sheltered sea areas for growing on. This increases the habitat area for attachment in the sea, and so increases yield. In the case of *Porphyra* in Japan, such manipulation of environmental conditions allows up to five generations per year from an area of coast where natural seasonal changes would only give one. It is not practical to grow the plants entirely on land in environmentally controlled seawater tanks because of the high cost of the facilities, but it is feasible to retain a small stock of, for example, shells infected with the *Conchocelis*-phase of *Porphyra* from which spores can be obtained on demand by manipulation of daylength and temperature. The technology exists to cultivate seaweeds entirely in land-based tanks should the economics be favorable in the future. For example, there are different genetic strains of *Chondrus crispus*, whose carrageenan yield and type can be controlled by nutrient and salinity conditions. Such tank culture may be useful in the future to produce high value fine chemicals for medical applications.

Seaweeds are the most obvious type of plant in the sea and are the main component of the phytobenthos but other algae and other marine plants are described elsewhere in this Encyclopedia.

## See also

**Coral Reefs. Eutrophication. Exotic Species, Introduction of. Phytoplankton Blooms. Primary Production Distribution. Primary Production Methods. Primary Production Processes. Rocky Shores. Salt Marsh Vegetation.**

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# PHYTOPLANKTON BLOOMS

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## Introduction

Among the thousands of species of microscopic algae at the base of the marine food chain are a few dozen that produce toxins. These species make their presence known in many ways, ranging from massive 'red tides' that discolor the water, to dilute, inconspicuous concentrations of cells noticed only because of the harm caused by their highly potent toxins. Impacts include mass mortalities of wild and farmed fish and shellfish, human illness and death, alterations of marine trophic structure, and death of marine mammals, sea birds, and other animals.

'Blooms' of these algae are commonly called red tides, since, in some cases, the tiny plants increase in abundance until they dominate the planktonic community and change the color of the water with their pigments (**Figure 1**). The term is misleading, however, since nontoxic species can bloom and harmlessly discolor the water, or can cause ecosystem damages as severe as those linked to toxic organisms. Adverse effects can also occur when toxic algal cell concentrations are low and the water is not discolored. Given the confusion surrounding the meaning of 'red tide', the scientific community now prefers the term 'harmful algal bloom' or HAB. This new descriptor includes algae that cause problems because of their toxicity, as well as nontoxic algae that cause problems in other ways. It also applies to macroalgae (seaweeds) which can cause major ecological impacts as well (**Figure 2**).

## Impacts

### Toxic Algae

HAB phenomena take a variety of forms, with multiple impacts (**Table 1**). One major category of impact occurs when toxic phytoplankton are filtered from the water as food by shellfish which then accumulate the algal toxins to levels which can be

**Figure 1** Dead fish and discolored water during a Florida red tide. (Photo credit: Florida Department of Environmental Protection.)

**Figure 2** Seaweed washed up on a Florida beach. Macroalgal blooms, although not toxic, can be quite harmful to coastal ecosystems and cause major economic problems to the tourist industry. (Photo credit: B. Lapointe).

lethal to humans or other consumers. The poisoning syndromes have been given the names paralytic, diarrhetic, neurotoxic, amnesic, and azaspiracid shellfish poisoning (PSP, DSP, NSP, ASP, and AZP, respectively). The symptomology and exposure route for each of these are presented in **Table 2**. Except for ASP, all are caused by biotoxins synthesized by a class of marine algae called dinoflagellates. A fifth human illness, ciguatera fish poisoning (CFP) is caused by toxins produced by dinoflagellates that attach to surfaces in many coral reef communities. Ciguatoxins are transferred through the food chain from herbivorous reef fishes to larger carnivorous, commercially valuable finfish. The final human illness linked to toxic algae is called possible estuary-associated syndrome (PEAS). This vague term reflects the poor state of knowledge of the human health effects of the dinoflagellate *Pfiesteria piscicida* and related organisms that have been linked to symptoms such as deficiencies in learning and memory, skin lesions, and acute respiratory and eye irritation – all after exposure to estuarine waters where *Pfiesteria*-like organisms have been present.

Another type of HAB impact occurs when marine fauna are killed by algal species that release toxins and other compounds into the water, or that kill without toxins by physically damaging gills or by creating low oxygen conditions as bloom biomass decays. Fish and shrimp mortalities from these types of HABs at aquaculture sites have increased considerably in recent years. HABs also cause mortalities of wild fish, sea birds, whales, dolphins, and other marine animals. To understand the breadth of these ecosystem impacts, think of the transfer of toxins through the food web as analogous to the flow of carbon. Just as phytoplankton are the source of fixed carbon that moves through the food web, they can also be the source of toxins which cause adverse effects either through toxin transmitted directly from the algae to the affected organism or indirectly through food web transfer.

A prominent example of direct toxin transfer was the death of 19 whales in Massachusetts in 1987 due to saxitoxin that had accumulated in mackerel that the whales consumed. Similar events occurred in Monterey Bay, California in 1998 and again in 2000 when hundreds of sea lions died from domoic acid (the ASP toxin) vectored to them via anchovies. In both cases, the food fish accumulated algal toxins through the food web and passed those toxins to the marine mammals.

Adult fish can be killed by the millions in a single outbreak, with obvious long- and short-term ecosystem impacts (**Figure 3**). Likewise, larval or juvenile stages of fish or other commercially important fisheries species can experience mortalities from algal toxins. Impacts of this type are far more difficult to detect than the acute poisonings of humans or

**Table 1** Some harmful effects caused by algae in coastal and brackish waters

| *Effect* | *Causative organisms* |
|---|---|
| *Human health syndrome* | |
| Paralytic shellfish poisoning (PSP) | *Alexandrium* spp., *Pyrodinium bahamense* var. *compressum, Gymnodinium catenatum, Anabena circinalis, Aphanizomenon* spp. |
| Diarrhetic shellfish poisoning (DSP) | *Dinophysis* spp., *Prorocentrum* spp. |
| Neurotoxic shellfish poisoning (NSP) | *Gymnodinium breve* |
| Azaspiracid shellfish poisoning (AZP) | *Protoperidinium crassipes* |
| Amnesic shellfish poisoning (ASP) | *Pseudo-nitzschia* spp. |
| Ciguatera fish poisoning (CFP) | *Gambierdiscus toxicus* |
| Respiratory problems and skin irritation | *Gymnodinium breve, Pfiesteria piscicida, Nodularia spumigena* |
| Possible estuary-associated syndrome (PEAS) | *Pfiesteria piscicida, P. shumwayae,* and possibly other *Pfiesteria*-like organisms |
| Hepatotoxicity | *Microcystis aeruginosa, Nodularia spumigena* |
| *Mortality of wild and cultured marine resources* | |
| Hemolytic, hepatotoxic, osmoregulatory effects and other unspecified toxicity | *Gymnodinium* spp., *Cochlodinium polykrikoides, Pfiesteria piscicida, Heterosigma carterae, Chattonella* spp., *Fibrocapsa japonica, Chrysochromulina* spp., *Prymnesium* spp., *Aureococcus anophagefferens, Microcystis* spp. |
| Mechanical damage to gills | *Chaetoceros* spp., *Leptocylindrus* spp. |
| Gill clogging and necrosis | *Phaeocystis* spp., *Thalassiosira* spp. |
| *Inhibition of tourism and recreational activities* | |
| Production of foams, mucilage, discoloration, repellent odors | *Noctiluca scintillans, Phaeocystis* spp., *Cylindrotheca closterium, Aureococcus anophagefferens, Nodularia spumigena* |
| Seaweed accumulation on beaches; coral reef overgrowth | *Cladophera*, *Codium*, *Ulva*, and many other species |
| *Adverse effects on marine ecosystems* | |
| Hypoxia, anoxia | *Noctiluca scintillans, Ceratium* spp., many others |
| Negative effects on feeding behavior, shading, reduction of water clarity | *Aureococcus anophagefferens, Aureoumbra lagunensis, Phaeocystis* spp. |
| Seaweed accumulation on beaches; coral reef overgrowth | *Cladophera, Codium, Ulva*, and many other species |
| Toxicity to marine organisms, including invertebrates, fish, mammals, and birds | *Gymnodinium breve, Alexandrium* spp., *Pseudo-nitzschia australis, Cocchlodinium polykrikoides* |

(Modified from Zingone and Enevoldsen, 2000.)

higher predators, since exposures and mortalities are subtle and often unnoticed. Impacts might not be apparent until years after a toxic outbreak, such as when a year class of commercial fish reaches harvesting age but is in low abundance. Chronic toxin exposure may therefore have long-term consequences that are critical with respect to the sustainability or recovery of natural populations at higher trophic levels. Many believe that ecosystem-level effects from toxic algae are more pervasive than are realized, affecting multiple trophic levels, depending on the ecosystem and the toxin involved.

## Nontoxic Blooms

Nontoxic blooms of algae can cause harm in a variety of ways. One prominent mechanism relates to the high biomass that some blooms achieve. When this biomass begins to decay as the bloom terminates, oxygen is consumed, leading to widespread mortalities of all plants and animals in the affected area. These 'high biomass' blooms are sometimes linked to excessive pollution inputs (see below), but can also occur in relatively pristine waters.

Large, prolonged blooms of nontoxic algal species can reduce light penetration to the bottom, decreasing densities of submerged aquatic vegetation (SAV). Loss of SAV can have dramatic impacts on coastal ecosystems, as these grass beds serve as nurseries for the food and the young of commercially important fish and shellfish. Such indirect ecosystem effects from a nontoxic HAB were seen with a dense brown tide that lasted for over 7 years in the Laguna Madre section of southern Texas. The dense, coffee-colored bloom reduced light penetration and dramatically altered the abundance of seagrasses over a wide area.

**Table 2** Human illnesses associated with harmful algal blooms

| *Syndrome* | *Causative organisms* | *Toxin produced* | *Route of acquisition* | *Clinical manifestations* |
|---|---|---|---|---|
| Ciguatera fish poisoning (CFP) | *Gambierdiscus toxicus* and others | Ciguatoxin, maitotoxin | Toxin passed up marine food chain; illness results from eating large, carnivorous reef fish | Acute gastroenteritis, paresthesias and other neurological symptoms |
| Paralytic shellfish poisoning (PSP) | *Alexandrium* species, *Gymnodinium catenatum, Pyrodinium bahamense* var. *compressum*, and others | Saxitoxins | Eating shellfish harvested from affected areas | Acute paresthesias and other neurological manifestations; may progress rapidly to respiratory paralysis and death |
| Neurotoxic shellfish poisoning (NSP) | *Gymnodinium breve* and others | Brevetoxins | Eating shellfish harvested from affected areas; toxins may be aerosolized by wave action | Gastrointestinal and neurological symptoms; respiratory and eye irritation with aerosols |
| Diarrhetic shellfish poisoning (DSP) | *Dinophysis* species | Okadaic acid and others | Eating shellfish harvested from affected areas | Acute gastroenteritis |
| Azaspiracid shellfish poisoning (AZP) | *Protoperidinium crassipes* | Azaspiracids | Eating shellfish harvested from affected areas | Neurotoxic effects with severe damage to the intestine, spleen, and liver tissues in test animal |
| Amnesic shellfish poisoning (ASP) | *Pseudo-nitzchia* species | Domoic acid | Eating shellfish (or, possibly, fish) harvested from affected areas | Gastroenteritis, neurological manifestations, leading in severe cases to amnesia, coma, and death |
| Possible estuary-associated syndrome (PEAS) | *Pfiesteria piscicida* and others | Unidentified | Exposure to water or aerosols containing toxins | Deficiencies in learning and memory; acute respiratory and eye irritation, acute confusional syndrome |

(Modified from Morris, 1999.)

**Figure 3** Toxic blooms can kill wild and aquacultured fish by the millions. (Photo credits: G. Pitcher and M. Aramaki).

Macroalgae (seaweeds) also cause problems. Over the past several decades, blooms of macroalgae have been increasing along many of the world's developed coastlines. Macroalgal blooms occur in nutrient-enriched estuaries and nearshore areas that are shallow enough for light to penetrate to the seafloor. These blooms have a broad range of ecological effects, and often last longer than 'typical' phytoplankton HABs. Once established, macroalgal blooms can remain in an environment for years unless the nutrient supply decreases. They can be particularly harmful to coral reefs. Under high nutrient conditions, opportunistic macroalgal species outcompete, overgrow, and replace the coral.

Nontoxic phytoplankton can also kill fish. The diatom genus *Chaetoceros* includes several species which have been associated with mortalities of farmed salmon, yet no toxin has ever been identified in that group. These species have long, barbed spines that lodge in gill tissues, leading to massive discharge of mucus that eventually results in lamellar degeneration and death due to the reduction in oxygen exchange. Why these diatoms have barbed spines is unknown. It is improbable that they have evolved specifically to kill fish, since the only mortalities from these species are caged fish that cannot escape the blooms. The problems now faced by the fish-farming industry are most likely the unfortunate result of an evolutionary strategy by certain *Chaetoceros* species to avoid predation or to stay afloat.

## The Toxins

The toxins produced by some HAB species are among the most potent natural poisons known. Saxitoxin, for example, is 1000 times more potent than cyanide, and 50 times stronger than curare. Many of the toxin classes are not single chemical entities, but instead represent families of compounds of similar chemical structure (**Table 2**). These toxins bind with high affinity to specific receptor sites such as ion channels of excitable cells (**Table 3**). Most binding is reversible, but dissociation times can be quite prolonged. Each derivative of the parent toxin is slightly different in structure, and thus binds to the target receptor with different affinity. Weaker toxins tend to dissociate more quickly, accounting for their reduced potency. The saxitoxin family binds to sodium channels, which are the protein 'tunnels' responsible for the flux of sodium in and out of nerve and muscle cells. The brevetoxins also bind to the sodium channel, but to a different site. Their effect is to cause continuous activation of the channel, rather than a blockage, as with saxitoxin. Domoic acid disrupts normal neurochemical transmission in the brain by binding to kainate receptors in the central nervous system. This results in sustained depolarization of the neurons, and eventually in cell degeneration and death. One unfortunate symptom in ASP victims is permanent, short-term memory loss due to lesions that form in portions of the brain such as the hippocampus where there is a high density of kainate receptors.

Man is exposed to algal toxins principally by consumption of contaminated seafood products, although one type of toxin (brevetoxin) also causes respiratory asthma-like symptoms because of aerosol formation due to wave action. Acute single-dose lethality of seafood toxins has been extensively studied, but chronic and/or repeated exposure to low toxin concentrations, which undoubtedly occurs, has not been adequately examined. There is also a serious lack of knowledge as to how the

**Table 3** Properties of major algal toxins (values in parentheses (n) indicate the number of toxin derivatives)

| *Toxin family* (n) | *Syndrome* | *Solubility* | *Action on* | *Pharmacology* |
|---|---|---|---|---|
| Saxitoxin (18) | PSP | Water soluble | Nerve, brain | Blockage of sodium channel |
| Brevetoxin (9) | NSP | Lipid soluble | Nerve, muscle, lung, brain | Selective sodium channel activators |
| Azaspiracid (3) | AZP | Lipid soluble | Nerve, liver, intestine, spleen | Not yet characterized |
| Okadaic acid (12) | DSP | Lipid soluble | Enzymes | Diarrhea by stimulating phosphorylation of a protein that controls sodium excretion of intestinal cells |
| Domoic acid (11) | ASP | Hot acid extraction | Brain | Binds to glutamate receptors in the brain, causing continuous stimulation of brain cells; lesions formed |
| Maitotoxin/ciguatoxin | CFP | Water/lipid soluble | Nerve, muscle, heart, brain | Activation of calcium/sodium channels |

toxins are distributed throughout the body and eliminated. Other important questions include how long the toxins circulate before elimination, and how they are metabolized by living organisms. These knowledge gaps prevent researchers from devising antidotes or effective treatments which may alleviate or lessen the symptoms. Therapeutic intervention is primarily limited to symptomatic treatment and life support.

The mechanisms by which fish are killed by toxic HABs are not well understood. Neurotoxins can rapidly act on specific fish tissues, significantly reducing heart rate and resulting in reduced blood flow and death from lack of oxygen. Other classes of compounds released by HABs such as reactive oxygen species, polyunsaturated fatty acids, and galactolipids are not toxins *per se*, but still can kill fish and other animals rapidly. Some have hemolytic activity, and others cause gill rupture, excessive production of mucus, hypoxia, and edema.

Scientists are searching for biochemical pathways within the algae to explain the metabolic role of the HAB toxins, but the search thus far has been fruitless. The toxins are not proteins, and all are synthesized in a series of chemical steps requiring multiple genes. Biosynthetic pathways have been proposed, but no chemical intermediates have been identified, nor have unique enzymes used only in toxin production yet been isolated. All we have are tantalizing clues that relate to toxin variability in individual cells, such as a 10-fold increase in saxitoxin production in cultured *Alexandrium* cells that are limited by phosphorus rather than nitrogen, or changes in the relative abundance of the different toxin derivatives produced by a particular dinoflagellate strain when growth conditions are varied. These observations indicate that the metabolism of the toxins is a dynamic process within the algae, but it is still not clear whether they have a specific biochemical role or are simply secondary metabolites. As with the nontoxic, spiny *Chaetoceros* species that kill fish, the illnesses and mortalities caused by algal 'toxins' may simply be the result of the 'accidental' affinity of those compounds for receptor sites on ion channels or tissues humans and in higher animals.

## Ecology and Population Dynamics

### Growth Features, Bloom Mechanisms

Impacts from HABs are necessarily linked to the population size and distribution of the causative algae. The growth and accumulation of individual algal species in a mixed assemblage of marine organisms are exceedingly complex processes involving an array of chemical, physical, and biological interactions. Given the diverse array of algae from several different classes that produce toxins or cause problems in a variety of oceanographic systems, attempts to generalize the bloom dynamics of HAB species are doomed to failure. Some common mechanisms can nevertheless be highlighted.

HABs are seldom caused by the explosive growth of a single species that rapidly dominates the water column. As stated by Ryther and co-workers many years ago, '… there is no necessity to postulate obscure factors which would account for a prodigious growth of dinoflagellates to explain red water. It is necessary only to have conditions favoring the growth and dominance of a moderately large population of a given species, and the proper hydrographic and meteorological conditions to permit the accumulation of organisms at the surface and to effect their future concentrations in localized areas.' In other words, winds, tides, currents, fronts, and other features can create discrete patches of cells of streaks of red water at all scales.

HABs in temperate and high latitudes are predominantly summer, coastal phenomena. They commonly occur during periods when heating or freshwater runoff create a stratified surface layer overlying colder, nutrient rich waters. Since the upper layer is quickly stripped of nutrients by other fast-growing algae, the onset of stratification often means that the only significant source of major plant nutrients lies below the interface between the layers – the pycnocline. This situation favors dinoflagellates and other motile algae, since nonmotile phytoplankton are unable to remain in suspension in the upper layer, and thus sink out of the zone where light permits photosynthesis. In contrast, motile algae are able to regulate their position and access the nutrients below the pycnocline. Many HAB species can swim at speeds in excess of 10 m $d^{-1}$, and some undergo marked vertical migration, in which they reside in surface waters during the day to harvest the sunlight and then swim to the pycnocline and below to take up nutrients at night. This strategy can explain how dense accumulations of cells can appear in surface waters that are devoid of nutrients and which would seem to be incapable of supporting such prolific growth.

Swimming can also allow a species to persist at some optimum depth in the presence of vertical currents. One striking observation about some HAB blooms is that highly concentrated subsurface layers of cells can sometimes be only tens of centimeters thick – so called 'thin layers' of cells. This is another

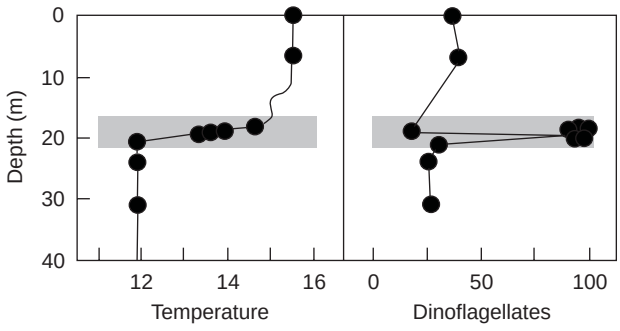


**Figure 4** Subsurface accumulation of 'thin layers' of HAB species are common, as shown in this dataset from La Rochelle, France. (Adapted with permission from Gentien *et al.* (1995)).

example of the importance of physical processes in determining organism distributions in stratified coastal systems. **Figure 4** shows a vertical profile of temperature and the corresponding thin layer of dinoflagellates that formed under those conditions along the coast of France. Similar thin layers are found throughout the world with scales as small as 10 cm in the vertical and 10 km in the horizontal.

Horizontal transport of blooms is also an important feature of some HABs, often over large distances. Major toxic outbreaks can suddenly appear at a site due to the transport of established blooms from other areas by ocean currents. This is the case in the western Gulf of Maine in the USA, where blooms of toxic dinoflagellates are transported hundreds of kilometers within a buoyant coastal current formed by the outflow of a major river system. An NSP outbreak in North Carolina in 1987 is another example of such long distance transport. The toxic *Gymnodinium breve* population that contaminated North Carolina shellfish that year and drove tourists and residents from beaches originated in a bloom off the south-west coast of Florida, nearly 1500 km away. That bloom was carried out of the Gulf of Mexico and up the south-east coast of the USA by a combination of current systems, culminating in the Gulf Stream. After approximately 30 days of transport, a filament of water separated from the Gulf Stream and moved onto the narrow continental shelf of North Carolina, carrying toxic *G. breve* cells with it. The warm water mass remained in nearshore waters and was identifiable in satellite images for nearly 3 weeks (**Figure 5**).

Another important aspect of physical/biological coupling relates to the enhanced phytoplankton biomass that can occur at hydrographic features such as fronts. This enhanced biomass is the result of the interaction between physical processes such as upwelling, shear, and turbulence, and physiological processes such as swimming and enhanced nutrient uptake. One example is the linkage between tidally generated fronts and the sites of massive blooms of the toxic dinoflagellate *Gyrodinium aureolum* (now called *Gymnodinium mikimotoi*) in the North Sea. The pattern generally seen is a high surface concentration of cells at the frontal convergence, contiguous with a subsurface chlorophyll maximum which follows the sloping interface between the two water masses beneath the stratified side of the front (**Figure 6**). The surface signature of the chlorophyll maximum (sometimes a visible red tide) may be 1–30 km wide. Chlorophyll concentrations are generally lower and uniform on the well-mixed side of the front. Lateral transport of the front and its associated cells brings toxic *G. aureolum* populations into contact with nearshore fish and other susceptible resources, resulting in massive mortalities. This is a case where small-scale physical/biological coupling results in biomass accumulation, and larger-scale advective mechanisms cause the biomass to become harmful.

## Life Histories

An important aspect of many HABs is their reliance on life history transformations for bloom initiation and decline. A number of key HAB species have dormant, cyst stages in their life histories, including *Alexandrium* spp., *Pyrodinium bahamense*, *Gymnodinium catenatum*, *Cocchlodinium polykrikoides, Gonyaulax monilata, Pfiesteria piscicida, Chattonella* spp., and *Heterosigma carterae*. Resting cysts remain in bottom sediments (sometimes termed 'seedbeds') when conditions in the overlying waters are unsuitable for growth. When conditions improve, such as with seasonal warming, the cysts germinate, inoculating the water column with a population of cells that begins to divide asexually via binary fission to produce a bloom. At the end of the bloom, often in response to nutrient limitation, vegetative growth ceases and the cells undergo sexual reproduction, whereby gametes are formed that fuse to form the swimming zygotes that ultimately become dormant cysts. **Figure 7** shows an example of the *Alexandrium tamarense* life history. Clearly, the location of cyst seedbeds can be an important determinant of the location of resulting blooms, and the size of the cyst accumulations can affect the magnitude of the blooms as well. It is generally believed, however, that environmental regulation of cell division is more important to eventual bloom magnitude than the size of the germination inoculum from cysts.

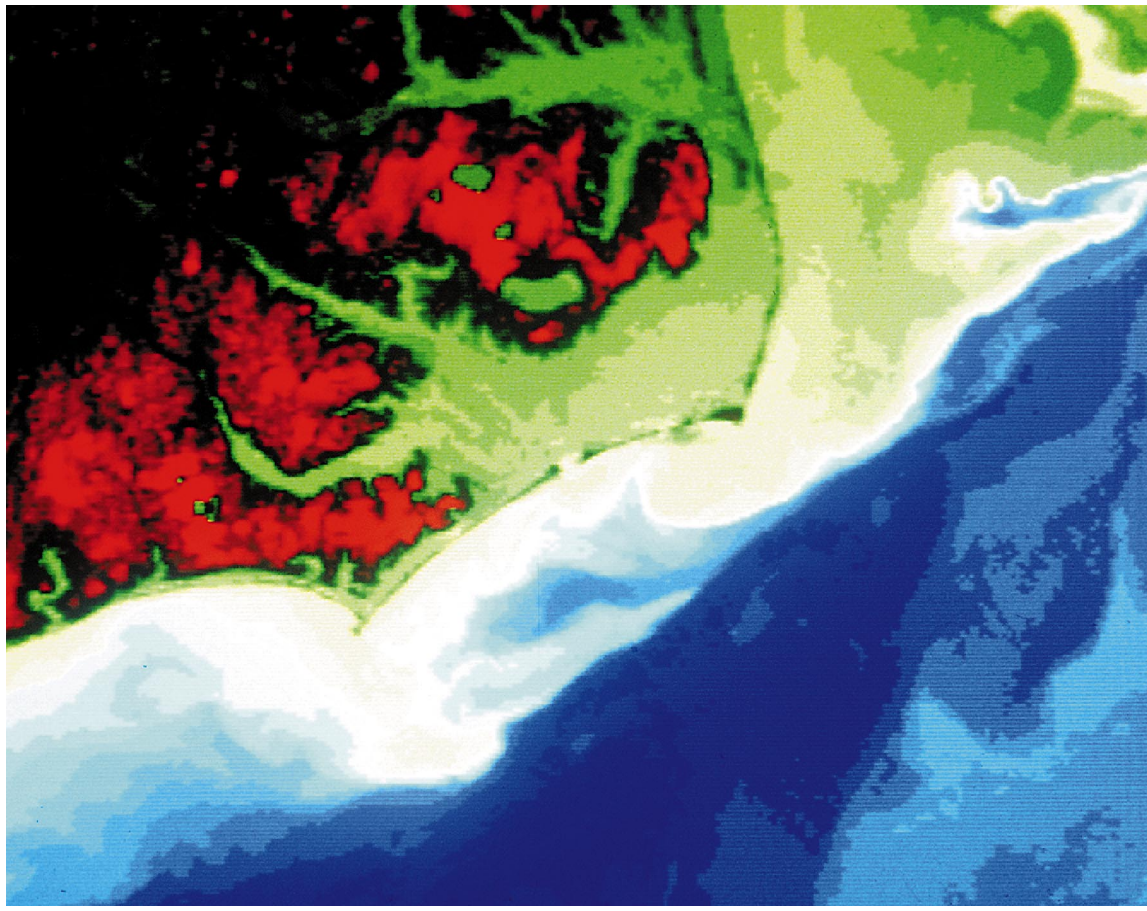

**Figure 5** Satellite image of sea surface temperature, showing the warm Gulf Stream (blue) off the coast of North Carolina, A filament of the Gulf Stream can be seen extending towards shore. This carried a toxic population of *Gymnodinium breve*; that population originated in the Gulf of Mexico, $>$1500 km away. (Photo credit: P. Tester.).

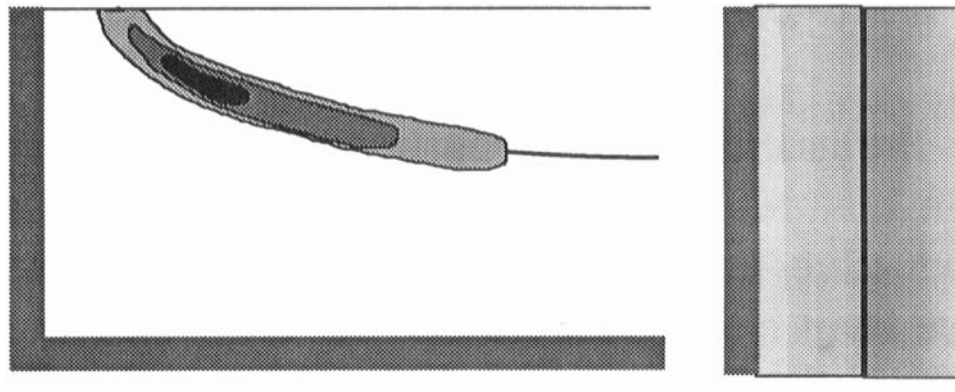


**Figure 6** Accumulation of HAB cells near a front. This schematic demonstrates how cells can accumulate at a frontal convergence, with a surface manifestation of the bloom at the frontal convergence, and a subsurface extension of the bloom that extends along the sloping pycnocline. (Adapted with permission from Franks (1992).)

Cysts are also important in species dispersal. Natural or human-assisted transport of cysts from one location to another (e.g. via ballast water discharge or shellfish seeding) can allow a species to colonize a region and extend its geographic range. In 1972, a hurricane introduced *Alexandrium fundyense* into southern New England waters from established populations in the Bay of Fundy. Since

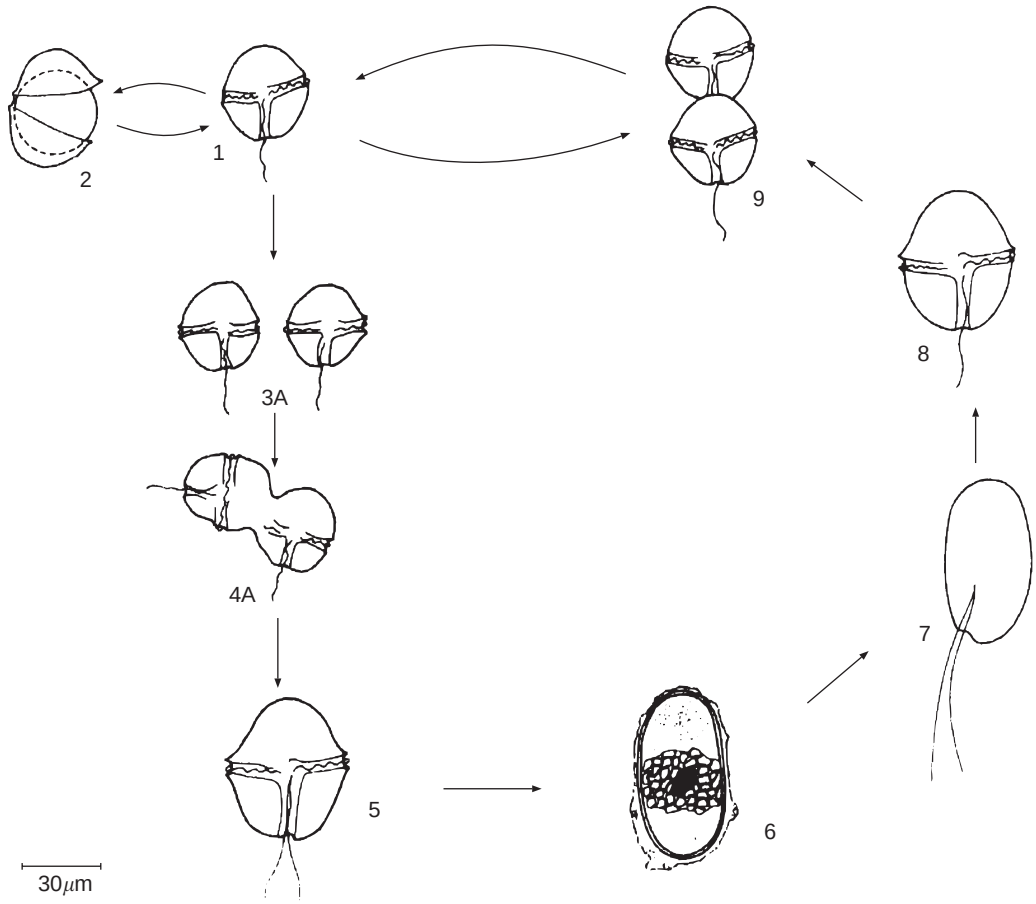


**Figure 7** Life cycle diagram of *Alexandrium tamarense*. Stages are identified as follows: (1) vegetative, motile cell; (2) temporary or pellicle cyst; (3) 'female' and 'male' gametes; (4) fusing gametes; (5) swimming zygote or planozygote; (6) resting cyst or hypnozygote; (7,8) motile, germinated cell or planomeiocyte; and (9) pair of vegetative cells following division. Redrawn from Anderson *et al.* 1998.).

that time, PSP has become an annually recurrent phenomenon in the region. Another example of human-assisted species introductions is the appearance of *Gymnodinium catenatu*m in Tasmania in the 1970s, coincident with the development of a wood chip industry involving commercial vessels and frequent ballast water discharge.

### Grazing Interactions

HAB cells can be food for herbivorous marine organisms (grazers). The extent to which grazing can control HABs depends upon the abundance of zooplankton, their ability to ingest the harmful algal species, and the effects of the HAB species on the grazers. In this context, one of the least understood aspects of toxic algal physiology concerns the metabolic or ecological roles of the toxins. Some argue that toxins evolved as a defense mechanism against grazing. Indeed, experimental studies demonstrate that zooplankton are affected to some degree by the algal toxins they ingest. Two different effects have been noted. The first is a gradual incapacitation during feeding, as if the zooplankton are gradually paralyzed or otherwise impaired. (One study even showed that a tintinnid ciliate could only swim backwards, away from its intended algal prey, after exposure to a toxic dinoflagellate culture.) The second response is an active rejection or regurgitation of the toxic algae by a grazing animal, as if it had an unpleasant taste. Either of these mechanisms would result in reduced grazing pressure on toxic forms, which would facilitate the formation of a bloom. However, this cannot be the sole explanation for the presence of the toxins, as nontoxic phytoplankton are abundant, and form massive blooms.

An example of these mechanisms is seen in **Figure 8**, which depicts the feeding behavior of the copepod *Acartia tonsa* in mixtures containing equal concentrations of toxic and nontoxic *Alexandrium*

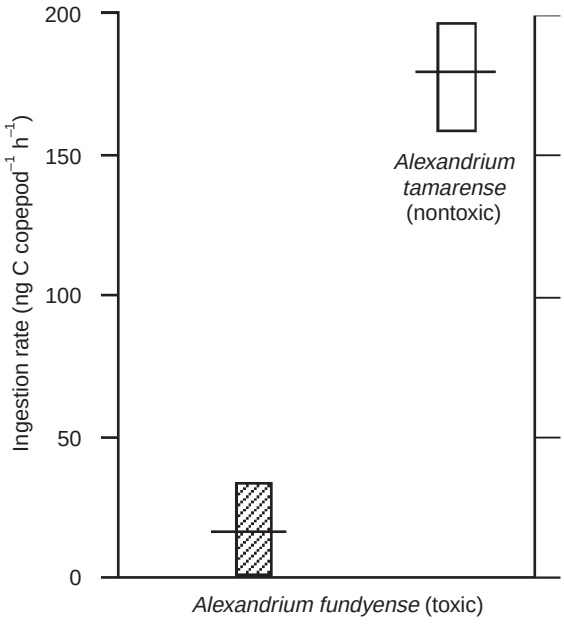


**Figure 8** Feeding behavior of the copepod *Acartia tonsa* in mixtures containing equal concentrations of toxic and non-toxic *Alexandrium* spp. dinoflagellates. Central bars denote mean rates, boxes indicate standard deviation. Redrawn with permission from Teegarden 1999.

spp. dinoflagellates. The copepods consumed nontoxic *Alexandrium* sp. cells at significantly higher rates than toxic cells, indicating chemosensory discrimination of toxic cells by the grazers.

Although there is clear evidence of the avoidance of certain HABs by grazers, or of incapacitation of the grazers themselves during feeding, it is also apparent that some toxins can be vectored through the food web via grazers. Fish kills of herring in the Bay of Fundy have been linked to PSP toxins from *Alexandrium* species that had been grazed by pteropods (small planktonic snails that are a favorite food of herring). Fortunately, in terms of human health, subsequent laboratory studies found that herring and other fish are very sensitive to these toxins and, unlike shellfish, die before they accumulate the toxins at dangerous levels in their flesh. Through similar food web transfer events, *Alexandrium* toxins have been implicated in kills of menhaden, sand lance, bluefish, dogfish, skates, monkfish, and even whales. Similar arguments can be made for other HAB toxins, especially the brevetoxins which have been linked to deaths of dolphins, and domoic acid (the ASP toxin), which has caused extensive bird and sea lion mortalities along the eastern Pacific coast of the USA and Mexico.

## Trends

The nature of the global HAB problem has changed considerably over the last several decades (**Figure 9**). Virtually every coastal country is now threatened by harmful or toxic algal species, often in many locations and over broad areas. Thirty years ago, the problem was much more scattered and sporadic. The number of toxic blooms, the economic losses from them, the types of resources affected, and the number of toxins and toxic species have all increased dramatically in recent years. Disagreement only arises with respect to the reasons for this expansion, of which there are several possibilities.

New bloom events may simply reflect indigenous populations that are discovered because of improved chemical detection methods and more observers. The discovery of ASP along the west coast of the USA in 1991 is a good example of this, as toxic diatom species were identified and their toxin detected as a direct result of communication with Canadian scientists who had discovered the same toxin 4 years earlier and developed new chemical detection methods for domoic acid.

Several other 'spreading events' are most easily attributed to natural dispersal via currents, rather than human activities. As described above, the first NSP event ever to occur in North Carolina was shown to be a Florida bloom transported over 1500 km by the Gulf Stream – a totally natural phenomenon with no linkage to human activities. Some believe that humans may have contributed to the global HAB expansion by transporting toxic species in ship ballast water (e.g. *Gymnodinium catenatum* in Tasmania) or by shellfish relays and seeding. Another factor underlying the global increase in HABs is that we have dramatically increased aquaculture activities worldwide (**Figure 10**), and this inevitably leads to increased monitoring of product quality and safety, revealing indigenous toxic algae that were probably always there.

Nutrient enrichment is another cause for the increasing frequency of HAB events worldwide. Manipulation of coastal watersheds for agriculture, industry, housing, and recreation has drastically increased nutrient loadings to coastal waters. Just as the application of fertilizer to lawns can enhance grass growth, marine algae grow in response to nutrient inputs. Shallow and restricted coastal waters that are poorly flushed appear to be most susceptible to nutrient-related algal problems. Nutrient enrichment of such systems often leads to excessive production of organic matter, a process known as eutrophication, and increased frequencies and magnitudes of phytoplankton blooms, including

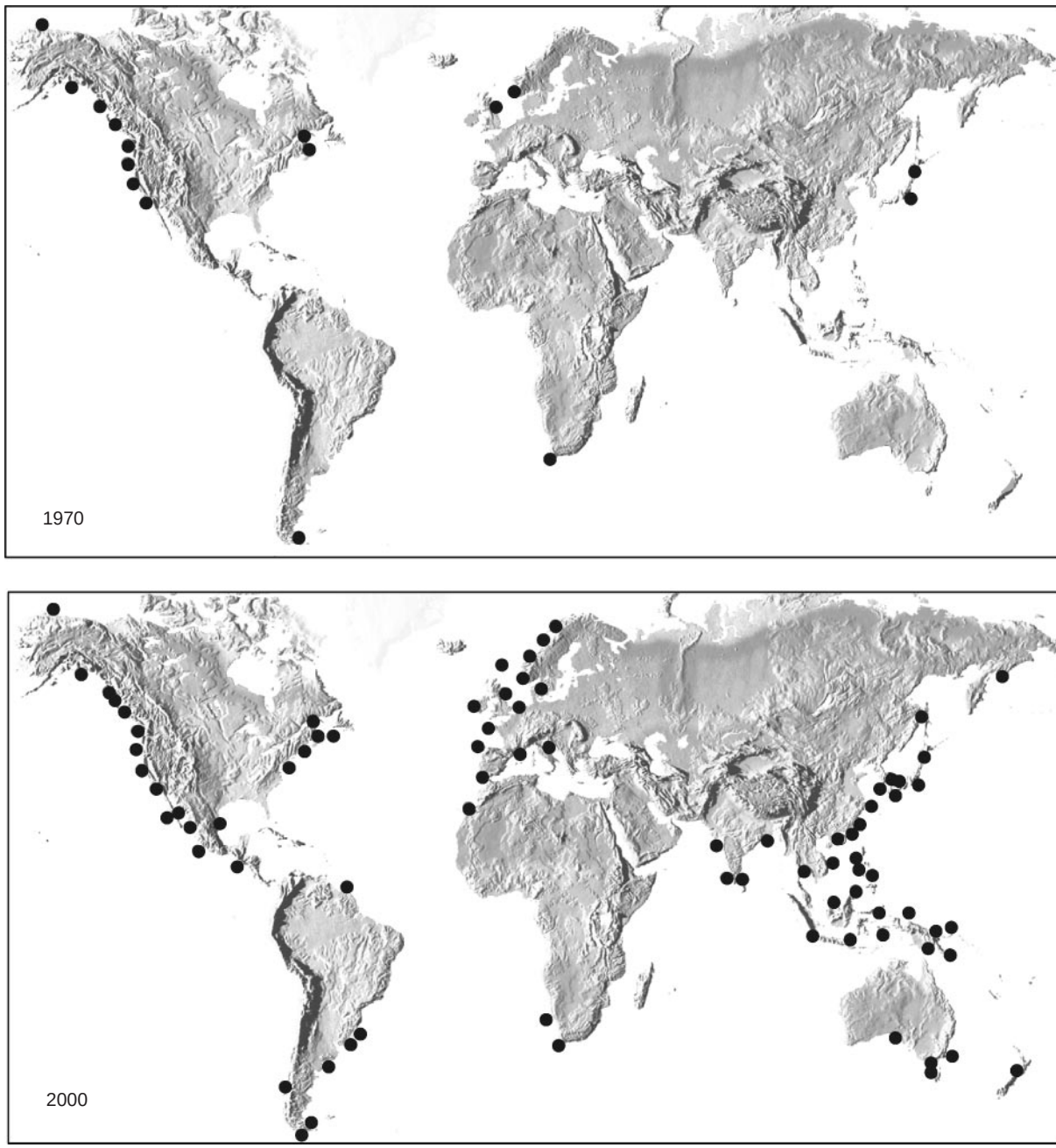


**Figure 9** Expansion of the PSP problem over the past 30 years. Sites with proven records of PSP-causing organisms are noted in 1970, and again in 2000. Modified from Hallegraeff 1993. ●, PSP.

HABs. There is no doubt that this is true in certain areas of the world where pollution has increased dramatically. It is perhaps real, but less evident in areas where coastal pollution is more gradual and unobtrusive. A frequently cited dataset from an area where pollution has been a significant factor in HAB incidence is from the Inland Sea of Japan, where visible red tides increased steadily from 44 per year in 1965 to over 300 a decade later, matching the pattern of increased nutrient loading from pollution (**Figure 11**). Effluent controls were instituted in the mid-1970s, resulting in a 70% reduction in the number of red tides that has persisted to this day. A related data set for the Black Sea documents a dramatic increase in red tides up to the mid-1990s, when the blooms began to decline. That reduction, which has also continued to this day, has been linked to reductions in fertilizer usage in

upstream watersheds by former Soviet Union countries no longer able to afford large, state-subsidized fertilizer applications to agricultural land.

Anthropogenic nutrients can stimulate or enhance the impact of toxic or harmful species in several ways. At the simplest level, toxic phytoplankton may increase in abundance due to nutrient enrichment, but remain as the same relative fraction of the total phytoplankton biomass (i.e. all phytoplankton species are affected equally by the enrichment). Alternatively, some contend that there has been a selective stimulation of HAB species by pollution. This view is based on the 'nutrient ratio hypothesis' which argues that environmental selection of phytoplankton species is associated with the relative availability of specific nutrients in coastal waters, and that human activities have altered these nutrient supply ratios in ways that favor harmful forms. For example, diatoms, the vast majority of which are harmless, require silicon in their cell walls, whereas most other phytoplankton do not. Since silicon is not abundant in sewage effluent but nitrogen and phosphorus are, the N:Si or P:Si ratios in coastal waters have increased through time over the last several decades. Diatom growth in these waters will cease when silicon supplies are depleted, but other phytoplankton classes (including toxic or harmful species) can continue to proliferate using the 'excess' nitrogen and phosphorus.

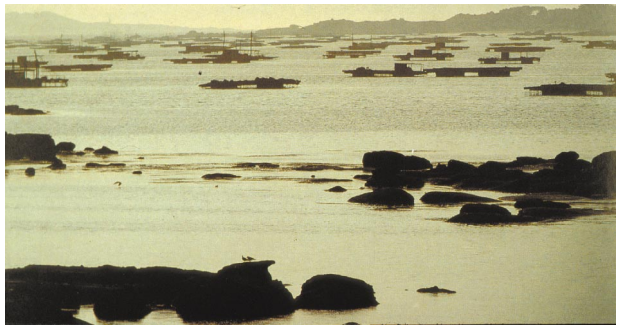

**Figure 10** One reason for the global expansion in HABs relates to the increase in aquaculture activities, as shown with the dense concentration of mussel rafts in Spain. Not only do these intense farming operations alter the nutrient loads and plankton composition of local waters, but the intense scrutiny of the product for quality control purposes can reveal cryptic toxins that may have always been present in the region. (Photograph courtesy of D.M. Anderson.).

This concept is controversial, but is supported by a 23-year time series off the German coast which documents the general enrichment of coastal waters with nitrogen and phosphorus, as well as a fourfold increase in N:Si and P:Si ratios. This was accompanied by a striking change in the composition of the phytoplankton community, as diatoms decreased and flagellates increased more than 10-fold.

Similar arguments hold for changes in N:P ratios, which have also changed due to the differences in removal of these nutrients with standard wastewater treatment practices. Some HAB species, such as *Phaeocystis pouchetii*, the species responsible for massive blooms that foul beaches with thick layers of smelly foam and slime, are not good competitors for phosphorus. As the N:P ratio of Dutch coastal waters decreased over time due to pollution effects, the size and duration of *Phaeocystis* blooms increased (**Figure 12**).

The global increase in HAB phenomena is thus a reflection of two factors – an actual expansion of

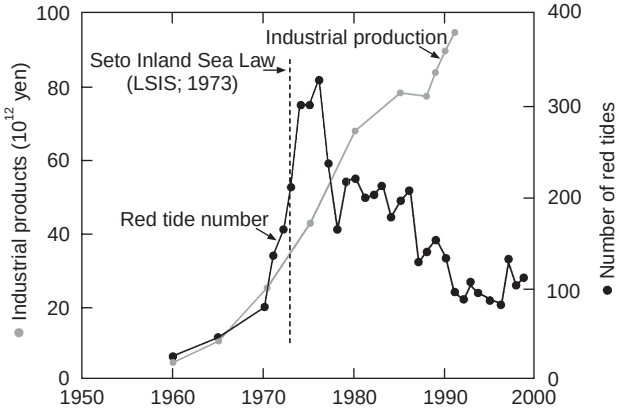


**Figure 11** Human pollution can directly affect the number of red tides and HABs. This figure shows how blooms increased dramatically during the 1960s and 1970s in the Seto Inland Sea of Japan, but declined after pollution control legislation was enacted. Redrawn from Okaichi 1998.

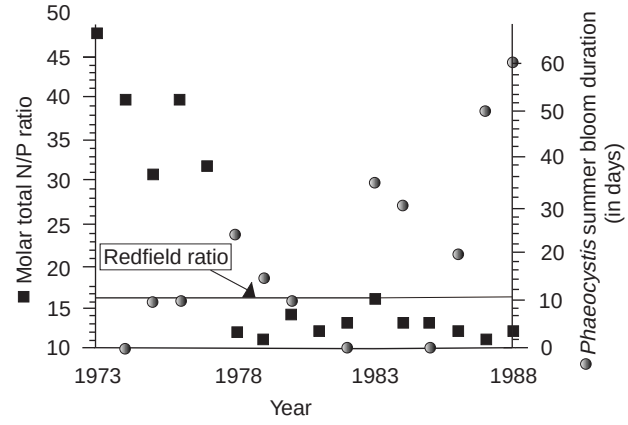


**Figure 12** Changes in the duration of *Phaeocystis* blooms as the N:P ratio of Dutch coastal waters declined. Redrawn from Riegman *et al.* 1992.

the problem, and an increased awareness of its size or scale. It is expanding due to pollution or other global change issues, but improved methods and enhanced scientific inquiry have also led to a better appreciation of its size or scale. The fact that part of the expansion is a result of increased awareness should not negate our concern, nor should it alter the manner in which we mobilize resources to attack it. The fact that it is also growing due to human activities makes our concerns even more urgent.

## Management Issues

Management options for dealing with the impacts of HABs include reducing their incidence and extent (prevention), stopping or containing blooms (control), and minimizing impacts (mitigation). Where possible, it is preferable to prevent HABs rather than to treat their symptoms. Since increased pollution and nutrient loading may cause an increased incidence of outbreaks of some HAB species, these events may be prevented by reducing pollution inputs to coastal waters, particularly industrial, agricultural, and domestic effluents high in plant nutrients. This is especially important in shallow, poorly flushed coastal waters that are most susceptible to nutrient-related algal problems. Other strategies that may prevent HAB events include: regulating the siting of aquaculture facilities to avoid areas where HAB species are present, modifying water circulation for those HABs where restricted water exchange is a factor in bloom development, and restricting species introductions (e.g. through regulations on ballast water discharges or shellfish and finfish transfers for aquaculture). Potential approaches to control HABs are similar to those used to control pests on land – e.g. biological, physical, or chemical treatments that directly target the bloom cells. However, more research is needed before these means are used to control HABs in natural waters. The most effective mitigation tools are monitoring programs that detect toxins in shellfish and/or monitor the environment for evidence of HAB events. These programs can provide advance warnings of outbreaks and/or indicate areas that should be closed to harvesting. Recent technological advances, such as remote-sensing and molecular techniques, have increased detection and characterization of HAB species and blooms, and are playing an increasing role in monitoring programs worldwide. A long-term goal of these HAB monitoring programs and tools is to develop the ability to forecast bloom development and movement.

## Summary

The HAB problem is significant and growing worldwide and poses a major threat to public health and ecosystem health, as well as to fisheries and economic development. The problems and impacts are diverse, as are the causes and underlying mechanisms controlling the blooms. The signs are clear that pollution and other alterations in the coastal zone have increased the abundance of algae, including harmful and toxic forms. All new outbreaks and new problems cannot be blamed on pollution, however, as there are numerous other explanations, some of which involve human activities, and some of which do not. As a growing world population increases its use of the coastal zone and demands more fisheries and recreational resources, there is a clear need to understand HAB phenomena and to develop scientifically sound management and mitigation policies.

## See also

**Molluskan Fisheries. Network Analysis of Food Webs. Primary Production Methods. Plankton.**

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# PINNIPEDS

*See* **MARINE MAMMAL OVERVIEW**

# PLANKTON

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The category of marine life known as plankton represents the first step in the food web of the ocean (and of large bodies of fresh water), and components of the plankton are food for many of the fish harvested by humans and for the baleen whales. The plankton play a major role in cycling of chemical elements in the ocean, and thereby also affect the chemical composition of sea water and air (through exchange of gases between the sea and the overlying atmosphere). In the parts of the ocean where planktonic life is abundant, the mineral remains of members of the plankton are major contributors to deep-sea sediments, both affecting the chemistry of the sediments and providing a micropaleontological record of great value in reconstructing the earth's history.

'Plankton' refers to 'drifting', and describes organisms living in the water column (rather than on the bottom – the benthos) and too small and/or weak to move long distances independently of the ocean's currents. However, the distinction between plankton and nekton (powerfully swimming animals) can be difficult to make, and is often based more on the traditional method of sampling than on the organisms themselves.

Although horizontal movement of plankton at kilometer scales is passive, the metazoan zooplankton nearly all perform vertical migrations on scales of 10s to 100s of meters. This depth range can take them from the near surface lighted waters where the phytoplankton grow, to deeper, darker and usually colder environments. These migrations are generally diurnal, going deeper during the day, or seasonal, moving to deeper waters during the winter months to return to the surface around the time that phytoplankton production starts. The former pattern can serve various purposes: escaping visual predators and scanning the watercolumn for food. (It should be noted that predators such as pelagic fish also migrate diurnally.) Seasonal descent to greater depths is a common feature for several copepod species and may conserve energy at a time when food is scarce in the upper layers. However, vertical migration has another role. Because of differences in current strength and direction between surface and deeper layers in the ocean, time spent in deeper water acts as a transport mechanism relative to the near surface layers. On a daily basis this process can take plankton into different food concentrations. Seasonally, this effective 'migration' can complete a spatial life cycle.

The plankton can be subdivided along functional lines and in terms of size. The size category, picoplankton (0.2–2.0 μm), is approximately equivalent to the functional category, bacterioplankton; most phytoplankton (single-celled plants or colonies) and protozooplankton (single-celled animals) are nano- or microplankton (2.0–20 μm and 20–200 μm, respectively). The metazoan zooplankton (animals, the 'insects of the sea') includes large medusae and siphonophores several meters in length. Size is more important in oceanic than in terrestrial ecosystems because most of the plants are small (the floating seaweed, *Sargassum*, being the notable exception), predators generally ingest their prey whole (there is no hard surface on which to rest prey while dismembering it), and the early life stages of many