Toxic Effects of Cyanide on Freshwater Fish

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Abstract

All types of living species may become toxic as a result of the discharge of various chemicals into inland and surface waters, land, and soil. "Cyanide" is one such hazardous and poisonous chemical. Wastewaters are frequently harmful, especially those produced by businesses like metal mining, electroplating, printed circuit board production, etc. Establishing the toxicity evaluation requires evaluating the toxicity of sodium cyanide in relation to aquatic biota, particularly fish. Aquatic creatures are known to quickly absorb sodium cyanide, and bioconcentration factors for different animals ranged from 135 to 4800. This study offers a platform for determining the acceptable limits and safe doses of harmful compounds like cyanide for the aquatic environment's biota as well as for preventing a residual imbalance in the cycle's aquatic bio-ecological, which aids in incorporating the monitoring of bio-detector.

Keywords: *Fish, Cyanide, Pesticides, Toxicity*

INTRODUCTION

Environmental degradation of the air, water, and soil has recently been brought on by the fast industrial development. All types of living species may become toxic as a result of the discharge of various chemicals into inland and surface waters, land, and soil. "Cyanide" is one such hazardous and poisonous chemical. Despite being well-known for being very hazardous, cyanide finds extensive use in a wide range of industrial processes, including metal mining (mostly for gold and silver), electroplating, steel manufacture, vehicles, carbonization, printed circuit board manufacturing, and chemical industries, among others. As a result, these sectors produce a significant amount of effluents that include cyanide. Wastewaters are frequently harmful, especially those produced by businesses like metal mining, electroplating, printed circuit board production, etc. Similar to this, valuable heavy metals such as copper, nickel, iron, zinc, cadmium, chromium, silver, and gold readily bond with free cyanide to form metal-cyanide complexes, which vary in stability and toxicity. The daily wastewater emitted by cyanide-using companies ranges from 200 to 1000 litres for small businesses and from 1-20 cubic metres or more for major businesses. In comparison to their acceptable (discharge) limitations of 0.2 and 0.01-5 mg/L, respectively, these effluents have total CN and metal contents that vary from 0.5-100 and 1-250 mg/L, respectively. Therefore, effluents containing metal-cyanide cannot be released into the environment without first receiving adequate treatment. Total cyanide levels in discharged liquid wastes are reduced to 0.2 mg/L (200 g/L) by the use of a number of physical-chemical treatment techniques. However, the majority of living forms are poisonous at even concentrations of 0.2 mg/L of cyanide in aqueous environments. According to some accounts, even cyanide concentrations as low as 0.01-0.1 mg/L can cause the death of some sensitive aquatic animal species. Fish play a significant role in toxicity testing and hazard assessment in the field of aquatic toxicology, just as the white rat and guinea pig do in mammalian toxicology. Fish are reportedly among the most vulnerable animals to the toxicity of cyanide. The purpose of the current review article is to highlight various scientific findings about the toxicity of cyanide in fishes while keeping this remark in mind [1-6].

Noteworthy Reports

Establishing the toxicity evaluation requires evaluating the toxicity of sodium cyanide in relation to aquatic biota, particularly fish. Aquatic creatures are known to quickly absorb sodium cyanide, and bioconcentration factors for different animals (fish, daphnia, and snails) ranged from 130 to 4,900. According to laboratory testing, sodium cyanide is extremely poisonous to aquatic creatures, with median lethal concentration values for freshly hatched prawns ranging from 0.02-1 g/L. According to the findings, *Labeo rohita* is poisonous to sodium cyanide. The edible freshwater fish *L. rohita*, sometimes known as the Indian large carp, is very significant economically [7].

The *L. rohita's* susceptibility to sodium cyanide (free cyanide) has recently been investigated1. The 33 g/L LC50 for 96 hours was discovered. Increased opercular movement, increased surface behaviour, loss of balance, change in body colour, increased mucus production, irregular swimming activity, quick jerk movement, partial jerk, and aggression were among the behavioural alterations seen in animals exposed to deadly concentrations of sodium cyanide. The swimming style was corkscrew-like and rotated on a horizontal axis. The fish's schooling habit was gradually interrupted and the ventilation rate was raised during sub-lethal treatment. The fish behaved normally on the 21st day after exposure, with balanced swimming and vigorous eating. Lethal concentrations saw a decline in oxygen consumption, although sublethal concentrations saw an improvement in the downward trend, which returned to normal levels by day 21 (-25.10 to -2.19%). Changes in oxygen consumption may be brought on by respiratory distress as a result of oxidative metabolism impairment. Stressed fish were discovered, however they were not killed by the sub-lethal dosage. The fish got agitated

and overexcited at the sodium cyanide concentrations below the fatal threshold. The fish began to swim erratically and jump, and eventually it flipped on its side. Loss of balance and mucus discharge were also seen. With brief, jerky movements, air surfacing and gulping, and erratically circular motions, they gradually become lethargic. They eventually came to rest at the bottom, losing their balance and rolling their bodies, and exhibiting spasms before passing away. Fish frequently surfaced in order to escape harmful environments. Additionally, gill inspection of deceased fish indicated that the red hue of the gill lamellae had turned to brown [8-10].

The peculiar conduct of *L. rohita* under cyanide stress conditions may be brought about by neurotransmitter malfunctions. The increased physiological activity in the stressful habitat caused the gill opercular motions to first rise, and then they may have reduced as a result of gill mucus buildup. Chemical molecules in tissues are directly impacted by the toxic stress of pesticides. The excessive release of mucus across the gills may prevent oxygen from diffusing during the exchange of gases. It implies that fish and other non-target creatures are not protected from the sodium cyanide.

Following the injection of sodium cyanide, *L. rohita* fish moved to the bottom of the tank, which is a response that has previously been seen in trout. The fish stop moving optically as soon as they are exposed to cyanide. The increase in fish surfacing frequency and concomitant increase in ocular movement show that fish have evolved to use aerial respiration to get atmospheric oxygen while avoiding contact with cyanide by using their gill chambers.

When *L. rohita* was exposed to sodium cyanide poisoning, the fish's increased ventilation rate was accompanied by fast, repetitive opening and closure of their mouth and opercular covers, as well as partly extended fins (coughing). The fish treated with sodium cyanide exhibited abnormal swimming patterns, which suggested that the brain area responsible for maintaining homeostasis should have been impacted.

When endosulfan and lidane were present at lethal and sub-lethal amounts, tilapia fish displayed anomalous behavior. Under abrupt, intense stress, the fish displayed erratic swimming, convulsion, spiralling, tremors, jerky movements, and quick opercular motions. The fish battled mightily to breathe, frequently swam to the surface to breathe atmospheric air, and attempted to swim away from the hazardous aquatic environment. After a few hours, the fish lost their balance and began to gently spiral upward in a vertical orientation. When they finally totally lost their balance, they were lying at the bottom. According to numerous authors the acetyl cholinesterase enzyme interference with the nervous system's capacity to function may be the cause of the fish's hyperexcitability, which occurs inevitably when they are exposed to free cyanide or pesticides, which are both known to kill fish when consumed. Acetylcholine builds up as a result, which might extend the excitatory post synaptic potential. The cholinergic system may initially be stimulated and then become blocked as a result of this. The buildup and increased mucus production in fish exposed to free cyanide may be adaptive reactions that offer extra defence against the pesticide's corrosive nature and prevent the toxicant from being absorbed by the fish's general body surface. Time affected the aberrant alterations in the fish exposed to the deadly dose of free cyanide. Since most fish breathe their own water, changes in the water's chemical composition may be reflected in the activity of the animal's ventilator, especially if the environment impacts respiratory gas exchange. Fish's respiratory systems are the major entry points for environmental toxins. Through pores and simple diffusion, toxins are likely taken up through the gills and subsequently absorbed through cell membranes. Under fatal and sub-lethal doses of sodium cyanide, *L. rohita's* oxygen consumption dropped, indicating the sort of compensatory mechanism, if any, that the animal may have used to cope with the toxin stress. The respiratory distress brought on by the impairment of oxidative metabolism may be the cause of the entire fishes' reduced oxygen consumption. Numerous publications have documented a comparable reduction in the total oxygen uptake of fish exposed to toxins. The primary respiratory organs are the gills, and every metabolic pathway depends on the effectiveness of the gill for its energy source. Therefore, damage to the gill of this important organ may set off a series of negative events that eventually result in respiratory distress. Due to the disruption of the respiratory system brought on by the gill's respiratory epithelium rupture, oxygen consumption decreased. Additionally, several mitochondrial enzymes exhibit lower activity as a result of the disruption of mitochondrial integrity. In addition to gill injury, a drop in haemoglobin levels and a reduction in tissue respiration can both obstruct the respiratory cycle and cause respiratory failure. As previously mentioned, the surfacing behavior of fish observed after cyanide exposure may be caused by the animal's hypoxic condition. The diffusion of oxygen during the process of gaseous exchange may be inhibited if the gills secrete an excessive amount of mucus as a result of cyanide stress. Mucus coagulation on the gills destroyed several critical functions, including blood circulation, nitrogen excretion, gas exchange, and salt balance. The internal action of cyanide would be an alternate explanation for the reduction in oxygen use. The reason why fish exposed to deadly concentrations consume less oxygen than fish exposed to sub-lethal concentrations may be due to both significant damage to the gill structure and increased mucus precipitation on gill filaments, which causes gill blockage. One of the causes of fish deaths at deadly concentration is likely the suffocation caused by the coagulated mucus film and necrosis on the epithelial and inter lamellar cells of gills. The increased damage done to red blood cells, as evidenced by the sharp decline in their number, may also have contributed to the fish's larger drop in oxygen consumption at the deadly dosage. In addition, fish exposed to sub-lethal cyanide concentrations showed significant metabolic reorganisation and enhanced anaerobic metabolism use. It has been determined that sodium cyanide is naturally poisonous to fish (like *L. rohita*), but there are various factors that affect how hazardous it is. This review also offers a platform for determining the acceptable limits and safe concentrations of toxic substances, such as cyanide, for the aquatic biota and to prevent residual imbalance in the aquatic bio-ecological cycles, which aids in involving the bio-detector monitoring and process, particularly for fish [11-17].

CONCLUSION

Fish are extremely susceptible to the toxicity of cyanide. Numerous industries produce large amounts of effluents that include cyanide. Even at concentrations as low as 0.01-0.1 mg/L, cyanide has the ability to harm some sensitive aquatic animal species. Aquatic creatures are known to quickly absorb sodium cyanide, and the bioconcentration factors for different animals including fish, snails, and daphnia range from 130-4900. As a result, farmers must be educated on the need to control agricultural pests using biological techniques rather than cyanides or other pesticides in order to safeguard the entire aquatic environment. This article makes recommendations on the safe cyanide concentrations and tolerated ranges for aquatic biota.

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REFERENCES

[1] Prashanth MS, Sayeswara HA, Goudar MA. Effect of sodium cyanide on behaviour and respiratory surveillance in freshwater fish, *Labeo rohita* (Hamilton). Rec. Res. Sci. Tech., 2011; 3(2): 24-30.

[2] Patil YB. Studies on biological detoxification of metal-cyanides containing industrial effluents. PhD Thesis. Pune, India: University of Pune; 1999.

[3] Chaudhary, Rathod V, Vankhede GN. Effect of water extract of the bark of *Buchanania langan* (Linn.) on behaviour and chromatophores of a freshwater fish, *Labeo rohita*. J. Enrion. Biol., 2001; 22(3): 229-31.

[4] Tilak KS, Yaeobu K. Toxicity and effect of fenvalerate on fish, *Ctenopharyngodon idellus.* J. Ecotoxi, Envrion. Moint**.,** 2002; 12(1): 9-15.

[5] Murthy AS. Sub-lethal effects of pesticides on fish. In: Toxicity of Pesticide to Fish, Vol. II. Boca Roton, USA: CRS Press; 1987; p. 55-100.

[6] Prashanth MS, Patil YB. Behavioural surveillance of Indian major carp *Catla Catla* (Hamilton) exposed to free cyanide. J. Curr. Sci., 2006; 9(1): 313-8.

[7] Deva Parkasa Raju B. Fenvalerate induced changes in protein metabolism of freshwater fish, *Tilapia mossambica*. PhD Thesis. Anantapur, AP, India: SK University; 2002.

[8] Thorat SR. Chronic effect of endosulfan on freshwater fish, *Catla catla*, J. Ecotoxicol. Envion. Moint., 2001; 11(4): 221-3.

[9] Prashanth MS, Neelgund SE. Free cyanide induced biochemical changes in nitrogen metabolism of the Indian major carp, *Cirrhinus mrigala*. J. Basic & Clin. Physiol. Pharmacol., 2008; 18(4): 277-87.

[10] Prashanth MS. Cypermenthrin induced physiological, biochemical and histopathological changes in freshwater fish, *Cirrhinus mrigala*. PhD Thesis. Dharwad, India: Karnataka University; 2003.

[11] David M, Mushigeri SB, Prashanth MS**.** Toxicity of fenvalerate to the freshwater fish, *Labeo rohita.* Geobios**,** 2002; 29(1): 25-7.

[12] Esther Joice P, Mallikaraj D, Parthi N, Natarajan GM, Sasikala G, Tamilselavi G, Kasthuri M. The effect of hypoxic stress on the bimodal respiration of *Macropodus cupanus.* J. Eco. Res. Biocon., 2001; 2(1&2): 76-80.

[13] Ravinder V. *In vivo* effects of decis on certain aspects of metabolism of freshwater fish, *Clarias batrachus*. PhD Thesis. Hyderabad, AP, India: Osmania University; 1988.

[14] Magare SR, Patil HT. Effect of pesticides on oxygen consumption. Red blood cell count and metabolites of fish, *Puntius ticto*. Environ. Eco., 2000; 18(4): 891-4.

[15] Sarkar SK. Effects of heavy metals (copper sulphate and cadmium sulphate) on the O2 consumption of fish, *Cyprinus carpio*. U.P. J. Zool., 1999; 19(1): 13-6.

[16] Radhaiah V, Jayantha Rao K. Behavioural response of fish, *Tilapia mossambica* exposed to fenvalerate. Environ. Ecol., 1988; 6(2): 2-23.

[17] Venkataramana, P. 1987. Studies on lethal and sublethal toxicity of copper in the freshwater teleost, *Labeo rohita*. PhD Thesis. Anantapur, AP, India: SK University; 1987.