Harmful and toxic algae

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Microscopic algae are important food for filter-feeding bivalves (oysters, muscles, scallops and clams) and for the larvae of commercially important crustaceans and fishes. Proliferation (algal blooms) of this algae up to a million cells/l, or cells/ml, is beneficial to aquaculture and wild fisheries operations. In some situations, however, algal blooms can cause severe economic losses to aquaculture, fisheries and tourism, and have major impacts on health and environment. There are 5,000 species of marine phytoplankton (Sournia et al. 1991). Some 300 species can, at times, occur in such high density that they discolor the surface of the sea (red tides). Only 40 species have the capacity to produce potent toxins that can find their way to fish and shellfish and, eventually, to humans.

The first written reference to harmful algal bloom appears in the Bible (1,000 years B.C). In Exodus 7:20-21 is written that at that time in Egypt all the waters in the river turned into blood, and all the fish in the river died, and the river stank, and the Egyptians could not drink the water in the river. In this case, a non-toxic bloom-forming alga became so densely concentrated that it generated anoxic conditions resulting in indiscriminate kills of both fish and invertebrates. Oxygen depletion developed due to high respiration by the algae (at night or in dim light during the day), but most probably, bacterial respiration during decay of the bloom caused it.

One of the first recorded fatal cases of human poisoning after eating shellfish contaminated with dinoflagellate toxins was in 1793 in Poison Cove in British Columbia. The seawater became phosphorescent due to dinoflagellate blooms. The causative alkaloid toxins, now called paralytic shellfish poisons (PSP) are so potent that about 500 micrograms of toxins, which can easily accumulate in just one serving of shellfish (100 gram), could be fatal to humans.

On a global scale, close to 2,000 cases of human poisoning (15% mortality) through fish or shellfish consumption are reported each year and, if not controlled, the economic damage through reduced local consumption and reduced export of seafood products can be considerable. Whales and porpoises can also become victims when they receive toxins through the food chain via contaminated zooplankton or fish. Poisoning of manatees by dinoflagellate brevetoxins contained in salps attached to seagrass, and of pelicans by diatom domoic acid contained in anchovies have also been reported.

Harmful algal bloom has become apparent only as a result of increased interest in intensive aquaculture systems for finfish. Some algal species can seriously damage fish either mechanically or through production of hemolytic sub-
stances. While wild fish stocks have the freedom to swim away from problem areas, caged fish appears to be extremely vulnerable to such noxious algal blooms.

In the Philippines, red tide was reported in 1908 in Manila Bay as due to Peridinium blooms. Thereafter, minor nontoxic red tide outbreaks became almost an annual event in Manila Bay, particularly in the Cavite Area. It was not until June 1983 that the first outbreak of a toxic red tide caused by Pyrodinium bahamense var. compressa occurred in Samar, Central Philippines.

In 1987, the presence of dinoflagellate blooms caused by Pyrodinium bahamense var was detected in the coastal waters of Masinloc, Zambales, extending from Subic to Santa Cruz. Almost simultaneously at that time, the toxic red tide recurred in Samar. Another case of paralytic shellfish poisoning (PSP) was reported on August 19, 1988 in Orion, Bataan, followed by another 28 cases in Limay within a four-day period.

### CONDITIONS THAT STIMULATE HARMFUL AND TOXIC ALGAL BLOOMS

#### Eutrophication

**Eutrophication due to upwelling of deep seawater**

Particulates of decayed phytoplankton may sink several meters a day. These particulates are mineralized by bacterial activities before they are deposited to the ocean bed. The nutrients released by decomposition of these particulates can drift below photic zone. For phytoplankton forming red tides, therefore, eutrophication of surface water by supplying nutrients, especially phosphorous is necessary. Eutrophication in the open sea is affected by upwelling whereby transport of deep water to the euphotic zone is driven by a force mainly originated from an eddy, enriching the coastal water column and initiating plankton blooms.

#### Terrestrial nutrition supplies

This is the amount of plant nutrients introduced with river discharge, effluents from aquaculture farms or a terrestrial run off from heavy rains that might provide enough nutrients to stimulate phytoplankton blooms.

#### Phosphate release from sediments

The increase in activities of sulfate reducing bacteria due to ascending temperature of bottom water that will lead to the production of large amount of \( \text{H}_2\text{S} \), liberating \( \text{P}_4\text{O}_{10}^{2-} \) from \( \text{Fe} \) bound \( \text{P} \) in the sediments, could induce phytoplankton red tides.

#### Nutrient circulation in surf zones

Surf zones are reasonably broad and shallow, cellular circulation predominates that tend to retain nutrients generated by the macrofauna and interstitial microfauna of the beaches. These nutrients may then cause blooms of surf zone phytoplankton, which in turn serves as food for macrofauna filter feeders. With the perimeters of the circulation cells of the surf zone forming its marine
boundary, beach plus surf zone may be denoted as an ecosystem with surf phytoplankton as the primary producers, beach macrofauna the consumers, and the interstitial fauna the decomposers.

**Nitrogen fixation in brackishwater**

In brackishwater, $N_2$ fixation by the blue-green algae takes place. But the fixed $N$ is mostly retained by the nitrogen fixers themselves, forming blooms. $N_2$ fixation decreased with depth in response to light, although other factors are involved. Rates of fixation decrease concurrently with bloom age, total soluble inorganic nitrogen and salinity. Maximum daily fixation occurs early morning.

**Vitamin B$_{12}$**

Unlike most centric diatoms studied, none has an absolute Vitamin B$_{12}$ requirement. However, Vitamin B$_{12}$ (5 mg l$^{-1}$) stimulates growth of most clones by eliminating or shortening the lag phase and increasing the growth rate. High population densities developed 4-54 days with Vitamin B$_{12}$ present. Several clones grown with Vitamin B$_{12}$ removed more than 80% of the Vitamin from the medium. Clearly, B$_{12}$ is important for quick growth and high density of the centric diatoms.

**Prolonged residence time of surface water**

**Stratification of water**

Stratification of water prolongs the residence period of the surface water, allowing enough growth for the red tide algae. But sometimes stratification inhibits growth because of lack of ingress of nutrients from outside. Thus, destratification, in some occasions, favors introduction of nutrients from outside and indirectly causes red tides.

**Change of direction of periodic wind**

Onshore winds carry offshore surface waters to the coast. The buoyancy of the surface water maintains phytoplankton at the surface for a longer period, causing dense blooms along the coast where terrestrial nutrients have been furnished.

**Downward migration of phytoflagellates**

Downward migration of phytoflagellates appears essential to maintain red tide blooms in estuarine embayment having intensive tidal flushes.

**Grazing pressure/depletion**

Grazing pressure due to zooplankton on phytoplankton is regarded as a great deterrent of phytoplankton blooms. Sometimes the grazing pressure by macrozooplankton depletion is exhibited on zooplankton, resulting in zooplankton depletion. Occasionally, zooplankton will avoid certain species and reduce grazing response to Gymnodinium splendens. Consequently, such phytoplankton grows massively, forming red tides.

These behavioral responses may help explain formation and persistence of dinoflagellate blooms such as red tides in coastal waters often dominated by diatoms with higher maximum growth rates.
In summer, phytoflagellates and diatoms stay temporarily in sediment as resting cells such as hypocites or cysts. In spring and for autumn, their germination is initiated with increase and or decrease in sediment temperature, and their swimming cells appear in the upperlying water.

**Lowered salinity stress**

Marine phytoflagellates and diatoms grow in marine waters with fairly high salinity and also in brackishwater with considerably low salinity. In coastal waters, estuarine surface waters are enriched with nutrients by river water, resulting in lower salinity and consequent phytoplankton stress. Species which can tolerate the stress may grow utilizing these nutrients and develop into red tide blooms. Table 8-1 shows the tolerance of phytoflagellates and diatoms to lowered salinity stress.

**Table 11-1.** Tolerance of phytoflagellates and diatoms to lowered salinity stress (from Hallegraef et al. 1995)

<table>
<thead>
<tr>
<th>Phytoplankton</th>
<th>Scientific name</th>
<th>Optimum range of Salinity (‰)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phytoflagellate</td>
<td>Masarita rotundata</td>
<td>24 – 30</td>
</tr>
<tr>
<td></td>
<td>Olisthodiscus luteus</td>
<td>10 – 36</td>
</tr>
<tr>
<td></td>
<td>Prorocentrum micans</td>
<td>27 – 36</td>
</tr>
<tr>
<td>Diatom</td>
<td>Skeletonema costatum</td>
<td>10 – 40</td>
</tr>
<tr>
<td></td>
<td>Chaetoceros spp.</td>
<td>4.4 – 40</td>
</tr>
<tr>
<td></td>
<td>Phaeodactylum tricornutum</td>
<td>8.8 – 40</td>
</tr>
</tbody>
</table>

Evidently, diatoms can tolerate lower salinity levels more than the dinoflagellates. They exploit this difference in utilizing nutrients in coastal waters. This may account for the succession of dinoflagellate blooms.

**Lowered silicate stress**

Diatoms can make their frutules with less amounts of silicate than usual when silicate in external media is depleted. Dinoflagellate blooms have so often appeared after diatom blooms that there must be a certain rule of succession between diatoms and dinoflagellates.

**Trace metal stress**

In waters containing metals in very low concentrations, phytoplankton accumulates these metals $10^3$–$10^4$ times higher than they would in ambient water. This might decrease the grazing pressure of zooplankton on phytoplankton having a high content of heavy metals.
Lowered nutrient stress

Ciliates called “Mesodinium” rubrum cause red tide and may die under poor nutritive conditions. However, not all of these ciliates may die, some of them may adapt themselves to the said situation, and settle to the bottom for some time. When there is a high supply of nutrient due to eutrophication of marine waters, these ciliates will utilize the nutrients for their growth and eventually will grow dense.

Increased scientific awareness of toxic species

Reports of harmful algal blooms associated with human illnesses or damage to aquaculture operations are receiving increased attention in newspapers, the electronic media and the scientific literature. As a result, more and more researchers are surveying their local waters for the causative organisms.

Increased utilization of coastal waters for aquaculture

Aquaculture operations act as sensitive bioassay systems for harmful algal species and can bring to light the presence of problem organisms in waters not known to exist before. The increase in shellfish farming worldwide is leading to more reports of paralytic, diarrhetic and neurotoxic or amnesic shellfish poisoning. On the other hand, increased finfish culture is drawing attention to algal species, which can cause damage to delicate gill tissues of fishes.

Increase algal blooms by cultural eutrophication

While some organisms such as the dinoflagellates Gymnodinium breve, Alexandrium and Pyrodinium appear to be unaffected by coastal nutrient enrichments, many other algal species appear to be stimulated by cultural eutrophication from domestic, industrial and agricultural wastes.

Unusual climatological conditions

The coincidental occurrence of Pyrodinium blooms and El Niño-Southern Oscillation (ENSO) climatological events presented strong circumstantial evidence on the possible impacts on algal bloom. El Niño is caused by an imbalance in atmospheric pressure and sea temperature between the eastern and western parts of the Pacific Ocean, which results in a shoaling of the thermocline. The 1991-1994 ENSO event and the recurrence of dinoflagellate blooms in the Philippines tend to substantiate these claims.

Transport of dinoflagellate cysts

Cargo ballast water was first suggested as a vector in the dispersal of non-indigenous marine plankton some 90 years ago. However, in the 1980s the problem of ballast water transport of plankton species gained considerable interest when evidence was brought forward that non-indigenous toxic dinoflagellate species had been introduced into Australian waters including sensitive aquaculture areas, without disastrous consequences for commercial shellfish farm operations. Another vector for the dispersal of algae (especially their resting cysts) is with the translocation of shellfish stocks from one area to another. The feces and digestive tracts of bivalves can be loaded with viable dinoflagellate cells and sometimes can also contain resistant resting cysts.
TYPES OF HARMFUL AND TOXIC ALGAL BLOOMS

**Type A species**

These are species that produce basically harmless water discoloration; however, under exceptional conditions in sheltered bays, the blooms can grow and cause indiscriminate kills of fish and invertebrates from oxygen depletion. The blooms are represented by dinoflagellate species (*Gonyaulax polygramma*, *Noctiluca scintillans*, and *Scrippsiella trochoidea*) and Cyanobacterium (*Trichodesmium erythraeum*) (Fig. 11-1).

**Type B species**

Type B species produce potent toxins that find their way through the food chain to humans, causing a variety of gastrointestinal and neurological illnesses such as:

- Paralytic Shellfish Poisoning (*PSP*) – Examples: dinoflagellates (*Alexandrium acatenella, A. catenella, A. colectula, A. fundyense, A. fraterculus, A. minutum, A. tamarense, Gymnodinium catenatum, and Pyrodinium bahamense var. compressum*) (Fig. 11-2a).
- Diarrhetic Shellfish Poisoning (*DSP*) – Examples: dinoflagellates (*Dinophysis acuta, D. acuminata, D. fortii, D. norvegica, D. mitra, D. rotundata, Prorocentrum lima*) (Fig. 11-2b).
- Neurotoxic Shellfish Poisoning (*NSP*) – Example: dinoflagellate (*Gymnodinium breve, G. cf. Breve, New Zealand*) (Fig. 11-2c).
- Amnesic Shellfish Poisoning (*ASP*) – Example: diatoms (*Pseudo-nitzschia multiseries, P. pseudodelicatissima, and P. australis*) (Fig. 11-2d).
- Ciguatera Fish Poisoning (*CFP*) – Example: dinoflagellate (*Gambierdiscus toxicus, Osteopsis spp., Prorocentrum spp.*) (Fig. 11-2e).
- Cyanobacterial Toxic Poisoning (*CTP*) – Example: cyanobacteria (*Anabaena circinalis, Microcystis aeruginosa, Nodularia spumigena*)

**Type C species**

Species of this type are non-toxic to humans, but harmful to fish and invertebrates (especially in intensive aquaculture systems) by damaging or clogging their gills.

Examples are diatoms (*Chaetoceros convolutus*), dinoflagellate (*Gymnodinium mikimotoi*), prymnesiophytes (*Chrysocromulina polylepis, Prymnesium parvum, P. patelliferum*), and raphidophytes (*Heterosigma carterae, Chattonella antiqua*).
Figure 11-1. Red tide microalgae (type A - useful, mostly harmless; type B - potentially harmful by oxygen depletion; type C - harmful, responsible for fish mass mortality)
Figure 11-2a. Species responsible for Paralytic Shellfish Poisoning

Figure 11-2b. Species responsible for Diarrhetic Shellfish Poisoning

Figure 11-2c. Gymnodinium breve, responsible for Neurotoxic Shellfish Poisoning

Figure 11-2d (far right). Pseudonitzschia spp., responsible for Amnesic Shellfish Poisoning

Figure 11-2e. Species responsible for and implicated in Ciguatera Fish Poisoning
EFFECTS OF HARMFUL AND TOXIC ALGAL BLOOMS TO FISH AND MARINE ENVIRONMENT

Red tide-inducing phytoplankton produce many lipids containing highly unsaturated fatty acids along with amino acids. Much carbohydrate is also released during the bloom. Decaying phytoplankton red tides sink slowly receiving heterotrophic decomposition due to bacteria and deposit on the seabed. If waters are shallow with low temperatures, phytoplankton deposits remain intact and benthos can utilize them over long periods. Low temperature of bottom water will cause rapid decomposition, including dissolved oxygen deficiency and the generation of H$_2$S. This situation will eventually damage the benthos.

Mass destruction of marine resources results from respiratory difficulty as evidenced by mortality of yellowtail Seriola quinqueradiata due to Chatonella antiqua red tide, and of whitefish Coregonus lavaretus due to Ceratium hirundinella bloom. Mass mortality of finfish and shellfish have occurred elicited by toxins produced by phytoflagellates and blue-green algae, notably brevetoxins by Ptychodiscus brevis and aphatoxin by Aphanizomenon flos-aqua blooms.

Phytoplankton that cause red tides affect distribution of heavy metals in marine waters by ion exchange of metals bound to phytoplankton cells. Heavy metals so bound are deposited with phytoplankton on the seabed. Phytoplankton red tide may increase the concentrations of heavy metals like Cd and Fe by producing humic acid. The remarkable sea foam accompanied by unusually high concentrations of dissolved organic matter manifested surfactant production due to a Phaeocystis pouchetti red tide. Similar productions were observed by species of Ballariophyceae, Cryptophyceae, Haptophyceae and Chlorophyceae. Association of Cu, Hg, and Pb with these surfactants was confirmed. Antimicrobial compounds were demonstrated in Gonyaulax tamarensis, which has a broad antimicrobial spectrum. Antimicrobial agents were also found in Gyrodinium cohnii, but not in axenic culture.

Toxin-producing algae are becoming an increasingly serious problem in both aquaculture and fisheries populations. The severity of these blooms has appeared to increase in recent years. Likewise, the number of geographic locations experiencing toxic algal blooms has also increased. Toxic algal blooms are worldwide problems affecting aquatic populations in both warm water and cold water environments. The great majority of the problems has occurred in near-coastal marine ecosystems and thus, may have the greatest potential impact on cage and net pen culture. They are also a threat to culturists obtaining water from sites prone to develop toxic algal blooms (e.g., land-based tank systems). In some cases, the introduction of toxic algae into a closed aquaculture system may result in devastating effects.

Increased eutrophication or nutrient enrichment has often been correlated with increased prevalence of many toxic algal blooms. But there appear to be other important risk factors besides simply increased nitrogen or phosphorous, which are essential for development of bloom. The problem of toxic algae is a cogent lesson on how the success of marine aquaculture is heavily dependent
upon a healthy, balanced natural environment and that aquaculturists have a vested interest in assuring that the marine environment is not degraded.

Many different toxic algae have been implicated in fish kills, although in most cases the precise toxins or mechanisms of toxicity are unclear. Nonetheless, many different types of toxins have been isolated from various algae and many of these are ichthyotoxic. Neurotoxins are especially common. Clinical signs of algal neurotoxicity include disorientation, loss of equilibrium, and sporadic hyperactivity. Other algae mechanically obstruct or damage the gills of fishes, causing hypoxia.

Dinoflagellates represent the predominant toxin producing algae. Many dinoflagellates have been implicated or suspected in fish kills. The red tide dinoflagellate (*Gymnodinium breve*) causes mass mortalities of fish and invertebrates in states bordering the Gulf of Mexico. Some dinoflagellate toxins are transferred up the food chain (e.g., *Alexandrium*) and have caused mortalities in wild fish (e.g., Atlantic herring *Clupea harengus*) that consume tainted zooplankton along the northwest Atlantic coast of the United States.

Other algal blooms associated with toxicity include the *Prymnesium parvum*, which causes mortality in brackishwater and marine pond fish (e.g., mullet *Mugil spp.*) in Europe and the Middle East. Diatoms of the genus *Chaetoceros* have been associated with mortality in seawater-cultured salmonids. The spines of other alga apparently cause it to become lodged on or in the gills. Diatoms may become embedded in gill tissue, inciting a foreign body reaction. Epithelial hyperplasia causes hypoxia. In some cases, hyperactive mucus production appears to be primarily responsible for the hypoxia. Other algae have been less commonly associated with ichthyotoxicity, although there is considerable speculation about possible sublethal effects.

The most well recognized economic impact of toxic algae is seafood contamination by their toxins (Table 11-2). This has caused major economic losses in shellfish, including cultured species. Other toxic algae are known to cause serious illness in persons exposed to their toxins. Thus, the presence of toxic algae poses a serious threat to the future success of marine aquaculture, and there is a need to have intensified efforts to address these problems.

The price of fish and invertebrates has dropped as a result of red tide scare, particularly in the Philippines. Prices of fishes caught from other areas free from red tide contamination have also been affected by the red tide scare.
Table 11-2. Causative organisms, clinical signs and potential treatments of various types of fish and shellfish poisoning among human consumers (Hallegraef et al. 1995)

<table>
<thead>
<tr>
<th>Type</th>
<th>Causative organisms</th>
<th>Symptoms</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>Paralytic shellfish poisoning (PSP)</td>
<td>Alexandrium catenella</td>
<td>Within 30 min: tingling sensation or numbness around lips, gradually spreading to face and neck; prickly sensation in fingertips and toes; headache, dizziness, nausea, vomiting, and diarrhea</td>
<td>Muscular paralysis; pronounced respiratory difficulty; choking sensation; death through respiratory paralysis may occur within 2-24 h after ingestion; Patient has stomach pumped and is given artificial respiration. No lasting effects</td>
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<tr>
<td></td>
<td>Alexandrium minutum</td>
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<td></td>
<td>Alexandrium tamarense</td>
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<tr>
<td></td>
<td>Gymnodinium catenatum</td>
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<td></td>
<td>Pyrodinium bahamense</td>
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<td></td>
<td></td>
<td>Mild case</td>
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<td>Extreme case</td>
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<td></td>
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<td>Treatment</td>
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<tr>
<td>Diarrhetic shellfish poisoning (DSP)</td>
<td>Dinophysis acuminata</td>
<td>After 30 min, to a few h (seldom more than 12 h); diarrhea, nausea, vomiting, abdominal pain</td>
<td>Chronic exposure may promote tumor formation in the digestive system; Recovery after 3 days, irrespective of medical treatment</td>
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<tr>
<td></td>
<td>Dinophysis fortii</td>
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<td>Dinophysis norvegica</td>
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<td>Prorocentrum lima</td>
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<td></td>
<td>Mild case</td>
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<td>Extreme case</td>
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<td>Treatment</td>
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<tr>
<td>Amnesic shellfish poisoning (ASP)</td>
<td>Pseudo-nitzschia multiseries</td>
<td>After 3-5 h; nausea, vomiting, diarrhea, abdominal cramps</td>
<td>Decreased reaction to deep pain; dizziness, hallucinations, confusion; short-term memory loss; seizures</td>
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<tr>
<td></td>
<td>Pseudo-nitzschia Pseudodelicateissima</td>
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<td>Pseudo-nitzschia australis</td>
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<td>Mild case</td>
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<td>Extreme case</td>
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<td>Treatment</td>
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<tr>
<td>Neurotoxic shellfish poisoning (NSP)</td>
<td>Gymnodinium breve</td>
<td>After 3-6 h; chills, headache, diarrhea, muscle weakness, muscle and joint pain; nausea and vomiting</td>
<td>Paraesthesia: altered perception of hot and cold; difficulty in breathing, double vision, trouble in talking and swallowing</td>
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<tr>
<td></td>
<td>G. cf. breve (New Zealand)</td>
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<td>Mild case</td>
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<td>Extreme case</td>
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<td></td>
<td></td>
<td>Treatment</td>
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<tr>
<td>Ciguatera shellfish poisoning</td>
<td>Gambierdiscus toxicus</td>
<td>Symptoms develop within 12-24 h of eating fish. Gastrointestinal symptoms; diarrhea, abdominal pain, nausea, and vomiting</td>
<td>Neurological symptoms: numbness and tingling of hands and feet; cold objects feel hot to touch; difficulty in balance; low heart rate and blood pressure; rashes. In extreme cases, death through respiratory failure; No antitoxin or specific treatment is available. Neurological symptoms may last for months and years. Calcium and mannitol may help relieve symptoms</td>
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<tr>
<td></td>
<td>?Ostreopsis siamensis</td>
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</table>

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STRATEGIES IN COPING WITH THE PROBLEM OF HARMFUL AND TOXIC ALGAL BLOOMS

Harmful and toxic algal bloom is one of the major public health and fishery problems in the Philippines and throughout the world. Its occurrence can cause serious social problems. The Philippine government, starting in 1984, has adopted some strategies for all red-tide affected areas in the country in an effort to detect a bloom at its early stage and minimize, if not totally eliminate, its effects on public health.

**Red Tide Monitoring** Weekly or twice monthly monitoring of plankton and shellfish samples should be collected in coastal areas, particularly with histories of *Pyrodinium* blooms. During red tide blooms, aerial surveillance from helicopters and light aircraft should be undertaken, in coordination with concerned agencies like the Department of Agriculture in the case of the Philippines, to determine the extent of the bloom and the movement of the visible red tide. Based on the information gathered from these aerial observations, the residents in affected areas are to be alerted.

Other hydrobiological parameters such as water temperature, salinity pH, dissolved oxygen, phosphate-phosphorus and cyst density should be determined once a month during neap tides to minimize the tidal effect on the water samples.

Meteorological parameters such as amount of rainfall, wind force and wind direction should be taken from the records of the Philippine Atmospheric, Geophysical and Astronomical Services Administration (PAGASA), in the case of the Philippines.

**Information Dissemination** Information dissemination on red tide to alert the public through print and broadcast media, including seminars and meetings, must be done whenever the toxicity in shellfish exceeds the regulatory limit of 80 microgram toxin per 100 g of shellfish meat. During the emergency, the people should be advised not to eat any kind of shellfish taken from red tide-infested waters.

**Regulation** A temporary ban on the harvesting, marketing and transporting of all kinds of marine shellfish from the red tide contaminated waters should be imposed upon information that the toxin level in shellfishes has exceeded the regulatory limit. Issuance of auxiliary invoices, a requirement in transporting fishery products from one place to another, should be suspended to prevent movement of the contaminated shellfish to non-affected areas. This should be matched by establishing checkpoints in strategic locations such as piers, airports and bus routes.
Medical Management of PSP Cases

There is no antidote for the PSP toxin. Treatment is largely symptomatic, as indicated by Halstead (1965). In the Philippines, as mentioned by Gonzales (1989), it is suggested that the first thing to consider in PSP management is to empty the stomach of the victim of the toxic material as quickly as possible. This may be done by giving the patient an oral emetic, or, when this is not available, by inserting a finger in the throat of the victim to induce vomiting. Since the toxin is water soluble, it is also recommended that the victim be given plenty of water to induce urination and minimize absorption of the toxin through the gastro-intestinal tract.

The Department of Health and the Philippine General Hospital follow a protocol in managing PSP cases as cited by Gonzales (1986). The universal antidote for all kinds of poisoning contracted through the oral route is pure coconut milk. Gacutan (1986) suggested that this local cure for PSP is effective. However, it should be emphasized, that coconut milk, or any drug for that matter, should not be given to patients starting to show symptoms of dysphagia and respiratory failure due to the risk of asphyxia.

Government Assistance Program

The occurrence of red tide, and the red tide scare itself, will adversely affect the income of fishermen and fish vendors, while others may not even earn a single centavo at all. Due to economic hardship suffered by fishermen during red tide occurrences, the government, through non-government organizations and fishermen's cooperatives, should grant red tide affected artisanal fishermen with emergency loans payable within a period of one year with affordable interest rate.

Recommendations

Efforts should be made to collect shellfish at regular intervals, e.g., weekly and subject this to mouse bioassay. Also, water samples must be collected weekly and analyzed for the presence of harmful and toxic algal blooms by the Bureau of Fisheries and Aquatic Resources. Efforts must also be exerted to develop the regional and provincial capabilities of the Department of Agriculture and the Department of Health on bioassay techniques to determine the presence or absence of toxin in shellfish samples. If PSP toxins are detected from the sample, a warning may be issued while waiting for the results of the confirmatory tests to be performed on the samples by the Bureau of Food and Drugs. This way, the government may be able to respond quickly should there be a recurrence of toxic red tide.
REFERENCES/SUGGESTED READINGS


White AW. 1980. Recurrence of kills of Atlantic herring (Clupea harengus) caused by dinoflagellate toxins transferred through herbivorous zooplankton. Canadian Journal of Fisheries and Aquatic Sciences 37. 2262-2265