Harmful Algal Blooms: Physiology, Behavior, Population Dynamics and Global Impacts- A Review

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ABSTRACT: Harmful, toxic algae are now considered as one of the important players in the newly emerging environmental risk factors. The apparent global increase in harmful algal blooms (HABs) is becoming a serious problem in both aquaculture and fisheries populations. Not only has the magnitude and intensity of public health and economic impacts of these blooms increased in recent years, but the number of geographic locations experiencing toxic algal blooms has also increased dramatically. There are two primary factors causing HABs outbreaks. The natural processes such as upwelling and relaxation, and the anthropogenic loading resulting in eutrophication. However, the influence of global climate changes on algal bloom phenomenon cannot be ignored. The problem warrants development of effective strategies for the management and mitigation of HABs. Progress made in the routine coastal monitoring programs, development of methods for detection of algal species and toxins and coastal modeling activities for predicting HABs reflect the international concerns regarding the impacts of

HABs. Innovative techniques using molecular probes will hopefully result in development of rapid, reliable screening methods for phycotoxins and the causative organisms.

KEYWORDS: Harmful algal blooms, phycotoxins, eutrophication, toxigenic algae, biotoxin, upwelling.

1. Introduction

Marine phytoplankton develops over various scales of space and time (Harris, 1986), one of which includes the massive, relatively sudden and local or geographically restricted outbursts of one or sometimes a few species. Such phenomena are given one of a few names: 'red tides', 'brown water', 'discoloured waters', 'colored waters'. All these terms imply that algal proliferation is so intense as to modify the appearance of the sea surface as perceived by human eye. Red tides refer to the bio optical phenomena and rely implicitly on a threshold of a plant biomass or light extinction; several attempts have been made to estimate this threshold in terms of cell numbers, total cell volume and carbon content (Kim *et al.* 1993) or chlorophyll content (Allen, 1946; Saunders and Glenn, 1969; Tett, 1987). On the other hand, blooms (originally, the regular spring growth in temperate waters) are ecophysiological rather than bio-optical in meaning; being typically harmless. Phyoplankton or single celled marine plants are organisms at the base of the food chain that are the food for many higher organisms. These microscopic planktonic algae are critical food for filter-feeding bivalve fish as well as the larvae of commercially important crustaceans and finfish. In most cases, the proliferation of planktonic algae, so-called ('algal blooms', up to millions of cells per litre) is therefore beneficial for aquaculture and wild fisheries operations (Hallegraeff, 1993).

In some situations algal blooms can have a negative effect, causing severe economic losses to aquaculture, fisheries and tourism operations and having major environmental and human health impacts. Red tides may be harmless or harmful to the surrounding fauna, man included, and they can be dramatically harmful when the causative organism produces a toxin (Sournia, 1995). However, toxin-producing microalgae may be deleterious even when present in low or moderate concentrations. Therefore:
(i) red tides are not all toxic
(ii) toxic phytoplankters do not all produce red tides.

2. Definition

Generally, approximately 1 million cells per liter of seawater equals a bloom which may also produce biotoxins. These events are termed harmful algal blooms or HABs. Attendees at the 1984 ICES Exceptional Plankton Blooms Meeting concluded that exceptional blooms should be defined in terms of the deviation from the normal cycle of phytoplankton biomass (Parker, 1987). HABs were defined as phytoplankton blooms where the effects are noticeable and or harmful. The term 'harmful' algal blooms (HABs) referred to as 'red tides", has been applied to a class of blooms increasingly thought to have unique properties (Smayda, 1997). This emergent bloom phenomenon is rapidly expanding in global coastal waters (Anderson, 1989; Smayda, 1990; Hallegraeff, 1993).

Richardson (1989) defined a bloom as an outburst resulting in 'high biomass', stating also that 'a red tide is an exceptional bloom'. According to Richardson (1997), phytoplankton (also called "microalgal or algal") bloom is the rapid growth of one or more species leading to an increase in biomass of the species. Exceptional phytoplankton blooms have also been defined as "those which are noticeable, particularly to the general public, directly or indirectly through their effects such as visible discoloration of water, foam production, fish or invertebrate mortality or toxicity to human (ICES,

1984). Reid *et al.* (1987) applied the term "exceptional" to blooms of microalgae, accompanied by 'deleterious side-effects.' Cellular abundance levels have also been used to define red tide blooms. Kim *et al.* (1993), classify blooms as 'red tides' (in Korean waters) when abundance of the causative species (>30 μm) exceeds 1000 cells ml^{-1}. This criterion results in about 200 diatom species being classified as red tide species, which represents about two thirds (65%) of the total number of species classified as such (Sournia, 1995).

Blooms, as a phenomenon collectively have properties other than biomass, numerical population density, and the potential of the bloom species to inflict harm. Harmful effects may therefore accompany very high (~10^8 cells l^{-1}) or very low (<10^3 cellsl^{-1}) biomass. Furthermore, bloom events may occur at very low (<10^3 cells l^{-1}) or very high (~10^8 cells l^{-1}) cellular abundance, and may be due to very small or very large species' size. In both cases same species may be simultaneously inimical to several trophic components or have multiple effects and for different reasons. The presence of a toxic species does not necessarily lead to a deleterious impact. Such effects require a threshold population density level, above which it becomes inimical. The population level at which this occurs need not be accompanied by seawater discolouration, contrary to anecdotal perceptions of red tides.

3. Harmful Algal Blooms

3.1 History

It is often cited that the first written record (1000 B.C.) of a harmful algal bloom appears in the Bible: '…all the waters that were in the river were turned to blood. And the fish that was in the river died; and the river stank, and the Egyptians could not drink of the water of the river' (Exodus: 20-21, Moore, 1977; Hallegraeff, 1995). However, there is fossil evidence that harmful algal blooms were occurring long before this. Noe-Nygaard *et al.* (1987) have suggested, on the basis of the distribution of dinoflagellate cysts and bivalve shells in fossil sediments taken from island of Bornholm in the Baltic Sea, that toxic dinoflagellates caused mass mortalities of bivalves on several occasions dating back to about 130 million years ago. Dale *et al.* (1993) have also studied the distribution of fossil dinoflagellate cysts and presented evidence suggesting bloom formation by the toxic dinoflagellate *Gymnodium catenatum* in the Kattegat-Skagerrak long before anthropogenic activities could have influenced these waters. The species first appeared in the region about 6000 years ago and achieved a minor peak in production about 4500 years ago. After that it occurred in relatively low numbers until about 2000 years ago. During the next 1500 years periodic blooms of the species took place. In the sediment records for the last 300 years these workers found no evidence of this dinoflagellate. The fossil records emphasize two important points with respect to exceptional algal blooms (Richardson, 1997):

i. The occurrence of such blooms is not a recent phenomenon i.e., anthropogenic perturbation of the environment is not a necessary prerequisite for all exceptional blooms

ii. Phytoplankton species are not necessarily permanent residents of a given body of water.

3.2 Causative Organisms

The numbers of species within each phytoplankton class implicated in the formation of harmful bloom or red tides have been estimated by Sournia (1995). According to him the dinoflagellates (*Dinophyceae*) comprise the class which is numerically the largest and it is also within this class that one finds the greatest numbers of both exceptional / harmful bloom forming and toxic species. Estimates show that 5.5–6.7% of the known phytoplankton species in the world's oceans, i.e. about 200 species, have been identified as causing exceptional or harmful blooms (Sournia, 1995; Skulberg *et al.* 1993; Steidinger, 1996; Hasle and Syvertsen, 1996; Throndsen, 1993; Taylor *et al.* 1995; Landsberg, 2002). About half of these species are dinoflagellates, while diatoms are probably the second most important algal class in terms of causing exceptional or harmful blooms (Sournia, 1995).

However, toxic species are about four times less numerous (60-78) than red tide species, but still represent 1.8-1.9% of the world flora (Sournia, 1995), with numbers increasing by not more than 20-30 species (Bomber, 1991; Shimizu, 1987; Elbrachter, 1990; Hawser *et al.* 1991). Approximately 75% of the species that have been identified as being toxic belong to the Dinophyceae and most of these belong to the order Peridiniale, Gymnodiniales and Dinophysales. Four genera (Alexandrium, Dinophysis, Gymnodium and Prorocentrum) dominate in terms of causing toxic blooms (White, 1979; Skulberg *et al.* 1993; Steidinger, 1996; Steidinger *et al.* 1996; Taylor *et al.* 1995).

3.3   Impacts of Harmful Algal Blooms

There is an increase in phytoplankton events in number, geographical extent, frequency, number of species, intensity and damages (Boalch, 1983; Anderson, 1989; Smayda, 1990; Sournia *et al.* 1991; Hallegraeff, 1993).

Three modes of negative impact have resulted in characterrising certain species as 'harmful'. They are described as : 'toxic' (Anderson *et al.* 1985), 'noxious' (Horner *et al.* 1990) or 'nuisance' (Hallegraeff, 1993). The three major impacts of the harmful blooms are:

1.   A number of toxins produced by phytoplankton that affect humans also affect fish (White, 1977; Gosselin *et al.* 1989; Riley *et al.* 1989; Robineau *et al.* 1991; Noga, 1998). In addition, however some bloom forming phytoplankton species that have not been shown to be toxic to human produce toxins that affect fish or other marine organisms (Moshiri *et al.* 1978; Granmo *et al.* 1988; Heing and Campbell, 1992; Eilertsen and Raa, 1994). Phytoplankton species which cause fish-poisoning (Ciguatera) and shell-fish vectored poisoning of humans are clearly harmful (Hallegraeff, 1993). Public health concerns over-bloom related, shellfish borne diseases affecting seafood safety are an emergent global concern.

2.   Species whose blooms induce die-offs of farmed fish and cultivated shellfish leading to aquacultural financial losses are also harmful. Gelatinous secretion during an unusual bloom of the diatom *Coscinodiscus wailessi* in the North Sea was so great that the weight of the slime tore fishing nets and impaired the fishery (Boalch and Harbour, 1977). Deflections of migratory routes (Savage, 1930), emigrations (Lenanton *et al.* 1985) and other avoidance reactions (Potts and Edwards, 1987) of commercially important fish during harmful blooms also occur.

3.   The third major category of impact-field and experimental evidence reveals at least eight different modes and mechanisms by which harmful species cause mortality or physiologically impair target victims (Table 1). These fall in two general types:

i.   Non-chemical effects: Effects, which lead to starvation or cause harmful mechanical and physical damage.

ii.   Chemical effects: Attributable to physical-chemical reaction, phycotoxins or other metabolites.

Starvation impaired growth and fecundity resulting from nutritional and prey size mismatches are well-known experimental results, (Smayda, 1997). Excess prey density ($> 250 \times 10^6 \, l^{-1}$) of the brown tide species, *Aureococcus anophagefferens*, led to a spawning failure and mortality of the mussel, *Mytilus edulis* (Tracey, 1988; Nuzzi and Waters, 1989; Smayda and Villareal, 1989). This organism has also been shown to reduce or stop filter feeding in some shellfish (Tracey, 1988; Gallager *et al.* 1989; Gainey and Shumway, 1991).

Mechanical damage leading to death of larval fish can result from collisions ("bumping") with dinoflagellates at high bloom population densities (Ho and Zubkoff, 1979). Lethal piercing of gill filaments of penned and feral population of fish and crustaceans by the setae of *Chaetoceros* diatoms also can occur (Horner *et al.* 1997; Rensel, 1993; Yang and Albright, 1992; Tester and Mahoney, 1995). Respiratory failure, haemorrhaging, or bacterial infection resulting from such particle irritation can cause mortality. Extra cellular polymer secretions of harmful species can induce mortality in three physical ways; as a viscosity barrier (Partensky *et al.* 1989; Potts and Edwards, 1987); by forming a gelatinous barrier (Taylor *et al.* 1985) and through mucoid layer reduction (Endo *et al.*

1985). Gill clogging, elevated shear stress, which tears gill filaments and osmoregulatory failure can lead to death (Endo *et al.* 1985, 1992).

Table 1. Mortality modes and impact mechanisms of harmful species and their blooms (Source : Smayda, 1997).

Modes	Impact
Starvation	NutritionL Mismatch Size mismatch Excess prey density
Mechanical	"Bumping" Particle irritation (*chaetoceros spp.*)
Physical	Viscosity barrier (*Gyrodinum areolum*) Gelatinous barrier (*Ceratulina pelagica*) Mucoid layer reduction (*Chattonella marina*)
Anoxia	*Ceratium* blooms
NH_4 toxicity	*Noctiluca* blooms
Phycotoxins	Direct vs. vectored toxicity Saxitoxin Brevetoxins Hemolysins Cytotoxins
Allelopathic	
Ambush predation	*Pfiesteria piscicida*
Unresolved	*Aureococcus anophaghagefferens*

Among the chemically harmful mechanisms die off due to anoxia or hypoxia following blooms of large, relatively ungrazed species such as *Ceratium* are well-known (Graneli *et al.* 1989; Mahoney and Steimle, 1979). *Noctiluca* produced a remarkably simple ichthyotoxin: NH_4 (Okaichi and Nishio, 1976). In contrast phycotoxins have much more elaborate effects. Mortality can occur through direct ingestion of endotoxin of the harmful species (Carmichael, 1996), upon exposure to secreted toxins (exotoxins; Hansen *et al.* 2000) or from toxin vectoring through the food web, accompanied by conformational changes in the toxic principles and their potencies (Shimizu, 1987; Shimizu *et al.* 1989; Yasumoto and Murata, 1993). The impact may be on a directly targeted predator or may lead to indiscriminate die-offs and physiological impairment during food web vectoring (Landsberg, 2002). Specific toxins are water or lipid soluble and have cytolytic, haemolytic (Hansen *et al.* 2000) or neurotoxic activity (Perovic *et al.* 2000; Daugbjerg *et al.* 2000). Allelopathic substances are distinguished from phycotoxins in being secondary metabolites (Subba Rao *et al.* 1995; Wright and Cembella, 1998; Naviner *et al.* 1999) and both can co-occur within a given harmful species. Allelopathic substances tend to be directly targeted and may physiologically impair, stun, repel, induce avoidance reactions or kill grazers (Plumley, 1997; Wyatt and Jenkinson, 1997, Fistarol *et al.* 2004). Examples of these diverse modes of impact have been demonstrated by Smayda (1992). A relatively newly discovered, remarkable ambush predatory behavior of the harmful dinoflagellate *Pfiesteria piscicida* has been presented by Burkholder and Glasgow (1995, 1997), Burkholder *et al.* (1992, 1999, 2001), and Berry *et al.* (2002) and Vogelbein *et al.* (2002).

3.4 Types of Harmful Algal Blooms

Harmful and toxic species are a small subset of the total phytoplankton flora and little is known about the inter-annual variability with respect to the relative frequency of occurrence of different phytoplankton species. The three different types of harmful HABs are:

1. In some cases, a non-toxic bloom forming alga becomes so densely concentrated that it can generate anoxic conditions resulting in indiscriminate kills of both fish and invertebrates due to oxygen depletion. For example, Dinoflagellates, *Gonyaulax polygramma* (Lam, 1988) *Noctiluca scintillans*. (Okaichi and Nishio, 1976; Chen and Qi, 1991; Ho and Hodgkins, 1992; Xie, *et al.* 1993; Hong and Huang, 1989; Qi *et al.* 1992, 1993, 1995; Qi and Li, 1994) and Cynobacterium: *Trichodesmium erythraeum* (Endean *et al.* 1993).

2. Species that produce potent toxins that can find their way through the food chain to humans (Table 2), cause a variety of gastrointestinal and neurological illnesses such as Paralytic Shellfish Poisoning (PSP; Lush and Hallegraeff, 1996). For example, Dinoflagellates, *Alexandrium acatanella* (Cembella *et al.* 1987), *Alexandrium minutum* (Chen and Chou, 2001) *Alexandrium fundyense* (Anderson *et al.* 1990). Most humans who experience paralytic shellfish poisoning have consumed toxic bivalves (Shumway, 1990) and toxic gastropods and crustaceans (Shumway, 1995). Whales and porpoises can also become victims when they receive toxins through the food chain via contaminated zooplankton or fish (Geraci *et al.* 1989).

3. The third type of harmful algal bloom has been apparent only as a result of the increased interest in intensive aquaculture systems for finfish. Some algal species can seriously damage fish gills, either mechanically (Berry *et al.* 2002) or through production of haemolytic substances (Hansen *et al.* 2000).

Table 2. Potency of some algal blooms (Source: Wyatt, 1998)

Toxin	Source	Toxicity
Cyanide		1
Muscarin	*Amanita muscaria*, fungus	9
Okadaic acid	Algae, dinflagellates (e.g. *Dinophysis* spp.)	50
Domoic acid	Algae, diatoms (*Pseudo-nitzschia* spp.)	80
Prymnesium	Algae, haptophytes (e.g. *Prymnesium parvum*)	350
Cobra toxin	Cobra snake	500
Saxitoxin	Algae, dinoflagellates (e.g. *Alexandrium* spp., *Pyrodinum bahamense*)	1 100
Ciguatoxin	Algae, dinoflagellates (*Gambierdiscus toxicus*)	22 000
Tetanus toxin	Bacterium (*Clostridium tetani*)	100 000 000

4. Harmful Algal Events

Upwards of ten thousand named species of plank-toxic algae are found in the upper layers of the oceans and continental waters. Excluding those which respond to eutrophication and associated processes and which can give rise to biomass problems, less than a hundred of these have so far been recorded as harmful (Wyatt, 1998).

Three distinct periods of harmful algal events can be distinguished:

1. Events due to accumulation of biomass of microplanktonic cells (larger than 20 μm in diameter), often associated with natural or anthropogenic eutrophication, which are normally composed of different species. In these cases, unrestrained growth of algae can lead to very high cell concentration. If these populations are neither grazed nor dispersed by winds and tides, their death and decay can lead to deoxygenation in the sediments where they settle

(Okaichi and Nishio, 1976; Onoue, 1990; Olsgard, 1993) in the lower water column and consequent mortalities of other components of the ecosystem are affected. Anoxia and or bacterial infection in combination with the damaged gill tissue leads to mortality of the fish (Jones and Rhodes, 1994; Tester and Mahoney, 1995). Hypoxia or anoxia resulting from the respiration or decay of dense blooms of phytoplankton can also, on its own, lead to fish or shellfish kills- especially of caged fish that are unable to swim from the affected area (Steimle and Sindermann1978; Taylor *et al.* 1985).

Events of this kind are common, often annual, in many parts of the world, especially in shallow, semi-enclosed and poorly furnished estuaries and lagoons but less frequently in open sea areas such as the Northern Adriatic Sea or the New York Bight, when the balance between nutrient supplies and flushing rates tips in favour of such events (Wyatt, 1998)

2. There are blooms of single microplanktonic species with particular morphological, physiological or biochemical features which interact in negative ways with other organisms in the environment, either directly or through the food web. For example, (i) some diatoms in the genus *Chaetoceros* cause much damage to the gills of caged fish (Ianora *et al.* 1995); (ii) the diatom *Coscinodiscus wailesii*, a water blooming species competes for nutrients with the red microalgae *Porphyra* which is farmed in Japan and other countries; and (iii) the dinoflagellate *Gyrodinium aureolum,* (frequently referred to as *Gymnodium*), (Mahoney *et al.* 1990) can cause potentially lethal respiratory distress in fish and other organisms brought about by secretion of large quantities of mucilaginous materials (Wyatt, 1998). But many harmful species in this group are poisonous and produce compounds which can cause paralytic, neurotoxic, diarrhetic and other effects when transmitted through the food web to vertebrates including man.

3. Events due to accumulation of biomass of ultraplanktonic cells (between 0.2 and 20 μm in diameter). Events of this kind are less common, but can be very persistent as in the case of the Texas 'brown tide' which has persisted in Laguna Madre for more than seven years (Wyatt, 1998).

5. Rise of Algal Blooms

The issue of a global increase in harmful algal blooms has been raised previously in Conference proceedings (Anderson 1989; Hallegraeff *et al.* 1990; Smayda, 1990), while the biology of toxic dinoflagellates has been reviewed by Steidinger (1993) while the facts of algal blooms on aquaculture have been summarized by Shumway (1990). Four possible explanations for the apparent rise of algal blooms are:

- Increased scientific awareness of toxic species
- Increased utilization of coastal waters for aquaculture
- Stimulation of plankton blooms by cultural eutrophication
- Unusual climatologic conditions and transport of dinoflagellate resting cysts either in ships ballast water or associated with movements of shellfish stocks from one area to another.

5.1 Increased Scientific Awareness of Toxic Species

Reports of harmful algal blooms associated human illnesses or damage to aquaculture operations are receiving increased attention in media and scientific literature. Concerns about aquatic biotoxin, particularly from micro-organisms, stems from an increased dependency on aquatic environments for food and potable water as well as on their potential source for drugs and other chemicals and modern aspects of genetic engineering (Colwell. 1983). These potent materials from natural sources are of more than just academic interest, since they may cause massive animal mortalities, as well as legal,

recreational and human health problems (Hashimoto, 1979: Carmichael, 1981, 1986; White, 1982; Baden, 1983; Ragelis, 1984; Van Dolah, 2000). Until 1988, the type of species *A.minutum* was known to be in Egypt only (Halim, 1960). But it has recently been reported in Australia, France, Spain, Portugal, Italy, Turkey and the east coast of North America (Hallegraeff *et al.* 1991).

5.1.1 Toxic Algal Blooms

Relatively few species among the primary producers produce potent materials which may affect organisms at other trophic levels in the food chain. In general a biotoxin is a harmful substance synthesized by one species and adversely affects other species when present in extremely small quantities. Toxins are characterized by their potency (μg toxin kg^{-1} body mass), their specificity of action (e.g. ion channels in membranes) and, in some cases, their reversibility. The latter characteristic has attracted the attention of physiologists and pharmacologists who study basic phenomena at the membrane level (Hashimoto, 1979; Adelman *et al.* 1982; Kao and Walker, 1982; Baden, 1983; Sasner *et al.* 1984).

In general, the major producers of toxins are the dinoflagellate and diatom groups in the marine environment and cyanobacteria in fresh and brackish waters. Dinoflagellates constitute the most prevalent of the toxic marine algae and their effects are widespread and disastrous. The effects may be generally classified as paralytic shellfish poisoning, diarrhetic shellfish poisoning, ciguatera poisoning and ichthyotoxicity.

5.1.2 Diarrhetic Shellfish Poisoning

The phenomenon of diarrhetic shellfish poisoning (DSP) was first documented in 1976 from Japan where it caused major problems for the Scallop Fishery (Yasumoto *et al.* 1978). The first dinoflagellate to be indicated was *Dinophysis fortii* (in Japan). It was soon followed by the *D. acuminata* (in Europe), *D. nutra*, *D. rotunda* and the benthic dinoflagellate *Prorocentrum lima* (Aune and Yndestad, 1993) and *Prorocentrum excavata* (Rausch de Traubenberg and Morlaix, 1995). The DSP toxins include Okadaic acid and polyether toxins, Dinophysistoxins (DTX1-4) (Aune and Yndestad, 1993), pectenotoxins and yessotoxins (Yasumoto, 1985; WHO, 1984). A number of dinoflagellate species (Hallegraeff, 1993), especially from the genus Dinophysis, have been identified as producing these toxins. The detection of DSP toxins in the heterotrophic dinoflagellates *Protoperidinium occanicum* and *P. pellucidum* (Lee *et al.* 1991) may reflect their feeding on Dinophysis.

The symptoms of DSP include stomach pain, nausea, vomiting and diarrhoea (WHO, 1984). No fatalities directly associated with DSP have ever been recorded (Richardson, 1997). However, some of the toxins associated with DSP may promote the development of stomach tumours (Suganuma *et al.* 1988).

5.1.3 Amnesic Shellfish Poisoning

Amnesic shellfish poisoning (ASP) gets its name from the fact that one of the symptoms of the poisoning is loss of memory. It was first recognized in 1987 on Prince Edward Island on Canadian East Coast when a serious incident occurred which caused three human fatalities and 105 cases of acute poisoning following the consumption of blue mussels *Mytilus edulis* (Bates *et al.* 1989). Clinical symptoms of ASP are nausea, vomiting, abdominal cramps, diarrhea, memory loss, decreased level of consciousness, seizures, confusion and disorientation (Wright *et al.* 1989; Perl *et al.* 1990; Todd, 1990; Nijjar and Nijjar, 2000). The cause of this poisoning was traced to a bloom dominated by the pinnate diatom, *Pseudo-nitzschia multiseries* (formerly *Pseudonitzschia pungens f. multiseries*) which produces the neurotoxin domoic acid (DA) (Subba Rao *et al.* 1998; Bates *et al.* 1989, 1995). Lundholm *et al.* (1994) have shown that the diatom *Pseudo-nitzschia seriata* also produces domoic acid at levels similar to those observed with *Pseudo-nitzschia multiseries*. This diatom is common in

colder waters of the Northern Hemisphere. There is also an indication in the literature that several other common diatom species may produce domoic acid e.g., *Fragilaria* spp (Pauley *et al.* 1993), *Amphora Coffeaeformis* (Maranda *et al.* 1990), *Nitzchia pungens .f. multiseries, N.pseudo delicatissima* and *N. pseudo seriata* (*Pseudonitzchia australis Frenguelli*) (Subba Rao *et al.* 1998, Martin *et al.* 1990, Work *et al.* 1992; 1993). The production of DA has been identified in nine species of *Pseudonitzschia* and one species of *Nitzschia* (Bates *et al.* 1989; Bates, 2000). These diatoms produce toxins only after the onset of the stationary growth phase which is induced by either phosphate or silicate deficiency (Bates *et al.* 1991).

5.1.4 Paralytic Shellfish Poisoning

Existence of paralytic shellfish poisoning (PSP) has been recognized for many centuries. This form of shellfish poisoning induces muscular paralysis and in severe cases can lead to death through paralysis of respiratory muscles. PSP is caused by toxins often referred to collectively as "PSP toxins". The structure of the most potent component of the PSP toxin is called saxitoxin and referred to as STX. Its structure was determined about 30 years ago. It is now known that PSP is caused not by just one toxin but a group of toxins, based on the backbone structure of the saxitoxin molecule (Cembella *et al.* 1987; Hall *et al.* 1990). PSP is caused by one or more of about 18 different toxins which, include saxitoxins, neosaxitoxins and gonyautoxins (Fremy, 1991). With the exception of the African and parts of the South American Coasts, PSP is a worldwide problem (Hallegraeff, 1993), although some evidence (Baddyr, 1992) suggests that PSP incidents have occurred along the Morrocan Coast during the 1980s and 1990s.

Paralytic shellfish toxins are produced by dinoflagellates – approximately 11 *Alexandrium* species, *Gymnodium catenatum*, and *Pyrodinium bahamense* var. *Compressum-* and by cyanobacteria *Anabena circinalis, Aphanizomenon flos-aquae, Cylindrospermopsis raciborskii, Lyngbya wollei* and *Planktothrix* sp. (Scholin, 1998; Balech, 1995). Anderson *et al.* (1990) examined different PSP producing dinoflagellates (*Alexandrium fundyense* and *A. tamarense*) and identified differences in toxin production that were directly related to changes in growth rate associated with the various stages of the life cycle.

Shellfish are the usual vectors of PSP to human consumers. However, crustaceans can also accumulate PSP toxins (Desbiens and Cembella, 1995). PSP toxins have been found in the zooplankton and guts of dead and diseased fish in the vicinity of blooms (White, 1977). Some PSP toxins have also been found in the muscle of the affected fish and there is at least circumstantial evidence that PSP toxins may be implicated in mortalities of marine animals (Hofman, 1989; Anderson and White, 1992). A number of studies have indicated reduced grazing rates by copepods on PSP containing phytoplankton (Ives 1985, 1987; Huntley *et al.* 1986; Turriff *et al.* 1995).

5.1.5 Neurotoxic Shellfish Poisoning

Blooms of the dinoflagellate *Ptychodiscus breve* (formerly *Gymnodium breve*) have been associated with human poisoning which is characterized by airborne bronchial irritation and neurological symptoms but no paralysis (Baden, 1989; Schulman *et al.* 1990). Brevetoxins, which have been isolated in pure form (Chou *et al.* 1985; Baden and Mende, 1982), have high molecular weight, and are lipid soluble, non-nitrogen containing complex polyethers.

5.1.6 Ciguatera

Ciguatera poisoning has been known for centuries in subtropical and tropical areas (Swift and Swift, 1993; Lewis *et al.* 2000). During the period, 1960-1984, over 24,000 cases were reported in French Polynesia alone (Hallegraeff, 1993). Ciguatera is caused by benthic dinoflagellates such as *Gambierdiscus toxicus*, (Faust, 1995; Chinain *et al.* 1999); *Ostreopsis siamensis, Coolia monotis, Prorocentrum lima* and related species (Anderson and Lobel, 1987; Hallegraeff, 1993, Hu *et al.*

1996). These toxins are transported through the food chain and usually reach humans through the consumption of finfish (Lewis and Holmes, 1993).

There are chemical similarities between ciguatoxin and brevetoxins and the toxic effects of both these types of toxins are apparently caused by the changes in sodium ion influx in the affected organisms (Baden, 1989, 1995; Trainer *et al.* 1990). Many of the symptoms associated with ciguatera poisoning also resemble NSP and in severe cases, death can result from circulatory collapse or respiratory failure due to NSP (WHO, 1984). Ciguatoxins have also been implicated in marine mammal kills (Hofman, 1989).

5.1.7 Cyanobacterial Toxins

Toxic cyanobacteria (blue-green algae) blooms are most often associated with fresh waters. However, it has been recently shown that blooms of the cyanobacterium *Trichodesmium thiebautii* occurring in the open Ocean of the US Virgin Islands can be toxic (Hawser and Codd, 1992). Toxicity tests conducted with *Trichodesmium* sp. cells on the copepod *Acartia tonsa* demonstrated that intact cells were not toxic to *A. tonsa,* but the homogenized cells were (Guo and Tester, 1994). Thus the authors suggested that the alga contained one or more intracellular biotoxins.

Brackish waters can also harbour toxic cyanobacteria blooms. Some Australian estuaries and inlets (Huber and Hamel, 1985; Lenanton *et al.* 1985; Blackburn and Jones, 1995) and the Baltic Sea (Kononen, 1992) are examples of brackish areas where the toxic cyanobacterium *Nodularia spumigena* frequently forms blooms. Nodularin, the toxin associated with *Nodularia spumigena* which is hepatotoxic (Kiviranta *et al.* 1991; De Mott *et al.* 1991) may promote tumours with chronic exposure (Falconer, 1991). Given the discovery that some cyanobacterial toxins may stimulate tumour formation (Falconer, 1991; Carmichael, 1992), it is likely that there will be increasing awareness directed towards this type of phytoplankton bloom in coming years.

5.1.8 Other Types of Toxic Bloom

Toxic blooms of *Prymnesiophytes* are frequently reported to be a problem for aquaculture facilities. For example, a massive bloom of *Chrysochromulina polylepis* in the Skaggerak / Kattegat during May-June, 1988, caused havoc with salmon raising facilities along the Swedish Norwegian Coasts (Moestrup, 1994). This bloom also affected wild stocks of a number of different genera (Olsgard, 1993) and in addition to causing mortalities of mature animals, it has been shown that the presence of the phytoplankton inhibits the reproduction process in the ascidian, *Ciona intestinalis* and the mussel *Mytilus edulis* (Granmo *et al.* 1988). Thus Granmo *et al.* (1988) suggested that the phytoplankton bloom might have had long term effects in the affected area by reducing larval settlement and recruitment for these and possibly other species. Other plankton organisms were also shown to be affected by this *Chrysochromulina* bloom (Nielsen *et al.* 1990). *Chrysochromulina leadbeatri* caused mortalities in caged salmon among the northern Norwegian coast in 1991 and the two *Prymnesium* (*P. patelliferum* and *P. parvum*) have also caused mortalities in aquaculture facilities for salmon and rainbow trout (Aune *et al.* 1992; Meldahl *et al.* 1994, 1995).

There can be mechanical interaction between phytoplankton and the gills which leads to gill damage and ultimately suffocation of the fish. Diatoms are often implicated in such events (Bell, 1961; Taylor *et al.* 1985; Farrington, 1988; Rensel, 1993; Kent *et al.* 1995; Tester and Mahoney, 1995). The raphidophyte *Heterosigma akashiwo* presents a serious problem for aquaculture in many parts of the world including Japan, North America, Asia and New Zealand (Haigh and Taylor, 1990; Change *et al.* 1990; Black *et al.* 1991; Mackenzie, 1991; Qi *et al.* 1993; Honjo, 1994). Affected salmon shows signs of gill and intestinal pathology and death has been attributed to impairment of the gills' respiratory and osmoregulatory functions (Change *et al.* 1990). It has been suggested that the toxic effects elicited by this organism on salmon may be brought about by the formation of toxic concentrations of oxygen radical and hydrogen peroxide (Yang and Albright, 1994). Dinoflagellate

Gyrodinium aureolum has been recognized as fish killer for many years (Tangen, 1977). Gill histopathology in affected fish has been reported to be similar to that observed in fish exposed to *Heterosigma* (Change *et al.* 1990).

It is now generally accepted that toxin production by *G. aureolum* is at least in some cases involved in mortalities caused by this organism (Boalch, 1983; Bullock *et al.* 1985; Heinig and Campbell, 1992). Jenkinson, (1989, 1993) suggested that *Gymnodium aureolum* and some other bloom forming flagellates may alter seawater characteristics through the production of extracellular organic material. This extracellular material should increase the viscosity of the medium surrounding the fish so that the energy expended in filtering water through the gills exceeds that which can be supported by the oxygen uptake. Phaecystis spp. are also considered to be harmful because they extrude protein-rich compounds that can be whipped into a stiff foam by wave action and which under certain hydrographic conditions can accumulate along beaches (Richardson, 1997).

A relatively new toxic dinoflagellate, *Pfiesteria piscicida* (Steidenger *et al.* 1996), was described by Burkholder *et al.* (1992). According to Burkholder *et al.* (1995) this dinoflagellate requires live finfish or their excrement in order to excyst and to release its potent neurotoxin. But more recently, research reports (Berry *et al.* 2002; Vogelbein *et al.* 2002) say that in the *Pfiesteria shumwayee,* the strain they have examined shows no signs of toxin. Instead, the single celled dinoflagellate alga kills by swarming over a fish and eating away its skin (Berry *et al.* 2002; Vogelbein *et al.* 2002; Kaiser, 2002; Rutherford, 2002).

5.1.9 Marine Algal Toxins: Biochemistry and Molecular Biology

The production of toxic compounds is a common, but not universal characteristic of harmful algal blooms (HABs). Only a few HAB toxins have been characterised and these are synthesised by only a limited number of algal species (Steidinger, 1993). Toxigenic algae may not have significant impacts on ecosystem processes as toxins affect viability, growth, fecundity and recruitment of a wide range of organisms.

The most widely known marine HAB toxins produce poisonous syndromes known as diarrhetic, paralytic, neurotoxic, and amnesic shellfish poisoning (DSP, PSP and ASP). Planktonic dinoflagellates synthesise the toxins associated with each of these poisoning events except that the ASP toxin, domoic acid is produced primarily by diatoms. Ciguatera, fish poisoning, another type of massive HAB toxin event is caused by benthic dinoflagellates.

From a chemical standpoint, the common marine HAB toxins are either alkaloids, poly ethers or substituted amines (Plumley, 1997). There are other toxins associated with HAB species such as superoxide and (or) hydroxyl radicals (Oda *et al.* 1992; Tanaka *et al.* 1992), lipoteichoic acids with hemagglutinin activity (Ahmed *et al.* 1995) and pentacyclic derivatives with a fuzed azine (Seki *et al.* 1995). Other toxic effects of HAB algae have been documented but the toxins remain to be elucidated (Burkholder and Glasgow, 1997). It is likely that many more marine HAB toxins will be identified and characterized as both utilization of marine resources and our ability to detect the toxins increases.

HAB toxins have complex chemical structures and they seem to be the end products of elaborate pathways and some of the enzymatic reactions are likely to entail highly unique and (or) specialized reactions (Plumley, 1997). Very little is known about the biochemistry of toxin synthesis. One of the difficulties is that HAB algae, which produce toxins are extremely difficult to culture (Shilo and Ashner, 1953) and remain largely resistant to most contemporary protocols routinely used with other microbes. At present rapid progress in understanding HAB algae at the molecular and biochemical levels is dependent on technological innovation. Some innovative analytical techniques developed promise to provide rapid and reliable detection of the causative organism and the toxins produced. Molecular probes are being applied to distinguish species-specific RNA and DNA sequences (Pierce and Kirkpatrick, 2001) for identification of HABs causing organisms.

5.2 Increased Utilisation of Coastal Waters for Aquaculture

With the increased problems of over fishing of coastal waters more and more countries are looking toward aquaculture as an alternative. Aquaculture operations act as sensitive 'bioassay systems' for harmful algal species and can bring to light the presence in water bodies of problem organisms known to exist there before. The increase in shellfish farming worldwide is leading to more reports of paralytic, diarrhetic and amnesic shellfish poisoning. On the other hand, increased finfish culture is drawing attention to algal species which can cause damage to the fishes delicate gill tissues (Taylor, 1988; Rensel *et al.* 1989). The diatoms long, hollow spines (setae) are studded with smaller barbs along their length. The setae can break off and penetrate the gill membranes of fish, with the smaller barbs preventing them from coming out (Tester and Mahoney, 1995; Rensel, 1993). Fish death may be caused by capillary haemorrhage, dysfunction of gas exchange at the gills, suffocation from an overproduction of mucus, or even from secondary infection of the damaged tissue (Bell, 1961, Bruno *et al.* 1989; Rensel, 1991; Yang and Albright, 1992, 1994).

A more widespread problem for fish farmers is the production by various algal groups of fatty acids or galactolipids (Bass *et al.* 1983; Bodennec *et al.* 1995) which damage the epithelial tissue of the gills. In experimental assay systems, these substances destroy red blood cells and therefore have been provisionally termed 'hemolysins' (Yasumoto *et al.* 1990). Algal species as diverse as the raphidophytes (*Hetersigma akashiwo* and *Chatonella antique*, the prymnesiophytes *Chrysochromulina polylepis* (Yasumoto *et al.* 1990) and *Prymnesium Parvum* (Kozaki *et al.* 1982) and the dinoflagellate *Gymnodium mikimotoi* (Arzul *et al.* 1995; Parrish *et al.* 1998) have been implicated (Change *et al.* 1990). The two prymnesiophyte flagellates *Chrysochromulina.* and *Prymnesium* produce substances that affect gill permeability leading to a disturbed ion balance (Yasumoto *et al.* 1990).

Toxicity by these species is promoted by phosphorous deficiency. A massive bloom (60000km^2, 10^7-10^8 cells l^{-1}) of *Chrysochromulina polylepis* occurred in May-June 1988 in the Skagerrak, the Kattegat, the Beltarea and the Sound between Denmark, Norway and Sweden (Rosenberg *et al.* 1988, Kaas *et al.* 1991). Fish deaths occurred due to damage of gill membranes which produced a lethal increase in the chloride concentration in the blood (Dahl and Tangen, 1993) and fish cages moved into less saline fjords were therefore less affected.

5.3 Increase of Algal Blooms by Cultural Eutrophication

Eutrophication as defined by Nixon 1995 "is an increase in the rate of supply of organic matter to an ecosystem". Eutrophication can occur via natural processes or as a result of human activities (cultural eutrophication). Correlations with eutrophication and human industrial development are of serious concern (Smayda, 1990, Sournia *et al.* 1991). A number of workers have suggested that there has been an increase in harmful blooms in recent years (Anderson, 1989; Smayda, 1990; Hallegraeff, 1993) and is often argued that the apparent increase in the occurrence of "harmful blooms" is linked to eutrophication. Indeed in some areas- especially those with limited water exchange such as fjords, estuaries and inland seas- there does seem to be good evidence for a stimulation of the number of algal blooms occurring by eutrophication. However, the relationship between the occurrence of harmful phytoplankton blooms and environmental conditions is complicated and anthropogenic perturbation of the environment is certainly not a pre-requisite for all harmful algal blooms (Richardson, 1997; Sellner *et al.* 2003). Thus, the occurrence of a harmful bloom may or may not have as one of its underlying causes a change in human activities or behavior.

Eutrophication from domestic, industrial and agricultural wastes can stimulate harmful algal blooms (Lam and Ho, 1989). In Hong Kong, this increase (mainly *Gymnodium nagaskiense*, *Gonyaulx polygramma*, *Nocticula scintillaus* and *Prorocentrum minutum*, shows a striking relationship with a 6-fold increase in human population in Hong Kong and the concurrent 2.5 fold increase in nutrient loading mainly contributed by untreated domestic and industrial waste (Okaichi, 1989, Lam and Ho, 1989). There is considerable concern (Ryther and Dunstan, 1971, Officer and

Ryther, 1980, Smayda, 1990) regarding the altered nutrient ratios in coastal waters. Improved wastewater treatment has been causing increases in the ammonia nitrate ratio of River Rhine discharge (Riegman and Noordeloos, 1991). The nutrient composition of treated wastewater is never the same as that of coastal waters in which it is being heavily discharged. Such altered nutrient ratios in coastal waters may favour blooms of nuisance flagellate species which replace the normal spring and autumn blooms of siliceous diatoms (Smayda, 1990).

The remarkable increase of foam producing blooms of the Prymnesiophyte *Phaeocystis pouchetti* which first appeared in Dutch Coastal waters in 1978 is probably the best studied example of this phenomenon (Lancelot *et al.* 1987). Algals species which are not normally toxic (*Chrysochromulina, Nitzschia pungens .f. multiseries, Prymnesium parvum*) may be rendered toxic when exposed to typical nutrient regimes (for example phosphate deficiency) resulting from cultural eutrophication (Shilo, 1981, Edvardsten *et al.* 1990, Bates *et al.* 1991). The 1998 bloom in the Kattegat of prymnesiophyte, *Chrysochromulina polylepi*s, is not usual in terms of biomass but in terms of its species composition and toxicity, has been related to a change in the nutrient status from nitrogen to phosphorus limitation (Maestrini and Graneli, 1991). In the Black Sea, a long term decrease in the Si:P ratio has been associated with an increase in the dinoflagellate blooms of *Prorocentrum cordatum* (Bodeanu and Usurelu, 1979). Changed patterns of land use, such as deforestation, can also cause shifts in phytoplankton species composition by increasing the concentrations of humic substances in land runoff. Acid precipitation can further increase the mobility of humic substances and trace metals in soils (Graneli and Moreira, 1990).

Agricultural run-off of phosphorus can also stimulate cyanobacterial blooms, for example *Nodularia spumigena* in the Baltic Sea and in the Peel Harvey Estuary, Australia. These species produce hepatotoxic peptides. *Nodularia microcystis* and neurotoxic alkaloids (*Anabena aphanizomenon*) can kill domestic and wild animals that drink from the shores of eutrophic ponds, lakes and reservoirs (Edler *et al.* 1985). Cyanobacteria also pose public health problems through the production of teratogens and tumour promoters, which are not acutely toxic (Carmicheal, 1989). The toxins accumulate in mussels and have been reported to contaminate drinking water. A neurotoxic factor has also been associated with some strains of the common marine, bloom forming tropical cyanobacterium *Trichodesmium thiebautii* (Hawser *et al.* 1991). While this organization can thrive under nutrient impoverished oceanic conditions, it is possible that coastal eutrophication can prolong or enhance bloom phenomena (Hallegraeff, 1993).

5.4   Stimulation of Algal Blooms by Unusual Climatological Conditions

The global change forcasts for the Period 1990-2100 in climate has been predicted to lead to a projected rise in temperature of 1.4-5.8°C with a 90% probability interval of 1.7-4.9% (IPCC 2001; Wigley and Raper, 2001). A direct consequence of global warming is thermal expansion of water and hence a rise in the sea level in 2100 by 0.1-0.9m (IPCC, 2001). Photosynthesis and plankton growth are basic processes to the marine ecosystem, and in general both rates double when temperature increases by 10°C (Raven and Geider, 1988). The qualitative conclusion from investigations by Peperzak (2003) on the effects of climate change in the marine ecosystem indicate that the risk of harmful dinoflagellate and raphidophyte blooms will increase rather decrease due to climate change.

Increased temperature leads to decreased viscosity (Vogel, 1996), which theoretically leads to (i) increased nutrient diffusion towards the cell surface, which is important when interspecific competition for nutrients takes place (ii) increased sedimentation rates, which will be important for nonflagellate species such as diatom. At the present, the dinoflagellate *Pyrodinium bahamense* is confined to tropical mangrove fringed coastal water of the Atlantic or Indo-West Pacific. According to McMinn (1989), a survey of fossil occurrences of its resting cyst *Polysphaeridium zokaryi* found in records going back to Eocene 50 million years ago, recorded by Bujak and others, indicated a much wider range of distribution in the past. At present, the dinoflagellate does not extend further south than

13

Papua New Guinea but in the Pleistocene, it ranged as far south as Sydney Harbour (McMinn, 1989). The first harmful implications of Pyrodinium blooms became evident in 1972 in Papua, New Guinea. Red brown water discolouration coincided with the fatal food poisoning of three children and mouse bioassays on shellfish from a house in the affected village subsequently established *Pyrodinium bahamense* as a source of paralytic shellfish poison (Maclean 1977). Maclean (1989) presented strong circumstantial evidence for a coincidence between Pyrodinium blooms and El Nino Southern Oscillation (ENSO) climatoligical events. Summer marine climate is dominated by the succession of periods of upwellings and other relaxation or downwellings. During the upwellings, water masses from as low as 60-70m are brought close to the surface, indicated by the lowering of the temperature to 23-25°C. These cool waters are also poor in oxygen (O_2) and rich in nutrients. Rich nutrient water is susceptible to trigger development of phytoplankton blooms (Claereboudt *et al.* 2001). Phytoplankton and other marine biota have relatively short life spans (roughly 1-3 days), under the right conditions they have the capacity to reproduce, or 'bloom' into exceptionally large numbers in a matter of days (Herring, 2002).

5.5 Transport of Dinoflagellate Cysts in Ship's Ballast Water or Associated with the Transfer of Shellfish Stocks

Dissemination of toxic species in the ballast water of commercial ships adds another concern (Hallegraeff and Bolch, 1991; Hallegraeff, 1993). Cargo vessel ballast water was first suggested as a vector in dispersal of non-indigenous marine plankton more than 90 years ago (Ostenfeld, 1908). The diatom *Odontella sinesis,* well known from the tropical and subtropical coasts of the Indo-Pacific, had not been reported in European waters until 1903, when it produced dense plankton blooms in the North Sea. Ostenfeld (1908) suggested that this species was introduced via the water or sediment contained in the ship's ballast tanks. Subsequently, Hallegraeff *et al.* (1990) confirmed this possibility by culturing the related diatom species *Odontella aurita* from a ballast water sample collected at the end of a voyage from Japan to Australia. The evidence of ballast water transfer of marine organisms other than microscopic algae is considerable and includes species of fish, crustaceans, polychaete worms and molluscs (Carlton, 1985).

The most effective measure to restrict spreading of dinoflagellate cysts via ship's ballast water would be to avoid ballasting during toxic dinoflagellate blooms in ports. Other options using heat or chemical treatment (chlorine, hydrogen peroxide) of ballast water either in hold or in onshore facilities have also been investigated (Rigby *et al.* 1991; Bolch and Hallegraeff, 1990).

Another anthropogenic vector for the dispersal of algae (especially their resting cysts) is with transfer of shellfish stocks containing phytoplankton in their digestive organs from one area to another (Hallegraeff, 1993).

6. Bloom Dynamics

6.1 Bloom Initiation

Generally, unusual wind/ weather/hydrographic conditions have been identified as factors preceding or accompanying the harmful algal blooms (Maestrini and Graneli, 1991; Rhodes *et al.* 1993). According to Fraga (1993) regions with coastal upwellings and river discharge areas are vulnerable to phytoplankton blooms. In some cases, wind, weather, tidal or other hydrographic conditions have been related to initiation of nearshore blooms by delivery of the organisms into the bloom region (Balch, 1981; Lindahl, 1983; Taylor *et al.* 1994). In some other cases, transport of phytoplankton blooms from offshore to inshore regions, longshore transport of blooms from one coastal region to another has also been documented (Franks and Anderson, 1992).

6.2 Bloom Composition

Individual phytoplankton species have different requirements and responses to the physical environment. Marine microalgae use inorganic nutrients such as nitrate, phosphates and sulphur, and convert them into the basic building blocks of living organisms- proteins, fats, carbohydrates. They also need trace elements such as silicon, iron and calcium (Kiørboe, 1993). The overall understanding of various phytoplankton species requirements and tolerances with respect to micronutrients is at a primitive stage. However, it is known that there are differences between species in terms of requirements for vitamin (Guillard *et al.* 1991; Graneli and Moreira, 1990; Graneli *et al.* 1993; Honjo, 1993) and trace metals (Price and Morel, 1991; Honjo, 1993).

6.3 Bloom Maintenance

Bloom maintenance requires the growth rate of the phytoplankton in a bloom to exceed the rate of loss of phytoplankton in a population. Phytoplankton loss usually occurs by grazing or through cell lysis sinking or advection away from the bloom site (Richardson, 1997).

Grazing impacts can be important in the development of harmful algal blooms. Limited grazing pressure often on toxic phytoplanktons may allow development of large biomasses of these phytoplankters. Grazing pressures can also be reduced by acting as a selection factor in phytoplankton species (Olsson *et al.* 1992) or by reduction in loss through advection (Crawford and Purdie, 1992) or by vertical migration of the phytoplankton in the water column.

The blooming organism can also be maintained by reduction of competition for limiting resources. If nutrients are limiting, then inhibition of other phytoplankters competing for the same nutrients will serve to optimize growth conditions for the bloom organism. Several harmful bloom forming species have been shown to have a repressing effect on other phytoplankton species, such as, *Gyrodniium aureolum* (Arzul *et al.* 1993); *Heterosigma akashiwo* (Honjo, 1993). Thus, more than one mechanism may be contributing towards maintenance of some phytoplankton blooms.

6.4 Bloom Termination

A phytoplankton bloom will terminate or decline when the loss factors exceed the increase factors. The immediate effect is usually a decrease in the growth rate of bloom forming phytoplankton species, brought about by a change in the environmental conditions, including light and / nutrients. The decaying phytoplankton cells can either sink out of the water column or lyse and release their contents to the surrounding water column (Richardson, 1997). Flagellate blooms are often degraded in the water column (Boekel *et al.* 1992), while diatom blooms often sink out of the water column (Cushing, 1992). Different diatoms have different sinking rates, which are probably influenced by cell volume and the physiological states, expressed as respiration rates (Waite *et al.* 1992).

According to Jackson (1990) a given phytoplankton population will grow exponentially until it reaches an equilibrium concentration : "critical concentration". At this stage, growth and coagulation will balance. Applying the coagulation theory to phytoplanktons, it has been suggested that aggregation may be an important mechanism in terms of sudden disappearance of diatom blooms (Jackson, 1990; Jackson and Lochmann, 1992). The phytoplankton cells are believed to possess a certain degree of adhesiveness or stickiness that allows them to aggregate or stick together (Kiørboe *et al.* 1990). Under laboratory conditions, different phytoplankton species exhibit different degrees of "stickiness" (Kiørboe *et al.* 1990). Moreover, age (physiological state) can influence stickiness within an individual species. A better understanding of these mechanisms will help to elucidate the bloom dynamics.

7. Summary and Conclusions

Marine phytoplankton blooms occur intermittently, within 100 km of coastal regions near all the major continents. The common denominators associated with phytoplankton blooms are (a) seed

population of organisms; (b) proper chemical environment of organic and inorganic nutrients; and (c) suitable physical and hydrographic conditions for concentration and maintenance of nutrient organisms. Phytoplankton blooms are natural phenomena that develop over various scales of space and time including the massive, sudden and local outbursts of one or more species. Such phenomena have also been termed "red tides", "brown tides", "colored waters" or "discolored waters". There are about 4000 different species which constitute the world's flora. Approximately 6% (about 200 species) have been indicated to cause red tides or are regarded as harmful. About 2% of the world's phytoplankton are implicated in toxin production. According to a number of workers, there has been a considerable rise in harmful algal blooms due to anthropogenic activities in recent years. These activities include eutrophication, distribution and transport of harmful species from one region to another via ballast water or in connection with aquacultural activities. Despite several common features HABs are very diverse in terms of causative organisms, dynamics of blooms and type of blooms.

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