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Causes, Human Health Impacts and Control of Harmful Algal Blooms: A Comprehensive Review

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Abstract. Harmful algal blooms (HABs) are increasingly attracting attention all over the world. A diverse set of algal species including diatoms, flagellates, chrysophytes and dinoflagellates can cause harmful blooms, and many produce toxins that harm other organisms and human health. Intensive cyanobacterial blooms, in particular, have been associated with high costs for society due to their potential toxicity. Algal blooms can produce different toxins. These toxins present a long-standing threat to human and environmental health. For example, the severe *Karenia* bloom of 2005 in the eastern Gulf of Mexico demonstrates the complexities and magnitude of the challenges of managing algal blooms for both environmental and public health. With increasing events and severe impacts of algal blooms on human health, it becomes necessary to monitor and manage toxic algal blooms. Hence this paper presents a review of causes, impacts and control of algal blooms.

1 Introduction

Harmful algal blooms (HABs) are increasingly attracting attention all over the world. The dynamics of rapid increase or almost equally, decrease of phytoplankton populations is known as a 'bloom'[1]. The term harmful covers those microalgal species, which can cause damage to marine living resources and ecosystems and create a negative impact on human welfare[2]. A harmful algal bloom (HAB) is thus defined as a bloom that has deleterious effects on plants, animals and/or humans[3]. Low nitrogen/phosphorus ratios and increased temperatures are known to be the most critical factors for increasing events of harmful algal blooms[4,5].

Algal blooms can produce different toxins. A diverse set of algal species including diatoms, flagellates, chrysophytes and dinoflagellates can cause harmful blooms, and many produce toxins that harm other organisms and human health. Intensive cyanobacterial blooms, in particular, have been associated with high costs to society due to their potential toxicity, with associated negative effects on recreational waters and on water supplies used for the preparation of drinking water [4]. With increasing events and severe impacts of algal blooms on human health, it becomes necessary to monitor and manage toxic algal blooms. Hence this paper presents a review of causes, impacts and control of algal blooms.

2 Causes of HABs

Unfortunately, the causes of HABs are uncertain to date. There is no conclusive report available for the causes of HABs. However, some of the factors which are thought to be responsible for causing HABs are succinctly described below.

2.1 Coastal Eutrophication

Coastal pollution resulting from various sources like domestic and industrial effluents is one of the most important factors in the development of HABs. On most occasions, eutrophication due to nutrient enrichment results in algal blooms, some of which are toxic to marine organisms and to humans. Recent research suggests that eutrophication and climate change are two processes that may promote the proliferation and expansion of cyanobacterial blooms[6]. Eutrophication or nutrient enrichment can alter

the species composition of an ecosystem[5]. Native biota may be displaced as the environment becomes enriched with nutrients such as nitrates and phosphates[7]. Coastal eutrophication and enhanced offshore nutrient concentrations occurring offshore due to vertical mixing have been linked with the development of large biomass, ultimately leading to anoxia, toxic or harmful impacts on ecosystems, human health or recreation and on fisheries resources. As eutrophication increases nitrogen and phosphorus inputs, the ratio of these nutrients to silicates becomes very high. This favours non-diatom species including several harmful species. There are some cases like that of the Black Sea, where an increase in eutrophication accompanied by a reduction in silicate concentrations has led to the proliferation of flagellates including diatom blooms[8]. Further, it is believed that high concentration of phosphorus, and a low total nitrogen to total phosphorus (TN:TP) TN: TP ratio, are favorable for the production of cyanobacteria blooms. According to the several studies, cyanobacteria usually dominate in lakes with low TN/TP ratio and are rare in lakes with high TN:TP ratios[9,10]. Cyanobacteria dominate in lakes where TN:TP mass ratio is below 29:1[10]. Lukatelich and McComb[11] found that summer blooms of the cyanobacterium *Nodularia* in the Harvey Estuary (Western Australia) were directly related to total riverine phosphorus loadings during the previous winter. Preece *et al.*[12] in their recent research also agree that toxin production by harmful cyanobacteria blooms constitutes a major, worldwide environmental threat to freshwater aquatic resources that is expected to expand in scale and intensity with global climate change. However, they further strongly suggest that increased monitoring and research efforts to understand, react to, and prevent ecological and health problems associated with the growing problem of toxic CyanoHABs in coastal environments are required[12]. They highlight a potential food-borne exposure route to humans by reviewing the growing body of evidence that shows microcystins can accumulate in coastal seafood in order to substantiate their arguments.

Nutrients derived from anthropogenic activities have resulted in the increase in HAB records. In some places, unusual heavy rains have resulted in blooms of *L. polyedrum* due to nutrient rich runoff into the coastal waters e.g. Santa Barbara to south of Mexican border[13]. Rainfall being one of the nutrient resources and in; places like Tolo bay, Hong Kong and the Seto Inland Sea in Japan have resulted in increased red tide episodes with increase in eutrophication[8]. Japanese waters also detected presence of new harmful species e.g. *Heterocapsa circularisquama*[8]. Blooms of dinoflagellate *Pfiesteria* are reported in estuaries of middle and southern Atlantic coasts. Water salinity, pH, nutrients and temperature are the main factors controlling cycles of dinoflagellates, but apart from these, studies in North California have illustrated that they thrive well near sources of organic phosphates released from sewage treatment plants[14]. According to Garcia-Hernandez *et. al.*[15], increased aquaculture activities like that of shrimp farming in Kun Kaak Bay, along coastal Sonora in Mexico, have resulted in a drastic change from blooms of diatoms to dinoflagellates and then Rhaphidophytes, which are again well-known indicators of eutrophication. This may be related to untreated nutrient-rich effluents discharged into Kun Kaak bay.

Similarly, Semeneh *et. al.*[16]studied the nitrogen uptake regime in relation to the biomass and structures of the phytoplankton community in the Atlantic and Indian sectors of the Southern Ocean. Southern Oceans fall in the category of High Nutrient Low Chlorophyll (HNLC) trophic category showing very low utilization of nutrients. In some of these parts, the net removal of silicate during the growth season exceeds that of nitrate and cycling of N as NH_3 leads to uncoupling of Si and N cycles on the surface of the water. As discussed by Semeneh *et. al.*[16], in the Coastal and Continental Shelf Zone (CCSZ) and Open Oceanic Zone (OOZ) of the Indian sector, a bloom can develop only when the rate of biomass increase exceeds the loss rate i.e. grazing rate and sedimentation rate. Once a bloom develops, it persists for a longer period under low growth rate if the loss rate is small. While the interactive effects of future eutrophication and climate change on harmful algal blooms are complex, much of the current knowledge suggests these processes are likely to enhance the magnitude and frequency of these events[6].

2.2 Dust Storms and Metals

Satellite studies carried out by the University of South Florida's College of Marine Science found that giant dust clouds that blow across the Atlantic Ocean from the Sahara Desert towards the Gulf of Mexico are responsible for red tides. Data revealed that these clouds fertilize the water off the West Florida Coast with iron. Cyanobacteria like species of *Trichodesmium* use Fe content from dust particles to fix atmospheric nitrogen in coastal water making the Gulf of Mexico a productive environment for toxic algae thus causing red tides[17]. Further studies showed that there was an increase in Fe content

in surface waters by 300%, thereby increasing the nitrogen content in this water. This change resulted in bloom of the toxic red alga *Karenia brevis* between the regions of Tampa Bay and Fort Myers, Florida[17].

Albert *et al.*[18] documented some common factors responsible for blooms of the cyanobacterium *Lyngbya majuscula* at coastal Queensland in Australia. Runoff during rain events introduces iron, phosphorus and dissolved organics into the coastal zone. A combination of iron and phosphorus is required to increase the photosynthetic activity of *L. majuscule,* thus causing a bloom. Fe plays a very important role in nitrogen acquisition. An increase in Fe may initiate blooms of *Aureococcus anophagefferens* resulting in brown tides. One good example is that of Peconic estuary, New York, USA[19]. Further, studies of Kun Kaak Bay, Mexico, by Garcia-Hernandez *et. al.*[15] have revealed higher concentrations of heavy metals such as Cd and Pb due to untreated nutrient-rich effluents from aquacultural activities, which are frequent along the Sonora coastline. This could be one good example for increase of metals in ocean due to aquacultural activities leading to HABs.

2.3 Climate Change and Increase in Temperature

According to Peperzak[20], climate change is one of the factors for HAB in the Dutch costal zone of the North Sea. Increase in temperature and in precipitation associated with climate change fall into wide ranges, and uncertainties exist in their effect on stratification and North Sea flushing. Dinoflagellates and raphidophytes increase considerably. Species of *D. acuminata, P. minimum, F. japonica and C. antiqua* are observed in this region regularly, indicating an increase in HAB[20]. Temperature variation affects circulation patterns, prolongs stratification periods and causes variations in the physical structure of water column that favours occurrence of HABs[8].

2.4 Upwelling of Oceans

Ascending or vertical motions caused due to oceanic circulation is known as 'Upwelling', which affects the environmental conditions, increases the nutrient content in euphotic zone thereby increasing the productivity of the region[21]. Sharma[21] has discussed various factors inducing upwelling off the south west coast of India. Further, according to Garcia-Hernandez *et. al.*[15], Kun Kaak Bay located in the south of Seri or Comcaac territory and the northwest of the fishing town of Kino bay in northern Sonora is known for its high productivity resulting from seasonal upwelling currents. Here, diatom blooms (e.g. *Stephanopyxis palmeriana*) were common in the past. Similarly, in the Gulf of California, non-toxic red tide algal blooms are common. *Mesodinium rubrum* is a common red tide species associated with upwelling in these areas. Ramana and Reddy[22] have carried out studies to find out the occurrence of the upwelling process along the Dakshina Kannada Coast of India and they report that upwelling was found to occur from March to October along the coast. This could be one of the factors for HABs along the southwest coast of India.

2.5 Ballast Water

Though there is limited research done in terms of the spread of harmful algal species through ballast water, according to Anil *et al.*[23], ballast water is considered to be one of the important vectors for spread of HAB species. For example, Untawale et. al.[24] documented that algal species like *Monostroma oxysporum*, which are native of northeast Atlantic and northwest Pacific, were introduced to the West Coast of India. Anil *et al.*[23], further suggest that India being one of the major maritime countries is susceptible to such type of bioinvasion from the rest of the world oceans and thus requires detailed research in terms of spread of HAB-causing species.

2.6 Unhealthy Coral Reefs

Unhealthy coral reefs play a very important role in the formation of blooms. According to Waldichuk[25], healthy coral reefs are free of external algal growth. Unhealthy conditions or death of corals due to oil pollution or depositions of sediments lead to encrustations of corals by calcareous materials and algae, and may in turn lead to the death of zooplanktons or higher fishes in the food web.

Endolithic algal bloom causes a disease called White Syndrome (WS), consisting of distinct lines between healthy corals and dead ones. These endolithic algae (primarily *Ostreobium* spp.) penetrate the coral tissues of tabular *Acropora* spp., which in turn may affect the corals leading to micro-lessions, and making them susceptible to infiltration by potential pathogens. One good example is that of *Gambierdiscus toxicus,* a benthic dinoflagellate that finds its way on dead corals. *G. toxicus* releases ciguatoxin thus causing Ciguatera Fish Poisoning (CFP). Thus Waldichuk[25] concludes that if a contaminant or a development affects water quality to such an extent that coral reefs are affected than *G.toxicus* is likely to bloom, causing extensive release of ciguatoxin.

3 Impacts of HAB

Harmful cyanobacterial blooms (cHABs) have significant socioeconomic and ecological costs, which impact drinking water, fisheries, agriculture, tourism, real estate, water quality, food web resilience and habitats, and contribute to anoxia and fish kills[26]. High biomass accumulation and degradation of algal blooms may lead to depletion of dissolved oxygen, light attenuation, clogging of fish gills etc. These effects can result in fish kills involving thousands of fish and other marine life, and lead to degradation of the ecosystem[27]. Nevertheless, the most critical impacts of algal blooms are on human health, which will be discussed in detail in this section. Worldwide HABs that produce toxins, which cause acute or chronic health effects in mammals (including humans) and other organisms[26].Toxins produced during harmful algal blooms are some of the most powerful natural toxic substances known. Toxins can directly kill fish or shellfish or other marine life or may accumulate in fish and sea food and lead to human poisoning after ingestion of contaminated sea food[3,27].

3.1 Human Health Impacts

As explained above, toxins produced during algal blooms may not be toxic to fish and marine life. However, they accumulate in fish and mollusks and move up the food chain to have a devastating impact on humans. Marine toxin diseases are categorized into two types based on their trans-vectors. Shellfish carry toxins that lead to paralytic shellfish poisoning, neurotoxic shellfish poisoning, diarrheic shellfish poisoning and amnesic shellfish poisoning. Poisoning through mollusks tends to occur during algal bloom episodes. Fish carry toxins that lead to ciguatera and tetrodotoxin poisoning. Fish poisoning is more localized and associated with parts of specific reefs or fish. Bloom Episodes of dinoflagellate *Pfiesteria* in estuaries of middle and Southern Atlantic coast suggest that anthropogenic stress on aquatic environment has caused fish kills and related health hazards in humans[14]. Species of *Pfiesteria* are also reported to cause lesions in fish.

Additionally, humans can be exposed to toxins that are directly released into water or air. This occurs naturally or as a result of cell disruption caused by turbulence or through human activities like water treatment. This phenomenon frequently occurs in the Gulf of Mexico where residents and beach goers are exposed to toxins through seas spray. Toxins can then be inhaled and lodged in the nose and throat or down into the lungs. Common symptoms associated with this are respiratory irritation, coughing, and other ailments[27]. Table 1 shows incidents of HABs described in the literature.

Table 1. Incidents of HABs described in the literature

Year of Incident	Geographical Location	Causative Organisms	Impacts	Literature
1879	Western Gulf of Mexico	*Karenia brevis*	Human respiratory irritation was first reported.	[28]
1900-1989	North America	PSP (paralytic shellfish poisoning) produced by several species	1399 people intoxicated and 90 deaths	[29]
1901-1992	Norway	PSP outbreak	32 people intoxicated and 2deaths	[29]
1917	Eastern Gulf of Mexico	*Karenia brevis*	Human respiratory irritation was first reported.	[30]

March 1942	Lake Hamana, Japan	*Prorocentrum. minimum*	114 of 324 affected people died after consuming oysters (*Venerupis semidecussata*) and short-necked clams *(Tapes semidecussata)*	[31]
			342 people affected after consumption of clams *Tapes japonica*	[32-35]
March 1943	Lake Hamana, Japan	*Prorocentrum. minimum*	Seventy-one deaths were attributed to ingestion of toxic oysters (*Crassostrea gigas*) from the same region	[31]
1946/ 1994	Portugal	PSP outbreak	132 people intoxicated and 7 deaths	[29]
1947	West coast of Florida	Brevetoxins released by red tide blooms	Respiratory irritation due to inhalation of aerosolized brevetoxins. Seawater containing red tide organisms was sprayed as an aerosol into the nose and throat of volunteers, coughing and burning sensation	[36]
March 1949	Lake Hamana, Japan	*Prorocentrum. minimum*	Ingestion of toxic clams. Symptoms included heavy liver injury (necrosis and fatty degeneration), hemorrhage diathesis with frenzy, unconsciousness and coma, and death occurring 24–48 h after symptoms appeared	[31]
1960	Florida waters	*Karenia brevis*	Human poisonings associated with bivalve shellfish consumption	[37,38]
1968	UK	PSP outbreak	78 people intoxicated	[29]
November 1972	East coast of Florida throughout Palm beach County	*K. brevis*- inhalation of aerosolized brevetoxins	People on beach (swimmers, workers, lifeguards) showed symptoms of acute eye and nose irritation, non-productive cough and respiratory distress.	[39]
1972/ 1997	Chile	PSP outbreak	329 people intoxicated and 23 deaths	[29]
1976	Malaysia	Blooms of *Pyrodinium Bahamense var. compressum*,	Affecting 202 people and causing death of 7 children's.	[40]
1976; 1977; 1987; May 1993	Off Mangalore Southwest coast of India	Blooms of *Noctiluca miliaris*	Not Known	[41]
1976-1977	Along Kerala coast	*Noctiluca Miliaris*	No impacts	[42]
1979-1981	Norway	*Prorocentrum. Minimum*	Mussels consumption caused human poisoning	[43, 44]
1981	Spain	DSP cases	500 cases of DSP were reported	[45]
1983	Mangalore, KT in India	Species not identified	Several hospitalized due to consumption of shell fish	[46]
1984 &1986	France	DSP cases	DSP 2000 cases	[45]

Since 1983, 1998-1999	Philippines Manila Bay and Malampaya Sound	*Pyrodinium bahamense var. compressum)*	Affects the shellfish industry but also endangers the health of the public through consumption of shellfish contaminated with toxins.	[47]
April 1985,	Tadri Estuary in India	Toxic dinoflagellate was not detected however PSP was detected in oysters	Not Known	[48]
1985-1986	Along the coast of Karnataka in India	Species of *D.caudate*-low levels of PSP detected	Caused DSP in infected shellfishes collected from the harvesting areas	[48]
1985	Long Island, USA,	*Prorocentrum. Minimum*	Clams, *Mercenaria mercenaria*, 3 humans affected	[49]
1987	Prince Edward Island, Canada	Diatom *Pseudo-nitzschia* caused ASP	Four Canadians lost their lives when exposed to toxic mussels	[45]
1987	Guatemala	PSP outbreak	187 people intoxicated and 26 deaths	[29]
October 1987	North Carolina Coast.	Outbreak of NSP secondary to red tide of *K.brevis* (then known as *P. brevis*).	48 persons were diagnosed with NSP following consumption of cooked and raw oysters at 20 different meals. Acutely 23 % of cases reported gastrointestinal and 39% neurologic symptoms.	[50]
1988 May-June	Kattegat and Skagerrak area (North Sea)	Blooms of *Chrysochromulina Polylepis*	Caused death of 900 tonnes of fish, including cod, salmon and trout	[8]
1990	Denmark	Mussels exported from Denmark to France	DSP poisoning in over 400 people.	[45]
October and November 1991	Oregon and Washington Coasts	Organism unknown DA (Domoic Acid) found Razor clams (*Siliqua patula Dixon*) and Dungeness crabs (*Cancer magister Dana*)	People became ill after consuming razor clams	[13]
1991 and 1992	Kattegat and Skagerrak area (North Sea)	Blooms of *prymnesiophyte*	Not Known	[8]
1992-1993	New Zealand	*Karenia mikimotoi* along with suspect species in bloom	Outbreak of NSP due to consumption of cockles and green shell mussels; oysters	[51]
1994	Hood Canal of Western Washington.	Blooms of *pseudo-nitzchia spp.* Mainly *P.multiseries, P.australis* and *P. pungens* persisted for more than 6 weeks	Mussels indicated the presence of domoic acid however impacts are not reported	[13]
1995-1996	West coast of Florida	*Karenia brevis*	Eye and respiratory irritation. Six person were hospitalized for illness attributed to red tide exposure	[52]

1996-1997	In estuaries of eastern shore of Maryland	Episodes of dinoflagellate *Pfiesteria*	Anthropogenic stress on aquatic environment have caused fish kills and related health hazards in humans – fatigue, headache, respiratory problems, diarrhea, weight loss, skin irritation and memory difficulties	[14]
1997	Kerala in India	Paralytic shellfish poisoning	Seven deaths, over 500 hospitalized after consuming mussels *Perna indica.*	[46]
(1998–2006)	Northwest Pacific (NWP) (Covering China, Korea, Japan and Russia)	Diatoms, Dinoflagellates, Ciliates and Raphidophytes : *Noctilula scintillans Skeletonema costatum Ceratium furca, Karenia digitata, Prorocentrum dentatum, Karania mikimotoi Mesodinium rubrum,Cochlodinium polykrikoides, Alexandrium tamarense, Alexandrium catenella, Gymnodinium mikimotoi,Heterosigma akashiwo, Gymnodinium catenatum, Exuviaella marina, Leptocylindrus danicus, Karania mikimotoi , Eucampia zoodiacus, Mesodinium rubrum, Skeletonema costatum* etc	Summer algal blooms causing fish mortality, shellfish poisoning, physiological impairment, and numerous ecological and health impacts	[53]
February 1999	Offshore red tide off the Gulf coast of Florida.	*Karenia brevis* red tide events,	Data suggest that people can experience upper and lower respiratory irritation and some inflammatory response after inhaling aerosolized brevetoxins during red tide events.	[54]
2001 and 2006,	Cuba	Not known	Symptoms of intoxication of Ciguatera Fish Poisoning (CFP) after consuming carangid (*Caranx latus).* Commonly called as “jurel” in Cuba. 570 reported cases of fish intoxication, 72% of which were due to CFP	[55]

2001 September - December 2002 same months	Florida waters, USA	*Karenia brevis* red tides exposure period No red tide bloom occurred	People located near the coast reflected higher or more chronic exposure to Florida red tide toxins with increase un rates of respiratory disease admissions to an hospital ER (emergency Room) in Sarasota, FL. Admissions to an ER were as compared to 2001 period were less.	[56]
2002	East Timor	PSP outbreak	1 people intoxicated and 1death	[29]
2002	South of Chile	Blooms of *A.catenella*	Area was closed	[40]
20th December 2002	Minnie bay of Port Blair in India	*Noctiluca scintillans* bloom	Not known	[57]
June 2003	Northumberland Strait (N S)	Not known	Closure in N S due to PSP toxins found in bar clams from southwestern Prince Edward Island.	[58]
2003 January to March	Flordia USA	*Karenia brevis blooms* present along shore.	Beach- goers exposed to brevetoxins	[59]
September 2004	South coast of Thiruvananthapuram in India	*Noctiluca* red tide	Not Known	[60]
16th and 17th September 2004	Kerala/ Malabar coast in India	*holococcolithophore* blooms resulted in massive fish kills *Cochlodinium polykrikoides, Karenia brevis*	A strong stench was reported and around 200 children's mostly below 15 years of age suffered from nausea, chest pain, respiratory problems, and many were even hospitalized. About 200 hospitalized, large number of fish kills	[61] [46]
2005	New England waters	Blooms of *Alexandrium fundyense*	Recorded levels of PSP toxins	[62].
2005	Puget Sound, Washington State	High density blooms of diatoms *Pseudo-nitzschia, and* Domoic acid detained in razor clams	Forced closure of recreational, commercial, and tribal subsistence shellfish harvesting in Puget Sound.	[63]
September 2006 for 3 months	Florida west Coast, US	Annual blooms of toxic din flagellate *Karenia brevis* causes red tide events	Exposure to toxic aerosols induces respiratory irritation; Beach Goers: eye irritation, nasal congestion and dry cough. Lifeguards: respiratory symptoms. Coastal residents had 54% increase in admissions in coastal hospital for respiratory diagnosis (i.e. asthma, pneumonia and bronchitis) during onshore red tide event	[64]
2008	French Atlantic Waters	Outbreaks of *Chrysochromulina* spp.	Not known	[55]

Annual occurrence	Gulf of Mexico	*Karenia brevis* blooms and aerosolized brevetoxins	Respiratory irritation in humans exposed at beach or boats during blooms	[37]
Annual occurrence	US Virgin Islands and French West Indies	Species of dinoflagelletes causing CFP (ciguatera fish poisoning)	3 % of the total population per year affected by CPF	[45, 27]
Not known	Florida waters	*Karenia brevis* ingestion of brevetoxins	Reported brevetoxins in urine from 3 persons who suffered from severe NSP after eating contaminated shellfish from Florida	[52]
2009	Washington State, 2009	*Akashiwo sanguinea*	Affected Bird (scoters) diversity	[65]
2009	Singapore	Dinoflagellate : *Gambierdiscus toxicus, Ostreopsis, Coolia* and *Prorocentrum*	Massive fish kill was observed for the first time in Singapore waters during late December 2009, killing 200,000 farm fish	[66]
2013	Singapore	Dinoflagellates: *Gambierdiscus toxicus, Ostreopsis, Coolia* and *Prorocentrum*	Around 90,000kg of fish were found dead in june 2013 in Lim Chu Kang.	[66]
2014	Singapore	Dinoflagellates: *Gambierdiscus toxicus, Ostreopsis, Coolia and Prorocentrum*	Massive fish kill was observed in waters during early Feb 2014, killing both farm fish and wild fish.	[66]
2014	Lake Erie,	*Microcystis aeruginosa. Aphanizomenon* spp., *An abaena* spp., *Cylindrosper mopsis* spp. and *Planktothrix* spp	Three days tap water was banned at Toledo, Ohio.	[67]
2015	off the Washington and Oregon coast	*Pseudo-nitzschia australis,*	highest levels of domoic acid contamination in the food web ever recorded for many species,	[68]
2015	Singapore	Dinoflagellates: *Gambierdiscus toxicus, Ostreopsis, Coolia and Prorocentrum*	Killed 600 tones of wild and farm fish in Feb 2015	[69]
2016	South west Florida	Red Tide: *Karenia brevis*	Fish kills reported	[70]

The most toxic algal species are recorded among dinoflagellates but diatoms and cyanobacteria also produce some toxic chemicals. Major classes of toxins produced by cyanobacteria include hepatotoxins (microcystins, nodularin), cytotoxin (cylindrospermopsin), neurotoxins (saxitoxins, anatoxins) and endotoxins (lypopolysaccharides). The hepatotoxic and neurotoxic metabolites of cyanobacteria are hazardous to humans and animals and accumulate in the liver and the kidney of the fish[71]. The toxins implicated in human illness are microcystins, cylindrospermopsin, anatoxins, saxitoxins, and methylamino alanine. Among the cyanotoxins, microcystins and cylindrospermopsins are more commonly produced by cyanobacteria.

Some of the crucial toxins produced by diatoms and dinoflagellates and their syndromes are described below:

3.1.1 Amnesic Shellfish Poisoning (ASP)/ Domoic Acid Poisoning (DAP)

1. Causative organism: Diatom *Pseudo-nitzschia*
2. Species: *Pseudo-nitzschia australis* & *Psedo-nitzschia multiseries*[13]
3. Toxins produced: Domoic acid
4. Toxins transferable through: clams oysters, crabs, anchovies, and sardines[72]

ASP is also called as DAP (Domoic Acid Poisoning) as these planktonic pinnate diatoms produce DA

(domoic acid) that causes ASP. Amnesic shellfish poisoning causes gastrointestinal and neurological disorders. Consumption of affected shellfish may lead to gastroenteritis showing symptoms of nausea, vomiting, diarrhoea, and abdominal cramps within 24 hours. Neurological symptoms like dizziness; headache, seizures, respiratory problems, short-term memory loss and coma usually appear within 48 hours of consumption of toxic shellfish. In the year 1987, four Canadians lost their lives when exposed to toxic mussels from Prince Edward Island, Canada. Along the west coast of the U.S in the year 1991, about 100 brown pelicans and cormorants in Monterey Bay died or suffered from neurological disorders. It was later identified that blooms of diatom *Pseudo-nitzschia australis* caused ASP. In the same year, in the month of October and November, DA was found in razor clams (*Siliqua patula Dixon*) and Dungeness crabs (*Cancer magister Dana*) on the Oregon and Washington Coasts. During the periods of 1991 and 1994, blooms of *P. australis* most commonly observed in US remained for a longer period of time until late summer and autumn. In the year 1994, blooms of *pseudo-nitzchia* sp. mainly *P. multiseries, P. australis* and *P. pungens* persisted for more than 6 weeks in the Hood Canal of Western Washington.

According to Stewart *et. al.*[73], DA is metabolized by bacteria of genera *Alteromona* and *pseudomonas,* present in Blue mussels (*M. edulis*). Scallops are reported not to contain these toxic DA. On the other hand O'Shea[74], reports that the Sea Otter is the only one animal known to avoid intoxication, probably recognizing toxic prey by their odour. Scholin *et al.*[75], as cited in Rue and Bruland[76], observed mass mortality of California sea lions in Monterey Bay due to blooms of *Pseudonitzschia australis*. Further, Rue *et. al.* [76] concluded that DA produced by species of *Pseudo-nitzschia* could bind iron and copper causing mortality of about 400 sea lions between the months of May and June 1998.

3.1.2 Paralytic Shellfish Poisoning (PSP)

1. Causative organism: Dinoflagellates like *Alexandrium* spp., *Gymnodium catenatum* and *Pyrodinium bahamense*[72]
2. Species: *A. acatenella, A. catenella, A. hiranoi , A ostenfeldii* and *A. tamrense*[13]
3. Toxins produced: saxitoxins (STX), neosaxitoxin (NEO), gonyautoxins (GTXs) and C-toxins. [77]
4. Toxins transferable through: saxitoxins accumulation in mussels, clams, oysters and fishes like sardines, herring and puffer fish (without harming them)[72]

Paralytic shellfish poisoning is caused due to a group of marine phycotoxins produced by several dinoflagellates. These toxins get accumulated in shellfish, and when consumed by vertebrates, block sodium channels leading to paralysis attack. Associated symptoms are mostly neurological. Other symptoms include numbness, giddiness, tingling, burning of perioral region, ataxia, drowsiness, fever, rash and staggering. Within 5 to 30 minutes of consumption, slight perioral tingling is observed progressing to numbness that spreads to the face and neck in moderate cases and further if medical support is not available within 24 hours it can cause complete paralysis leading to a severe respiratory attack.

In 1976, Malaysia was subjected to toxic blooms of *Pyrodinium bahamense* var. *compressum*, affecting 202 people and causing death of 7 children. In 2002, blooms of *A. catenella* had hit south of Chile to such an extent that the President declared closure of this area[40].In India incidences of PSP are reported along the east coast as well as along the west coast.

3.1.3 Diarrhetic Shellfish Poisoning (DSP)

1. Causative organism: Dinoflagellates like *Dinophysis* spp., *Prorocentrum* spp.,
2. Species: *D. acuminata, D. acuta, D. fortii, D. norvegica*, [13] and *D. caudate* [48]
3. Toxins produced: Okaidic acid
4. Toxins transferable through: mussels, scallops, clam etc.

DSP causes gastrointestinal illness without neurological clams disorders. Gastrointestinal symptoms include diarrhoea, nausea, vomiting, abdominal cramps and chills within 30 min to 12 hrs from ingestion. However recovery can occur within 3 days of medical treatment. DSP is caused by certain species of dinoflagellates. The first reported outbreak of DSP took place in the 1960s in the Netherlands, followed by Japan in 1970s. Since then many cases have been reported from several parts of world, including Europe, South America, North America Asia Australia and New Zealand (Mak *et. al.*, 2005)[78]. About 500 cases of DSP were reported in Spain in 1981 with 2000 cases in France in the years 1984 and 1986. In 1990, mussels exported from Denmark to France caused DSP poisoning in over 400 people.

Karunasagar *et al.* [48] observed species of *D. caudate* causing DSP in infected shellfishes collected from the harvesting areas along the coast of Karnataka in India.

3.1.4 Ciguatera Fish Poisoning (CFP)

1. Causative organism: Dinoflagellates like *Gambierdiscus toxicus*, *Prorocentrum* sp., *Ostreopsis* spp., *Coolia monotis*, *Thecadinium* sp. and *Amphidinium* sp.
2. Species: *Amphidinium carterae*
3. Toxins produced: Ciguatoxins / maitotoxin
4. Toxins transferable through: small-algae eating fish (e.g. reef fish) and passed up to food chain to predators such as barracuda, grouper snapper, jacks and kingfish (without harming)[72]

CFS causes neurological symptoms like paresthesias, pain in the teeth, pain on urination, blurred vision, temperature reversal, gastrointestinal effects like that of diarrhoea, abdominal cramps and vomiting, and cardio-vascular symptoms like heart block and arrhythmias. All these symptoms are likely to occur within few hours of ingestion. Ciguatera can also be transmitted sexually. Exposure of any pregnant woman to it may lead to premature labour and spontaneous abortion, and effects on the foetus and newborn child through placental and breast milk transmission have been reported. Reports from the US Virgin Islands observe that 3% of the total population per year is affected. This is similar to that of the French West Indies.

3.1.5 Neurotoxic Shellfish Poisoning (NSP)

1. Causative organism: Dinoflagellates like *Gymnodinium* spp.[72,75]
2. Species: *Kerenia brevis* (formerly known as *Gymnodinium breve*)
3. Toxins produced: Brevetoxins
4. Toxins transferable through: accumulates in shellfishes, sea grasses (without harming)[72]

NSP produces symptoms identical to that of CFP, which includes gastrointestinal, and neurological symptoms. In addition to this, it produces asthma-like symptoms through aerosols. It also causes massive fish kills and other respiratory problems. Airborne toxins may cause irritations in eyes, nose, and throat and cause sinus infection. It poisons manatees, dolphins, sea turtles and sea birds as well as humans on inhalation or through contaminated seafood. The causative species of *Kerenia brevis* produces 2 types of lipid soluble toxins which are hemolytic and neurotoxic, out of which neurotoxic toxins are known as brevetoxin.

Since its recognition as the cause of massive fish kills and initial description in 1948)[79], blooms of *Karenia brevis* have been documented almost annually along Florida's southwest coastal region[37]. Cases have been reported in other coastal regions of the Gulf from Alabama to Texas and the eastern coast of Mexico ([28]as well as along the southeast Atlantic coast to North Carolina[80]). The commercial shellfish industry experienced repeated and prolonged shellfish bed closures due to the presence of *K. brevis* throughout the year during the severe *Karenia* bloom of 2005 in the eastern Gulf of Mexico[30].

4 Control of Algal Blooms

Control of algal blooms is crucial to maintain health of coastal and marine ecosystems. Reducing nutrient loads from various land based activities into coastal systems will help in control of algal blooms. Managing nutrient inputs is the critical factor for managing algal blooms. However, this is not the sole approach for management of algal blooms. This section presents some approaches for control of algal blooms.

4.1 Control of HABs by Managing Nutrient Inputs

Martins *et al*[81] suggest that reductions in nitrogen loading should, in many cases, be geared to reductions in phosphorus, so that N: P loading ratios are kept sufficiently high to discourage N2-fixing cyanobacteria. Further, coastal marine simulation models by Kiirikki *et al.* [82] suggest that phosphorus removal at St. Petersburg, Russia, would reduce the biomass of nitrogen-fixing cyanobacteria in the entire Gulf of Finland. Similarly, Schernewski and Neumann[83] observe that reducing phosphorus loads in the most polluted rivers could be the most cost-effective way of improving water quality in the Baltic Sea. Thus, a high N: P ratio in estuaries or riverine waters may be the key factor in controlling HABs in

coastal and marine ecosystems.

4.2 Control of HABs Through Biomanipulating

The possibility of controlling the symptoms of eutrophication in North American lakes by 'biomanipulating' higher members of aquatic food chains instead of controlling nutrient inputs was pioneered by Joseph Shapiro and his students[80,84,85]. They suggest that eutrophication can be intensified by the removal of piscivorous predators such as bass, pike and walleye by overfishing. This causes an increase in zooplanktivores, which in turn reduce grazing herbivores to low levels, allowing phytoplankton to flourish. Thus removal of piscivorous predators could cause a lake to move from a low algal phase to a high algal phase at the same nutrient loading. Similar results have been demonstrated by Carpenter and Kitchell[86] and Elser *et al.*[87]. Further, some grazers may increase the supply of nitrogen relative to phosphorus, by selective excretion of nitrogen[87,88]. The high N: P ratio in grazer-excreted nutrients in some systems thus can allow nitrogen-fixing cyanobacteria to be outcompeted by other species.

4.3 Control of HABs through Chemical Treatment

It is observed that internal regeneration of nutrients may hinder recovery from eutrophication. This hindrance may correspond to the iron concentration in the system. Though the mechanism is not yet fully known, a possible reason for this phenomenon is that in high-iron systems, phosphorus coprecipitates with ferric hydroxide when oxygen is abundant in the water column. If iron concentration is low, phosphorus is free from control by iron coprecipitation, and is free to diffuse into overlying water, where it facilitates increased algal growth[81]. Recycling of phosphorus from sediments can be avoided by treatment with iron or alum. It is found that addition of iron or alum can prevent the internal recycling of phosphorus and thus maintain a higher N: P ratio, which, in turn, may control HABs.

5 Conclusions

The increasing occurrence of toxic algal blooms has been reported worldwide in the past few decades. These increasing incidents of algal blooms are contributed by various causes such as eutrophication of the coastal areas, dust storms and metals, climate change and increase in temperature, upwelling of oceans, ballast water from ships, unhealthy coral reefs etc. The human ingestion of marine organisms contaminated with algal toxins provokes pathological symptoms termed biopoisonings, and may even lead to high levels of mortality. Hence, there is an urgent need for control of harmful algal blooms. Reductions in phosphorus inputs in estuarine or riverine waters may be the key factor in controlling HABs in coastal and marine ecosystems.

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