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Comprehensive Insights into the Impact of *Microcystis* on Aquaculture: Challenges and Future Perspectives

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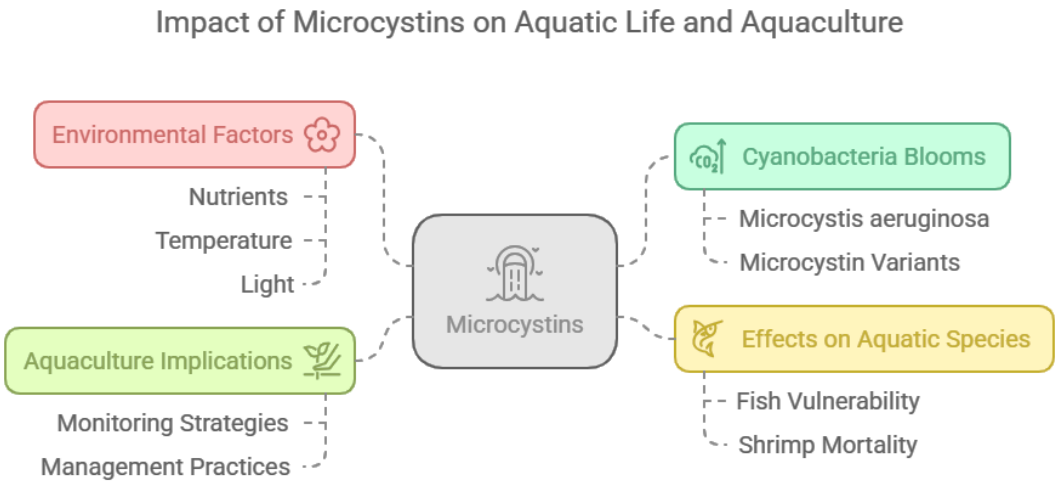
ABSTRACT

The occurrence of harmful blue-green algae blooms is becoming more frequent worldwide. In recent years, the incidence has increased globally in frequency, severity, and duration. Many cyanobacteria species produce a group of toxins known as microcystins. The species most commonly associated with microcystin production is *Microcystis aeruginosa.* Succession of toxic cyanobacterial species and fluctuation in biomass, which is influenced by seasonal changes in various environmental factors including nutrients, grazing, light and temperature, is believed to affect the concentration of microcystin in the field. More than 200 Microcystin variants have been identified in cyanobacteria blooms and cultures, among which microcystin-LR, RR, and YR are the most common. Microcystins can cause liver damage that can lead to death in dogs and livestock. No known deaths have been reported in humans from the ingestion of microcystins. Regardless of species, the mechanism of action is the same, the inhibition of protein phosphatase which causes primarily liver damage, but also affects other organs. Cyanotoxins cause serious poisoning in aquatic species and humans. Ingestion of Microcystins 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. Microcystin has great toxic effect on shrimps. High dose of *M. aeruginosa* cells would exert great mortality in shrimps, and low dose depressed the immunity of shrimp, including antioxidant, detoxification and antimicrobial activity. Hence, a comprehensive review on impact of microcystins, water quality dynamics and practical management of aquaculture ponds is very important and provide tangible benefits to fish and shrimp producers. In order to

minimize the unnecessary losses in the aquaculture sector, an effective approach for aquaculture monitoring must be developed.

Keywords: *Microcystis aeruginosa,* Microcystins, Bloom, Toxicity, Fish, Shrimp.



INTRODUCTION

Cyanobacteria, also known as blue-green algae, are a family of single-celled algae that proliferate in water bodies such as ponds, lakes, reservoirs, and slow-moving streams when the water is warm and nutrients are available. Studies evaluating the diversity and dynamics of blue-green algae have recently been conducted in Japan (Mitsuhiro *et al.*, 2007), Finland (Vaitomaa *et al.*, 2003), Germany (Kurmayer *et al.*, 2003), Canada and the United States (Rinta- Kanto & Wilhelm, 2006; Gobler *et al.*, 2007). Currently, more than 65 countries worldwide including Thailand, Vietnam, Philippine, Singapore and Malaysia have recorded the detection of toxic cyanobacteria in the water environment (Sinang *et al.*, 2015; Mohamad *et al.*, 2016).

Blue-green algae blooms can cause severe water quality deterioration including scum formation, toxin production, hypoxia, foul odours and tastes (Paerl & Huisman, 2009: Guzman-Guillen *et al.*, 2013). Sometimes they grow to large populations known as 'blooms' which are mostly harmful due to the fact that certain species are capable of producing toxins (Qiao *et al.*, 2013). The term Harmful Algal Bloom (HAB) is used to describe a proliferation of algae or phytoplankton. HABs in the aquaculture industry can cause serious economic losses. A preliminary study on the effect of the bloom to the United States economy reported that the country lost more than USD 40 million per year and at least USD 1 billion per decade (Landsberg, 2002). Algal toxins can cause problems in the freshwater aquaculture of both vertebrates (fish) and invertebrates (shellfish). The production of cyanotoxins in blue-green algae is the primary concern because it can pose lethal and sub-lethal effects in both humans, animals, and fishes (Landsberg, 2002; Sangolkar *et al.*, 2009; Chen *et al.*, 2014). Toxic blue-green algae poisonings have been reported in animals such as birds, cattle, and sheep (Carmichael *et al.*, 2001) and have caused over 350 cases of suspected or confirmed poisonings or deaths in the U.S. between the 1920s and 2012 (Backer *et al.*, 2013).

Algal toxins are organic molecules produced by a variety of algae in marine, brackish and fresh waters, as well as on wet soils (Falconer, 1993). They are a problem in aquaculture when they are produced in sufficient quantities, with sufficient potency, to kill cultured organisms, decrease feeding and growth rates, cause food safety issues, or adversely affect the quality of the product (Shumway, 1990).

Microcystins are among the most common and dangerous cyanotoxins (Singh *et al.*, 2012), produced by some cyanobacteria genera, including *Microcystis*, *Anabaena* and *Planktothrix* (DeFigueiredo *et al.*, 2004). The species most commonly associated with microcystin production is *Microcystis aeruginosa* [Dai *et al.*, 2008]. Microcystin enters the fish body through gills, diet and food chain (Poste, 2011; Schmidt, 2013), destroys the liver tissues and causes fish death (Falconer, 2008). Besides, this cyanotoxin also can accumulate in fish/shrimp tissues and pose a health risk to the human when consumed (Peng, 2010). In addition, some cyanobacteria are also capable to synthesise two highly odorous compounds called geosmin and 2- methylisoborneal (MIB) that can cause earthy musty taste on fish (Wnorowski, 1992; Tucker, 2000).

In this review, an emphasis is placed on the effects of microcystins in aquaculture especially fish and shrimps.

2. Role of cyanobacteria algal blooms in the toxicity of aquaculture

The production of algal toxins is normally associated with algal blooms or the rapid growth and exceptionally dense accumulation of algae. Severe blooms of even non-toxic algae can spell disaster for cultured animals, because blooms deplete the oxygen in the shallow waters of many aquaculture systems. The number of HABs around the world is increasing (Shumway, 1990; Sunda *et al.*, 2006), especially in the U.S. where almost every coastal state is now threatened, in some cases by more than one species of harmful algae. Scientists are unsure why this trend is occurring. The causes may be natural (species dispersal) or human related (nutrient enrichment, climate change, and/or transport of algae in ship ballast water) (Johnk, *et al.* 2008; Sunda *et al.*, 2006). Cyanobacteria can colonize and rapidly grow to great masses in aquaculture ponds. Factors that affect their growth are nutrient status, salinity or ionic strength, light conditions, turbulence and mixing, temperature and herbivory (Sunda *et al.*, 2006). In aquaculture situations, eukaryotic algae (green, diatoms, etc.) can often grow faster than cyanobacteria. However, cyanobacteria can out-compete algae for nutrients, thrive with low dissolved oxygen, and photosynthesize more efficiently at low light levels. Cyanobacteria are less affected by turbidity, high concentrations of ammonia and warm temperatures. They can seize the advantage in eutrophic aquaculture situations.

The effects of algal blooms vary widely. Some algae are toxic only at very high densities, while others can be toxic at very low densities (a few cells per liter). Some blooms discolor the water while others are almost undetectable with casual observation (Shumway, 1990). HABs can affect public health and ecosystems when filter-feeding shellfish (clams, mussels, oysters, scallops) feed on toxic phytoplankton and accumulate harmful toxins that are passed up the food chain; fish, shellfish, birds and even mammals are killed by eating organisms that have consumed algal toxins; light cannot penetrate the water, thus changing the function and structure of the aquatic ecosystem; discoloration makes water aesthetically unpleasant; the decaying biomass of a bloom depletes dissolved oxygen (especially critical in aquaculture); or blooms kill other algae important in the food web (Codd *et al.*, 2005b; Landsburg, 2002). Cyanobacteria can affect the production of zooplankton and consequently the production of fish. They also produce allelochemicals that can inhibit competing algae and invertebrate grazers (Gross, 2003; Berry *et al.*, 2008). Cyanobacteria can rapidly overtake an aquaculture pond and contribute to unstable conditions. Cyanobacteria blooms can decrease fish production and kill fish because of oxygen depletion. Cyanobacteria can also cause off-flavor and objectionable odor in fish.

Microcystis sp. is a very tolerant algae growing in high salinity water (Soedarsono *et al.*, 2013). The status of pond waters that have optimum salinity levels and optimal temperature conditions will allow plankton to grow optimally (Ariadi and Mujtahidah, 2022). *Microcystis* sp. in pond waters has a high level of dominance because of its cosmopolitan nature. In trophic waters several types of plankton such as Oscillatoria sp., Microcystis sp. and Anabaena sp. prone to periodic blooming (Aliviyanti *et al.*, 2017). Based on the results analysis of the diversity index and uniformity index, it was shown that the waters in the research ponds were still quite good. This status correlates closely with the water quality profile and plankton abundance level in pond waters. The dominance and abundance of plankton will follow by water quality dynamics in the waters (McQuatters Gollop *et al.*, 2019). The plankton dominance and abundance level is also greatly influenced by aquatic

productivity rate (Xiong *et al.*, 2020). *Microcystis* sp. is a plankton that tends to be adaptive with water condition changes (Huang *et al.*, 2014). Then indirectly, *Microcystis* sp. abundance will greatly affect the grazing process and the food chain in the pond ecosystem.

3 Types of toxins produced by Cyanobacteria

Cyanobacterial toxins can be classified several ways. They may be classified according to their chemical structures as cyclic peptides (microcystin and nodularin), alkaloids (anatoxin-a, anatoxin-a(s), saxitoxin, cylindrospermopsin, aplysiatoxins, lyngbyatoxin-a) and lipopolysaccharides. However, cyanotoxins are more commonly discussed in terms of their toxicity to animals. While there are several dermatotoxins (e.g., lyngbyatoxin and aplysiatoxins), which are produced primarily by benthic cyanobacteria, most cyanotoxins are either neurotoxins or hepatotoxins (Codd *et al.*, 2005a).

3.1 Neurotoxins

Neurotoxins are organic molecules that can attack the nervous systems of vertebrates and invertebrates. Three primary types of neurotoxins have been identified: 1) anatoxin-a, an alkaloid, inhibits transmissions at the neuromuscular junction by molecular mimicry of the neurotransmitter acetylcholine (blocks post-synaptic depolarization); 2) anatoxin-a(s) blocks acetylcholinesterase (similar to organophosphate pesticides); 3) saxitoxins are carbamate alkaloids that act like carbamate pesticides by blocking sodium channels. Neurotoxins are produced by several genera of cyanobacteria including *Anabaena, Aphanizomenon, Microcystis, Planktothrix, Raphidiopsis, Arthrospira, Cylindrospermum, Phormidium and Oscillatoria*. Neurotoxins produced by *Anabaena* spp., *Oscillatoria* spp. and *Aphanizomenon flos-aquae* blooms have been responsible for animal poisonings around the world (Carmichael, 1997; Briand *et al.*, 2003). Neurotoxins usually have acute effects in vertebrates, with rapid paralysis of the peripheral skeletal and respiratory muscles. Other symptoms include loss of coordination, twitching, irregular gill movement, tremors, altered swimming and convulsions before death by respiratory arrest.

3.2 Hepatotoxins

Hepatotoxins are produced by many genera of cyanobacteria and have been implicated in the deaths of fish, birds, wild animals, livestock and humans around the world (Briand *et al.*, 2003; Carmichael, 1997). The cyclic heptapeptides, or microcystins, inhibit eukaryotic protein phosphatases type 1 and type 2A, resulting in excessive phosphorylation of cytoskeletal elements and ultimately leading to liver failure (Codd, 2005b). These toxins target the liver by binding the organic anion transport system in hepatocyte cell membranes.

3.3 Microcystins

Microcystins are the largest group of cyanotoxins, with more than 70 structural variants (Malbrouk and Kestemont, 2006). Microcystin is the only cyanotoxin for which the biosynthetic pathway and gene cluster have been identified (Huisman *et al.*, 2005).

4. Environmental effects on toxin production

The effects of environmental factors on toxin production are much studied and widely disputed (Codd, 2000; Codd *et al.*, 2005a). Blooms in the same body of water can be toxic or non-toxic from one year to the next. A different strain composition (i.e., toxic versus non-toxic), which cannot be distinguished microscopically if belonging to the same species, is a common explanation for this occurrence. However, some species are known to produce high or low levels of toxicity under different laboratory conditions. The stimulus for toxin production in such species is not known.

Environmental parameters such as light intensity, temperature, nutrients and trace metals have been mimicked under laboratory conditions and their effect on cyanotoxin production investigated. Studies on light intensity are not definitive, but it is known that intense light increases the cellular uptake of iron, which may be responsible for more toxin production. However, low concentrations of iron lead to higher microcystin concentrations (Huisman *et al.*, 2005). Nutrients such as nitrogen and phosphorus are essential for cyanobacterial growth. Phosphorus is usually the limiting factor in ponds, so small increases in this nutrient may influence toxin production simply as a result of increasing algal growth. Generally, decreased amounts of microcystin (produced by *Anabaena, Microcystis* and *Oscillatoria*) and anatoxin-a (produced by *Aphanizomenon*) have been reported under the lowest phosphorus concentrations tested (Watanabe *et al.*, 1995).

5. Effects of Microcystins on Fish

Microcystins are toxic to fish at concentrations as low as a few micrograms per liter (µg/L) or possibly even fractional µg/L (Malbrouck and Kestemont, 2006). Considering that microcystins has been measured in concentrations up to 25,000 µg/L in waters with cyanobacterial blooms (WHO, 1999), it is not surprising that potential impacts on fish are receiving increased attention. Fish typically either ingest cyanobacteria or prey that have fed on cyanobacteria (Fischer *et al.*, 2000). To a lesser extent, they can absorb the toxins directly from the water (Phillips *et al.*, 1985). As with mammals, microcystins are actively taken up by the liver in fish where they disrupt normal cellular activity by inhibiting protein phosphatases (Boaru *et al.*, 2006). Inhibition of these enzymes in fish can ultimately result in widespread cellular death and loss of liver structure (Malbrouck and Kestemont, 2006). Protein phosphatases are particularly important during fish embryonic development because they regulate critical developmental processes (Gotz *et al.*, 2000). Due to the limited capacity of fish to detoxify microcystins, they easily succumb to the toxic effects of increased microcystin concentrations (Jayaraj *et al.*, 2006).

Field observations of impacts on fish coincide when blooms are abundant. However, aquatic ecosystems are complex and it can be very difficult to discern the exact cause of the impacts. For example, fish kills following a bloom could be caused by microcystin released from dying cells, but are more likely due to the decreased oxygen and pH levels caused by the decaying bloom (Ibelings and Havens, 2007). Consequently, the toxic effects of microcystins in fish have been studied experimentally using several different fish species and exposure routes.

Like small mammals, most studies on the immediate (acute) lethality of microcystins in fish have utilized IP injections of extracted microcystins to determine the dose that is lethal to half the test population (LD50). Reported LD50 values of microcystins in fish range from 20 to 1500 µg microcystin LR/kg body weight (Malbrouck and Kestemont, 2006). The large range of values could reflect variation between fish species, or differences in toxin extraction, purification, or measurement methods. As a group, mature fish are less sensitive to acute microcystin toxicity than mammals. Data from these acute studies are useful to make general comparisons between species. However, IP injections of microcystins are not analogous to field exposures since the toxin is absorbed faster and metabolized differently when administered into the abdominal cavity (as with the IP route) as compared to oral administration (Ibelings and Havens, 2007). For example, IP injection of 50 µg MC/kg in carp killed all test fish while an oral dose of 250 µg MC/kg in similar carp resulted in no lethality and minimal liver damage (Carbis *et al.*, 1996). No oral LD50 values were found for microcystins in fish. When developing loach were immersed in solutions of isolated MC-LR (over multiple days), the median lethal concentrations (LC50) were 164.3 µg/L in embryos and 593.3 µg/L in small hatched juveniles.

In nature, fishes are most likely subject to sublethal impacts resulting from exposure to microcystins over days or weeks. Several studies have observed severe liver damage in fish following oral administration of microcystins, usually in the form of freeze-dried cyanobacterial cells. The sublethal microcystin concentrations shown below are commonly found in food items of fish during blooms. For example, a diet containing greater than 130 to 2,500 µg MC/kg diet wet weight (ww) for two or more weeks may result in sublethal effects in carp (based on 5 kg fish consuming 2% body weight/day). Microcystin concentrations in cyanobacterial blooms commonly reach 20,000 µg MC/kg algae and have been reported as high as 129,000 µg MC/kg algae [ww, converted from dry weight, 1].

Mussels, snails and zooplankton collected from areas with blooms have contained microcystin concentrations up to 2,500, 2,900 and 13,700 µg MC/kg body weight (bw), respectively [ww, converted from dw,]. These estimates indicate that fish exposed to typical microcystin producing blooms may be experiencing sublethal toxic effects (i.e., liver damage). This is in agreement with Carbis *et al.* (1996), where the majority of common carp sampled from a lake with 22,000 – 40,000 µg MC-LR/kg bloom material (ww, converted from dry) exhibited widespread liver damage consistent with microcystin toxicity.

Additional sublethal effects of microcystins have been described in fish including effects on kidney, gill, growth, immune status and cardiac function (Best *et al.*, 2001). Developing fish appear to be very sensitive to chronic exposures to microcystins. In general,

exposure of embryos and larvae to environmentally relevant concentrations of microcystins has resulted in oxidative stress, reduced growth, developmental defects, and lethality, as well as the lack of significant impacts. Fish embryos can take up significant levels of dissolved microcystins from the surrounding water (Wiegand *et al.*, 1999). Exposures as low as 0.25 µg/L resulted in oxidative stress to zebrafish embryos (Pietsch *et al.*, 2001). Immersion of embryos and larvae in solutions of 0.5 - 50 µg MC/L for up to 30 days resulted in interferences with hatching, developmental defects, liver damage and/or increased mortality in several species including chub, carp, loach, trout and zebrafish.

Reported concentrations of microcystins in water (not cells) during blooms range from trace amounts to 1,800 µg/L [median was 2 ug/L, 1]. Maternal transport of microcystins from the female to developing eggs may be an additional exposure route to developing fish. Although this route has not been demonstrated for microcystins, experiments indicate that developing fish embryos would be more sensitive to maternal transport of microcystins compared to uptake from water. Microinjection of minute amounts of microcystin into medaka embryos significantly reduced survival rates (Jacquet *et al.*, 2004). Similar experiments in zebrafish resulted in significant disruption of development and reduced survival (Wang *et al.*, 2005). These studies reveal potential impacts of microcystin maternal transport. The precise mechanisms of exposure and effects in fish embryos have not been fully determined.

Extracts from cyanobacteria, with or without microcystins present, disrupt development and growth of fish Most studies have utilized purified cyanotoxins to isolate specific toxicity thresholds and effects. However, most natural blooms contain more than one cyanobacteria species, many of which produce more than one toxin [reviewed by 1]. Typically, crude extracts of cyanobacteria elicit more severe effects in fish embryos and larvae than purified microcystins. Observed effects of exposure to crude extracts include increased oxidative stress, liver damage, gross malformations, osmoregulatory imbalance, and decreased survival [Pietsch *et al.*, 2001, Palikova 2003 and 2007).

Symptoms of poisoning in fish include flared gills because of difficulty breathing and weakness or inability to swim. Channel catfish, Ictalurus punctatus, can become intoxicated at ~50 to 75 µg microcystin/L (Zimba *et al.*, 2001). All fish may be killed within 24 hours of exposure. At necropsy, severe lesions may be observed in liver tissues. One potent hepatotoxin, cylindrospermopsin, is produced by Cylindrospermopsis raciborskii, a relatively small cyanobacterium. Cylindrospermopsin is an alkaloid that suppresses glutathione and protein synthesis. C. raciborskii has been in the South and Southeast for decades and is becoming more widespread. Mammals (such as humans) are relatively sensitive to cylindrospermopsin and may be affected when they eat fish that have been exposed to the toxin. A study reporting the bioaccumulation of cylindrospermopsin in muscle tissue of the redclaw crayfish (Cherax quadricarinatus) and visceral tissues of rainbow fish (*Oncorhynchus mykiss*) shows that exposure could occur from farm-raised freshwater aquatic foods. Fish are generally more tolerant of algal toxins than mammals and tend to accumulate them over time (Carson, 2000).

6. Effect of Microcystins on Shrimp culture

In Mexico (Maria, *et al.*, 2016), the effects of toxic cyanobacteria from Chapultepec Lake, Mexico were investigated by performing, short-term acute toxicity tests on white shrimp (L. vannamei) postlarvae in low-salinity water, using a natural cyanobacterial bloom. The main objective was to evaluate the possible effects of Microcystis aeruginosa on the invertebrate L. vannamei to study the effect of toxins, intact cells from a natural cyanobacterial bloom were taken, and the colonies were disaggregated by ultrasound for 3 minutes. Histological analysis of exposed shrimps revealed lesion development in antennal gland, gills, hepatopancreas, lymphoid organ, muscle and dorsal cecum in Litopenaeus vannamei postlarvae, such kind of lesions may interfere with food absorption, respiration, excretion, locomotion and mortality. Hence, Microcystis aeruginosa blooms in shrimp ponds may jeopardize the culture by mortality and slowing shrimp growth rate. The acute test with postlarvae of the white shrimp were effective in indicating the toxicity of cyanobacteria and in prognosticating the toxic effects of cyanobacterial blooms, at least on some usual components of the aquatic community, such crustaceans and micro crustaceans.

In Malaysis, (Lim Mui Hua, 2019), a total of 17 shrimp farms in Sarawak were assessed for the abundance of blue-green algae (cyanobacteria) and the levels of

microcystin in the tissue of shrimps using enzyme-linked immunosorbent assay (ELISA). There was a high cell count of Microcystis sp. at 6.77 x 10^8 cells/ L in Muara Tebas, *Anabaena* sp. at 4.99 x 10^7 cells / L in Telaga Air and *Pseudanabaena* sp. at 1.69 x 10^8 cells/ L in Kuala Baram. Microcystin was detected in most of the shrimp samples collected from the 17 farms in Sarawak throughout the study. The highest level of microcystin was 0.448 ppb, which was detected in Selabat whereas a value below 0.15 ppb was detected in Bandar Baru Semariang, Santubong and Oya. This study demonstrated that microcystin was detected in aquaculture samples collected from shrimp farms in Sarawak.

In China (Yu zu *et al.*, 2022), *Microcystis aeruginosa* is a common kind of harmful bloom algae, which was also frequently found as a dominant microalgae specie in shrimp breeding ponds. And it was found that blooms always induced massive death of shrimp, but the toxic effects of M. aeruginosa on Litopenaeus vannamei are still not completely understood. When the toxicity of M. aeruginosa cells to L. vannamei was examined, and the toxic components in the cells were analyzed through high-pressure liquid chromatography (HLPC). In addition, the immune response of shrimp to the microalgal extract was assessed by measuring the activity of immune-related enzymes, as well as the transcription of the relevant genes. Overall, both M. aeruginosa cells and the algal extract resulted in a 100% mortality rate in shrimp, whereas the cell-free culture medium was ineffective. And HPLC analysis results revealed the presence of microcystin-LR (MC-LR) at a concentration of 190.40 mg/kg of cells.

7. Management of Toxins

7.1 Monitoring and diagnosing the problem:

While not all blooms of toxin-producing cyanobacteria result in toxin production, most do. Once a bloom is observed, the onset of toxicity will be rapid (hours to a day or two). To confirm the problem, a diagnostician will need fresh samples (unpreserved) of the water containing the suspected cyanobacteria (Rottmann *et al.*, 1992). A sample of both sick and dead fish will also be needed, along with information on fish behavior and any other symptoms observed. Young fish are generally more sensitive than older fish. The diagnostician may look for lesions on fish livers, although this is inconclusive as the sole method of diagnosis (Zimba *et al.*, 2001).

7.2 Chemical and biological control of Microcystis (Kumar and Sinha, 2014)

Most of the time, managing a pond specifically to prevent toxic blue-green algae blooms is not justified, and the treatments themselves are risky. An algicide should not be applied without considering the size of the affected pond, the number and type of fish at risk, the age and condition of the fish, the sensitivity of the cyanobacterium to treatment, and the cost of the treatment. Non-chemical treatments include 1) physical mixing and aeration, 2) increasing flow rate or flushing to decrease hydraulic retention time, and 3) decreasing or altering nutrient content and composition.

Bloom like situation was recorded during summer and was observed only when its population density was > 2.5x10^4 cells/cm^3 . *Ochromonas danica*, a golden brown Chrysophytean alga engulfs and digests *Microcystis aeruginosa* colonies, when water sample was examined microscopically. The population density of *M. aeruginosa* in BRL-III medium inoculated with different concentration of culture suspension of *Ochromonas danica* was studied. In vitro results related to biological control indicated that a population density of 9.9 x 10^4 cells/cm^3 (1.5 ml) to 16.5 x 10^4 cells/cm^3 (2.5 ml) of *O. danica* caused a rapid decline in the population density of *M. aeruginosa* to almost nil only after 6 or 9 days of incubation.

Investigations related to growth response of toxic strains of *M. aeruginosa* in BRL-III medium supplemented with different concentrations of Copper sulphate, Potassium permaganate, Quinine, Urea, Potassium permanganate, Ammonia, Simazine, Calcium hypochlorite, Ferric alum and Cupricide indicated that CuSO4, KMnO4 and Quinine were more toxic to *M. aeruginosa* in comparison to urea and ammonia. Copper sulphate and potassium permagnate caused a rapid decline in population density of *M. aeruginosa* to almost nil following 15 days of incubation; the same concentration of quinone brings this effect within 6 days. Quinones was more toxic to Microcystis aeruginosa followed by copper sulphate, potassium permagnate, urea and ammonia. Among calcium hypochlorite, ferric alum and cupricide, calcium hypochlorite showed maximum inhibitory effect on the growth of M. aeruginosa.

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