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Cyanobacteria toxicity in aquaculture system and its impact on fish physiology

Abstract

Algae and some Cyanobacteria, being a core part of primary production, act as a food organism for many fishes and other aquatic organisms. But they can also be responsible for fish kill or illness. Review on Cyanobacteria effect on fish growth, survival, and recruitment is the need of the hour. The mechanisms of toxicity of cyanotoxins and their toxic metabolites in fish have been scarcely covered. The effects may be sublethal on growth, physiology, survival, recruitment, and in long run, it may have a role in the fish's adaptive response to abiotic and other biotic stressors. Around 46 species from genera of *Microcystis, Cylindrospermopsis, Synechococcus, Anabaena, Lyngbya, Oscillatoria,* etc. have been shown to cause toxic effects in aquatic system. The bloom of these cyanobacteria is primarily associated with altered temperature and nutrient load in water bodies due to effluents from municipal discharge and aquaculture. Their acute or chronic toxic effects may vary depending on the species, type of toxin produced, and concentration. The various cyanotoxins are grouped as hepatotoxins like microcystin, nodularin cylindrospermopsin, neurotoxins; like anatoxins, homoanatoxins, dermatotoxins; like aplysiatoxin, debromoaplysiatoxins, lyngbyatoxins, and pyrogenic component; like lipopolysaccharides (LPS). The concentration of the specific cyanotoxin in the fish body and the water along with other factors such as the length of exposure, fish metabolic processes, water parameters like dissolved oxygen and temperature, are likely to impact cyanotoxin toxicity in freshwater fish. The impact of such toxicity may be reflected on the individual species level, ecosystem level, and even at the culture system level.

Keywords: cyanobacteria, cyanotoxins, aquatic system, fish physiology, toxicity, aquaculture

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Md Aklakur,[1] Subham Bakli,[2] Ashutosh D Deo,[2] D K Singh,[3] G H Pailan[3]

[1]Regional Research & Training Centre (RRTC) Motipur of ICAR-CIFE, India
[2]ICAR- Central Institute of Fisheries Education, India
[3]Central Institute of Fisheries Education, India

Correspondence: Md. Aklakur, Regional Research & Training Centre (RRTC) Motipur of ICAR-CIFE Mumbai, Motipur, Muzaffarpur, Bihar, 843111, India, Tel +91 9004862708, Email aklaku@cife.edu.in

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Introduction

The term 'algae' is a diversified informal term for microscopically small, unicellular photosynthetic organisms which form colonies and are shown as minute green particles. These tend to distribute smoothly throughout the water and cause more turbidity with higher density. However, algae are the core part of primary production in the aquatic system and they act as a food organism for many fishes and food organisms, being in the food chain of fish. The algae have very good nutritional quality and they make a major part of phytoplanktonic biomass in a pond and other aquatic systems. But some of these algae have been known as causing death or illness in cultures fishes and as well as different aquatic mammals, birds, etc. through, the toxicity of Cyanophyta. Cyanophyta or Cyanobacteria are nothing but a mixture of bacteria and some of the algae which contain blue-green (c-phycocyanin) and green pigments (chlorophyll- a), carotenes, xanthophylls, and a red pigment (c- phycoerythrin) that can execute photosynthesis.

The toxic effect of the cyanophyta is one of the major concerns for humans and other animals. However, its effect on fishes, their growth and survival, recruitment is the need of the hour especially in the present condition of anthropogenic activity leading to community change in the aquatic system and natural ponds.

Most of the papers were published based on toxic freshwater algae and Prescott[1] reported that toxicity of algae has tended to kill fish. Similarly, Olson[2] published a brief summary of poisonous plankton, and Scott[3] presented a general paper on fresh-water algal poisons. So, the theories of most of the studies are based on human activities (agriculture runoff, sewage treatment, chemical fertilization, etc.) which led to an unrestricted proliferation of algae due enrichment of water in nutrients. Proliferation is a consequence of eutrophication). The Cyanobacteria of freshwater bodies are very common to cause bloom and show a huge impact upon recreational and culture water bodies.

In temperate regions, cyano bacterial dominance is most pronounced during the summer months. But in tropical climates, spring and post-monsoon seasons are the actual time of eutrophication of such blooms. The cyano bacterial toxins are the most studied algal toxin in the freshwater system. The understanding of the mechanisms of toxicity of cyanotoxins and their toxic metabolites from algae in fish has been rarely covered.

Similarly, marine species of various genera often contain toxins and are responsible for toxic bloom in coastal water leading to fish kills. So, it is reasonable to expect toxic species among these groups in freshwater aquatic systems like lakes, chaurs (shallow water bodies in wetlands), ponds, and other aquaculture systems. We can say this review may focus primarily on the freshwater fish health impacts of cyanobacteria, while many species of freshwater algae grow rapidly in eutrophic waters, the toxin does not accumulate which is hazardous to the health of livestock.[4] When we understand the toxic effect, it may be a sublethal impact like, on growth, physiology, survival, and recruitment in long run, which may have a role on the adaptation of the fish in response to abiotic and other biotic stressors. The toxic effect and sub lethal levels are something of great concern as it may have role in growth reduction and a stress inducing factor for cultured fish. Therefore on farm several chemical like Cupper sulphate, hydrogen peroxide etc. have been used and evaluated.[5] Similar study by Kang et al., reflects such initiatives.[6,7]

Toxic cyanobacteria

Cyanobacteria can be benthic or pelagic, and their form makes them one of the most varied bacterial phyla. Cyanobacteria have been

discovered classified into five distinct morphological parts, including unicellular colonies with binary fission, uni-colonies having numerous fissions, multicellular colonies, differentiated cells in multicellular colonies, and branching colonies clusters of developed cells that are multicellular in nature.[8]

The cyano bacterial colonies are large enough to be seen from the naked eye. When they are in abundant amount and form bloom, it alters the colour of water and sometimes they form thick scum on the surface of the water. Consumption of fish from such waters is an important route of human exposure to cyanotoxins.[9] As much as 40% of global finfish production comes from freshwater,[10] and cyanotoxins have been detected in freshwater fish from around the world.[11] However, the accumulated toxins give an impact on other animals eating the fish, but fishes are the first target of such toxins. There is enough chance of toxic effect on fish and other aquatic communities, when these toxic compounds or their metabolites leach to water.

At least 46 species (approx. 60% of cyano bacterial samples) have been shown to cause toxic effects in vertebrates.[12] The most common toxic cyanobacteria in fresh water are *Microcystis* spp., *Cylindrospermopsis raciborskii*, *Planktothrix* (syn. *Oscillatoria*) *rubescens*, *Synechococcuss*, *Anabaena* spp., some *Oscillatoria* spp. etc. *Anabaena*, *Aphanizomenon*, *Oscillatoria*, and *Microcystis* grow best at temperatures ranging from 25 °C to 35 °C. The bloom of cyanobacteria is primarily associated with altered temperature, nutrient load in water bodies due to effluents from municipal discharge and aquaculture. Secchi disc transparency in fish ponds provides a rough estimate of plankton abundance. The most important factor influencing pond productivity is dissolved oxygen.

Cyanotoxins and cyano bacterial prevalence

Microcystins and neurotoxins are the most common cyano bacterial poisons or cyanotoxins. There are dangerous and harmful strains in most species, however, nontoxic strains do exist. Microcystins is produced by *Microcystis*, the most common bloom-forming genus,[13] is virtually always harmful in the wild as their blooms resemble a greenish, thick, paint-like (sometimes granular) material that accumulates along shores of ponds or lakes. While the conditions that contribute to cyanobacteria proliferation are well established,[4] and varies widely. The factors that lead to harmful strain dominance are the favourable circumstances, such as high temperature, strong light, and pH, and increased nutrient availability. The cyanobacteria can proliferate swiftly in such surface waters and create blooms.[14]

Because certain cyano bacterial species create extremely poisonous secondary metabolites known as cyanotoxins so these blooms can be dangerous to people, other animals, and aquatic life. Blooms can persist anywhere from a few days to several months. They have grown in size and frequency in most locations, and are typically caused by an excess of nutrients in the environment. The water contains the two most prevalent macronutrients, which are nitrogen and phosphorus, and their fixation from agricultural and industrial sources, low circulation and other variables have a major role like increased water temperature.[15] Hazardous cyano bacterial species may benefit from global climate change. As they can tolerate adverse climatic condition and boost their growth rate, dominance, persistence, geographical dispersion, and activity.[16,17]

Toxins produced by some cyanobacteria species or strains may threaten aquatic life or fish. The probable acute toxic effect[18] of cyanotoxins may be among a few of following: decreased survival and reduced growth; reduced swimming motions or activity; lethargy and gasping; full paralysis; gastroenteritis; hepatotoxicity and hepatic oxidative stress; hyperplasia and histopathological damage to vital organs. Similarly, chronic impacts may be witnessed on the population or community level, are: lower fertility and population growth rate; poor recruitment performance; reduce biological growth potential; reduced reproductive performance; change in community structure of the aquatic ecosystem; altered species or trophic dominance. These effects can also occur as a result of sub-lethal doses. Effects, on the other hand, vary depending on the species, type of toxin produced, and concentration.[19]

Formation of cyano bacterial bloom

The bloom occurs in aquaculture system and their load in water produce scum. The vertical movement of these algae is triggered by buoyancy regulation via intracellular gas vesicles in several species. Changing the amount of gas in the vesicles to regulate buoyancy is a slow process. They are driven to leeward side by light breezes, where they produce scums.

In extreme circumstances, these agglomerations can become extremely dense and even gelatinous.[4] Dying and lysing cells (i.e., cell rapture) release their toxins into the water, where pigments can take on a coppery hue. Wave motion can swiftly break scums and re-disperse them with new wind mixing. However, scum material may take a long time to disperse, especially in shallow bays or ponds, due to either wave wash or cell disintegration. However, Cyanobacteria mats, also known as benthic mats, are typically caused by planktonic species living in eutrophic waters (which are relatively low in plant nutrients), but they can also be caused by pollution and a lack of oxygen. During sunny days, their photosynthesis may result in high rates of oxygen production, forming bubbles that loosen and drive parts of the mats to the surface. Beach mats of benthic cyanobacteria washed to the shore and scavenged by dogs have been lethal[20] and cyanobacteria may also be responsible for cattle deaths on Swiss alpine meadows.[21]A similar kind of threat is for aquatic species as a target or non-target species.

However, it is essential to have knowledge on the potential toxicity of such mats in aquatic systems especially associated with aquaculture practice or nearby areas. Toxic blooms may reappear periodically in aquaculture environments, and fishes under culture may be chronically exposed to cyanotoxins at relatively low doses. The concentration of the specific cyanotoxins in the body and the water, as well as the length of exposure and fish metabolic processes, water parameters like temperature, are likely to impact cyanotoxin concentrations in freshwater fish.[22] The impact of such toxicity may be reflected on the individual species level, ecosystem-level, and even at system level in particular aquaculture system.

Toxicity on freshwater system

Microcystins are cyclic heptapeptides that are commonly produced by different cyano bacterial genera those are discussed earlier. They are also called hepatotoxin, named from the fact that the liver is their principal target (Table 1). In most places, anthropogenic nutrient over-enrichment (eutrophication), as well as other variables such as intense agriculture growth, fast industrialization, and urbanization, appears to increase the incidence of microcystins-producing HABs.

Microcystins, nodular in, and cylindrospermopsins are examples of known toxins that can induce liver and kidney damage, as well as cytotoxicity, neurotoxicity, skin toxicity, gastrointestinal disorders, and other issues. These effects may arise immediately after exposure or may take days to manifest. Among them Microcystin-LR is the most toxic, well studied and abundant variant and is one of the most important algal toxins that has received worldwide attention. Micro-

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cystin poisoning has different clinical symptoms depending on the method of exposure, the quantity of exposure, and the combination of components involved in the exposure. In most exposures, low to moderate levels of exposure of mammals to toxic blooms are linked to irritating effects in the skin, respiratory system, and gastrointestinal system, leading to inflammatory reactions. Higher amounts of exposure, particularly oral exposures cause liver damage, and if the liver damage is severe, it can be fatal.[23]

Table 1 Cyanotoxins their target organ of toxicity and nature (source: derived from Geoffrey A. Codd, Steven G. Bell and William P. Brooks Department of Biological Sciences, University of Dundee, Dundee DDI 4HN, U.K.)

Name of the toxin	Toxin class/primary target organ of the toxin	Nature of compound
Microcystin	Hepatotoxin Liver Tumour promotion	Cyclic heptapeptide
Nodularin	Hepatotoxin Liver Tumour promotion	Cyclic pentapeptide
Cylindrospermopsin	Hepatotoxin/cytotoxin Liver and kidney	Alkaloid
Anatoxin-a Homoanatoxin-a	Neurotoxin Neurons	Alkaloid
Anatoxin-a(S)	Neurotoxin Neurons	Alkaloid (organophosphate)
Saxitoxins	Neurotoxins Neurons	Alkaloid
Aplysiatoxin	Dermatotoxin Skin Tumor promotion	Alkaloid
Debromoaplysatoxin	Dermatotoxin Skin Tumor	Alkaloid
Lyngbyatoxin-a	Dermatotoxin Skin	Alkaloid
LPS	Pyrogenic Unspecific health effects such as fever	Call wall component

Liver edema and elevated concentrations of liver enzymes like alanine aminotransferase and aspartate aminotransferase in the blood are the first indicators of liver injury. Inappetence, sadness, and vomiting are common early symptoms, followed by diarrhoea that can become severe and haemorrhagic within minutes to hours of contact. Inappetence and sadness get worse with time. Recumbency and coma may be connected with the latter stages of fatal poisoning.[24] Microcystin--LR has the potential to release carcinogens to the human's liver, testes after consuming toxin affected fishes.[25] It can progress colorectal carcinoma[26] It can also stimulate epithelial-mesenchymal transitions (EMT), which regulate cancer cell's motility and invasive properties that can lead to metastasis in embryonic development and cancer progression.[27] Such symptoms of toxicity mostly based on freshwater system-based toxicity are directly linked to humans. However, such impact may have on fishes and is a matter of widespread study and delineation in species-specific manner.

Microcystin exposure causes cellular damage in fish, notably liver damage, comparable to the consequences reported in mammals[28] When compared to young fish, mature fish are more resistant to microcystin toxicosis. Although fish have the capacity to avoid areas of toxic algal growth, sublethal liver injury in fish is connected with microcystin accumulation in fish food items. Mussels, snails, and zoo plankton.[29] Fish are equally vulnerable to low oxygen levels during such bloom.

Water oxygen demand is induced by algal bloom respiration as well as with algal degradation of scum, and this impact may potentially contribute to fish fatalities[30] The impact of lower and greater doses of microcystin-LR has been reflected at cellular or tissue level in form of apoptosis and necrosis in lymphocytes, reorganization of the actin cytoskeleton, cell shrinkage, and the absence of filopodia were also detected in phagocytes, and finally the immune system dysfunction.[31] Kozdeba et al.[32] reported that microcystin irritated the skin after epidermal exposure and in situations of skin injury, quicker penetration to deeper cell layers and absorption into the systemic circulation might be predicted. Additionally, only after lengthier skin exposures, the persistent harmful effects such as keratinocyte migration and cytoskeleton were observed.

Toxicity effect on fish physiology

Histopathological examinations of fish killed during cyano bacterial blooms revealed that the cause of death was mostly related to gill, digestive system, and liver damage. The gill injury was most likely caused by the high pH created by cyano bacterial photosynthetic activity before to bloom collapse. Increased microcystin intake may have led to liver necrosis. Other pathological symptoms associated with toxic blooms include liver, heart, kidney, skin, gills, and spleen damage[12] Malbrouck and Kestemont[29] reported the occurrence of disturbances in the main development processes can often result in death. Fish in the early stages of life are more sensitive to toxic compounds than adults and juveniles, probably due to their thin epithelial layer combined with a relatively large body surface area or the limited ability of the metabolic process to neutralize the cyanotoxins. Liu et al.[33] observed that mortality in juvenile loach (*Misgurunsmizolepis*) was dependent on the developmental stage of loach embryos. Similarly, the toxicity of different cyano bacterial blooms on the embryo and larval development of carp *Cyprinus carpio* were investigated. Microcystis spp. was found to dominate in two samples. Both the sam-

ples of complex cyano bacterial biomass, in that the aqueous extracts, showed embryo toxic effects such as significant fish mortality and delayed hatching. According to their findings, the individual carp exposed to higher concentrations of some fractions from all biomasses showed missing eye pigmentation and incomplete filling of the air bladder after 120 hours.

Tencalla et al.[34] studied the effects of MC-LR on yearling rainbow trout where microcystins travel primarily through the gastrointestinal tract by force-feeding in trout, and that toxicity manifests as massive hepatic necrosis followed by fish deaths, whereas immersion of adults and juveniles in contaminated water did not cause toxic effects. *M. aeruginosa* concentrations of 8-16 mg freeze-dried algae/L were not found to be toxic to trout when added to water. When 1.440 mg of lyophilized algae/kg of body weight was passed through the gills for 18 hours, the trout died within 96 hours. Oberemm et al.[35] discovered that exposure to concentrations of 50 and 5 mg/L MC-LR reduced embryonic survival rates by 40% and 20%, respectively, as well as growth and weight by 25%, in a study on the effects of microcystins in *Danio rerio*. Other evidence suggests that sensitivity differences while exposure of toxins between species can be significant: goldfish were found to be nearly 30 times less susceptible to i.p. microcystin than mice.[36]

The gill damage was most likely caused by the high pH caused by cyano bacterial photosynthesis activity prior to bloom collapse, as well as the higher level of ammonia caused by cyano bacterial decomposition (Table 2). However, gill damage may have increased microcystin uptake, leading to liver necrosis. Dissolved microcystin-LR has been shown in Tilapia and trout to cause gill damage.[37]

Table 2 Toxicity of microcystin for some freshwater fish species

Species	Toxic concentration	Effects	References
Loach (*Misgurunsmizolepis*)	0–500 µg MC-LR/L, during 30 days	Chronic exposures are shown. Mortality and abnormalities include pericardial edoema and tubular heart, bradycardia, homeostasis, inadequate yolk resumption, a tiny head, a curved body and tail, and improper hatching.	Liu et al.[33]
Chub (*Leuciscus cephalus*)	0.5, 5, 50 µg MC-LR or MC-RR/L (Microcystin-LR OR RR strain)	Dose-dependent decrease of survival rate.	Oberemm et al.[35]
Zebra fish (*Danio rerio*)	0.5, 5, 50 µg MC-LR or MC-RR/L; 5, 50 µg MC-LR/L	Survival and growth rates have slowed. The survival rate has been cut by 40%, and the weight has been reduced by 25%.	Oberemm et al.[35]
Medaka (*Oryziaslatipes*)	Microinjection MC-LR 1–10 µg /mL (0.1–2 pg or 1–20 pg of toxin injected)	Embryo mortality is dose dependant. Late-stage hepato biliary damage includes hepato biliary enlargement, hepatic bleeding, and necrosis.	Jacquet et al.[23]
Rainbow trout (*Oncorhynchus mykiss*)	0.5, 5, 50 µg MC-YR, MC-RR/L and 50 µg MC-LR/L	Stimulated hatching	Oberemm et al.[35]
	8–16 mg freeze-dried algae (MC-LR)/L	The trout died within 96 h when 1.440 mg of lyophilized algae/kg of body weight passed through the gills for 18 h., toxicity manifests itself as massive hepatic necrosis.	Tencalla et al.[34]
Common carp (*Cyprinus carpio*)	5 and 0.5 g L-1 MC-LR or MC-RR	Embryo toxic effects such as delayed hatching and suppression in embryonic development. Missing eye pigmentation at 48 h post fertilization and incomplete filling of air bladder after 120 h	Palíková et al.[46]
	Suspensions of 10^5 and 10^7 cell/mL lyophilized cyanobacteria containing anatoxin-a	Rapid opercular movement and abnormal swimming. This interference with fish behaviour caused by anatoxin-a	Ossvald et al.

ERodger et al. [38] and Rabergh et al.[39] conducted experiments on trout and carp, respectively. The latter study focused on degenerative changes in the kidney tubules and glomeruli. Carbis et al.[40] described the effect of microcystins on *Cyprinus carpio*, in natural field conditions in Australia as hepatocyte atrophy, gills with pinpoint necrosis, epithelial ballooning, folded lamellar tips, and exfoliation of the lamellar epithelium, elevated aspartate amino transferase activity, and serum bilirubin concentrations. Laboratory studies show that dissolved microcystins can affect fish embryos and behaviour. [41,42] Anatoxins also caused behavioural changes in juvenile *Cyprinus carpio*, such as rapid opercular movement and abnormal swimming, and lateral malformations and increased mortality in *Danio rerio* embryos.[43] Such physiological impact has been reported in fishes and been found to be quite wide spread. Therefore it creates concern wide aspect.

Conclusion

Cyanobacteria are vital in a variety of habitats, but when conditions are good, they may dominate aquatic environments and multiply excessively. Human activities are mostly to blame for the abundant supply of nutrients in bodies of water, which contributes to the increased severity of cyano bacterial blooms. Cyanotoxins cause serious poisoning in aquatic species and humans. Ingestion of Microcystis cells has negative consequences in a variety of aquatic species. In the case of fish, the toxic effects induced disruptions in the primary development processes, making the early life stages more vulnerable to microcystin. Anatoxins also caused behavioural changes in young *C. carpio,* such as fast opercular movement and aberrant swimming. Saxitoxins caused lateral abnormalities in *D. rerio* embryos and incre-

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ased mortality. Due to these toxic impacts the concern of cyanophyta and its control in aquaculture is gaining very much importance. Effort in this regard has been made through several studies like Buley et al. [44] investigated different product to control the bloom in aquaculture. Similarly Drummond et al.[45,46] studied other strategies to control the cyano bacterial blooms in reservoirs. However, given the great potential of cyanobacteria for the production of compounds important in the discovery of new medicines, there is also a need for additional efforts to identify biomolecules, develop new methods to isolate cyano bacterial toxins, and conduct new research on anticancer compounds obtained from cyanobacteria.

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Conflicts of interest

We declare that there is no conflict of interest among the authors or with any one related to the present work.

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