# 1

# Harmful algal blooms: a global overview

G. M. Hallegraeff

The microscopic planktonic algae of the world's oceans are critical food for filter-feeding bivalve shellfish (oysters, mussels, scallops, clams) as well as the larvae of commercially important crustaceans and finfish. In most cases, the proliferation of plankton algae (so-called 'algal blooms'; up to millions of cells per litre) therefore is beneficial for aquaculture and wild fisheries operations. However, 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. Among the 5,000 species of extant marine phytoplankton (Sournia *et al.*, 1991), some 300 species can at times occur in such high numbers that they obviously discolour the surface of the sea (so-called 'red tides'), while only 80 or so species have the capacity to produce potent toxins that can find their way through fish and shellfish to humans (Table 1.1).

It is believed that the first written reference (1000 B.C.) to 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 7: 20–1). 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 can be due to high respiration by the algae (at night or in dim light during the day) but more commonly is caused by bacterial respiration during decay of the bloom. Essentially non-toxic bloom formers sometimes can evoke major ecosystem impacts, however, and unsightly dead fish, slime and foam deter tourism and recreational activities.

One of the first recorded fatal cases of human poisoning after eating shellfish contaminated with dinoflagellate toxins was in 1793 when Captain George Vancouver and his crew landed in British Columbia in an area now known as Poison Cove. He noted that for local Indian tribes it was taboo to eat shellfish when the seawater became bioluminescent due to dinoflagellate blooms (Dale and Yentsch, 1978). The causative alkaloid toxins, now called paralytic shellfish poisons (PSP) (see Chapter 7), are so potent that a pinhead-size quantity (about 500 µg), which can easily accumulate in just one 100 g serving of shellfish, 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 (Geraci *et al.*, 1989). Poisoning of manatees by dinoflagellate brevetoxins contained in salps attached to seagrass (in Florida: Anderson and White, 1989) and of pelicans and

sealions by diatom domoic acid contained in anchovies has also been reported (in California: Scholin *et al.*, 2000; Work *et al.*, 1993).

TABLE 1.1 Different types of harmful algal bloom

1. Species that produce basically harmless water discolorations; however, under exceptional conditions in sheltered bays, blooms can grow so dense that they cause indiscriminate kills of fish and invertebrates through oxygen depletion.
Examples: dinoflagellates *Akashiwo sanguinea, Gonyaulax polygramma, Noctiluca scintillans, Scrippsiella trochoidea*; cyanobacterium *Trichodesmium erythraeum*

2. Species that produce potent toxins that can 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 catenella, A. cohorticula, A. fundyense, A. fraterculus, A. leei, A. minutum, A. tamarense, Gymnodinium catenatum, Pyrodinium bahamense* var. *compressum*)

- diarrhetic shellfish poisoning (DSP)
(Examples: dinoflagellates *Dinophysis acuta, D. acuminata, D. caudata, D. fortii, D. norvegica, D. mitra, D. rotundata, D. sacculus, Prorocentrum lima*)

- amnesic shellfish poisoning (ASP)
(Examples: diatoms *Pseudo-nitzschia australis, P. delicatissima, P. multiseries, P. pseudodelicatissima, P. pungens* (some strains), *P. seriata*)

- ciguatera fish poisoning (CFP)
(Examples: dinoflagellate *Gambierdiscus toxicus*,? *Coolia* spp.,? *Ostreopsis* spp.,? *Prorocentrum* spp.)

- neurotoxic shellfish poisoning (NSP)
(Examples: dinoflagellate *Karenia brevis* (Florida), *K. papilionacea, K. selliformis, K. bicuneiformis* (New Zealand))

- cyanobacterial toxin poisoning
(Examples: cyanobacteria *Anabaena circinalis* (freshwater), *Microcystis aeruginosa* (freshwater), *Nodularia spumigena*)

- estuarine associated syndrome (through aerosols from dinoflagellates *Pfiesteria piscicida, P. shumwayae*)

3. Species that are non-toxic to humans but harmful to fish and invertebrates (especially in intensive aquaculture systems) by damaging or clogging their gills.
Examples: diatoms *Chaetoceros concavicorne, C. convolutus*; dinoflagellates *Karenia mikimotoi, K. brevisulcata, Karlodinium micrum*; prymnesiophytes *Chrysochromulina polylepis, Prymnesium parvum, P. patelliferum*; raphidophytes *Heterosigma akashiwo, Chattonella antiqua, C. marina, C. verruculosa*

The third type of harmful algal bloom has become apparent only as a result of our increased interest in intensive aquaculture systems for finfish. Some algal species can seriously damage fish gills, either mechanically or through production of hemolytic substances, whereas other algae kill fish through the production of extracellular neurotoxins. Whereas wild fish stocks have the freedom to swim away from problem areas, caged fish appear to be extremely vulnerable to such noxious algal blooms. In





1972 in Japan, a bloom of the raphidophyte flagellate *Chattonella antiqua* thus killed US$500 million worth of caged yellowtail fish in the Seto Inland Sea (Okaichi, 1989).

Table 1.1 summarizes the above three types of harmful algal bloom problems, together with representative examples of causative algal species, ranging from dinoflagellates, diatoms, prymnesiophytes and raphidophytes to cyanobacteria. Clinical symptoms of various types of fish and shellfish poisoning are listed in Table 1.2 and the diversity of chemical structures of algal toxins is discussed in Chapters 7–11. Unfortunately, there is no clear-cut correlation between algal concentrations and their potential harmful effects. Dinoflagellate species such as *Dinophysis*, *Alexandrium* and *Pyrodinium* can contaminate shellfish with toxins, even at very low cell concentrations. The prymnesiophyte *Chrysochromulina polylepis* produces only moderate biomass levels but has a very high toxic potency. Finally, the prymnesiophyte *Phaeocystis* is basically non-toxic but its nuisance value is caused by very high biomass levels.

## 1.1 GLOBAL INCREASE OF ALGAL BLOOMS

While harmful algal blooms, in a strict sense, are completely natural phenomena that have occurred throughout recorded history, in the past two decades the public health and economic impacts of such events appear to have increased in frequency, intensity and geographical distribution. One example, the increased global distribution of paralytic shellfish poisoning, is illustrated in Fig. 1.1. Until 1970, toxic dinoflagellate blooms of *Alexandrium (Gonyaulax) tamarense* and *Alexandrium (Gonyaulax) catenella* were only known from temperate waters of Europe, North America and Japan (Dale and Yentsch, 1978). By 1990, this phenomenon was well documented throughout the Southern Hemisphere, in South Africa, Australia, New Zealand, India, Thailand, Brunei, Sabah, the Philippines and Papua New Guinea. Other species of the dinoflagellate genus *Alexandrium,* such as *A. cohorticula* and *A. minutum*, as well as the unrelated dinoflagellates *Gymnodinium catenatum* and *Pyrodinium bahamense* var. *compressum* have now also been implicated. Unfortunately, there are very few long-term records of algal blooms at any single locality. Probably the best dataset refers to the concentration of PSP toxins (μg saxitoxin equivalent/100 g shellfish meat) in Bay of Fundy clams, which has been monitored by mouse bioassay since 1944 (White, 1987). Shellfish containing more than 80 μg PSP/100 g shellfish meat are considered unfit for human consumption. Fig. 1.2 shows evidence of a cyclic pattern of toxicity at this site with increased frequency of toxic blooms in the late 1940s, early 1960s, late 1970s and early 1980s, and possibly beginning again in the mid-1990s (not shown). The importance of such long-term datasets is discussed in Chapter 21.

The issue of a global increase in harmful algal blooms has been a recurrent topic of discussion at all major conferences dealing with harmful algal blooms (Anderson, 1989; Hallegraeff, 1993; Smayda, 1990). Four explanations for this apparent increase in algal blooms have been proposed: increased scientific awareness of toxic species; increased utilization of coastal waters for aquaculture; stimulation of plankton blooms by cultural eutrophication and/or unusual climatological conditions; and transportation of dinoflagellate resting cysts either in ships' ballast water or associated with translocation of shellfish stocks from one area to another.

TABLE 1.2 Clinical symptoms of various types of fish and shellfish poisoning

| **Paralytic shellfish poisoning (PSP)** | **Diarrhetic shellfish poisoning (DSP)** | **Amnesic shellfish poisoning (ASP)** | **Neurotoxic shellfish poisoning (NSP)** | **Ciguatera fish poisoning** |
|---|---|---|---|---|
| Causative organism | | | | |
| *Alexandrium catenella* | *Dinophysis acuminata* | *Pseudo-nitzschia multiseries;* *Pseudo-nitzschia pungens* (some strains) | *Karenia brevis* (Florida); *K. papilionacea* *K. selliformis* *K. bicuneiformis* (New Zealand) | *Gambierdiscus toxicus* |
| *Alexandrium minutum* | *Dinophysis acuta* | *Pseudo-nitzschia pseudodelicatissima* | | *?Ostreopsis siamensis* |
| *Alexandrium tamarense* | *Dinophysis fortii* | *Pseudo-nitzschia australis* | | *?Coolia monotis* |
| *Gymnodinium catenatum* | *Dinophysis norvegica* | *Pseudo-nitzschia seriata* | | |
| *Pyrodinium bahamense* var. *compressum* | *Prorocentrum lima* | *Pseudo-nitzschia delicatula* | | |
| Symptoms | | | | |
| Mild case | | | | |
| 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, diarrhoea. | After 30 min to a few hours (seldom more than 12 hours): diarrhoea, nausea, vomiting, abdominal pain. | After 3–5 hours: nausea, vomiting, diarrhoea, abdominal cramps. | After 3–6 hours: chills, headache, diarrhoea; muscle weakness, muscle and joint pain; nausea and vomiting | Symptoms develop within 12–24 hours of eating fish. Gastrointestinal symptoms: diarrhoea, abdominal pain, nausea, vomiting. |
| Extreme case | | | | |
| Muscular paralysis; pronounced respiratory difficulty; choking sensation; death through respiratory paralysis may occur within 2–24 hours after ingestion. | Chronic exposure may promote tumour formation in the digestive system. | Decreased reaction to deep pain; dizziness, hallucinations, confusion; short-term memory loss; seizures | Paraesthesia; altered perception of hot and cold; difficulty in breathing, double vision, trouble in talking and swallowing | 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. |
| Treatment | | | | |
| Patient has stomach pumped and is given artificial respiration. No lasting effects. | Recovery after three days, irrespective of medical treatment. | | | No antitoxin or specific treatment is available. Neurological symptoms may last for months or years. Calcium and mannitol may help to relieve symptoms. |

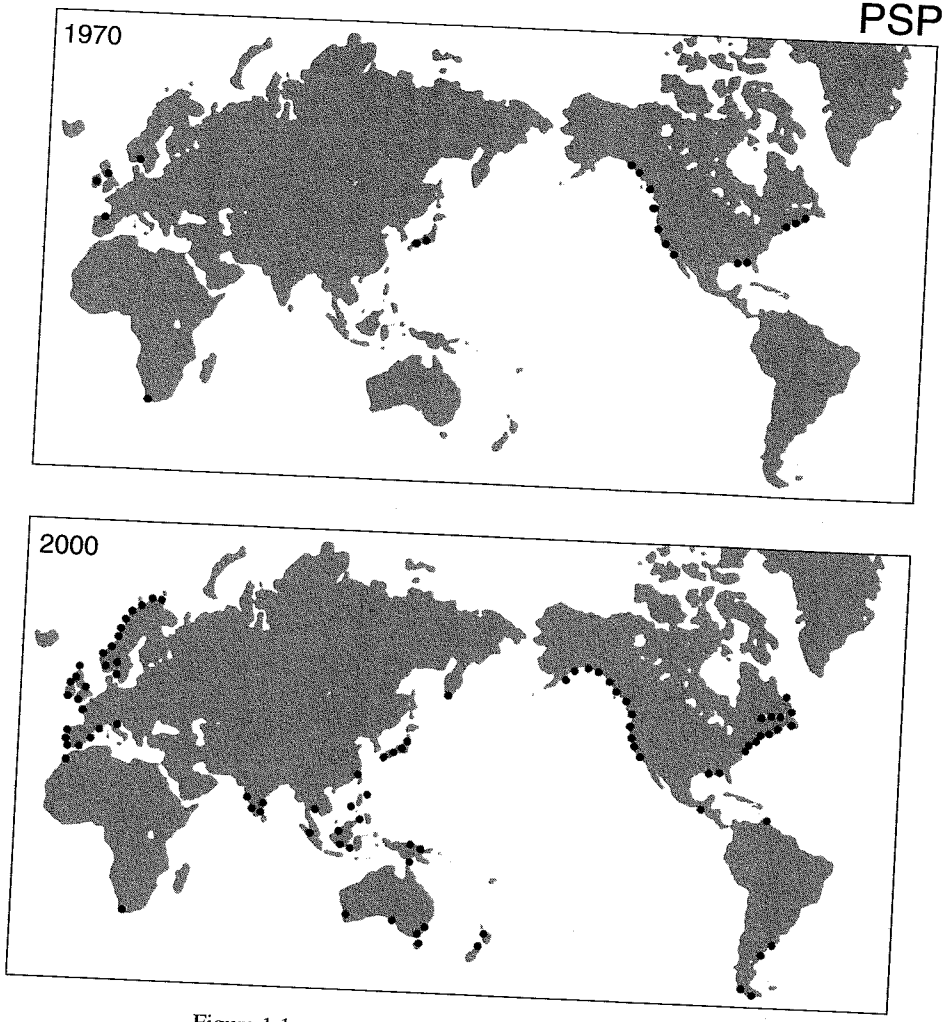


Figure 1.1
Known global distribution of paralytic shellfish poisoning (PSP) in 1970 and 2000.

## 1.2 Increased scientific awareness of toxic species

Reports of harmful algal blooms, associated human illnesses or damage to aquaculture operations are receiving increased attention in the press, electronic media and scientific literature. Fig. 1.3 illustrates the doubling of annual literature on algal blooms every two to two-and-a-half years. As a result, more and more researchers are now surveying their local waters for the causative organisms. Increased reports



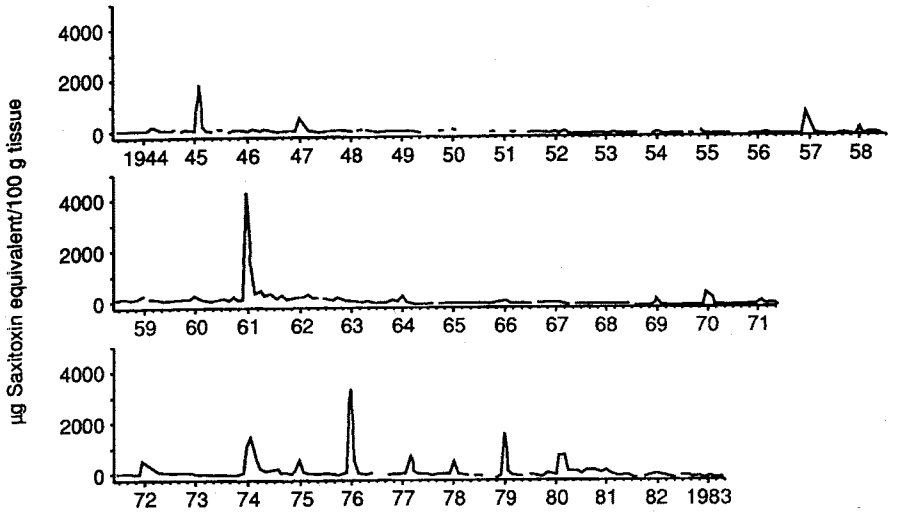


Figure 1.2
Concentration of PSP toxins in Bay of Fundy clams (µg saxitoxin equivalent/100 g tissue) in 1944–1983.
*Source:* White (1987).

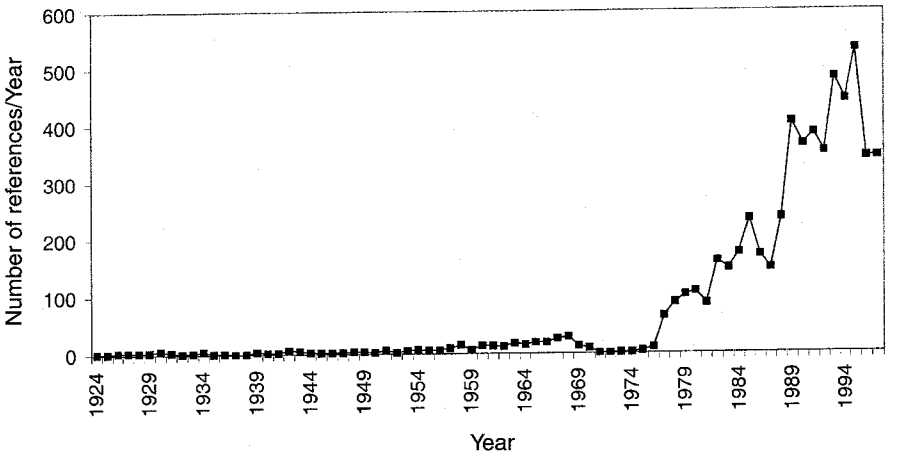


Figure 1.3
Growth of literature on harmful algal blooms, illustrated by analysing some 6,000 HAB publications from Aquatic Sciences and Fisheries Abstracts (ASFA) from about 1978 to 1998, with pre-1970 literature based on University of Copenhagen records.
*Source:* data courtesy of Gert Hansen.

on the occurrence of dinoflagellates of the genus *Alexandrium* are a good example. Until 1988, the type species *A. minutum* was only known from Egypt (Halim, 1960); it has now been reported from Australia, Ireland, France, Spain, Portugal, Italy, Turkey, the east coast of North America, Thailand, New Zealand, Taiwan and Japan (Hallegraeff *et al.*, 1991; Yuki, 1994). Other examples are the recent description of the newly recognized phenomena of diarrhetic shellfish poisoning (since 1976), amnesic shellfish poisoning (since 1987) and azaspiracid poisoning (AZP, since 2000, so far only documented from Ireland, England and Norway).

### 1.2.1 Diarrhetic shellfish poisoning (DSP)

This phenomenon 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 implicated was *Dinophysis fortii* (in Japan), soon followed by *D. acuminata* (in Europe), *D. acuta, D. norvegica* (in Scandinavia), *D. mitra, D. rotundata* and the benthic dinoflagellate *Prorocentrum lima*. Between 1976 and 1982, some 1,300 DSP cases were reported in Japan, in 1981 more than 5,000 cases were reported in Spain and in 1983 some 3,300 cases were reported in France. In 1984 in Sweden, DSP problems caused a shutdown of the mussel industry for almost a year. The clinical symptoms of DSP (Table 1.2) may often have been mistaken for those of bacterial gastric infections and the problem may be much more widespread and serious than previously thought. Unlike PSP, no human fatalities have ever been reported and patients usually recover within three days. However, some of the polyether toxins involved (okadaic acid, dinophysis toxin-1; see Chapter 8) may promote stomach tumours (Suganuma *et al.*, 1988) and thus produce chronic problems in shellfish consumers. Shellfish containing more than 2 µg okadaic acid and/or 1.8 µg dinophysis toxin-1 per gram of hepatopancreas are considered unfit for human consumption (Lee *et al.*, 1987). Increasing problems caused by pectenotoxins have also been noted. The known global distribution of DSP (Fig. 1.4) includes Japan, Europe, Chile, Thailand, Canada (Nova Scotia), Australia and New Zealand.

### 1.2.2 Amnesic shellfish poisoning (ASP)

This phenomenon was first recognized in 1987 in Prince Edward Island, Canada, where it caused three deaths and 105 cases of acute human poisoning following the consumption of blue mussels. The symptoms (Table 1.2) include abdominal cramps, vomiting, disorientation and memory loss (amnesia). Most unexpectedly, the causative toxin (the excitatory amino acid domoic acid; see Chapter 9) is produced by a diatom and not by a dinoflagellate. Shellfish containing more than 20 µg domoic acid per gram of shellfish meat are considered unfit for human consumption. The diatom species *Pseudo-nitzschia australis* (= *N. pseudoseriata*), *P. delicatissima, P. multiseries, P. multistriata, P. pseudodelicatissima, P. seriata* and occasionally *P. fraudulenta, P. pungens* and *P. turgidula* have been implicated (Bates *et al.*, 1989; Garrison *et al.*, 1993; Martin *et al.*, 1990; Rhodes *et al.*, 1998). To date, reports of domoic acid in seafood products have been mainly confined to North America (Bay of Fundy, California, Oregon, Washington, Alaska) and Canada (Prince Edward Island, British Columbia), whereas only insignificant concentrations have been detected in other parts of the world such as Europe, Australia, Japan and New Zealand (Fig. 1.5). Of further concern is the demonstration of domoic acid production by *Nitzschia navis-varingica* from a tropical shrimp aquaculture pond (Lundholm and Moestrup, 2000).





## 1.3 Increased utilization of coastal waters for aquaculture

With increased problems of overfishing of coastal waters, more and more countries are looking towards aquaculture as an alternative. Indeed, fisheries scientists predict that, within the next 10–20 years, the increasing value of world aquaculture production may well approach the decreasing value of the total catch of wild fish and shellfish. Aquaculture operations act as sensitive 'bioassay systems' for harmful algal

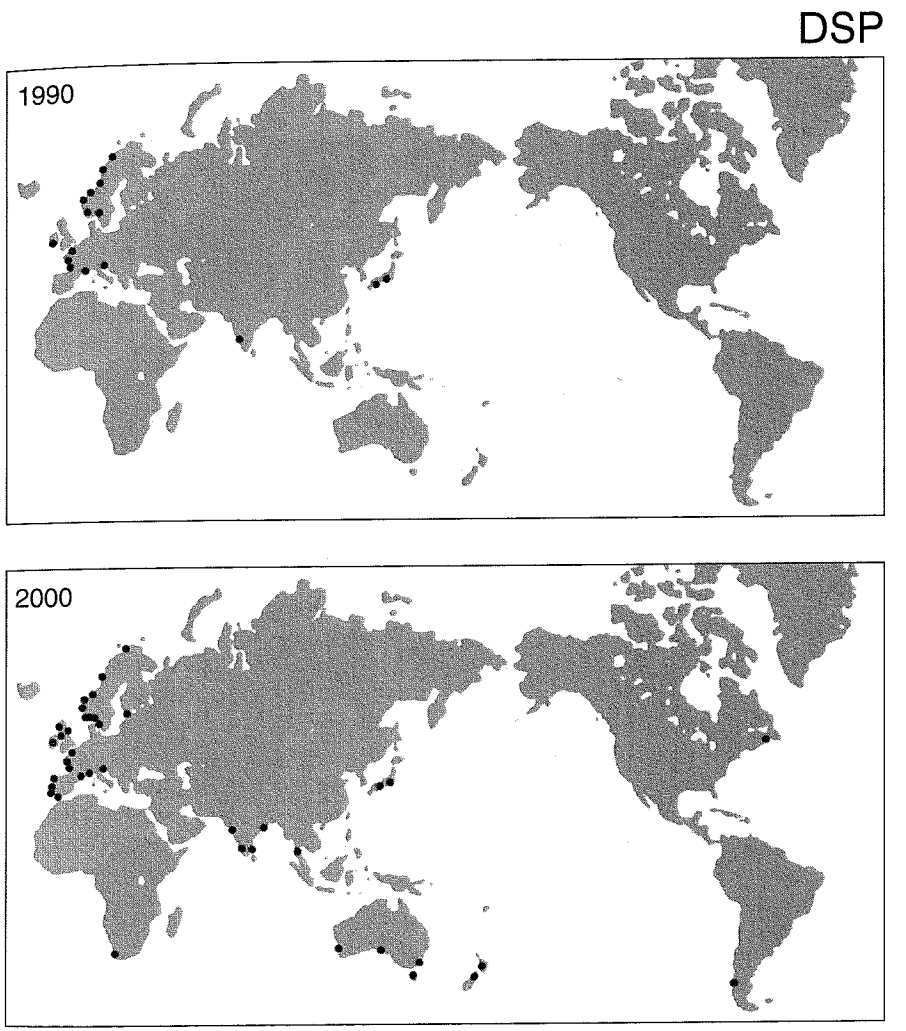


Figure 1.4
Known global distribution of diarrhetic shellfish poisoning (DSP) in 1990 and 2000.

species and can bring to light the presence in water bodies of problem organisms not previously known to exist there. The increase in shellfish farming worldwide is leading to more reports of paralytic, diarrhetic, neurotoxic or 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.

In fish pens in British Columbia, deaths of lingcod, sockeye, coho, chinook and pink salmon have been caused by dense concentrations (5,000 cells per litre) of the diatoms *Chaetoceros convolutus* and *C. concavicornis*. The diatom's long hollow

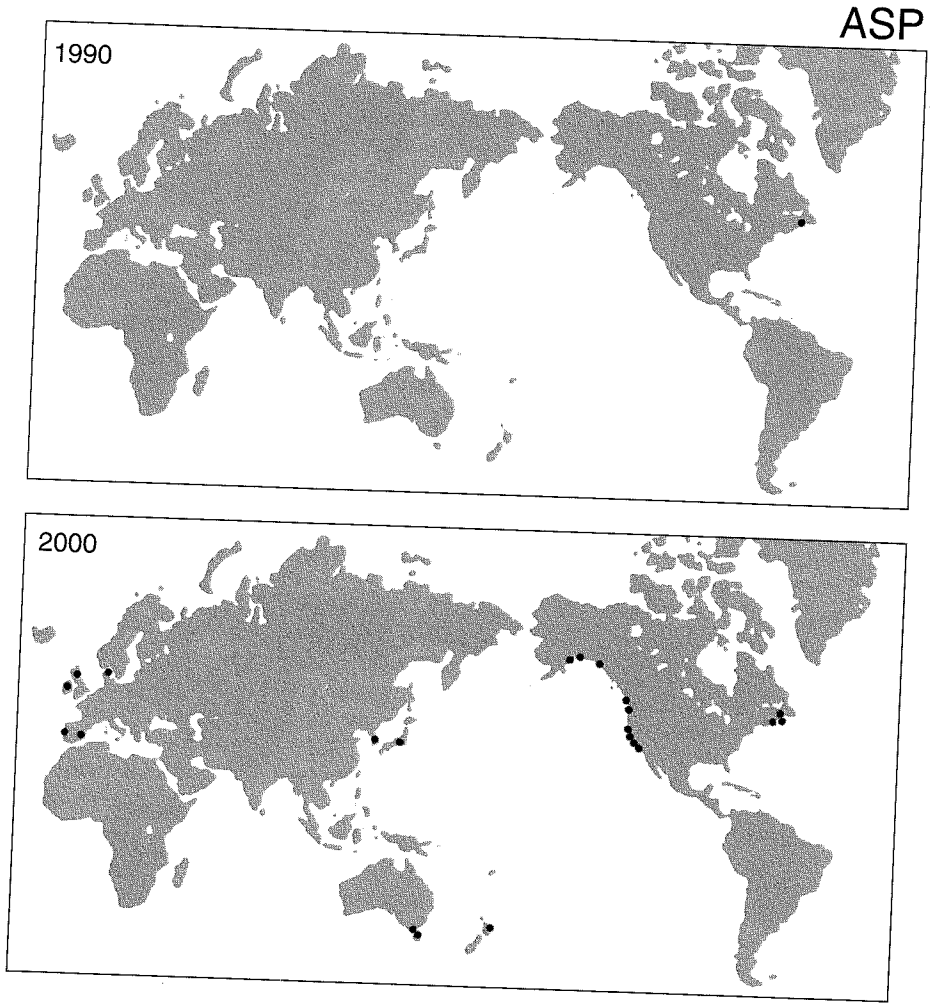


Figure 1.5
Known global distribution of amnesic shellfish poisoning (ASP) in 1990 and 2000.



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. 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; Rensel, 1993; Yang and Albright, 1992).

A more widespread problem for fish farmers is the production by various algal groups of fatty acids or galactolipids which damage the epithelial tissues 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 *Heterosigma akashiwo* and *Chattonella antiqua/marina* (see Chapter 18), the prymnesiophytes *Chrysochromulina polylepis* and *Prymnesium parvum* (see Chapter 16), and the dinoflagellate *Karenia mikimotoi* (= *Gymnodinium nagasakiense;* see Chapter 15) have been implicated. *Heterosigma* has killed caged fish in Japan, Canada, Chile and New Zealand, whereas *Chattonella* is a fish killer known from Japan (Seto Inland Sea), South East Asia, Australia and Europe. With these two raphidophyte flagellates, physical clogging of gills by mucus excretion or gill damage by hemolytic substances or the production of oxygen radicals, free fatty acids and breve-like neurotoxins may be involved. In January 1989, a *Heterosigma* bloom in Big Glory Bay, Stuart Island (New Zealand), killed cage-reared chinook salmon worth NZ$12 million (Chang *et al.*, 1990), and *Chattonella marina* killed Aus$45 million of cultured blue-fin tuna in South Australia in 1996 (Hallegraeff *et al.*, 1998). The two prymnesiophyte flagellates *Chrysochromulina* and *Prymnesium* produce substances that affect gill permeability, which leads to a disturbed ion balance. Toxicity by these species is promoted by phosphorus deficiency. A massive bloom (60,000 km$^2$; $10^7$ cells l$^{-1}$) of *Chrysochromulina polylepis* occurred in May–June 1988 in the Skagerrak, the Kattegat, the Belt and the Sound between Denmark, Norway and Sweden (Rosenberg *et al.*, 1988). The deaths of 900 tons of fish, including cod, salmon and trout, occurred due to damage of gill membranes that produced a lethal increase in the chloride concentration in the blood; fish cages moved into less saline fjords were therefore less affected. *Prymnesium parvum* has caused mortality of *Tilapia* fish in brackish water culture ponds in Israel (Shilo, 1981), as well as mortality of salmon and rainbow trout in net-pens in Norway. However, probably the greatest problem for Norwegian fish farms are blooms of the unarmoured dinoflagellate *Karenia mikimotoi* (as *Gyrodinium aureolum*) (first reported in 1966; Tangen, 1977). Similar dinoflagellates are common in Ireland and Scotland, as well as Japan and Korea. Characteristic histopathological symptoms in fish are a severe necrosis and sloughing of epithelial tissues of the gills and digestive system (Roberts *et al.*, 1983). A bloom of related gymnodinioid dinoflagellate species in Hong Kong waters in April 1998 caused over US$20 million damage to finfish aquaculture.

Sophisticated monitoring systems using buoys with fibre-optical sensors and data transfer by satellite (the MARINET system) are in place on the Norwegian coast to allow cages to be towed away from bloom-affected areas. During the 1988 *Chrysochromulina* bloom, more than 26,000 tons of fish in 1,800 cages were thus moved from their permanent site into inland fjords. Fish losses in cages can also be reduced by not feeding the fish, as feeding attracts them to the surface and increases oxygen demand. In some cases, pumping of water to dilute the algal concentration, the administration to fish of mucolytic agents or immediate harvesting of marketable

fish before they can be killed by algal blooms may also be an option. The hemolytic toxins do not accumulate in fish flesh. Virtually all algal blooms, even of non-toxic species, reduce the fishes' appetite and reduced oxygen concentrations stress the fish and make them more vulnerable to disease (see Chapter 25).

Finally, ichthyotoxic 'ambush predator' dinoflagellates *Pfiesteria piscicida* and *P. shumwayae* were first recognized in North Carolina in 1991 and later in Chesapeake Bay (Burkholder *et al.*, 1992). Their ephemeral presence (cysts germinate in the presence of live fish and encyst again after fish death) may explain many mysterious fish kills along the south-east coast of the USA and has also been associated with human health impacts (estuarine associated syndrome). *Pfiesteria* has now also been documented from northern Europe, New Zealand and Australia.

## 1.4 Increase of algal blooms by cultural eutrophication

While some organisms such as the dinoflagellates *Karenia (Gymnodinium) brevis, Alexandrium, Dinophysis* and *Pyrodinium* appear to be unaffected by coastal nutrient enrichments, many other algal bloom species appear to be stimulated by 'cultural eutrophication' from domestic, industrial and agricultural wastes. Fig. 1.6 illustrates an 8-fold increase in the number of red tides per year in Tolo harbour, Hong Kong, in the period 1976–1986 (Lam and Ho, 1989). This increase (mainly *Karenia mikimotoi, Gonyaulax polygramma, Noctiluca scintillans* and *Prorocentrum minimum = cordatum)* shows a striking relationship with the 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. Red-tide events in Hong Kong harbour were less frequent in 1989–1997 until the major bloom year of 1998. A similar experience was noted in the Seto Inland Sea, one of the major fish-farm areas in Japan (Okaichi, 1989) (Fig. 1.7). Between 1965 and 1976, the number of

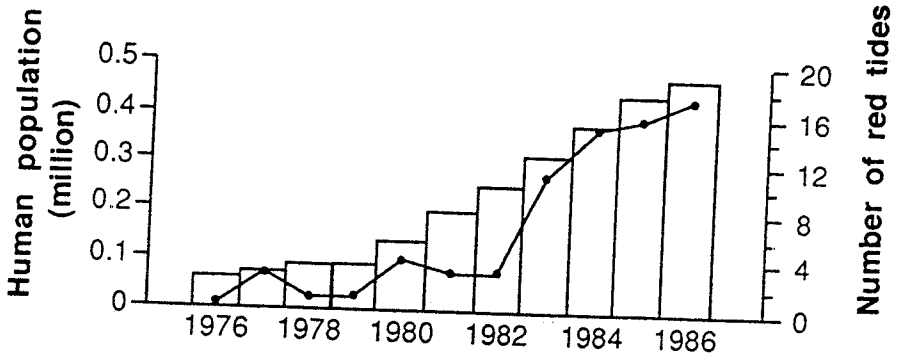


Figure 1.6
Correlation between the number of red-tide outbreaks per year in Tolo Harbour (continuous line) and the increase in the human population in Hong Kong (bar diagram), 1976–1986.
*Source:* Lam and Ho (1989).





confirmed red-tide outbreaks (mainly *Chattonella antiqua*, since 1964; and *Karenia mikimotoi,* since 1965) progressively increased 7-fold, concurrent with a 2-fold increase in the COD (chemical oxygen demand) loading, mainly from untreated sewage and industrial waste from pulp and paper factories. During the most severe outbreak in 1972, a *Chattonella* red tide killed 14 million cultured yellow-tail fish. Effluent controls were then initiated to reduce the chemical oxygen demand loading by about half, to introduce secondary sewage treatment and to remove phosphate from household detergents. Following a time-lag of four years, the frequency of red-tide events in the Seto Inland Sea then decreased about 2-fold to a more stationary level.

A similar pattern of a long-term increase in nutrient loading of coastal waters is evident for the North Sea in Europe (Smayda, 1990) (Fig. 1.8). Since 1955, the phosphate loading of the River Rhine has increased 7.5-fold, whereas nitrate levels have increased 3-fold. This has resulted in a significant 6-fold decline in the Silicon: phosphorus ratio, because long-term reactive silicate concentrations (a nutrient derived from natural land weathering) have remained constant. More recently, improved wastewater treatment has been causing increases in the ammonia:nitrate ratio of River Rhine discharge (Riegman *et al.*, 1992). The nutrient composition of treated wastewater is never the same as that of the coastal waters into which it is being discharged. Furthermore, atmospheric deposition of nitrogen also needs to be included in budgets of anthropogenic nutrient input. There is considerable concern (Officer and Ryther, 1980; Ryther and Dunstan, 1971; Smayda, 1990) that 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.

The remarkable increase in foam-producing blooms of the prymnesiophyte *Phaeocystis pouchetii,* which first appeared in Dutch coastal waters in 1978, is probably the best-studied example of this phenomenon (Lancelot *et al.*, 1987). The 1988 bloom in the Kattegat of the prymnesiophyte *Chrysochromulina polylepis*,

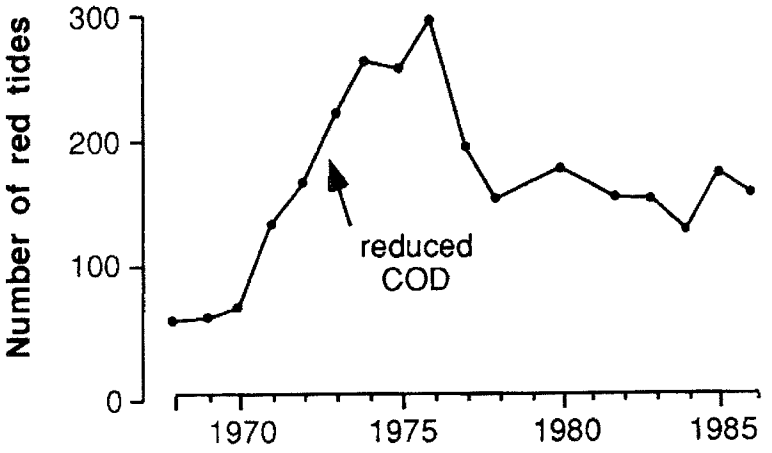


Figure 1.7
Long-term trend in the frequency of red-tide outbreaks in the Seto Inland Sea, Japan, 1965–1986. COD: chemical oxygen demand.
*Source:* Okaichi (1989).

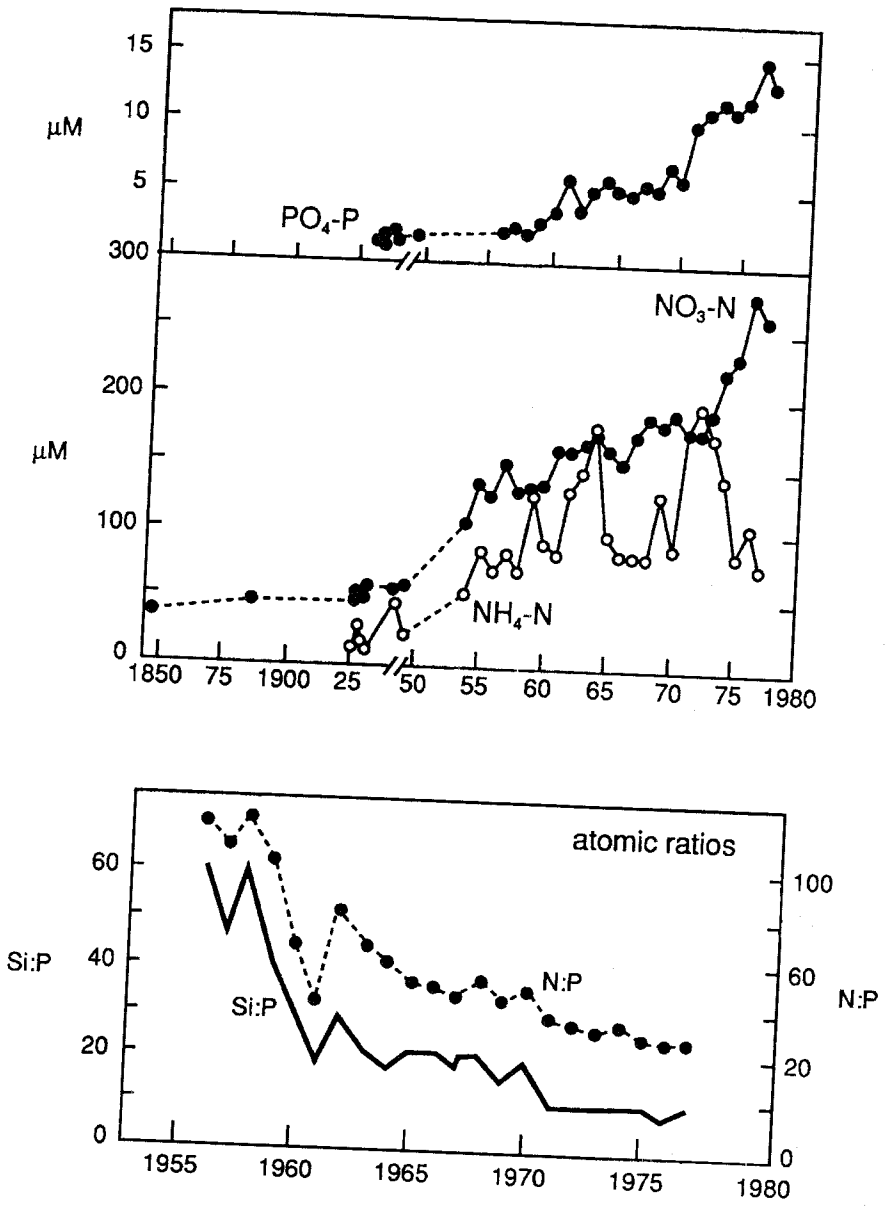


Figure 1.8
Long-term trend in the phosphate, nitrate and ammonia loading of the River Rhine (above) and concurrent changes in the N:P and Si:P nutrient ratios (below).
*Source*: Smayda (1990), using data by Van Bennekom and Salomons (1981).





not unusual in terms of biomass but rather 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 Granéli, 1991). As in Hong Kong and Japan, several North European countries have now agreed to reduce phosphate and nitrate discharges by 50% in the next several years, but their efforts will almost certainly be in vain if neighbours continue polluting. Furthermore, such indiscriminate reductions in nutrient discharges are not addressing the problem of changing nutrient ratios of coastal waters. 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. Experimental evidence from Sweden indicates that river water draining from agricultural soils (rich in N and P) stimulates diatom blooms but that river water draining from forest areas (rich in humic and fulvic acids) can stimulate dinoflagellate blooms of species such as *Prorocentrum minimum* (Granéli and Moreira, 1990). Agricultural runoff of phosphorus can also stimulate cyanobacterial blooms, for example of *Nodularia spumigena* in the Baltic Sea and in the Peel-Harvey Estuary, Australia (Fig. 1.9). These species produce hepatotoxic peptides (*Nodularia, Microcystis*) and neurotoxic alkaloids (*Anabaena, Aphanizomenon)* which can kill domestic and wild animals drinking from the shores of eutrophic ponds, lakes and reservoirs (for example, during a 1,000 km long *Anabaena circinalis* bloom in the Darling River, Australia, in 1991). Toxicity problems from freshwater cyanobacteria have been documented from Australia, Bangladesh, China, Europe (12 countries), India, Israel, Japan, Latin America, North America, South Africa, Thailand and the former Soviet Union (Carmichael, 1989). The toxins can accumulate in the digestive system of shellfish (Falconer *et al.*, 1992) but contamination of drinking water with teratogens and tumour promoters is a more common public health risk. Human fatalities have resulted when microcystin-contaminated lake water was offered to patients in a haemodialysis clinic (Pouria *et al.*, 1998). A neurotoxic factor has also been associated with some strains of the

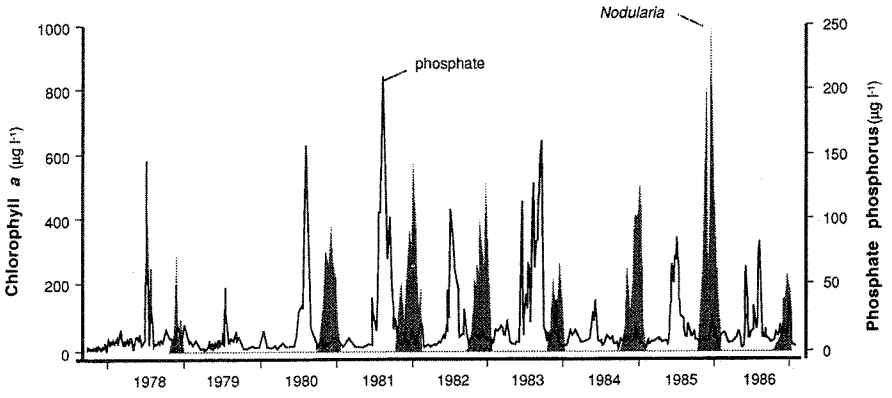


Figure 1.9
Relationship between *Nodularia spumigena* cyanobacterial blooms (as chlorophyll concentration) in the Peel-Harvey Estuary, Australia, and its relationship to riverine phosphate loading from agricultural runoff.
*Source*: Hillman *et al.* (1990).

common marine, bloom-forming tropical cyanobacterium *Trichodesmium thiebautii* (Hawser *et al.*, 1991: see Chapter 11).

A much more complex 'cultural eutrophication' scenario has emerged in coastal waters of New Jersey, New York and Rhode Island, USA, where an unusual 'brown tide' (caused by the chrysophyte picoplankton *Aureococcus anophagefferens*) has been related to the discharge of chelators (such as citric acid) in detergents and lawn treatments, together with a suppression of zooplankton grazing by pesticides (Cosper *et al.*, 1989, 1993). This bloom was responsible for a reduction in the extent and biomass of eelgrass beds and caused starvation and recruitment failure in commercial scallop populations. Suppression of zooplankton grazing by the overexploitation of piscivorous fish can similarly release HAB species from grazing. Eutrophication problems like this cannot be readily diagnosed by routine monitoring programmes that focus on macronutrients or algal chlorophyll biomass alone (see Chapter 21).

### 1.4.1 Ciguatera fish poisoning and coral reef disturbance

Ciguatera is a tropical fish food-poisoning syndrome well known from coral reef areas in the Caribbean, Australia, and especially French Polynesia (Fig. 1.10). Humans consuming contaminated fish such as red bass, chinaman fish, moray eel, and paddle tail can suffer from gastrointestinal and neurological illnesses and in extreme cases can die from respiratory failure (Table 1.2; Gillespie *et al.*, 1986). The causative organisms are benthic dinoflagellates such as *Gambierdiscus toxicus*, and possibly *Ostreopsis siamensis*, *Coolia monotis* and related species, that live in epiphytic association with bushy red, brown and green seaweeds (up to 200,000 cells/100 g of algae) and also occur freely in sediments and coral rubble. These dinoflagellates produce the potent neurotoxins gambiertoxin and maitotoxin (see Chapter 10),

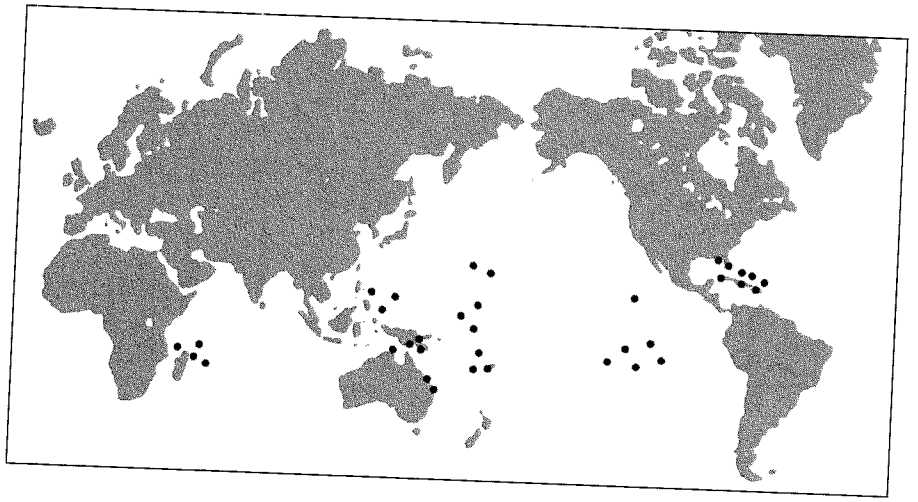

Figure 1.10
Global distribution of ciguatera fish poisoning (CFP).





which accumulate through the food chain, from small fish grazing on the coral reefs into the organs of bigger fish that feed on them (the principal toxin fraction in fish is ciguatoxin). While in a strict sense this is a completely natural phenomenon (Captain Cook suffered from this illness when visiting New Caledonia in 1774), from being a rare disease two centuries ago ciguatera now has reached epidemic proportions in French Polynesia. In the period 1960–1984 more than 24,000 patients were reported from this area, which is more than six times the average for the Pacific as a whole. Evidence is accumulating that reef disturbance by hurricanes, military and tourist developments, as well as coral bleaching (linked to global warming), are increasing the risk of ciguatera by increasing benthic substrate for dinoflagellate growth (Bagnis *et al.*, 1985).

## 1.5 Stimulation of algal blooms by unusual climatological conditions

### 1.5.1 Toxic Pyrodinium bahamense blooms in the tropical Indo-West Pacific

At present the dinoflagellate *Pyrodinium bahamense* is confined to tropical, mangrove-fringed coastal waters of the Atlantic and Indo-West Pacific. A survey of fossil occurrences of its resting cyst *Polysphaeridinium zoharyi* (Fig. 1.11) (records go back to the Eocene, 50 million years ago) indicates a much wider range of distribution in the past. For example, in the Australasian region at present the dinoflagellate does not extend further south than Papua New Guinea, but in the Pleistocene it ranged as far south as Sydney Harbour (McMinn, 1989). There is genuine concern that, with an increased greenhouse effect and warming of the oceans, this species may return to Australian waters. In the tropical Atlantic, in areas such as Bahia Fosforescente in Puerto Rico and Oyster Bay in Jamaica, this species forms persistent luminescent blooms which are a major tourist attraction. Both plankton bloom material and oysters and mussels attached to mangrove roots in Bahia Fosforescente appeared at one time to be non-toxic (Hallegraeff; Oshima, unpublished data). The first harmful implications of *Pyrodinium* blooms became evident in 1972 in Papua New Guinea. Red-brown water discolorations 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 poisons (MacLean, 1977). Since then, toxic *Pyrodinium* blooms have apparently spread to Brunei and Sabah (1976), the central Philippines (1983), the northern Philippines (1987) and Indonesia (North Mollucas). MacLean (1989) presented strong circumstantial evidence for a coincidence between *Pyrodinium* blooms and El Niño-Southern Oscillation (ENSO) climatological events. El Niño is caused by an imbalance in atmospheric pressure and sea temperature between the eastern and western parts of the Pacific Ocean and results in a shoaling of the thermocline. The 1991–1994 ENSO event and recurrence of dinoflagellate blooms in the Philippines tend to substantiate these claims (Fig. 1.12).

*Pyrodinium* is a serious public health and economic problem for the tropical countries that are affected, as they depend heavily on seafoods for protein and have little prior experience in toxic dinoflagellate research. In the Philippines alone, this organism has now been responsible for more than 2,000 human illnesses and

100 fatalities resulting from the consumption of contaminated shellfish, as well as planktivorous fish such as sardines and anchovies. Most unexpectedly, during a *Pyrodinium* bloom in 1987 on the Pacific coast of Guatemala, 187 people had to be hospitalized and 26 died. In 1989 another bloom swept northward along the Pacific coast of Central America, again causing illness and death. The Guatemala populations are morphologically more similar to the Indo-West Pacific populations (sometimes distinguished as a separate variety *compressum)* than to the Caribbean morphospecies (var. *bahamense)* (Rosales-Loessener *et al.*, 1989).

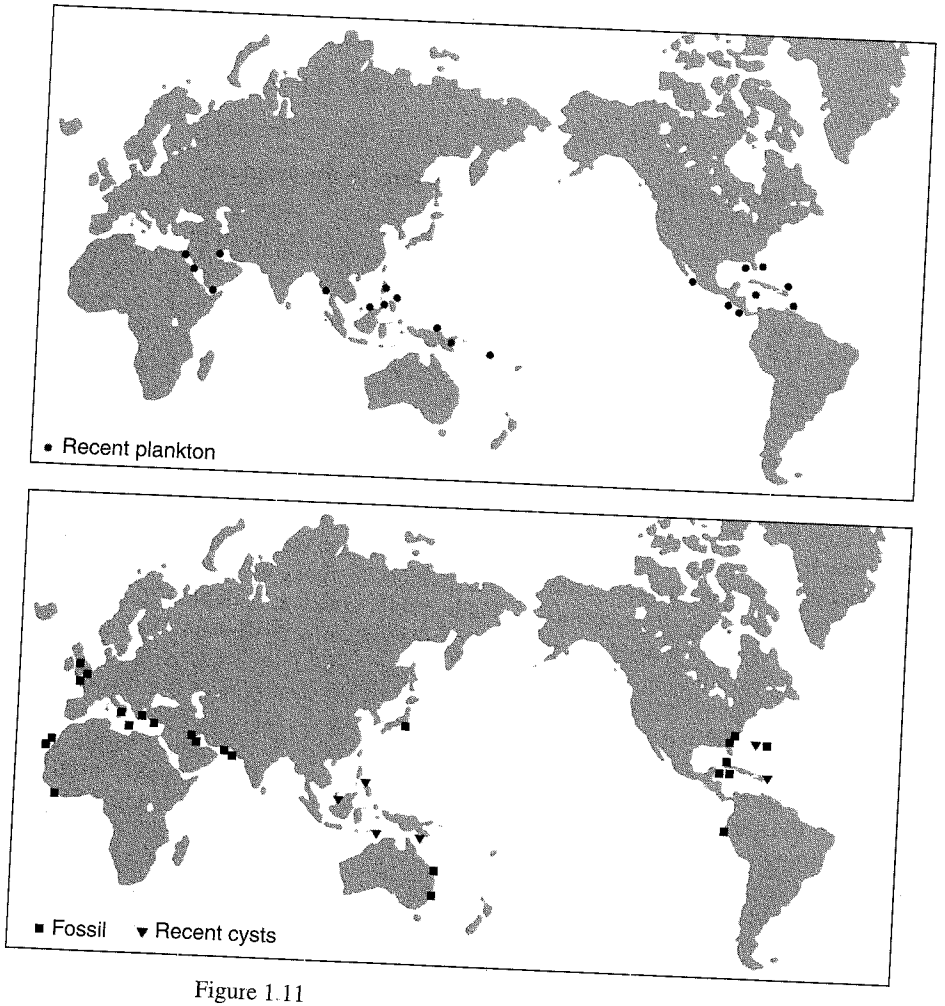


Figure 1.11
Global distribution of the tropical dinoflagellate *Pyrodinium bahamense* in recent plankton (above) and of the fossil cyst *Polysphaeridinium zoharyi* (below).
*Source:* Hallegraeff and MacLean (1989).





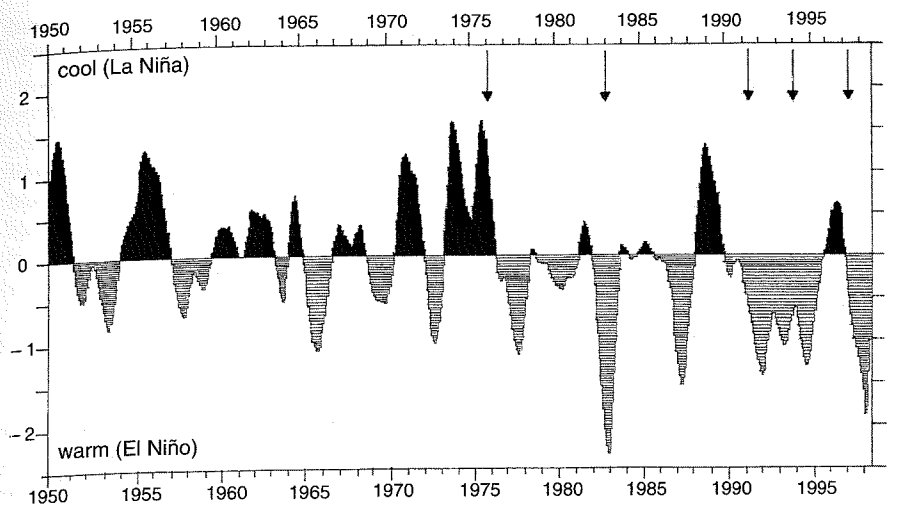


Figure 1.12
Relationship between *Pyrodinium bahamense* blooms (arrows) in the tropical Indo-West Pacific and El Niño–Southern Oscillation (ENSO) climatological events in 1970–1998. The graph shows mean surface temperature anomalies over the near-equatorial eastern Indian and western Pacific Oceans. Strong positive anomalies are indicative of ENSO events.
*Source:* Azanza and Taylor (2001)

### 1.5.2 Neurotoxic shellfish poisoning in Florida (USA) and New Zealand

Until recently, neurotoxic shellfish poisoning (NSP: Table 1.2), caused by polyether brevetoxins produced by the unarmoured dinoflagellate *Karenia brevis*, was considered to be endemic to the Gulf of Mexico and the east coast of Florida, where 'red tides' had been reported as early as 1844. An unusual feature of this organism is the formation by wave action of toxic aerosols that can lead to respiratory asthma-like symptoms in humans. In 1987 a major Florida bloom event was dispersed by the Gulf Stream northward into North Carolina waters, where it has since continued to be present (Tester *et al.*, 1991). Unexpectedly, in early 1993 more than 180 human shellfish poisonings were reported from New Zealand, caused by a number of species similar to *Karenia brevis*, now newly described as *K. bicuneiformis, K. brevisulcata, K. papilionacea, K. selliformis* (Jasperse, 1993; Haywood and Steidinger, in prep.). Most likely, this was a member of the hidden plankton flora (previously present in low concentrations), which developed into bloom proportions triggered by unusual climatological conditions (higher than usual rainfall, lower than usual temperature) coinciding with an El Niño event.

### 1.5.3 Fossil blooms of *Gymnodinium catenatum* in the Kattegat-Skagerrak

The present-day distribution of the paralytic shellfish poison-producing dinoflagellate *Gymnodinium catenatum* includes the Gulf of California, Gulf of Mexico, Venezuela, Argentina, Japan, Korea, China, the Philippines, Palau, Tasmania (Australia),

New Zealand, the Mediterranean and the Atlantic coast of Spain, Portugal and Morocco (Hallegraeff and Fraga, 1997). The microreticulate cysts of a closely related (apparently nontoxic) species *Gymnodinium nolleri* were present in unusually large amounts in pollen records from Kattegat sediments (Nordberg and Bergsten, 1988). A multi-disciplinary study (Dale and Nordberg, 1993) to reconstruct the prevailing paleoenvironment has suggested the following scenario: the migration of this organism into the area about 5000 B.P.; its establishment as part of the local plankton; a major blooming phase about 2000–500 B.P. of a magnitude that has not been seen since; and its disappearance during the 'Little Ice Age'.

## 1.6 Transport of dinoflagellate cysts in ballast water or during translocation of shellfish stocks

Cargo-vessel ballast water was first suggested as a vector in the dispersal of non-indigenous marine plankton some ninety 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 sensitive aquaculture areas of Australian waters, with disastrous consequences for commercial shellfish farm operations (Hallegraeff and Bolch, 1992). While the planktonic stages of diatoms and dinoflagellates show only limited survival during the voyage in dark ballast tanks, their resistant resting spores are well suited to survive these conditions. One single ballast tank was thus estimated to contain more than 300 million toxic dinoflagellate cysts which could be germinated into confirmed toxic cultures. Paralytic shellfish poisoning was unknown in the Australian region until the 1980s when the first outbreaks appeared in the ports of Hobart (*Gymnodinium catenatum*), Melbourne (*Alexandrium catenella)* and Adelaide (*A. minutum*). In Hobart, Tasmania, an examination of historical plankton samples and cyst surveys in dated sediment depth cores (McMinn *et al.*, 1997) provided strong circumstantial evidence that the toxic dinoflagellate *G. catenatum* was introduced after 1973. Furthermore, in Melbourne and Adelaide, genetic fingerprinting using rRNA sequencing provided circumstantial evidence for the genetic affinities between Australian and Japanese strains of *A. catenella* and Australian and European strains of *A. minutum* (Scholin *et al.*, 1993; de Salas *et al.*, 2000). The toxic dinoflagellate *Pfiesteria* has been confirmed from ballast water entering North America from Europe (P. Rublee, unpublished).

The evidence of ballast-water transfer of marine organisms other than microscopic algae is considerable and includes species of seaweeds, fish, crustaceans, polychaete worms, starfish and molluscs (Carlton, 1985). As of 1 November 1991, the International Maritime Organization (IMO) has ratified the introduction of voluntary guidelines for ballast-water handling procedures by bulk-cargo vessels. These measures aim to reduce the risk of harmful introductions by encouraging a range of practices such as reballasting at sea (only feasible for vessels up to 40,000 dead weight tonnage), ballasting in deep water and disposal of ballast-tank sediments away from sensitive aquaculture or marine park areas. The most effective measure to prevent the spreading of dinoflagellate cysts via ships' ballast water would be to avoid ballasting during toxic dinoflagellate blooms in ports. Other options using heat, electrical shock or chemical treatment (chlorine, hydrogen peroxide) of ballast





water, either in hold or in onshore facilities, have also been explored (Hallegraeff, 1998).

Another vector for the dispersal of algae (especially their resting cysts) is with the translocation of shellfish stocks from one area to another. The faeces and digestive tracts of bivalves can be loaded with viable dinoflagellate cells and sometimes can also contain resistant resting cysts (Scarratt *et al.*, 1993; Schwinghamer *et al.*, 1994). The Japanese seaweeds *Sargassum muticum* (United Kingdom, Netherlands, Norway), *Undaria pinnatifida* and *Laminaria japonica* (Mediterranean) thus are thought to have been introduced into European waters via sporophyte stages contained with introduced Japanese oyster spat.

## 1.7 Conclusions

Whether the apparent global increase in harmful algal blooms represents a real increase is a question that we will probably not be able to answer conclusively for some time to come. There is no doubt that the growing interest in utilizing coastal waters for aquaculture is leading to greater awareness of toxic algal species. What we are faced with today in the field of harmful algal bloom research is that the effects on public health and the economic impact of harmful algal blooms are showing signs of a truly global 'epidemic' and we should start to respond to this problem. In countries that pride themselves on their disease- and pollution-free status for aquaculture, every effort should be made to quarantine sensitive aquaculture areas against the unintentional introduction of non-indigenous harmful algal species. Furthermore, no aquaculture industry can avoid having to monitor for an increasing number of harmful algal species in the water column and for an increasing number of algal toxins in seafood products.

Most importantly, those responsible for management decisions on pollutant loadings of coastal waters (including decisions on agricultural and deforestation activities in catchment areas) should be made aware that one probable outcome of increased nutrient loading will be an increase in harmful algal blooms. Finally, global climate change studies (El Niño, greenhouse, ozone depletion) need to consider possible impacts on algal-bloom events. A number of new international programmes have been created to study and manage harmful algal blooms and their links to environmental changes in a manner consistent with the global nature of the phenomena involved (see Appendix B). It is hoped that this *Manual on Harmful Marine Microalgae* will facilitate these international efforts.

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