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Review

Microalgal toxin(s): characteristics and importance

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Prokaryotic and eukaryotic microalgae produce a wide array of compounds with biological activities. These include antibiotics, algicides, toxins, pharmaceutically active compounds and plant growth regulators. Toxic microalgae, in this sense, are common only among the cyanobacteria and dinoflagellates. The microalgal toxins is either important as material for useful drugs or one of the great mysteries in the world of biotoxicology. The aquatic poisons have long remained one of the great mysteries in the world of biotoxicology. There is evidence that these toxic organisms are on the increase, perhaps as a result of increased global pollution. The ability of cyanobacterial populations to produce potent toxins and annual examples of associated human and animal health problems have raised the position of cyanobacteria in the priorities for the management and protection of water quality in countries where health problems associated with the toxins have been perceived. The purpose of this review is to discuss the present understanding of microalgal toxins from microalgae in a manner that will stimulate interdisciplinary research with these microorganisms.

Key words: Toxin, cyanobacteria, microalgae, dinoflagellate.

INTRODUCTION

Recently, microalgae have become targets for screening programmers in search of novel compounds of potential medicinal valve. These secrete vitamins, amino acids, fatty acids, siderophores, simple carbon hydrates and other nutrilites that are essential or support growth of other microbes. When the processes with this are better understood, microalgae might become economic sources of new drugs and other specialty chemicals because production can be optimized in controlled culture (Metting and Pyne, 1986). Detrimental and beneficial properties of prokaryotic and eukaryotic microalgae may be qualifications giving them possibilities for their biotechnological utilization (Skulberg, 2000).

Numerous compounds have been isolated from prokaryotic and eukaryotic microalgae, and may have been tested for different types of bioactivity with positive effects.

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However, only a small fraction has yet reached the store shellfish. The future role of microalgal compounds in drug discovery is especially in the priority areas for development of new medicines, namely to fight viral infections and cancer, and to combat infections from antibiotic resistant bacteria and fungi.

Extensive growth of prokaryotic and eukaryotic microalgae can create considerable problems, including water quality deterioration and health hazards. The practical problems involved for water supply, fisheries, recreation etc. are manifold and intricate. Consequently, the negative characteristics of microalgae have usually gained primary research attention, as well as the practical means of how to control their growth when they are undesirable. However, the same specific properties making the relevant microalgae of general harmful significance may be just the qualifications that give them possibilities for their positive economic utilization (Knutsen and Hansen, 1997).

Microalgae compose the aquatic phytoplankton and are common inhabitants of nearly all terrestrial and sub-terial surfaces, including extreme environments in hot and cold deserts. Interest in pharmaceuticals from microalgae has

Table 1. Cyanobacterial toxins and general features (Metting and Pyne, 1986; Chorus and Bartram, 1999).

Cyanobacterium	Toxin(s)	Structure	Primary target organ in mammals
Microcystis aeruginosa	Microcystin	Cyclic peptide	Liver
	Microcystis-type-c	Peptide	Liver
	2 Microcystin-like-toxins	Peptides	Liver
	Microcystin-like	Peptide	Liver
Aphanizomenon flos-aquae	Aphantoxins	Alkaloids	
	Neosaxitoxin	Alkaloids	Nerve axons
	Saxitoxin	Alkaloids	Nerve axons
Anabaena flos-aquae	Anatoxin-a	Alkaloids	Nerve synapse
	Anatoxin-b	?	Nerve synapse
	Anatoxin-c	?	Nerve synapse
	Anatoxin-c	?	Nerve synapse
Schizothrix calcicola	Aplysiatoxins	Alkyl phenols	Skin
Lyngbya gracilis	Debromoaplysiatoxin	Alkyl phenols	Skin, gastrointestinal tract
L. majuscule	Debromoaplysiatoxin	Alkyl phenols	Skin, gastrointestinal tract
	Lygbyatoxin	?	Skin, gastrointestinal tract
Oscillatoria nigroviridis	Aplysiatoxin	Alkyl phenols	Skin
Calothrix crustacean	Aplysiatoxin	Alkyl phenols	Skin
Nostoc muscorum	Aplysiatoxin	Alkyl phenols	Skin
S. muscorum	Aplysiatoxin	Alkyl phenols	Skin
Nodularia	Nodularin	Peptides	Liver
Cylindrospermopsis	Cylindrospermopsins	Alkaloids	Liver
All	Lipopolysaccharides (LPS)	Alkaloids	Potential irritant: affect any exposed tissue

benefited from the resurgent interest in ethnobotany. Also stimulating were discoveries by ecologists in the 1930s and 1940s that suggested and then demonstrated the production of antibiotic and autotoxic substances from microalgae (Metting and Pyne, 1986). These are a biochemical diverse assemblage of microorganisms amenable to fermentation and mass culture. Including the cyanobacteria and nearly a dozen eukaryotic classes, microalgae produce a wide array of compounds with biological activity. These include antibiotics, algicides, toxins, pharmaceutically active compounds and plant growth regulators (Codd, 2000).

The purpose of this article is to discuss the present understanding of microalgal toxins from microalgae in a manner that will stimulate interdisciplinary research with these microorganisms.

MICROALGAL TOXINS

The use of the term toxin is restricted to substances poisonous to animals in addition to or exclusive of antimicrobial properties. Toxic microalgae, in this sense, are common only among the cyanobacteria and

The cyanotoxins are a diverse group of natural toxins, both from the chemical and the toxicological points of view. In spite of their aquatic origin, most of the cyanotoxins that have been identified to date appear to be more hazardous to terrestrial mammals than to aquatic biota. Cyanotoxins fall into three broad groups of chemical structure: cyclic peptides, alkaloids and lipopolysaccharides (LPS) (Chorus and Bartram, 1999). The specific toxic substances within these broad groups that have been identified to date from different genera of cyanobacteria, together with their primary target organs in humans, are given Table 1.

Hepatotoxic cyclic peptides

Cyanobacterial hepatotoxins cause death by liver hemorrhage within a few hours of the acute doses (Chorus and Bartram, 1999). These toxins, which target the liver due to specific binding of the organic anion transport system in hepatocyte cell membranes, have been implicated in the deaths of birds, wild animals, agricultural livestock and fish, and have been responsible for human illness and death, reported from India, China, Australia and Brazil (Kaebernick and Neilan, 2001). Microcystins have been characterized from planktonic *Anabaena, Microcystis, Oscillatoria, Nostoc* and *Anabaenopsis* species, and from terrestrial *Hapalosiphon*

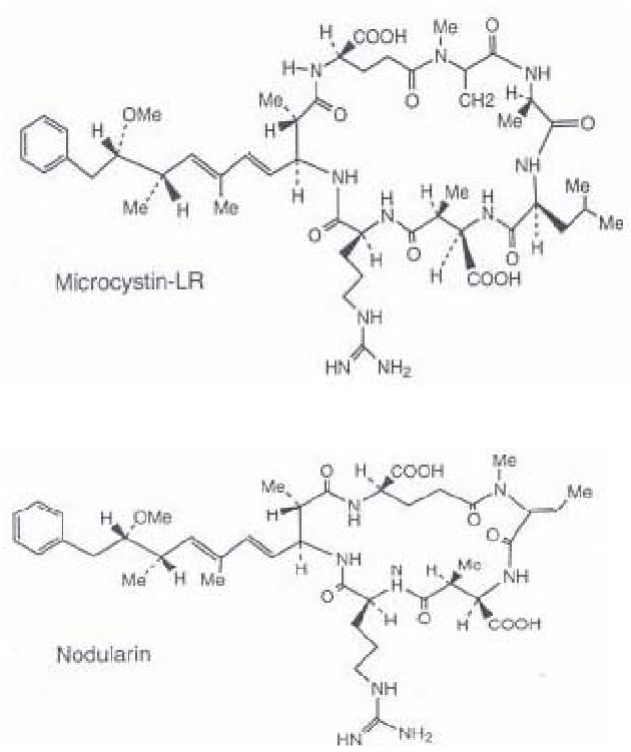

Figure 1. Chemical structure of hepatotoxic cyclic peptides (Kaebernick and Neilan, 2001).

genera. Nodularin has been characterized only from *Nodularia spumigena.*

The cyclic peptides are comparatively large natural products, molecular weight ≈ 800-1,100, although small compared with many other cell oligopeptides and polypeptides (Figure 1). The first chemical structures of cyanobacterial cyclic peptide toxins were identified in the early 1980s and the number of fully characterised toxin variants has greatly increased during the 1990s.

These compounds were first isolated from the cyanobacterium *Microcystis aeruginosa* and therefore the toxins were named microcystins (Carmichael et al., 1988). The microcystins are tumour-promoters. Disruption of liver structure and function occurs with haemorrhage into the liver and death by respiratory arrest. At least 65 microcystin variants and six nodularin variants are known (Codd, 2000).

In one species of brackish water cyanobacterium, an identically acting and structurally very similar, cyclic pentapeptide occurs. It has been named as nodularin after its producer, *Nodularia spumigena*. In the marine sponge, *Theonella swinhoei* , a nodularin analogue called motuporin has been found. It differs from nodularin only by one amino acid. The toxin might be cyanobacterial in origin because the sponge is known to harbour cyanobacterial symbiyonts (Chorus and Bartram, 1999).

Numerous toxic agents induce apoptosis (i.e. programmed cell death), specially in proliferating cells.

This phenomenon can be important in carcinogenesis by retarding tumor growth, which is suggested to result from an imbalance between proliferation and apoptosis. Carcinogenesis experiments (Fujiki et al., 1996) have demonstrated that microcystin-LR, as a protein phosphatase inhibitor, is a potent tumor promoter. In test using aquatic plants, microcytins have caused increases in glutathione sutransferase activities of plants (Pflugmacher et al., 1997; Pflugmacher et al., 2001) and inhibited growth.

Neurotoxic alkaloids

The substances of this group are rapidly acting alkaloids causing death after exposure to vertebrates by paralysis of skeletal muscles and then respiratory muscles, leading to respiratory arrest within a few minutes to a few hours (Skulberg, 1999). Three families of cyanobacterial neurotoxins are known:
anatoxin-a and homoanatoxin-a, which mimic the effect of acetyl choline. Anatoxin-a(s), which is an anticholinesterase saxitoxins, also known as paralytic shellfish poisons (PSPs) in the marine literature, which block nerve cell sodium channels.

Alkaloids, in general, are a broad group on heterocyclic nitrogenous compounds usually of low to moderate molecular weight (<1,000). The non-sulphated alkaloid toxins of freshwater cyanobacteria (anatoxins and saxitoxin) are all neurotoxins. The sulphated PSPs, C-toxins and gonyautoxins are also neurotoxins, but the sulphated alkaloid cylindrospermopsin blocks protein synthesis with a major impact on liver cells. Some marine cyanobacteria also contain alkaloids (lyngbyatoxins, aplysiatoxins) which are dermatoxins (skin irritants), but have also been associated with gastroenteritis and more general symptoms such a fever (Chorus and Bartram, 1999; Kaebernick and Neilan, 2001; Rivasseau et al., 1998).

Several freshwater bloom forming cyanobacterial genera including *Anabaena, Aphanizomenon, Oscillatoria* and *Cylindrospermum* produce the neurotoxin, anatoxin-a, an alkaloid with a high toxicity to animals (Mitrovic et al., 2004).

Anatoxin-a is a commonly encountered toxin that has been found throughout much of Europe, as well as Canada and Japan. The neurotoxin anatoxin-a is a patent post-synaptic depolarizing neuromuscular blocking agent that causes death within minutes to hours in animals (Carmichael and Falconer, 1993). Gorham et al. (1964), isolated anatoxin-a from algae, calling it *Anabaena* “very fast death” factor (Metting and Pyne, 1986). The toxin is an alkaloid with LD_{90} (mouse) of 0.3 mg/kg (4-5 min). Anatoxin-a ($C_{10}H_{15}NO$) (Mw=165) and anatoxin-a(s) ($C.H.N_4O_4P$) (Mw=252) inhibit transmissions at the neuromuscular junction by molecular mimicry of the neurotransmitter acetylcholine and inhibition of acetylcholinesterase activity, respectively. Similar to

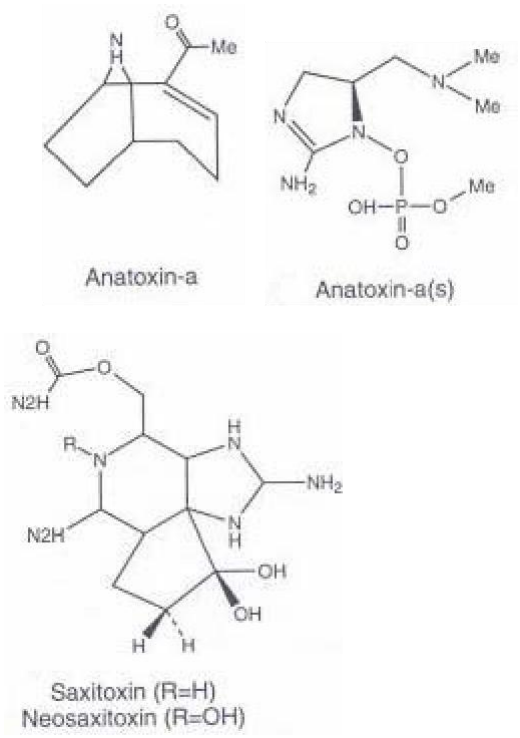

Figure 2. Chemical structure of neurotoxic alkaloids (Kaebernick and Neilan, 2001).

cylindrospermopsin, both are believed to be synthesized via arginine derivatives involving a retro-Claisen condensation (Kaebernick and Neilan, 2001). Anatoxin-a (s), an organophosphate neurotoxin, causes hypersalivation in animals with death due to the inhibition of acetylcholinesterase (Codd, 2000).

Neurotoxins, produced in association with *Anabaena* spp. and *Aphanizomenon flos-aquae* blooms, have been responsible for several animal poisoning around the world. Carmichael and Gerhman (1978) also identified the existence of three other toxic principles from *Anabaena flos-aquae*, distinguishable on the basis of pathogenic symptoms in animals (Metting and Pyne, 1986).

Anatoxin-b is similar in its effects on mice, but not birds. Anatoxin-c appears to be similar to microcystin anatoxin-d is six times as potent as the others and shows anticholinesterase properties that induce symptoms in animals and birds in addition to those for the other anatoxins.

Homoanatoxin-a is a neurotoxin produced by strains of some oscillatorialean blue-greens. The toxin has been isolated, its molecular structure elucidated and its toxicity investigated. This toxin is a low molecular weight bicyclic secondary amine. It has potent cholinergic properties and a high toxicity. Toxicosis in the lethal dose range leads to severe body paralysis, convulsions, and death by respiratory arrest in 2 to 12 minutes (Skulberg, 1999).

Alam et al. (1973) first demonstrated that aphantoxin, a toxin from *Aphanizomenon flos-aquae*, is a mixture of several compounds. One of these toxins is saxitoxin that is more commonly associated with dinoflagellates. Toxic blooms of *Aphanizomenon flos-aquae* in nature are rare. On the other hand, case histories of animal poisoning by *Anabaena flos-aquae* are common (Metting and Pyne, 1986).

The saxitoxins are responsible for paralytic selfish poisoning and are the major toxic components present in numerous dinoflagellate. Chemically, the saxitoxins are a group of heterocyclic guanidines, which have the basic molecular structure shown in Figure 2. The saxitoxins are a complex of more than 21 congers of toxins that have similar molecular structures (Halstead, 2002; Mitrovic et al., 2004).

The paralytic selfish poisons, saxitoxin, neosaxitoxin, decarbamoyl saxitoxin, and gonyautoxins 1,3 and 4, derivatives of saxitoxin, were found in freshwater cyanobacteria *A. flos-aquae*, *Anabaena circinalis, Lyngbya majuscula* , and *Oscillatoria mougeotti* in Portugal (Ferreira et al., 2001). There are a number of derivatives of the saxitoxins known as gonyautoxins, the semantics of which become somewhat complex. The synonymy of saxitoxins and gonyautoxins presented by Shimizu (1988) is helpful in sorting out the chemical terminology (Halstead, 2002).

Saxitoxins (carbamate alkaloid neurotoxins) are well known in a marine context as products of dinoflagellate red tides that accumulate in shellfish to cause paralytic shellfish poisoning when contaminated shellfish are consumed. The same family of neurotoxins can be produced by a range of freshwater cyanobacteria and about 20 saxitoxin variants have been described to date (Codd, 2000; Codd et al., 1999).

The neurotoxins, including the C-toxins and gonyautoxins, involved in paralytic shellfish poisoning (PSP) and conrequently classified as PSPs. These toxins, which inhibit nerve conduction by blocking neuronal sodium channels, are also produced by dinoflagellate species, *Alexandrium* spp*., Gymnodinium catenatum, Pyrodinium bahamense* var*. compressum*, manifesting as toxic “red tide” events (Carmichael, 1994).

Cytotoxic alkaloids

Another cyanatoxin, cylindrospermopsin, has also been found to inhibit seeding growth in the terrestrial plant Synapsis (Vasas et al., 2002). This suggests other caynotoxins may have adverse effects on aquatic plants. Cylindrospermopsin is a cyclic guanidine alkaloid that is hepatotoxic (Figure 3). This toxin is a protein synthesis inhibitor. As with other classes of cyanobacterial toxins, it is likely that several variants of cylindrospermopsin will emerge (Codd, 2000).

The tricyclic alkaloid cylindrospermopsin has been reported to be produced by *Cylindrospermopsis*

Figure 3. Chemical structure of cytotoxic alkaloids (Kaebernick and Neilan, 2001).

raciborskii strains from Australia. This compound has been found to cause severe liver damage in the mouse bioassay with symptoms clearly distinguishable from those of some other cyanobacterial hepatotoxins including microcystins and nodularin. Clylindrospermopsin has been implicated in outbreaks of human sickness and cattle mortality and has been suggested by Humpage et al. (2000) to have carcinogenic activity (Saker et al., 2003).

Dermatotoxic alkaloids

Benthic marine cyanobacteria such as *Lyngbya, Oscillatoria* and *Schizothrix* may produce toxins causing severe dermatitis among swimmers in contact with the filaments. The inflammatory activity of *Lyngbya* is caused by aplysiatoxins and debromoaplysiatoxin which are tumour promoters and protein kinase C activators. *Lyngbya majuscule* has caused dermatitis and severe oral and gastrointestinal inflammation. It was found to contain lyngbyatoxin-a. Debromoaplysiatoxin along with other toxic compounds has also been isolated from other *Oscillatoriaceae* such as *Schizothrix calcicola* and *Oscillatoria nigroviridis* (Chorus and Bartman, 1999).

Lipopolysaccharides

Lipopolysaccharides are generally found in the outer membrane of the cell wall of gram negative bacteria, including cyanobacteria, where they form complexes with proteins and phospholipids and can elicit irritant and allergenic responses in human and animal tissues that come in contact with the compounds (Chorus and Bartman, 1999).

Many cyanobacteria contain lipopolysaccharide endotoxins (LPS) in their outer cell layers. It is thought that cyanobacterial LPS may contribute to waterborne health incidents, although this possibility has not been adequately investigated. Although apparently not as toxic as *Salmonella* LPS, the LPS of cyanobacteria may be of health significance in water bodies due to the ability of cyanobacteria to develop and accumulate to high population levels (Codd, 2000).

Although comparatively poorly studied, cell wall components, particularly LPS endotoxins from cyanobacteria may contribute to human health problems associated with exposure to mass occurrences of cyanobacteria. More studies are needed to evaluate the chemical structures and health risks of cyanobacterial LPS (Chorus and Bartman, 1999).

Other toxic compounds

More than a dozen toxic dinoflagellates have been identified; most are icthyotoxic and may cause different kinds of paralytic shellfish poisoning (PSP) of mammals including humans (Table 2).

Tetrodotoxin: Tetrodotoxin is phylogetically the most widely contributed nonprotein biotoxin in nature. Its toxicity is similar to that of saxitoxin, and its molecule consists of a positively charged guanidine group made up of three nitrogen atoms and a pyrimidine ring (Halstead, 2002). Ogata and colleagues (1987) and Kodama and Ogata (1988) found, in their investigations of the toxicity of the dinoflagellate *Protogonyaulax tamarensis*, that there was a marked fluctuation in their toxicity. *Protogonyaulax* species produce complex mixtures of neurotoxins that vary in their bioactivities (Metting and Pyne, 1986).

Ciguatoxins

The generic term “ciguatoxin” refer to a complex of toxins, some of which are among the most toxic nonprotein poisonous substances known. Ciguatoxin was first isolated by Scheuer and colleagues in 1967, but the molecular structure was determined by Murata and colleagues in 1989 and 1990 (Halstead, 2002). Ciguatoxin is a polyether lipophilic toxin (Figure 4). Gambiertoxin, derived from the dinoflagellate *Gambierdiscus toxicus* , is the precursor of ciguatoxin. *G. toxicus* produces two classes of polyether toxins, the ciguatoxins and maitotoxins (Holmes et al., 1991). Ciguatoxins are lipophilic and are accumulated through the food web. Maitotoxins are similar in structure to ciguatoxins. They are transfused polyether water-soluble toxins. These toxins responsible for ciguatera fish poisoning (Halstead, 2002).

Polytoxin

Maeda and colleagues (1984) obtained palytoxin or a

Table 2. Toxic dinoflagellates (Metting and Pyne, 1986).

Microalga	Toxin(s)	Disease
Protognonyaulax catenalla	Saxitoxin	PSP*
	Neosaxitoxin	
	Gonyautoxins 1-5	
P. acatenella	Saxitoxin/others	PSP
P. tamarensis	Saxitoxin/others	PSP
P. monilata	?	Icthyotoxic
P. polyedra	?	Icthyotoxic
Goniodoma sp.	Goniodomin	Icthyotoxic
Dinophysis fortii	Dinophysitoxin-1	Diarrhoetic SP
Ptychodiscus brevis	2 Neurotoxins/1	Icthyotoxic
Peridinium polonicum	Glenodine	Icthyotoxic
Pyrridinium phoneus	?	PSP
Gymnodinium veneficum	Water-soluble neurotoxin	Icthyotoxic
Noctiluca scintillans	?	Icthyotoxic
Prorocentrum minimum	Venerupin	PSP
Amphidinium spp.	Choline esters	Icthyotoxic
Gambierdiscus toxicus	Ciguatoxin+ 2 others	Ciguatera fish poisoning
Prymnesium parvum	Prymnesin glycolipid	Icthyotoxic

PSP*, paralytic shellfish poisoning

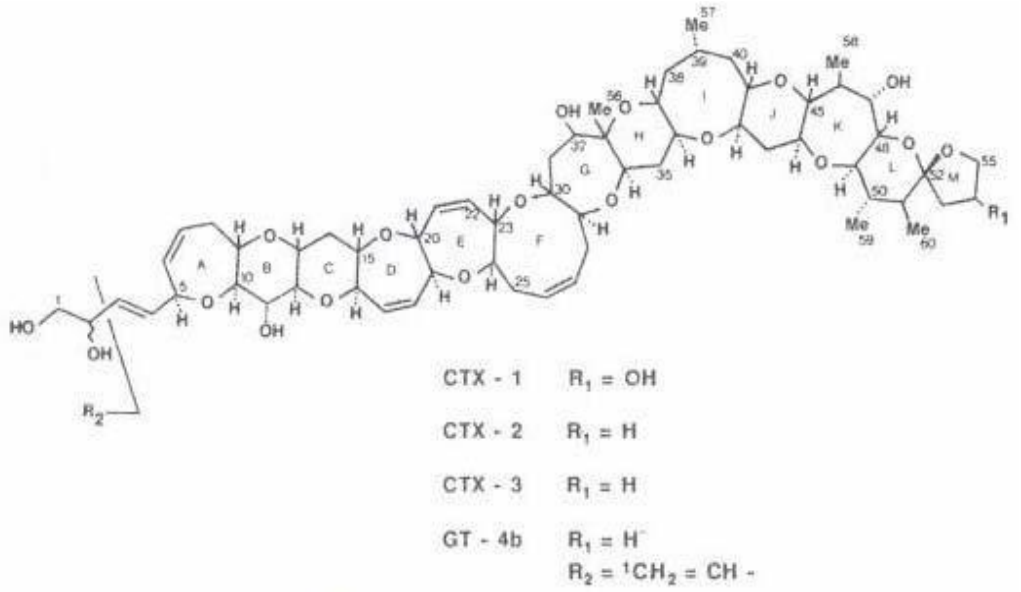

Figure 4. Chemical structure of ciguatoxin and gambiertoxin (Halstead, 2002).

closely related compound from the red alga *Chondria crispus* (Halstead, 2002). The toxic dinoflagellata *Ostreopsis siamensis* has been implicated in clupeotoxism in which the causative poison was found to be palytoxin or one of its analogs (Onuma, 2001). Palytoxin has produced ciguatera-like symptoms in humans that have eaten mackerel in Hawaii.

Icthyotoxin production has been implicated or onstrated for *Amphidinium carteri, A. klebsii* and *A. rhynchocephalum, Gymnodinium veneficum, Goniodoma* sp.,*Peridinium polonicum, Noctiluca scintillans* and *Prorocentrum minitans.* Chemical and toxicological data about these dinoflagellates are meagre. Several groups have isolated a number of toxins from *Ptychodiscus brevis* from Florida red tides. The structures of some P. brevis toxins have been elucidated. *Prymnesium parvum*

is a brackish-water golden-brown phytoflagellate which produces an icthyotoxin called prymnesin (Metting and Pyne, 1986).

Okadaic acid and dinophysistoxins are produced by some marine unicellular algae from the plankton and also benthic microalgae and may accumulate in shellfish. The phycotoxins are involved in a gastrointestinal syndrome called diarrhetic shellfish poisoning (DSP), which occurs in humans after consumption of bivalve molluscs.

DSP toxins fall into three groups according to their carbon skeleton= the okaddic acid (OA) group involving OA and dinophysistoxins (DTXs), the pectenotoxin group (PTXs) including PTX 1-7 and yessotoxin group (YTXs) including yessotoxin and homoyessotoxin. Whereas OA group toxins and PTXs are produced by dinoflagellates belonging to planktonic *Dinophysis* and benthic *Prorocentrum* species, YTXs share a different origin, since they are produced by *Protoceratium reticulatum*.

Some epidemiological studies following outbreaks show that DSP toxin amounts as low as 40 ∝g OA equivalent per body are sufficient to induce diarrhetic syndrome to humans. Moreover, OA and DTX1 are potent inhibitors of protein phosphates, and display tumour promoting activity may also be genotoxic. In addition of hazards for human health, the closure of shellfish forms during toxic blooms causes an economic loss (Fremy et al., 1999).

Environmental Effects on Toxin Production

The stimulus for toxin production in such species is currently unknown. Environmental parameters such as light intensity, temperature, nutrients and trace metals have been mimicked under laboratory conditions and investigated with respect to their effect on microalgal toxins production.

High light intensities increase cellular iron uptake which may ultimately be responsible for higher toxin production. In contrast, low concentration of iron, implicated in slower cell growth, has led to higher microcystin concentration.
Nutrients, such as nitrogen and phosphorus are essential for cyanobacterial growth. Phosphorus is usually the limiting factor in lakes, and hence small changes in this nutrient may influence toxin production merely as a result of influencing growth. Generally, decreased amounts of microcystin (produced by *Anabaena*, *Microcystis* and *Oscillatoria*), anatoxin-a (produced by *Aphanizomenon*) and nodularin (produced by *Nodularia*) have been reported under the lowest phosphorus concentrations tested (Metting and Pyne, 1986; Kaebernick and Neilan, 2001).

Anabaena, Aphanizomenon, Nodularia and *Cylindrospermopsis* strains, all capable of nitrogen-fixation show highest levels on microcystin, anatoxin-a, or nodularin when in a nitrogen-free medium. *Microcystis* and *Oscillatoria* strains show highest levels of toxin at high levels of nitrogen (Sivonen and Jones, 1999). Toxin produced by *Aphanizomenon flos-aquae* stability varied with the temperature and pH of the medium. Toxin production was found to correlate with the age of the culture, temperature and light intensity but not with nitrogen source (Metting and Pyne, 1986).

Temperature effects on saxitoxin production have not been investigated, but their concentration in dinoflagellate *Alexandrium* sp. is increased at low temperatures and phosphorus limitation. Different temperatures can also be correlated with different chemical forms of toxin produced. At temperatures below 25°C, *Anabaena* spp. produces microcystin-LR, instead of microcystin-RR which is preferentially synthesized at higher temperatures.

In many cases, highest toxin concentrations are reported under conditions which are optimal for cell growth. Such correlation was not observed for the production of cylindrospermopsin by *Cylindrospermopsis raciborskii* (Kaebernick and Neilan, 2001).

Toxin production by dinoflagellate *Protogonyaulax tamarensis* has recently been shown to vary with nutrient limitation. A gradual decrease during stationary phase under nitrogen limiting condition was judged due to the ability of the alga to recycle nitrogen available in the toxin. In contrast, toxin production increased and remained drastically elevated in phosphate-limited culture. The toxicity of the chrysomonad *Prynesium parvum* changed with salinity and glycerol content of the growth medium (Metting and Pyne, 1986).

Perspective

The understanding of the phenomena with toxic water blooms and extensive intoxications in relation to ecological, recreational and public health problems needs a new synthesis of the relevant knowledge. Several biotoxins from a variety of microalgae-including blue-greens- are of chemical similar nature. The toxins produce analogous physiology and toxicological effects. An understanding is emerging that suggest a general pattern of chemical and physiological characteristics among the microalgal toxins. This is driving a disciplinary convergence, bringing biologists and ecologists together with specialists working in the realm of public health and with people from a wide range of professions concerned with water quality, conservation of nature and the planning and improvement of the physical environment. The knowledge and scientific confidence of the biological nature and implications of microalgal toxins will exhibit large progress by such an interdisciplinary exploration of the complex life system of microalgae.

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