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REVIEW OF THE IMPACT OF HARMFUL ALGAE BLOOMS AND TOXINS ON THE WORLD ECONOMY AND HUMAN HEALTH

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ABSTRACT

The most common type of harmful algal blooms (HABs) is referred to as a "Red Tide" because the bloom discolors the water, making it appear red. However, HABs may also be yellow, orange, brown, green, white, or pink, depending on which one of the three primary types of phytoplankton are responsible for the problem; dinoflagellate, diatoms, or blue-green algae. Of the more than 4,400 marine phytoplankton species, only 50 to 60 are believed to be toxic. Many HABs (harmful algal blooms), produce vividly colored blooms of cells that accumulate on surface water. These high biomass blooms can cause hypoxia, can contribute to toxicity of fish and shellfish, and can cause other environmental problems. They occur in waters all over the globe, and have been called red tides, brown tides or yellow tides. Because these descriptors tend to vary, it's more accurate to call these natural phenomena harmful algal blooms (HABs). Red tides, whether they are harmful or not, have been found throughout the world. Scientific studies indicate a global increase in occurrence and geographic extent of harmful red tides. The aim of the review is to increase the awareness of the scientific community as well as the general public of the aspects and effects of such a phenomenon, seeking the search for novel techniques and tools for limiting its influences on various water bodies. The harmful effect of the Red Tide phenomenon on the health of man, methods for its detection and means to overcome it will be intensely discussed. The most common of the toxic syndromes are Paralytic Shellfish Poisoning (PSP) and Diaffhetic Shellfish Poisoning (DSP). More recently, or since late 1987, Amnesic Shellfish Poisoning (ASP) has become a problem in eastern Canada.

1. INTRODUCTION

Red tide phenomenon naturally occurs world-wide and its seasonal occurrence depends on environmental conditions and meteorological factors. Evidence indicates that red tides usually occur between February and May. From the viewpoint of seasonal occurrence, the causative species are divided into three types: 1. All season type; 2. summer type; and 3. winter type. Data on seasonal occurrence and distribution of each species, particularly toxic ones, would enable better planning of preventive measures and management actions in different coastal regions. Red tides and associated catastrophic mortalities of organisms occur world-wide in fresh and marine water habitats. Knowledge on toxicity of different species would facilitate the initial assessment of risks involved. Public health problems arising directly or indirectly as a result of exposure to the noxious metabolites produced during red tides are global in occurrence. Part of the problem is establishing a proper diagnosis and therapy program and in developing specific assay kits for red tide toxins depending on their diverse geographic location. The increased trend of toxic dinoflagellate blooms poses potential health

risks of Paralytic Shellfish Poisoning (PSP) and Diarrhetic Shellfish Poisoning (DSP) to humans through ingestion of red tide contaminated shellfish, (Arzul, 1993; Campbell, *et. al.*, 2004; Carreto, *et. al.*, 2001; Cembella, *et. al*., 2005; Cembella, *et. al*., 2003; Climent, and Lembeye, 1993; Clément, *et. al*., 2001).

The literature on toxic algal blooms and red tides documents a global increase in the frequency, magnitude, and geographic extent of these events over the last two decades. Some of this increase is undoubtedly a result of the increased awareness and analytical capabilities of the scientific community, but a strong correlation between the number of red tides and the degree of coastal pollution or utilization of coastal waters for aquaculture argue that there are other contributing factors. It also appears likely that toxic algal species have spread within regions over spatial scales of hundreds of kilometers, moving with major water currents and storms, (John, *et. al.*, 2005; Kim, 1999; Klöpper, *et. al*., 2003; Leikin and Paloucek, 1998).

It is the responsibility of the World Health Organization to take proper consideration of the problem, establish a proper diagnosis and universal therapy program develop assay kits for red tide toxins specific to their diverse geographic locations, and provide chemical tests that detect toxins in suspected food sources.

In this review, a discussion is presented on the Impact of the Harmful Algae Blooms economically and human health problems.

2. RED TIDE PHENOMENON

Red tides are harmful algal blooms (HABs) that occurs when toxic microscopic algae in seawater proliferate to higher-thannormal concentrations (bloom), often discoloring the water red, brown, green, or yellow. Climatic events and biophysical sequences involved in red tide formation have been characterized by mnemonic symbols and applied to geographical regions with spatial scales from meters to hundreds of kilometers and temporal scales ranging from daily to annual. A frontal convergence zone at the seaward edge of upwelling regions can exist and be a source of dinoflagellates transported into near shore waters below a pycno-cline. The physical hydrographic integrity of red tide surface patches enhances transport within geostrophic surface current jets with minimal dilution losses, (Carreto, *et. al*., 2001; Cembella, *et. al*., 2005; Cembella, *et. al.,* 2003; Clément and Lembeye 1993; Climent *et. al*., 2001; Clément, 1999; Cohen, 1974; Cosper, *et. al.*, 1989).

Dinoflagellate dominated red tides were most common during transitions in the synoptic weather patterns which resulted in diminished upwelling activity and increased thermal stratification. Red tides represented spectacular localized accumulations, usually within embayment, of widespread seasonal dinoflagellate blooms, (Currie, 2000; Eschbach, *et. al*., 2005; Fraga, and Sánchez, 1985; Gentien, and Raffin, 1998; Gerdts, *et. al*., 2002; Guzmán, *et. al*., 1999; Guzmán, *et. al*., 1975; Haddad, and Lee, 1998; Hallegraeff, *et. al.*, 2003; Hansen, *et. al*., 2000; Haywood, *et. al*., 1996).

Blooms are of such great extent that there is little hope of eliminating them once they have developed but, if human activities are contributing to their occurrence, these can be identified and changed. Accurate prediction is a primary research goal which can allow steps to be taken to ameliorate the effects, such as the towing of salmon pens or increased shellfish toxin monitoring. Methods of protecting marine life need to be improved as they are still very primitive at present. Antidotes do not exist for several of the toxins harming humans, (John, *et. al*., 2005; Kim, 1999; Klöpper, *et. al*., 2003; Leikin, and Paloucek, 1998; Lembeye, *et. al*., 1993; Lembeye, 1981; Luckas, *et. al*., 2005; MacKenzie *et. al*., 1996; Mahoney, *et. al*., 1990).

The term brown tide is too much broad for characterizing a phytoplanktonic bloom, since brown water discolorations can be

produced by microorganisms as different as *Aureococcus anophagefferens* Hargraves *et* Sieburth (*Aureoumbra lagunensis* or *Gymnodinium mikimotoi* Miyake *et* Kominami *ex* Oda, all of them with singular peculiarities. For these reasons comparison must be restricted to species or genus levels. Brown tides produced by *Gymnodinium* are known in Europe, Australia, Japan and Korea. *Gymnodinium cf. nagasakiense* has been described as a common blooming species from Norway (70°N) to the coast of Spain (40°N) and especially in the coast of Brittany. The features evaluated in these *Gymnodinium* did not permit the assignment to any particular species, since this genus is highly complex and comprises more than 200 species with high morphological variability. Moreover, morphological changes occur in preserved material reducing the possibility of accurate identification. However, we consider both morphs as distinct and not the result of different stress, since they were obtained from the same samples. Both share features with *G. mikimotoi* and with others species like *Gymnodinium* sp. The cell outline of *Gymnodinium* sp.1 differed from *G. mikimotoi* especially by the length/wide. *Gymnodinium* sp. 2 presented a more similar outline to *G. mikimotoi*, but differed notably in the apical groove. Both morphs were larger than *G. mikimotoi*, altough they overlap in the upper range of the larger *G. mikimotoi* isolate. In *Gymnodinium* sp. 1 and *Gymnodinium* sp. 2 the position of the nucleus was also different from the above mentioned species of *Gymnodinium*, since it was more spherical and centrally located. Both morphs could represent new species in this highly complex genus, but the alternative of different stages of the same species can not be discarded. Occasionally this species has been observed in waters with low temperatures (<10°C) and low salinities. The mean temperature and salinity at Punta Carrera, recorded during the sampling period, were 8.40°C and 30.06 psu respectively, and both are normal values during the autumn. In Chiloé, higher concentrations of *Gymnodinium* were associated with temperatures above 13.5 °C and salinities above 33.2 psu. Another known characteristic of the ecology of these species is that when blooms arise, they are often associated with an off shore tidal front, spreading later to the coastal zone. This situation seems to be coincident with the observations in the Patagonic fjords. It was evident both in Chiloé and in the Magellan Region that brown patches were well developed in the oceanic entrance of the fjord system and later the microorganisms were detected in the inner waters. These observations and the other common features such as the quasi-simultaneous blooming and the presence of the same genus as a major component, indicate that both episodes could represent a manifestation of one major event. However, the lack of sampling in the geographic space in between, and the different methods used to fix the material, preclude a definitive conclusion. At Chiloé, various sites were evaluated and a maximum of 4,000,000 cell / L was detected. In the Magellan Region, the brown patches observed in Canal Abra indicated high concentrations of the microorganism but the survey made by IFOP showed a maximum of 43,000 cell / L. Two days before this sampling, a windy front reached the Magellan Region and seemed to be the most probably factor that dissipated the brown tide. The lower values detected at Punta Carrera indicate a limited dispersion into the inner waters. However, it must be considered that higher concentrations could be reached since the high motility of dinoflagellates permit them to concentrate at specific depths, (Arzul, 1993; Campbell, *et. al*., 2004; Carreto, *et. al*., 2001; Cembella, *et. al*., 2005; Cembella, *et. al*., 2003; Climent, and Lembeye. *et. al*., 1993).

3. ECONOMIC IMPACT

Scientists use the term, harmful algal bloom, HAB, to refer to such high density algal populations that contain toxins or that

because negative impacts. A small number of algal species produce potent hepatotoxins or neurotoxins that can be transferred through the food web where they may kill other life forms such as zooplankton, shellfish, fish, birds, marine mammals and even humans that feed, either directly or indirectly, on them. The term red tide is a misnomer because they are not associated with tides; they are usually not harmful and those species that are harmful may never reach the densities required to discolor the water. There are also marine brown tides caused by brown algal species. Fortunately, most blooms are short-lived. An affected area will likely be safe again in anywhere from a few days to a week or two. However, contaminated shellfish which have concentrated the toxins may take a very long time, up to a year, to cleanse themselves to the point where they are safe for people to eat, The relationship between nitrogen and phosphates in the sea is expressed as a ratio, N/P, which is normally about 15. The algae were exposed to N/P ratios ranging from 2.5 to 100. The *Chrysochromulina polylepis* growth rate was not very sensitive to the N/P ratio, while other algae tested in the same way obviously grew more slowly when the N/P ratio was high. This means that *Chrysochromulina polylepis* is at a competitive advantage when the nutrient content of the ocean is dominated by N-salts. This is one factor that helps to explain the huge algal bloom of 1988. It killed practically all other life-forms in the upper layers of the water, (John, *et. al*., 2005; Kim, 1999; Klöpper, *et. al*., 2003; Leikin, and Paloucek, 1998; Lembeye, *et. al*., 1993; Lembeye, 1981; Uribe, *et. al*., 2001; Uribe, 1992; Villareal, *et. al*., 1997; Widdows, *et. al*., 1979; Windust, and Quilliam, 2004; Wommack, *et. al*., 2003).

In 1999, the world's seafood (fish, crustaceans, mollusks) production reached 106.8 million mt, with 92.9 million mt derived from capture fisheries and 13.9 million mt from marine / brackish water aquaculture. Out of the total seafood production, mollusks comprised 13.6 million mt, of which 3.4 million mt (25.2%) were from capture fisheries. Seafood is one of the most highly traded commodities in the world market, and experienced a doubling of trade volume between 1984 and 1994. For 1999, the world's total value of seafood imports was US$57,492,816,000, with the international exports recording a value of US$52,882,533,000. Lobster continued to be the most valuable species landed in the region and the state with Maine accounting for 70.6% of the revenues ($286 million) from the 2004 lobster harvest. Other species of importance include the ground fish complex, soft-shell clam, Atlantic herring, sea urchin, and blood worm Maine's saltwater recreational fishery is valued in excess of $27 million by about 362,000 anglers as striped bass and other species have rebounded. The aquaculture industry is undergoing a period of change but is still a valuable industry, including the culture of Atlantic salmon, oysters, mussels, trout, and freshwater species, (John, *et. al*., 2005; Kim, 1999; Klöpper, , *et. al*., 2003; Leikin, and Paloucek, 1998; Lembeye, *et. al*.,1993; Lembeye, 1981; Uribe, *et. al*., 2001; Uribe, 1992; Villareal, *et. al*., 1997; Widdows, *et. al*., 1979; Windust, and Quilliam, 2004; Wommack, *et. al*., 2003).

Fish production from aquaculture reached 29.1% of total fish production by weight in 2001 (FAO, 2003). Worldwide aquaculture has been growing at an average compounded rate of 9.2% since 1970, exceeding the growth rate of capture fisheries (1.4%) and farmed terrestrial animals (2.8%) in the same period. The most impressive growth in aquaculture production has occurred in Asia, and in particular in China; per capita supply of aquaculture fish outside China has also increased from 0.6 kg in 1970 to 2.3 kg in 2000. Different forecast scenarios analyzed by FAO agree on the continued increasing contribution of aquaculture to total fish supply in the coming decades, with probably not less than a doubling of the current volume of production by 2030, (Tunik, and Goldfrank, 1998; Uribe *et. al*., 2001; Uribe,

1992; Villareal *et. al*., 1997; Widdows *et. al*., 1979; Windust, and Quilliam, 2004; Wommack *et. al*., 2003).

In 1987, four victims died after consuming toxic mussels from Prince Edward Island, Canada. Since that time, Canadian authorities have monitored both the water column for the presence of the causative diatom and shellfish for the presence of the toxin, domoic acid. Shellfish beds are closed to harvesting when the domoic acid concentration reaches 20 µg / g in shellfish meat. Fish and crab viscera can also contain domoic acid so the risk to human consumers and animals in the marine food chain is more significant than previously believed, (Kim, 1999; Klöpper *et. al*., 2003; Leikin, and Paloucek, 1998; Lembeye *et. al*., 1993; Lembeye, 1981; Luckas *et. al*., 2005; MacKenzie *et. al*., 1996; Mahoney *et. al*., 1990; McKenzie *et. al*., 2003; Nielsen, and Tønseth 1991; Partensky, and Sournia, 1986).

Toxic mechanisms of the red tide flagellate, *Chattonella marina*, collected in 1985 from Kagoshima Bay, Japan, were studied at the sub cellular level. *C. marina* was found to reduce ferricytochrome-c at a rate related to the concentration of plankton cells. Ca. 50% of the cytochrome-c reduction was inhibited by the addition of l00 µ superoxide dismtase/ml. Moreover, a small amount of hydrogen peroxide was detected in the *C. marina* suspension using the fluorescence spectrophotometric assay method. The identity of the hydrogen peroxide was confirmed by its reaction with 500 µ catalyses / ml. Epicubenol, alphacadinol and cubenol, was identified as characteristic volatile compounds from both field and cultured red tides of *Gymnodinium nagasakiense*. They were excreted from the phytoplankton into the media. The amount of cubenol increased from the logarithmic phase to the stationary phase during culture of the plankton, and was shown to cause cell burst of swimming plankton such as *G. nagasakiense*, *Heterosigma akashiwo*, *Chatonella marina* and *Chatonella antique* at 5 ppm, (Schnorf *et. al*., 2002; Tangen, 1977; Taylor *et. al*., 1995; Tillmann, 2004).

Much has occurred in red tide research since the 1972 incident when a large-scale bloom of the toxic dinoflagellate, *Gonyaulax tamareasis*, stimulated wide-spread public and scientific interest and concern. In 1972, only one toxin, saxitoxin was identified as the toxicant responsible for paralytic shellfish poisoning (PSP): now more than 12 toxins are known to play a part. The structures of most of the toxins have been elucidated, and the information has proven useful in the study of the action of toxin on the nervous system. The biology of these organisms has also been made clearer. A study has been made of the distribution and the toxicity variance of the *Gonyaulax* species along the northeast coast of the United States. The results reveal the presence of weakly toxic organisms in the areas where PSP problems have never previously been reported. This information should help in selecting sites for shell fish aquaculture and identifying areas where shellfish are potentially toxic. The PSP toxicity of popular eating shellfish from various parts of the Hong Kong territorial waters has been monitored using the mouse bioassay method since 1984, in view of the frequent red tide occurrences in local waters such as Tolo Harbour and the potential risk of public health hazards caused by toxic red tides. A very low toxicity level below 2000 mouse units per kg tissue was prevalent in the shellfish samples tested. The mean monthly toxicity level appears to follow an increasing trend with time over the past years from 1984-1987. The presence of taxi toxin in green-lipped mussels (*Perna viridis*) from Tolo Harbour has been identified by RPLC analysis. It is not known what is the origin of this baseline toxicity, (Arzul, 1993; Campbell *et. al*., 2004; Carreto *et. al*., 2001; Cembella *et. al*., 2005; Cembella *et. al*., 2003; Climent, and Lembeye, 1993; Clément *et. al*., 2001; Clément 1999; Cohen, 1974; Cosper *et al*, 1989; Currie *et. al*., 2000).

Amnesic Shellfish Poisoning (ASP) causative organisms:

Pseudo-nitzschia australis, Pseudo-nitzschia pungens.

Toxins produced: Domoic Acid.

ASP can be a life-threatening syndrome. It is characterized by both gastrointestinal and neurological disorders. Gastroenteritis usually develops within 24 hours of the consumption of toxic shellfish; symptoms include nausea, vomiting, abdominal cramps, and diarrhea. In severe cases, neurological symptoms also appear, usually within 48 hours of toxic shellfish consumption. These symptoms include dizziness, headache, seizures, disorientation, short-term memory loss, respiratory difficulty, and coma. In 1987, four victims died after consuming toxic mussels from Prince Edward Island, Canada. Since that time, Canadian authorities have monitored both the water column for the presence of the causative diatom, and shellfish for the presence of the toxin, domoic acid. Shellfish beds are closed to harvesting when the domoic acid concentration reaches 20 µg/g shellfish meat. Fish and crab viscera can also contain domoic acid, so the risk to human consumers and animals in the marine food chain is more significant than previously believed, (Rafuse *et. al*., 2004; Schnorf *et. al*., 2002).

Diatoms produce domoic acid. Bivalve shellfish and some fin fish filter these diatoms from the water. In most cases domoic acid accumulates in the viscera of these animals. In razor clams domoic acid also accumulates in the meat. Unsafe levels of domoic acid have been found in anchovies, mussels, razor clams and crab viscera but not crab meat. Many other species have not yet been investigated.

Amnesic shellfish poisoning is generally associated with the consumption of molluscan shellfish from the northeast and northwest coasts of North America. It has not yet been a problem in the Gulf of Mexico although the alga that produces the toxin has been found there. ASP toxins have recently been identified as a problem in the viscera of Dungeness crab, tanner crab, red rock crab and anchovies along the west coast of the United States.

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Ciguatara Fishfood Poisoning (CSP).

Causative organisms: *Gambierdiscus toxicus, Prorocentrum concavum, Prorocentrum hoffmannianum, Prorocentrum lima, Ostreopsis lenticularis, Ostreopsis siamensis, Coolia monotis, Thecadinium* and *Amphidinium carterae*

Toxins produced: Ciguatoxin, Maitotoxin

Ciguatera poisoning in humans and domestic animals is caused by potent neurotoxins produced by benthic dinoflagellates including *Gambierdiscus toxicu*. In the tropics and subtropics toxic dinoflagellates living on coral reefs are eaten by small herbivorous fish grazing on coral which in turn are eaten by larger carnivores. The poisons move up the food chain into the organs of larger top-order predators such as coral trout, red bass, chinaman fish, mackerels and moray eels and cause ciguatera fish poisoning, CFP, in people who eat these fish. Over 400 non-lethal cases of ciguatera food poisoning have been recorded in Australia and tropical countries such as French Polynesia report thousands of cases every year (Kim, 1999; Klöpper *et. al*., 2003; Leikin, and Paloucek, 1998).

Diarrhetic Shellfish Poisoning (DSP).

Causative organisms: *Dinophysis, Prorocentrum, Dinophysis fortii, Dinophysis*

acuminata, *Dinophysis norvegica*, *Dinophysis acuta*.

Toxin produced: Okadaic Acid.

DSP produces gastrointestinal symptoms, usually beginning within 30 min to a few hours after consumption of toxic shellfish. The illness, which is not fatal, is characterized by incapacitating diarrhea, nausea, vomiting, abdominal cramps, and chills. Recovery occurs within three days, with or without medical treatment, (Climent, and Lembeye, 1993; Climent, *et. al*., 2001; Clément, 1999; Cohen, 1974; Cosper *et. al*., 1989; Currie *et. al*., 2000).

Neurotoxic Shellfish Poisoning (NSP).

Causative organism: *Gymnodinium breve*, *Karenia brevis.*

Toxins produced: Brevetoxins

NSP produces an intoxication syndrome nearly identical to that of ciguatera. In this case, gastrointestinal and neurological symptoms predominate. In addition, formation of toxic aerosols by wave action can produce respiratory asthma-like symptoms. No deaths have been reported and the syndrome is less severe than ciguatera, but nevertheless debilitating. Unlike ciguatera, recovery is generally complete in a few days. Monitoring programs (based on *K. brevis* cell counts) generally suffice for preventing human intoxication, except when officials are caught off-guard in previously unaffected areas (Passow, 1991; Perez *et. al*., 2001; Rafuse *et. al*., 2004; Schnorf *et. al*., 2002; Tangen, 1977; Taylor *et. al*., 1995).

Paralytic Shellfish Poisoning (PSP)

Causative organisms: *Alexandrium excavatum*, *Alexandrium monilata*, *Alexandrium tamarense*,

Gymnodinium catenatum, *Pyrodinium bahamense*

Toxins produced: Saxitoxins.

PSP, like ASP, is a life threatening syndrome. Symptoms are purely neurological and their onset is rapid. Duration of effects is a few days in non-lethal cases. Symptoms include tingling, numbness, and burning of the perioral region, ataxia, giddiness, drowsiness, fever, rash, and staggering. The most severe cases result in respiratory arrest within 24 hours of consumption of the toxic shellfish. If the patient is not breathing or if a pulse is not detected, artificial respiration and CPR may be needed as first aid. There is no antidote, supportive therapy is the rule and survivors recover fully. PSP is prevented by large-scale proactive monitoring programs (assessing toxin levels in mussels, oysters, scallops, clams) and rapid closures to harvest of suspect or demonstrated toxic areas.

Paralytic shellfish poisoning (PSP) has been reported to occur after eating puffer fish, filter feeding shellfish and mollusks. There are approximately 10 outbreak-associated PSP cases reported to the CDC each year.[9] Algae of the genus *Alexandrium* are responsible for seasonal outbreaks of PSP along the New England coast from Maine down to New Jersey and on the West Coast from Alaska to Northern California. If ingested by humans, PSP produces neurologic symptoms such as tingling and burning of the mouth and tongue, numbness, drowsiness and incoherent speech. These symptoms occur within 30 minutes to two hours after ingestion and in severe cases cause ataxia, muscle weakness, respiratory paralysis and death. The Toxic Exposure Surveillance System of the American Association of Poison Control Centers (TESS) has identified 10 illnesses of presumed puffer fish poisoning due to exposure from PSP after eating puffer fish from the area of Titusville, Florida, (Klöpper, *et. al*., 2003; Leikin and Paloucek, 1998; Lembeye *et. al*., 1993; Lembeye, 1981; Luckas *et. al*., 2005; MacKenzie *et. al*., 1996; Mahoney *et. al*., 1990).

Ciguatera Fish Poisoning (CFP)

Causative organisms: *Gambierdiscus toxicus*, *Prorocentrum* spp., *Ostreopsis* spp., *Coolia monotis*, *Thecadinium* sp. and *Amphidinium carterae*.

Toxins produced: Ciguatoxin/Maitotoxin

CFP produces gastrointestinal, neurological, and cardiovascular symptoms.

Generally, diarrhea, vomiting, and abdominal pain occur initially, followed by neurological dysfunction including reversal of temperature sensation, muscular aches, dizziness, anxiety, sweating, and numbness and tingling of the mouth and digits. Paralysis and death have been documented, but symptoms are usually less severe although debilitating. Recovery time is variable, and may take weeks, months, or years. Rapid treatment (within 24 hours) with manitol is reported to relieve some symptoms. There is no antidote, supportive therapy is the rule, and survivors recover. Absolute prevention of intoxication depends upon complete abstinence from eating any tropical reef fish, since there is currently no easy way to measure routinely ciguatoxin or maitotoxin in any seafood product prior to consumption, (Nielsen, and Tønseth, 1991; Partensky and Sournia, 1986; Partensky *et. al*., 1988; Partensky *et. al*., 1991; Passow, 1991; Perez *et. al.*, 2001; Rafuse *et. al*., 2004; Schnorf *et. al*., 2002; Tangen 1977; Taylor *et. al*., 1995; Tillmann, 2004).

4. CONCLUSION

World Public Health authorities must assume responsibility to take proper consideration of the problem, establish a proper diagnosis and universal therapy program, and develop specific assay kits for red tide toxins invested in the wide variety of toxins and in their disperse geographic localization, and chemical test capable of detecting all toxins in any suspected food source. The demonstration of specific antibodies against brevetoxins suggested that immunoassays were possible for detecting these toxins in marine food sources.

Methods of protecting marine life need to be improved as they are still very primitive at present. A complete picture of red tide can be produced if water quality data meteorological data and bioassay results are suitably integrated. Scientific report of red tide from the world as international news including the area affected by the red tide is needed. In RO and MSF plants conditions there is no risk of drinking water from toxic red tide organisms and their toxins products.

Aircraft and Satellite observations with minimal need for sea-truth data are the preferred method of remote sensing. It is very important to develop special channel on the internet as database information worldwide from scientific and managerial expertise on red tide for rapid access to international and information assistance when red tide catastrophes occur. There for:

1. Public health problems arising directly or indirectly as a result of exposure to the noxious metabolites produced during red tides are global in occurrence. Part of the problem is establishing a proper diagnosis and therapy program and in developing specific assay kits for red tide toxins and in their dispersed geographic localization.
2. Governments must encourage more detailed studies of PSP and DSP red tides in order to avoid or minimize future blooms and their effects.
3. World Public Health authorities must assume responsibility to take proper consideration of the problem, establish a proper diagnosis and universal therapy program, and develop specific assay kits for red tide toxins invested in the wide variety of toxins and in their disperse geographic localization, and chemical test capable of detecting all toxins in any suspected food source
4. Aircraft observations with minimal need for sea-truth data are the preferred method of remote sensing. It is very important to develop special channel on the internet as database information worldwide from scientific and managerial expertise on red tide for rapid access to international and information assistance when red tide catastrophes occur.

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