide dismutase (SOD), glutathione levels, and total antioxidant capacity (TAC) were significantly higher in the control and 4.5 μg/L groups compared to the higher concentrations. Conversely, malondialdehyde (MDA) levels, a marker of lipid peroxidation, were elevated in fish exposed to sub-lethal concentrations of abamectin. The findings suggest that low concentrations of abamectin (4.5 μg/L) activate the fish's antioxidant defense system, enhancing the activity of antioxidant enzymes to combat free radicals. However, at higher concentrations (5 and 5.5 μg/L), there is a suppression of antioxidant enzyme activity, a reduction in glutathione and TAC levels, and an increase in oxidative stress markers. Overall, abamectin exposure leads to oxidative stress in Rainbow trout, characterized by diminished antioxidant defenses and increased lipid peroxidation.

Article history:
Received 19 March 2025
Accepted 8 June 2025
Available online 25 June 2025

Keywords:
Toxicity
Abamectin
Antioxidant capacity
Oxidative stress

Introduction

The widespread and unregulated application of agricultural chemicals and pesticides for pest control and crop yield enhancement worldwide poses significant risks to human health, non-target organisms, and the environment (Kadiru et al., 2022). Avermectin or abamectin, a widely used acaricide, insecticide, and nematicide, is particularly prevalent in the agricultural practices of Kerman Province, Iran, especially within pistachio orchards, where it is employed to manage the pistachio psyllid (*Agonoscena pistaciae*). This insect, native to Iran, is currently recognized as the most significant pest affecting pistachio cultivation in the region. Avermectin inhibits gamma-aminobutyric acid (GABA) receptors in both invertebrates and vertebrates (Novelli et al., 2016). Upon entering an organism, abamectin disrupts the transmission of the GABA neurotransmitter.

The ongoing application of abamectin, along with its environmental persistence, raises potential ecological concerns across various ecosystems, particularly in aquatic environments. Furthermore, abamectin has been approved for therapeutic use in aquaculture to combat aquatic parasites (Hong et al., 2020b). In addition to its direct application in aquaculture, residues from agricultural use can infiltrate aquatic ecosystems, leading to toxic effects on aquatic life. Although abamectin degrades quickly in water through photolysis, its widespread use contributes to environmental accumulation (Halley et al., 1993; Tišler and Kožuh Eržen, 2006). It is identified as highly toxic to particular aquatic species, with long-term implications, as it can penetrate the blood-brain barrier in specific aquatic organisms and accumulate in fish (Wang et al., 2011; Novelli et al.,

[*]Correspondence: Amir Sattari
E-mail: amirsatari@uk.ac.ir
DOI: https://doi.org/10.22034/ijab.v13i3.2497

2012, 2016). Hence, laboratory toxicity studies are crucial for predicting the effects of such chemicals on aquatic ecosystems (Novelli et al., 2012). Investigating the ecotoxicological effects of abamectin on aquatic environments is crucial during bioconversion; an increase in aerobic metabolism results in the production of reactive oxygen species (ROS), including superoxide radicals (O^{2-}) and hydroxyl radicals (OH), along with other pro-oxidant compounds such as hydrogen peroxide (Sanches et al., 2024).

Pesticides, particularly abamectin, contribute to oxidative stress in aquatic organisms by generating excess ROS (Hong et al., 2020b; Huang et al., 2020). This oxidative stress arises from a disruption in the balance between oxidants and antioxidants at the cellular level (Hong et al., 2020a). The overproduction of ROS during this process can damage essential biological macromolecules, including DNA (Lushchak, 2011). The liver is vulnerable to pesticide toxicity due to its role in major metabolic pathways and the accumulation of metabolites; exposure to insecticides can result in liver damage mediated by ROS (El-Shenawy, 2010). Given the insufficient assessment of antioxidant defenses in rainbow trout (*Oncorhynchus mykiss*) in the region when exposed to the abamectin toxin commonly used in gardens and agriculture, this work aimed to evaluate the toxicity levels and the resulting physiological impacts on rainbow trout.

Materials and Methods

In this study, juvenile rainbow trout, weighing approximately 7 grams, were obtained from the Sirch fish farm in Kerman, Iran. They were acclimatized to laboratory conditions for 10 days in 100-liter plastic tanks at the Aquatic Laboratory of the Faculty of Veterinary Medicine at Shahid Bahonar University of Kerman. The fish were fed commercial feed (Biomar, France) at a feeding rate of 3% of their body weight, administered in three meals daily. Furthermore, 20% of the tank water was replaced daily with well water, and aeration was maintained using a central air pump and air stones.

The commercial abamectin, with a purity of 1.8% (equivalent to 18 grams of active ingredient per liter in an emulsifiable form), was procured from Gol Sam Company in Gorgan, Iran. To assess the acute toxicity of abamectin using a static method, an appropriate concentration range for the toxin was established based on prior research on fish (Tišler and Kožuh Eržen, 2006; Hong et al., 2020a) and preliminary tests. A stock solution of abamectin was prepared by dissolving it in acetone at a concentration of 0.5 mg/L (Hong et al., 2020a). A total of 168 juvenile rainbow trout were distributed across eight tanks, each containing 100 liters of water, with a working volume of 50 liters. The fish were exposed to a range of abamectin concentrations, specifically 0, 40, 50, 60, 70, 80, 90, and 100 μg/L, with the control group receiving no toxins. Each tank housed 21 juvenile fish. The acetone concentration in the control group was equivalent to that of the highest abamectin concentration tested (Hong et al., 2020a).

In the second stage, to examine the impact of the abamectin on the antioxidant defense mechanisms of rainbow trout, feeding was halted 24 hours prior to the experiment and throughout the exposure period. To prevent the fish from leaping out of the tanks, mesh panels covered them. A total of 180 rainbow trout were randomly distributed into 12 tanks, each with a capacity of 100 liters (with a working volume of 50 liters, and 15 fish per tank), and were subjected to sub-lethal concentrations of abamectin for 14 days. The concentrations tested included 0 (control), 4.5, 5, and 5.5 micrograms per liter. The control group was maintained in water free of the toxin but contained acetone at the same concentration as that used in the highest treatment group (5.5 μg/L). Consequently, the experimental design consisted of four treatment groups, each with three replicates. The fish were provided with commercial feed (Biomar, France) at a feeding rate of 3% of their body weight, administered in three meals daily. Water was changed every two days, replacing 40% of the tank water with fresh water containing the specified concentrations of abamectin (Hong et al., 2020a), and the tanks were continuously aerated. Throughout the experiment, the

Table 1. Mortality rate of rainbow trout in acute avermectin toxicity test (n = 21).

Concentration (µg/L)	24h	48h	72h	96h
Control (0)	0	0	0	0
40	0	0	0	0
50	2	9	17	18
60	7	11	17	20
70	9	15	19	21
80	15	17	21	0
90	17	21	0	0
100	21	0	0	0

Table 2. Lethal concentration (LC) of abamectin in rainbow trout during 96 hours of exposure.

Mortality Level	Mortality concentration (µg/L)	Confidence interval
LC1	38.26	29.90-44.20
LC5	45.51	37.86-50.83
LC10	49.91	42.88-54.85
LC20	55.82	49.72-60.29
LC30	60.51	55.13-64.77
LC50	69.15	64.60-73.81
LC70	79.02	74.02-86.02
LC80	85.66	79.66-95.21
LC90	95.80	87.69-110.24
LC95	105.08	94.66-124.76
LC99	124.97	108.93-157.89

environmental and water quality parameters across all tanks remained consistent, with a water temperature of 1.1±13 degrees Celsius, pH levels ranging from 4.0 to 7.3, dissolved oxygen levels between 7-8 mg/L, total ammonia below 0.1 mg/L, and a light cycle of 13 hours of light followed by 11 hours of darkness.

After 14 days of experiments, the fish were dissected, and their livers were removed. Liver samples were homogenized in a buffer composed of 100 millimolar potassium phosphate, 100 millimolar potassium chloride, and 1 millimolar EDTA at a pH of 7.4, using a volume-to-weight ratio of 1:10, while kept on ice. Then, the samples were centrifuged at 20,000 g for 25 minutes at a temperature of 4°C, and the resulting supernatant was utilized to assess antioxidant indices (Atli and Canli, 2010). The concentration of soluble protein in the liver extracts was determined via the Bradford method (Bradford, 1976). The activities of catalase and superoxide dismutase (SOD) enzymes in the fish liver were evaluated using commercial kits from Randox (UK). The liver's glutathione (GSH) levels were quantified through a reaction with DTNB, measuring absorbance at 412 nanometers (Liu et al., 2018). Total antioxidant capacity (TAC) was assessed using a commercial kit from ZellBio (Veltlinerweg, Germany), following the manufacturer's instructions. Additionally, the concentration of malondialdehyde (MDA) in the liver was determined through spectrophotometric analysis (Buege and Aust, 1978).

To determine the 50% lethal concentration (LC_{50}) of abamectin, a probit analysis was conducted with a 95% confidence interval. The normality of the data was verified using the Kolmogorov-Smirnov test, and one-way ANOVA was employed to analyze the antioxidant response data. Duncan's post hoc test was applied for mean comparisons across treatments. Results were presented as mean ± standard deviation, with significance evaluated at the 5% level. Statistical analyses were performed using SPSS version 23, and graphical representations were created in Microsoft Excel.

Results

Table 1 details the fish mortality rates observed in the acute toxicity assessment of abamectin, while Table 2 illustrates the lethal concentration of abamectin after 96 hours of exposure. The 96-hour lethal concentration that resulted in 50 percent mortality (LC_{50}) for rainbow trout was 15.69 µg/L.

The impact of different sublethal concentrations of

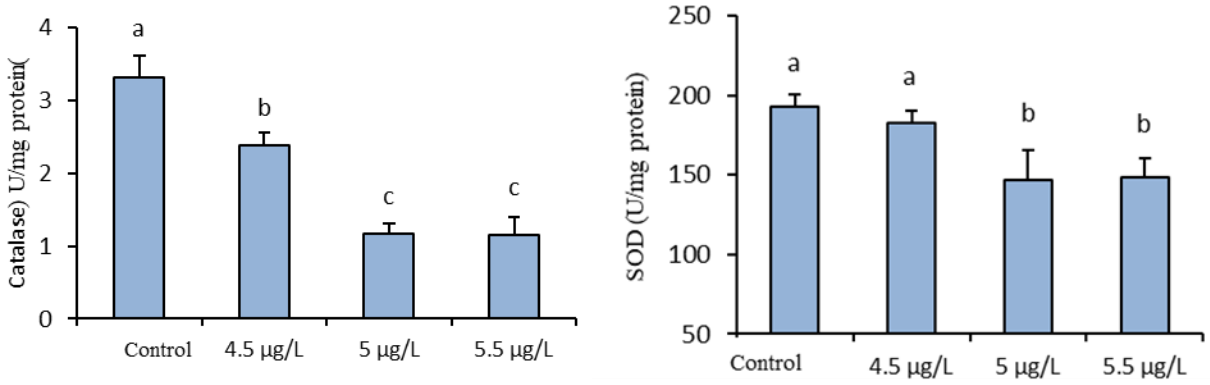

Figure 1. Effects of different concentrations of Abamectin on the activity of Catalase (CAT) and Superoxide Dismutase (SOD) enzymes in the liver of rainbow trout after 14 days of exposure. Different lowercase letters above the columns indicate significant differences among the treatments (P<0.05).

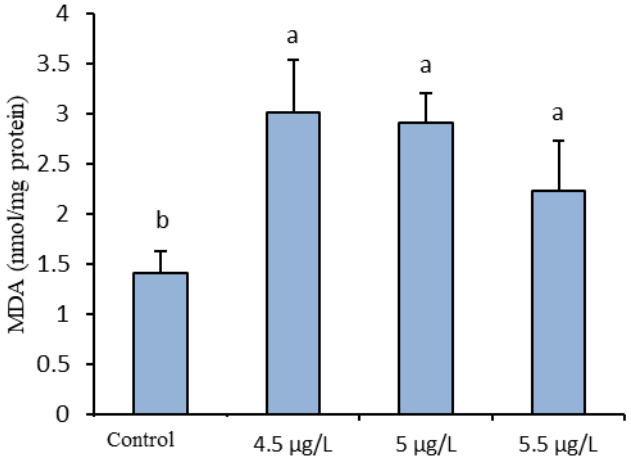

Figure 2. Effects of different concentrations of abamectin on the level of malondialdehyde (MDA) in the liver of rainbow trout after 14 days of exposure. Different lowercase letters above the columns indicate significant differences among treatments (P<0.05).

abamectin (4.5, 5, and 5.5 µg/L) on the activity of the antioxidant enzymes catalase and superoxide dismutase (SOD) in the liver of rainbow trout after a 14-day exposure period is shown in Figure 1. The control group exhibited the highest catalase activity, followed by the group exposed to 4.5 µg/L of abamectin. In contrast, the lowest enzyme activity was recorded in the groups treated with 5 and 5.5 µg/L of abamectin (P<0.05). Similarly, after 14 days, SOD activity in the liver was significantly greater in both the control and 4.5 µg/L treatments compared to those at 5 and 5.5 µg/L (P<0.05).

The impact of different sublethal concentrations of abamectin on lipid peroxidation levels, specifically malondialdehyde (MDA), in the liver of rainbow trout following a 14-day exposure to this toxin is shown in Figure 2. The MDA levels in fish subjected to sublethal doses of abamectin (4.5, 5, and 5.5 µg/L) were significantly elevated compared to the control (P<0.05).

Discussions

The widespread use of abamectin has led to the contamination of many natural water bodies, resulting in documented negative effects on the growth and development of non-target species (Wang et al., 2025). This chemical is a significant environmental concern due to its persistence and high toxicity to aquatic life, particularly fish (Wu et al., 2023). While it is not very toxic to mammals, it poses a serious threat to small crustaceans and fish (Tišler and Kožuh Eržen, 2006; Campbell, 2012; Novelli et al., 2012). Even in small amounts, abamectin can be highly harmful to daphnia and fish (Tišler and Kožuh Eržen, 2006).

In the current study, the 50% lethal concentration (LC_{50}) of abamectin for a 96-hour exposure in rainbow trout was determined to be 15.69 µg/L. The investigation by Jenčič et al. (2006) demonstrated that acute toxicity in rainbow trout occurred after a 58-hour exposure to abamectin concentrations ranging from 0.6 to 4.5 micrograms per liter, resulting in an LD75 of 4 µg/L. Observations of degenerative organ changes confirmed the direct toxic effects of abamectin on rainbow trout (Jenčič et al., 2006). For zebrafish (*Danio rerio*), the reported 96-hour LC_{50} for abamectin toxicity was 1.55 µg/L (Tišler and Kožuh Eržen, 2006). Additionally, another study found that

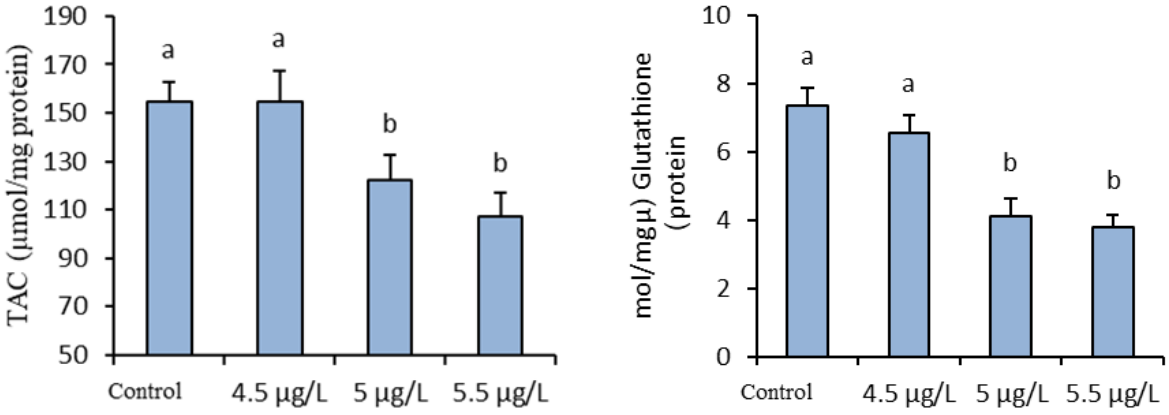

Figure 3. Effects of different concentrations of abamectin on the amount of glutathione and total antioxidant capacity (TAC) in the liver of rainbow trout after 14 days of exposure. Different lowercase letters above the columns indicate a significant difference among the treatments (P<0.05).

the LC_{50} values for zebrafish larvae at 24, 48, 72, and 96 hours were 58.29, 53.34, 48.31, and 47.50 µg/L, respectively (Wang et al., 2025). In addition, fish mortality rates tend to increase with longer exposure times and higher concentrations of abamectin (Wang et al., 2025). A study by Novelli et al. (2012) showed that abamectin is highly toxic to freshwater species, with a 48-hour LC50 value of 33 µg/L for juvenile zebrafish. For the freshwater fish *Schizothorax prenanti*, the lethal concentration values after 48 and 96 hours of exposure to abamectin were found to be 33.32 and 15.98 µg/L, respectively (Hong et al., 2020a). The 96-hour LC50 for abamectin, when formulated as an emulsifiable concentrate, in Nile tilapia was reported to be 10 µg/L (Ahmed and Reda, 2021). In common carp that had been on a multi-enzyme Natosim diet for eight weeks, the lethal concentration of abamectin was recorded at 0.305 mg/L after 96 hours (Froohar Vajargh et al., 2016). The differences in lethal concentration values of abamectin across various studies may be influenced by factors such as fish species, age and developmental stage, sex, body size, environmental conditions, exposure duration, and dietary composition.

The current study found that higher concentrations of abamectin (5 and 5.5 µg/L) resulted in a decrease in the activity of antioxidant enzymes, such as catalase and superoxide dismutase (SOD). There was also a decrease in glutathione levels and overall antioxidant capacity. As a stimulant for ROS, abamectin can potentially reduce the activity of antioxidant enzymes in fish (Wu et al., 2023). Supporting our findings, Wu et al. (2023) reported that carp exhibited a decline in antioxidant enzyme activity after exposure to abamectin. In addition, Feng et al. (2023) highlighted that abamectin negatively impacts the gill structure of common carp, causing oxidative stress by reducing antioxidant enzyme activity and increasing malondialdehyde (MDA) levels. Furthermore, Guan et al. (2024b) investigated the toxicity of abamectin in the red swamp crab P. clarkii, administering doses of 0, 3, and 6 µg/L over a 28-day period. Their findings indicated that extended exposure to abamectin, especially at higher concentrations, resulted in lower expression levels of important genes and reduced activity of enzymes involved in digestion, antioxidant responses, and immune functions (Guan et al., 2024). In the study of Hong et al. (2020a), *S. prenanti* were subjected to sub-lethal levels of abamectin (0.5, 2, or 8 µg/L) for 8 days, resulting in liver cell death at all tested concentrations. This effect was linked to increased ROS production and caspase activity, which depended on both the dose and the duration of exposure. Additionally, there were significant decreases in the activities of GPx, SOD, and catalase, accompanied by higher levels of malondialdehyde (Hong et al., 2020a).

In a different study, Nile tilapia exposed to just one-tenth of the 96-hour lethal concentration for half of the population (LC_{50}) of abamectin (20.73 µg/L) showed a decrease in plasma glutathione levels and overall antioxidant capacity (Mahmoud et al., 2021).

Similarly, zebrafish exposed to polyethylene terephthalate microplastics along with the pesticide abamectin also experienced a reduction in glutathione levels (Hanachi et al., 2021). Additionally, Nile tilapia that received sub-lethal doses of abamectin had significantly lower glutathione concentrations in their liver and brain tissues, along with reduced GPx activity in the brain (Reda et al., 2023). Glutathione is essential for detoxifying hydrogen peroxide (H_2O_2). Therefore, the decrease in glutathione levels leads to an increase in H_2O_2 production, which subsequently heightens lipid peroxidation levels (Mahmoud et al., 2021).

The results of the present study showed that the activities of CAT and SOD, along with glutathione levels and the overall antioxidant capacity in the livers of rainbow trout exposed to 4.5 µg/L of abamectin for 14 days, were significantly higher than those in groups that were exposed to 5 and 5.5 µg/L of abamectin. This suggests that a concentration of 4.5 µg/L of abamectin can effectively enhance the antioxidant system in rainbow trout, unlike higher concentrations. The increased activity of antioxidant enzymes suggests that the antioxidant defense mechanism is activated to combat free radicals (Karimzadeh et al., 2017). Similarly, low doses and short exposure to abamectin triggered the defensive system in the red swamp crayfish *P. clarkii* (Guan et al., 2024). However, as oxidative damage increased, the antioxidant and immune defense mechanisms started to falter (Guan et al., 2024). An imbalance between the production of ROS and the effectiveness of antioxidant defenses can lead to oxidative stress, as indicated by higher levels of lipid hydroperoxides and malondialdehyde (MDA) (Sanches et al., 2024).

In the current study, rainbow trout exposed to sub-lethal concentrations of abamectin (4.5, 5, and 5.5 µg/L) for 14 days showed significantly elevated MDA levels. This suggests that sub-lethal doses of abamectin significantly increase lipid peroxidation in the exposed fish, leading to oxidative stress. Additionally, Sanches et al. (2024) found a significant rise in MDA levels in the gills of zebrafish exposed to the commercial formulation of abamectin for 48 hours. Furthermore, abamectin has been shown to cause oxidative stress in common carp, as indicated by a notable increase in MDA levels in fish exposed to 12.5% of the LC_{50} compared to unexposed groups (Rohmah et al., 2022). The accumulation of ROS in fish subjected to abamectin can disrupt the balance of the oxidative system, leading to oxidative stress (Wu et al., 2023).

Our findings reveal that rainbow trout exposed to 4.5 µg/L of abamectin boost their antioxidant defense systems. As the levels of abamectin rise to 5 and 5.5 µg/L, however, a decrease in the activity of antioxidant enzymes, such as catalase and superoxide dismutase (SOD), occurs. Additionally, there is a drop in glutathione and total antioxidant capacity (TAC) levels. In conclusion, abamectin induces oxidative stress in rainbow trout by inhibiting the activity of antioxidant enzymes, decreasing glutathione and TAC levels, and increasing MDA concentrations.

References

Ahmed F.A.G., Reda R.M. (2021). Comparative Acute exposure study of abamectin different formulations inducing physiological and oxidative stress biomarkers in nile tilapia, (*Oreochromis niloticus*). Egyptian Academic Journal of Biological Sciences, B. Zoology, 13: 223-238.

Asifa K., Vidya P., Chitra K. (2016). Assessment of median lethal concentration (LC50-96h) and behavioural modification of nonylphenol in the cichlid fish, *Etroplus maculatus* (Bloch, 1795). International Journal of Advanced Life Sciences, 9: 10-15.

Atli G., Canli M. (2010). Response of antioxidant system of freshwater fish *Oreochromis niloticus* to acute and chronic metal (Cd, Cu, Cr, Zn, Fe) exposures. Ecotoxicology and Environmental Safety, 73: 1884-1889.

Bradford M.M. (1976). A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. Analytical Biochemistry, 72: 248-254.

Buege J.A., Aust S.D. (1978). Microsomal lipid peroxidation, Methods in Enzymology, 302-310.

Campbell W.C. (2012). Ivermectin and abamectin. Springer Science and Business Media. 363 p.

El-Shenawy N.S. (2010). Effects of insecticides

fenitrothion, endosulfan and abamectin on antioxidant parameters of isolated rat hepatocytes. Toxicology in Vitro, 24: 1148-1157.

Feng H., Zhou P., Liu F., Zhang W., Yang H., Li X., Dong J. (2023). Abamectin causes toxicity to the carp respiratory system by triggering oxidative stress, inflammation, and apoptosis and inhibiting autophagy. Environmental Science and Pollution Research, 30: 55200-55213.

Guan T., Wang L., Hu M., Zhu Q., Cai L., Wang Y., Xie P., Feng J., Wang H., Li J. (2024). Effects of chronic abamectin stress on growth performance, digestive capacity, and defense systems in red swamp crayfish (*Procambarus clarkii*). Aquatic Toxicology 268, 106861.

Halley B.A., VandenHeuvel W.J., Wislocki P.G. (1993). Environmental effects of the usage of avermectins in livestock. Veterinary Parasitology, 48: 109-125.

Hanachi P., Kazemi S., Zivary S., Karbalaei S., Abolghasem Ghadami S. (2021). The effect of polyethylene terephthalate and abamectin on oxidative damages and expression of vtg and cyp1a genes in juvenile zebrafish. Environmental Nanotechnology, Monitoring and Management, 16: 100565.

Hong Y., Huang Y., Yang X., Zhang J., Li L., Huang Q., Huang Z. (2020a). Abamectin at environmentally-realistic concentrations cause oxidative stress and genotoxic damage in juvenile fish (*Schizothorax prenanti*). Aquatic Toxicology, 225: 105528.

Hong Y., Yin H., Huang Y., Huang Q., Yang X. (2020b). Immune response to abamectin-induced oxidative stress in Chinese mitten crab, *Eriocheir sinensis*. Ecotoxicology and Environmental Safety, 188: 109889.

Huang Y., Hong Y., Huang Z., He H. (2020). Cytotoxicity induced by abamectin exposure in haemocytes of Chinese mitten crab, *Eriocheir sinensis*. Environmental Toxicology and Pharmacology, 77: 103384.

Jenčič V., Černe M., Eržen N.K., Kobal S., Cerkvenik-Flajs V. (2006). Abamectin effects on rainbow trout (*Oncorhynchus mykiss*). Ecotoxicology, 15: 249-257.

Kadiru S., Patil S., D'Souza R. (2022). Effect of pesticide toxicity in aquatic environments: A recent review. International Journal of Fisheries and Aquatic Studies, 10: 113-118.

Liu G., Ye Z., Liu D., Zhao J., Sivaramasamy E., Deng Y., Zhu S. (2018). Influence of stocking density on growth, digestive enzyme activities, immune responses, antioxidant of *Oreochromis niloticus* fingerlings in biofloc systems. Fish and Shellfish Immunology 81, 416-422.

Lushchak V.I. (2011). Environmentally induced oxidative stress in aquatic animals. Aquatic Toxicology, 101: 13-30.

Mahmoud H.K., Reda F.M., Alagawany M., Farag M.R. (2021). The stress of abamectin toxicity reduced water quality, growth performance, immunity and antioxidant capacity of Oreochromis niloticus fish: Modulatory role of *Simmondsia chinensis* extract as a dietary supplement. Aquaculture, 534: 736247.

Novelli A., Vieira B.H., Braun A.S., Mendes L.B., Daam M.A., Espíndola E.L.G. (2016). Impact of runoff water from an experimental agricultural field applied with Vertimec® 18EC (abamectin) on the survival, growth and gill morphology of zebrafish juveniles. Chemosphere, 144: 1408-1414.

Novelli A., Vieira B.H., Cordeiro D., Cappelini L.T.D., Vieira E.M., Espíndola E.L.G. (2012). Lethal effects of abamectin on the aquatic organisms *Daphnia similis*, *Chironomus xanthus* and *Danio rerio*. Chemosphere, 86: 36-40.

Reda R.M., Helmy R.M.A., Osman A., Ahmed F.A.G., Kotb G.A.M., El-Fattah A.H.A. (2023). The potential effect of Moringa oleifera ethanolic leaf extract against oxidative stress, immune response disruption induced by abamectin exposure in Oreochromis niloticus. Environmental Science and Pollution Research, 30: 58569-58587.

Rohmah M.K., Salahdin O.D., Gupta R., Muzammil K., Qasim M.T., Al-qaim Z.H., Abbas N.F., Jawad M.A., Yasin G., Mustafa Y.F., Heidary A., Abarghouei S. (2022). Modulatory role of dietary curcumin and resveratrol on growth performance, serum immunity responses, mucus enzymes activity, antioxidant capacity and serum and mucus biochemicals in the common carp, Cyprinus carpio exposed to abamectin. Fish and Shellfish Immunology, 129: 221-230.

Sanches A.L.M., da Silva Pinto T.J., Daam M.A., Teresa F.B., Vieira B.H., Reghini M.V., de Almeida E.A., Espíndola E.L.G. (2024). Isolated and mixed effects of pure and formulated abamectin and difenoconazole on biochemical biomarkers of the gills of *Danio rerio*. Aquatic Toxicology, 273: 106978.

Tišler T., Kožuh Eržen N. (2006). Abamectin in the aquatic environment. Ecotoxicology, 15: 495-502.

Wang F., Chen J., Cheng H., Tang Z., Zhang G., Niu Z., Pang S., Wang X., Lee F.S.-C. (2011). Multi-residue

method for the confirmation of four avermectin residues in food products of animal origin by ultra-performance liquid chromatography–tandem mass spectrometry. Food Additives and Contaminants: Part A, 28: 627-639.

Wang Y., He J., Li M., Xu J., Yang H., Zhang Y. (2025). Abamectin at environmentally relevant concentrations impairs bone development in zebrafish larvae. Comparative Biochemistry and Physiology Part C: Toxicology and Pharmacology, 287: 110039.

Wu X., Ma Y., Li X., He N., Zhang T., Liu F., Feng H., Dong J. (2023). Molecular mechanism of kidney damage caused by abamectin in carp: oxidative stress, inflammation, mitochondrial damage, and apoptosis. Toxicology, 494: 153599.