Impact of Temperature on Contaminants Toxicity in Fish Fauna: A Review

Kamal Mehta*
Department of Zoology, J.C.D.A.V College, Dasuya – 144205, Punjab, India; kamal.davzoo@yahoo.in

Abstract

Objective: Toxicity of contaminants in the aquatic ecosystems is found to be affected by rise in temperature due to global warming. The main objective of the present study was to investigate the toxicity effects of some specific organic and inorganic contaminants like iron, copper, endosulfan, chlorpyrifos, phenol, chlorine, cyanides, nitrites and ammonia at different temperatures to fish fauna. Methodology: secondary data concerning with the impact of temperature fluctuations on toxicology of contaminants in aquatic ecosystems especially with reference to fish fauna was collected to review and predict their correlation. Findings: This review study indicated that toxicity of contaminants is greatly affected by climate change and it may have adverse effects on biology of fish fauna. The concentration of toxic contaminants which is not active at low and moderate temperature proved lethal with increase in water temperature. Application: This review study may sensitize the ecologists to draw correlation between temperature and toxicity of contaminants for effective management of aquatic ecosystems and fish fauna. This will also essentially helpful in understanding of basic mechanisms that allow the aquatic fauna to survive in the contaminated waters. Conclusion: The study of interaction mechanisms of aquatic toxicants with environmental factors and their management in aquatic ecosystems, hence, is the need of hour otherwise; they can lead to loss of aquatic biodiversity and can disturb ecological balance.

Keywords: Aquatic Fauna, Contaminants Toxicity, Heavy Metals, Pesticides, Temperature

1. Introduction

Aquatic ecosystems are home to a vast diversity of flora and fauna. They act as sinks for a great variety of anthropogenic contaminants, many of which are toxic. These toxic contaminants are broadly classified into three main types-heavy metals, metalloids and pesticides. A few of heavy metals like Zn, Cu, Co, Mn, Fe and Ni are needed for regulation of biological metabolism in trace amounts and hence, biologically important. They are found to be toxic in their higher doses. Heavy metals like Pb, Hg, Cd and As are potentially toxic at any concentration and hence, are not biologically useful. The fourth IPCC report published in 2007 revealed that anthropogenic activities are largely responsible for the observed and projected changes in the environment that may adversely affect life and these changes may get more intensified in the near future. There are several environmental variables like temperature and pH that have marked effect on the toxicity of contaminants. Temperature is a key factor that can have serious physiological effects on living systems and directly influences the tolerance limits of toxic substances in the aquatic biota. It has long been known to cause significant alterations in the chemistry of a number of chemical pollutants that effects aquatic fauna. Rise in temperature is found to increase the rate of uptake of pollutants with increase of metabolic rate and decrease in oxygen solubility. It has been reported that the upper temperature tolerance limits are decreased in the presence of certain organic chemicals for a variety of freshwater fish species. At optimal temperature range, growth and reproduction rate is high and abundance of the species will be more. Outside this range, there are zones of physiological stress that may affect distribution as well as activities of organisms by producing stress and discomfort. Temperature tolerance ability of fish may be affected by presence of contaminants in the water bodies, their concentration and time of exposure. It has been

* Author for correspondence
reported that high temperature tolerance ability of fish is directly reduced by chronic exposure to contaminants. High temperature may further augment the toxicity of contaminants in aquatic ecosystems. It may directly affect the growth and development of fish. Developmental abnormalities especially during osteogenesis may be induced by such thermal changes. Many substances like cyanides, phenol, zinc, ammonia, nitrites exhibit increased toxicity at elevated temperatures. The purpose of this review paper is to survey the present knowledge of contaminant toxicity to fish with relation to temperature parameter and is an indicator of the fact that climate changes may have ill effects on fish biology.

2. Impact of Water Temperature Fluctuations on Fish Biology

Fish are cold-blooded animals; hence their body temperature vary with the surrounding water temperature. They are abundant in those water bodies whose temperature show minor fluctuations with respect to environmental temperatures. The fish body temperature is almost isothermal with the surrounding water temperature with minor fluctuations of ± 0.5 to 1°C. Water temperature directly influences the fish metabolism. Higher the water temperature greater is the fish body metabolism. This is applicable particularly to fish residing in the warm water. Certain fish like salmonids and whitefish residing in the cold water show normal metabolic activity at comparatively low temperatures, in contrast to high water temperatures. When the water temperature is above 20°C, they become passive and even take the little food. Elevations in water temperature effects aquatic life by decreasing the solubility of dissolved oxygen, thereby, increasing the oxygen demand of fish and metabolism. Rise in temperature increases the solubility of many toxic contaminants in the water bodies. It also increases the permeability of tissue towards toxic contaminants, with increase in their basal metabolic rate, water and oxygen consumption. Immune power of majority of fish is also affected by water temperatures showing optimal activity at 15°C. Minimal and maximal temperature tolerance range in the majority of fish is 0°C and 20–30°C respectively. Abrupt seasonal changes in temperature and shifting of fish to new water body whose temperature is high or low than the native aquatic ecosystem may lead to temperature shock in fish. It may lead to paralysis of respiratory and heart muscles followed by death of the fish. Hence, a concentration of a toxicant which is not active at low and moderate temperature proved lethal with an increase in temperature in a water body. There are a number of factors on which temperature tolerance limits of an adult fish depends like fish species, prior acclimatization, oxygen concentration and the synergistic effects of other contaminants. Researches carried on fish toxicology indicate increased toxicity of contaminants with rise in temperature and revealed that rate of absorption of inorganic contaminants such as lead, mercury and zinc and their biomagnifications in the tissue will increase with temperature and may prove lethal at their excessive concentrations. The rate of accumulation for mercury and DDT has been shown to increase with temperature. In contrast, studies on other organic contaminants have shown toxicity to decrease with increasing temperatures.

2.1 Effect of Temperature on Pesticide Toxicity

Toxicity of contaminants in the water bodies is found to be augmented by global warming and it initiated the complex interactions between contaminants and aquatic creatures. A study was conducted in order to evaluate the effects of temperature on acute toxicity of pesticides like endosulfan; chlorpyrifos and phenol. The three warm water species of fish namely silver perch, rainbow fish, and western carp gudgeon and one cold water species of rainbow trout were taken for this study. It was found that an elevation in temperature significantly affects the pesticide toxicity. It is evident by endosulfan toxicity impact values at low and high temperature to silver perch and rainbow trout. Its toxicity is reported to increase at 30°C and 35°C than at low temperature range from 15°C to 25°C during short exposures of 24 hours, but, temperature had no effect on toxicity at 96 hours. Chlorpyrifos show more toxicity to all species with increasing temperature. A decrease and increase in toxicity of phenol is observed in all fish species at low to intermediate temperatures and toward the upper thermal limit respectively. On entering the aquatic environment, the original active ingredient of a pesticide (like parathion) may undergoes biochemical degradation and changes into secondary pollutants (paraoxon) which may prove more fatal to fish than the original active primary pollutant. Trichlorphon based organo-phosphorus chemical insecticides are generally used to kill predatory cyclopids in natural water bodies.
and fish cultivation sites before stocking the pond with juvenile stages of fish. These may further decompose into highly toxic secondary stage pollutant namely dichlorvos. High water temperature increases the permeability of fish tissue towards pesticide and hence, facilitates the transport of pesticides in the fish tissue. Majority of organic Pesticides like DDT, carbamates, pyrethroids and diazines are found to show biological magnification in fish fauna by concentrating in their tissues, thereby, reducing and altering the tissue functionality. The condition may prove lethal at higher concentrations of pesticide in water bodies as well as with increase in water temperature.

2.2 Ammonia Toxicity in Relation to Water Temperature to Fish Fauna

Aquatic ecosystems are generally contaminated with ammonia which exists in water in molecular and/or ionic form (\(\text{NH}_3/\text{NH}_4^+\)). The concentration of molecular and ionic forms of ammonia depends on the pH and temperature of the water bodies. Out of these two chemical forms, ammonia can readily pass through the biomembranes of fish and is, therefore, potentially toxic to fish. It has been found that acidity in the fish tissue and high temperature of surrounding water promotes the transport of ammonia from outside water to fish tissue interior. Hence, ammonia transport in the tissue is pH and temperature dependent. If acid-base balance is altered at water and tissue interface, additional molecular ammonia will be attracted on the side that is towards the low pH water. Furthermore, low oxygen concentration in water also facilitates ammonia toxicity. Ammonia poisoning is found to cause toxic necrosis of the gills. It greatly affects brain; hence, an ammonia poisoned fish shows nervous symptoms indicated by restlessness, rapid movements, loss of balance, leaping out of water, increased rate of respiration as reported in carp and cyprinids. It is followed by spasmodic opening of the mouth and gill opercula. Apparent recovery phase is reported thereafter, as indicated by normal swimming. But soon after apparent recovery, the body surface turns pale and the fish die. Post death signs of ammonia poisoned fish include color change, increased production of mucus over the skin, visceral dysgenesis, hemorrhage of pectoral fin gills and ocular cavity. Fish epithelium showed more water permeability under conditions of ammonia poisoning. It is evidenced from increase in the urine output. Disturbances in water balance may induce oedema. Acute toxicity tests yield LC\(_{50}\) values for ammonia that vary from species to species. A study report submitted by centre for aquatic ecology, Illinois Natural History Survey indicated the species specific effects of cold temperature (3-5°C) on ammonia toxicity. The study was conducted on three fish species and highlighted the fact that sensitivity of fish fauna towards ammonia is different.

2.3 Nitrite Toxicity in Relation to Water Temperature to Fish Fauna

Nitrogen fertilizers and sewage effluents are the main sources of nitrite pollutant in the water bodies. It is produced as an intermediate during the oxidation of ammonia to nitrates. Since they are quite unstable, hence, found in lower concentration in surface water. Its toxicity to fish varies considerably and determined by a number of physiochemical factors prevailing in the aquatic media like pH, temperature, ion and oxygen concentration along with duration of exposure, fish species, fish size, age and individual fish susceptibility. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport. Nitrite poisoning produces haematological effects besides acting as a disrupter of multiple physiological functions including ion regulatory, respiratory, cardiovascular, endocrine and excretory processes. It resulted into oxidation of haemoglobin to methaemoglobin, which is indicated by browning of blood and gills. This affects oxygen transport. Nitrite poisoning proved lethal when the amount of methaemoglobin in the blood exceeds 50%. Before death, fish become torpid, lose their orientation and fail to give response to stimuli. Lethality can be reversed if the affected fish is transferred to the nitrite free water due to reductase enzyme activity which can convert methaemoglobin back to haemoglobin and gills. This affects oxygen transport.
2.4 Chlorine Toxicity Relations with Temperature

Industrial effluents discharged into water bodies act as sources of chlorine contamination. In addition to this, Chlorine containing salts are also used as disinfectants in fish culture bodies especially for the fish stock affected from gill disease. Concentration factor for chlorine in this regard is of vital concern as excess of chlorine is deleterious to fish fauna. Chlorine toxicity is directly influenced by the water temperature. Temperature effects on chlorine toxicity has been evaluated on carp and results clearly revealed that if water temperature ranges 3-7°C containing chlorine concentration of 3.5 mg per litre, it proved sub lethal to carp and an increase of temperature upto 15-20°C proved lethal to the carp at same concentration of chlorine. Furthermore, it also depends upon the time period for which the fish is exposed to the chlorinated water. Chronic exposure may prove lethal even at low concentrations of chlorine. Chlorine poisoning may show localized or systemic effects in fish fauna. Localized effects involve skin and gills of the fish and resulted in pale color of skin, hypersecretion of mucus, congestion of gills followed by haemorrhage of gill. The systemic effects are executed through brain tissue deterioration that causes restlessness, unilateral leaping movements, sustained contractions of the muscles and spasmodic contractions of the mouth, fins and tail. The sudden involuntary muscular contractions of the mouth region hinder respiration by producing suffocation and ultimately death in fish.

2.5 Cyanides Toxicity Relations with Temperature

Cyanide is added to aquatic ecosystems due to discharge of effluents mainly from metal plating industrial plants. It is found in both free and combined states in water in the form of cyanide ion and hydrogen cyanide respectively. It also occurs in the form of co-ordination complexes with iron, cobalt, nickel and other metals. It is found to be more toxic in the ionic state. Cyanides are extremely toxic and lethal to the fish species even at the low concentrations of 0.03 to 0.5 mg per litre. Its toxicity is dependent upon water quality parameters like pH, water temperature and oxygen concentration of the water. An increase in water temperature and a decrease in its pH or concentration of dissolved oxygen increase the chances of cyanide toxicity. An increase in water temperature causes rapid dissociation of cyanide compounds and hence, increases the incidence of its toxicity to aquatic ecosystems. Cyanide is recognized as a potent respiratory enzyme inhibitor and hence executes its toxicity by inhibition of respiratory enzyme specifically cytochrome oxidases. Due to its inhibition of this key respiratory enzyme, the transfer of oxygen from the blood to the tissues stops, thereby, reducing tissue respiration and results in tissue asphyxia. Cyanide poisoned fish may show morphologically visible symptoms like change of color of the gills to cherry red color and physiological effects like nervous breakdown and loss of equilibrium. Cyanide toxicity may get reversed in the early stages of its toxicity, if the fish are shifted to clean and cyanide free water.

3. Effect of Temperature on Heavy Metal Toxicity

The presence of hazardous heavy metals in aquatic ecosystems is an important ecological problem. A few of them are biologically useful in their minimum concentrations metals like Zn, Cu, Co, Mn, Fe and Ni, however, their higher dose may show toxic effects and lethality. While others, such as Pb, Hg, Cu, Cd and As are recognized as potentially harmful heavy metals in this review, copper and iron toxicity with respect to temperature fluctuations is briefly discussed.

3.1 Copper Toxicity Relations with Temperature

Copper toxicity in water bodies depends largely on the physio-chemical properties of the water as well as fish species under exposure. It is found both in free ionic State and in the form of soluble as well as insoluble copper salts in water bodies. Its toxicity effects include labored breathing, leaping movements out of water, over secretion of mucilage on the body surface especially on the respiratory surfaces of gills. A study was conducted to evaluate the copper toxicity effects on juvenile stages of the Neotropical freshwater fish prochilodus scorfa at two different temperatures with low and high pH. In the first stage of the experiment, acclimatization of Fry and fingerling stages of fish was done at 20°C and 30°C and in the second stage of the experiment, they were exposed to copper in water with pH 4.5 and 8.0 respectively. The 96 h-LC50 were determined at each temperature and pH. The results of the study indicated that when the pH value
of the water varies between 4.5 and 8.0, the 96 h-LC$_{50}$ for copper was significantly dissimilar, but no difference was reported in these values when pH kept constant at different temperatures. The physiological changes reported in this case include changes in blood parameters due to disturbances in the ion regulatory mechanisms and hence, respiratory trouble that imply an increase in energy consumption to restore homeostasis. Copper toxicity has been evaluated not only in fish but also in the related ecological fauna. The erratic heart rate was interpreted as a response to enhanced copper toxicity at higher temperature (25°C) in case of common shore crab *Carcinus maena*.

3.2 Iron Toxicity Relations with Temperature
Iron occurs in both ionic and combined states in surface waters. In combined state, iron occurs in Fe$^{2+}$ state in the form of soluble compounds or Fe$^{3+}$ states in the form of insoluble compounds. The percentage of these two forms of iron in the water bodies is dependent upon the physiochemical characteristics of water like oxygen concentration, pH and temperature. High water temperature decreases the solubility of gases like oxygen as per Henry’s law. Poorly oxygenated waters with a low pH increases the probability of adverse effects of soluble iron compounds on fish populations. These soluble compounds having ferrous ion may get oxidized into insoluble ferric compounds that covers surface of gill lamellae and inhibit respiration. Gills may show deposition and proliferation of filamentous iron bacteria at low temperature which oxidize iron compounds and cause their precipitation over the gill surface. It reduces the respiratory surface area of the gills, damages the respiratory epithelium and results in suffocation. Eggs and juvenile stages of fish may get affected in a similar way due to precipitation of ferric compounds over their which then die due to a lack of oxygen.

4. Conclusion
The present review may sensitize the ecologists about the toxicity impacts of contaminants which are greatly affected by climate change and may adversely affects biology of fish fauna. The principle outcome of this study is that it is necessary to consider the effects of temperature when assessing the toxic effects of organic and inorganic contaminants on the survival of aquatic fauna. High and low temperature that is lethal to individual organism of a species also determines the biogeography and abundance of its populations. The study of interaction mechanisms of aquatic toxicants with environmental factors and their management in aquatic ecosystems, hence, is the need of hour otherwise; they can lead to loss of aquatic biodiversity and can disturb ecological balance. This will also essentially helpful in understanding of basic mechanisms that allow the aquatic fauna to survive in the contaminated waters.

5. References

1. Yadav A, Gopesh A, Pandey RS, Rai DK, Sharma B. Fertilizers industry effluent induced biochemical changes in fresh water teleost Channa staiatus (Bolch). Bulletin of Environmental Contamination and Toxicology. 2007; 79, 588-595. Crossref
2. Gautama RK, Sharma SK, Mahiyab S, Chattopadhyaya MC. Chapter 1: Contamination of heavy metals in aquatic media: transport, toxicity and technologies for remediation in heavy metals in water: Presence, removal and safety; 2014. p. 1–24. Crossref
3. IPCC, 2007. Climate change 2007: the physical basis – summary for policy makers [Internet]. Available from: Crossref
4. Bryant V, Newbery DM, McLusky DS, Campbell R. Effect of temperature and salinity on the toxicity of Nickel and Zinc to two estuarine invertebrates (Corophiumvolutator, macomabaltilhica). Marine Ecology Progress Series. 1985; 24:139–53. Crossref
5. Jones MB. Synergistic effects of salinity, temperature and heavy metals on mortality and osmoregulation in marine and estuarine isopods (Crustacea). Marine Biology. 1975; 30:13–20. Crossref
6. Schiedek D, Sundelin B, Readman JW, Macdonald RW. Interactions between climate change and contaminants(review). Marine Pollution Bulletin. 2007; 54:1845–56. Crossref
7. Patra RW, Chapman JC, Lim RP, Gehrke PC, Sunderum RM. Interactions between water temperature and contaminant toxicity to freshwater fish. Environmental Toxicology and Chemistry. 2015; 34(8):1809–17. Crossref
8. Cossins AR, Bowler K. Temperature biology of animals. Chapman and Hall, London; 1987. Crossref
9. Fry FEJ. Effect of environment on animal activity. University of Toronto. Stud., Biol. Ser. No. 55, Pub. Ont. Fish. Res. Lab. 1947; 68:1–62.
10. Brown VK. Test animals. Acute Toxicity: Theory and Practice. John Wiley & Sons, Chichester; 1980. p. 33–67,117
11. Chauhan RSS, Sexsena KK. Effect of temperature on the toxicity of some metals to Heteropneustes fossilis (Bi.). Pollution Research. 1992; 11(3):131–4.
12. Bhadja P, Vaghela AK. Effect of temperature on the toxicity of some metals to Labeo buta (Hamilton, 1822). International Journal of Advanced Life Sciences. 2013; 6(3):252–4.
13. Somero GN, Chow TJ, Yancey PH, Snyder CB. Lead accumulation rates in tissues of the estuarine teleost fish (Gillichthys mirabilis); salinity and temperature effects. Archives of Environmental Contamination and Toxicology. 1977; 6:337–48. Crossref
14. Terzi, Verep B. Effects of water hardness and temperature on the acute toxicity of mercuric chloride on rainbow trout (Oncorhynchus mykiss). Medline. Crossref
15. Boudou A, Ribeyre F, Delachre A, Marty R. Bioaccumulation and bioamplification des derive du mercure par un consommateur de troisieme ordre: Salmo gairdneri-incidences du facteur temperature. Water Research. 1980; 14:61–5. Crossref
16. Cember H, Curtis EH, Blaylock BG. Mercury biconcentration in fish:temperature and concentration effects. Environmental Pollution. 1978; 17:311–19. Crossref
17. Kumaraguru AK, Beamish FWH. Lethal toxicity of permethrin to rainbow trout (Salmo gairdnen) in relation to body weight and water temperature. Water Research. 1981; 15:503–05. Crossref
18. Viswanathanand PN, Murti CRK. Effects of temperature and humidity on ecotoxicology of chemicals. Ecotoxicology and Climate. Bourdeau P, Haines JA, Klein W, Murti CRK, editors. SCOPE: John Wiley & Sons Ltd; 1989.
19. Emerson K, Russo RC, Lund RE, Thurston RV. Aqueous ammonia equilibrium calculations: Effect of pH and temperature. Journal of the Fisheries Research Board of Canada. 1975; 32(12):2379–83. Crossref
20. Reinbold KA, Pescitelli SM. Effects of cold temperature on toxicity of ammonia to rainbow trout, bluegills and fathead minnow. Aquatic Ecology Technical Report, Illinois Natural History Survey; 1990. p. 1–26.
21. Lewis WM, Morris DP. Toxicity of nitrite to fish: A review. Transactions of the American Fisheries Society. 1986; 115:183–95. Crossref
22. Kroupova H, Machova J, Svobodova Z. Nitrite influence on fish: a review article. Vet. Med. – Czech; 2005; 2005(11):461–71.
23. Colt J, Tchobanoglous G. Evaluation of short term toxicity of nitrogenous compounds in the channel catfish, Ictalurus punctatus. Aquaculture. 1976; 8:209–24. Crossref
24. Colt J, Ludwig R, Tchobanoglous G, Cech JJ. The effect of nitrite on the short-term growth and survival of channel catfish, Ictalurus punctatus. Aquaculture. 1981; 24:111–22. Crossref
25. Huey DW, Simco BA, Criswell DW. Nitrite induced methaemoglobin formation in channel catfish. Transactions of the American Fisheries Society. 1980; 109:558–62. Crossref
26. Huey DW, Beitinger TL. A haemoglobin reductase system in channel catfish Ictalurus punctatus. Canadian Journal of Zoology. 1982; 60:1511–13. Crossref
27. Huey DW, Beitinger TL, Wooten MC. Nitrite induced methaemoglobin formation and recovery in channel catfish (Ictalurus punctatus) at three acclimation temperatures. Bulletin of Environmental Contamination and Toxicology. 1984; 32:674–81. Crossref
28. Scott GR, Sloman KA. The effects of environmental pollutants on complex fish behavior: Integrating behavioral and physiological indicators of toxicity. Aquatic Toxicology. 2004; 68:369–92. Crossref
29. Carvalho CS, Fernandes MN. Effect of temperature on copper toxicity and haematological responses in neotropical fish Prochilodus scorf a at lower and high pH. Aquaculture. 2006; 251(1):109–17. Crossref