THE EXPANDING GLOBAL PROBLEM OF HARMFUL ALGAL BLOOMS

DONALD M. ANDERSON
Biology Department, Woods Hole Oceanographic Institution
Woods Hole, MA 02543 USA

INTRODUCTION

Over the last several decades, countries throughout the world have experienced an escalating and worrisome trend in the incidence of problems associated with blooms of harmful and toxic algae, commonly called "red tides," but now termed "harmful algal blooms" (HABs) by scientists (Anderson[1]). Impacts of these phenomena include mass mortalities of wild and farmed fish and shellfish, human illness and death from contaminated shellfish or fish, death of marine mammals, seabirds, and other animals, and alteration of marine habitats or trophic structure through shading, overgrowth, or adverse effects on life history stages of fish and other marine organisms. Several decades ago, relatively few countries were affected by HABs, but now most coastal countries are threatened, in many cases over large geographic areas and by more than one harmful or toxic species (Anderson;[2] Hallegraeff[3]). It is still a matter of debate as to the causes behind this expansion, with possible explanations ranging from natural mechanisms of species dispersal to a host of human-related phenomena such as pollution-related nutrient enrichment, or transport of algal species via ship ballast water (Anderson;[2] Smayda;[4] Hallegraeff[3]). Whatever the reasons, coastal regions throughout the world are now subject to an unprecedented variety and frequency of HAB events. Many countries are faced with a bewildering array of toxic or harmful species and impacts, as well as disturbing trends of increasing bloom incidence, more impacted resources, larger areas affected, and higher economic losses.

BACKGROUND

HAB events are characterized by the proliferation and occasional dominance of particular species of toxic or harmful algae. In some cases, the cells can increase in abundance until their pigments discolor the water—hence the common use of the term "red tide" to describe these phenomena. There are, however, "blooms" of species which are not in high cell concentrations and which do not discolor the water, but which still cause harm, typically because of the potent toxins produced by those algae. For this reason, the term "harmful algal bloom" is generally used. The term is very broad however, and covers blooms of many types. HABs have one unique feature in common: they cause harm, either due to their production of toxins or to the manner in which the cells' physical structure or accumulated biomass affect co-occurring organisms and alter food-web dynamics.

HAB phenomena take a variety of forms, with multiple impacts. 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 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). The symptomology and exposure route for each of these are presented in Table 1. Except for ASP, all are caused by biotoxins synthesized by a class of marine algae called dinoflagellates. The ASP toxin, domoic acid, is produced by diatoms that until recently were thought to be free of toxins. A sixth human illness, ciguatera fish poisoning (CFP) is caused by ciguatoxins produced by dinoflagellates that attach to surfaces in many coral reef communities (reviewed in Anderson and Lobel[6]). 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 (Burkholder and Glasgow;[7] Burkholder[8]).

Another type of HAB impact occurs when marine fauna are killed by algal species that release toxins and other compounds into the water. 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, seabirds, whales, dolphins, and other marine animals, typically as a result of the transfer of toxins through the food web. A poorly defined but potentially significant concern relates to sublethal, chronic impacts from toxic HABs that can affect the structure and function of entire ecosystems. Adult fish can be killed by the millions in a single outbreak, with obvious long- and short-term ecosystem impacts (Fig. 1). Likewise, larval or juvenile stages of fish or other commercially important fisheries species can experience mortalities from algal toxins. Impacts of this latter type are far more difficult to detect than the acute poisonings of humans or 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 we realize, affecting multiple trophic levels, depending on the ecosystem and the toxin involved.

Non-toxic 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, but can also occur in relatively pristine waters.

Fig. 1. Dead fish from a Texas red tide. (Credit: Brazosports.)

Large, prolonged blooms of non-toxic 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. 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 sea floor. 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 (Fig. 2). Under high nutrient conditions, opportunistic macroalgal species outcompete, overgrow, and replace the coral.

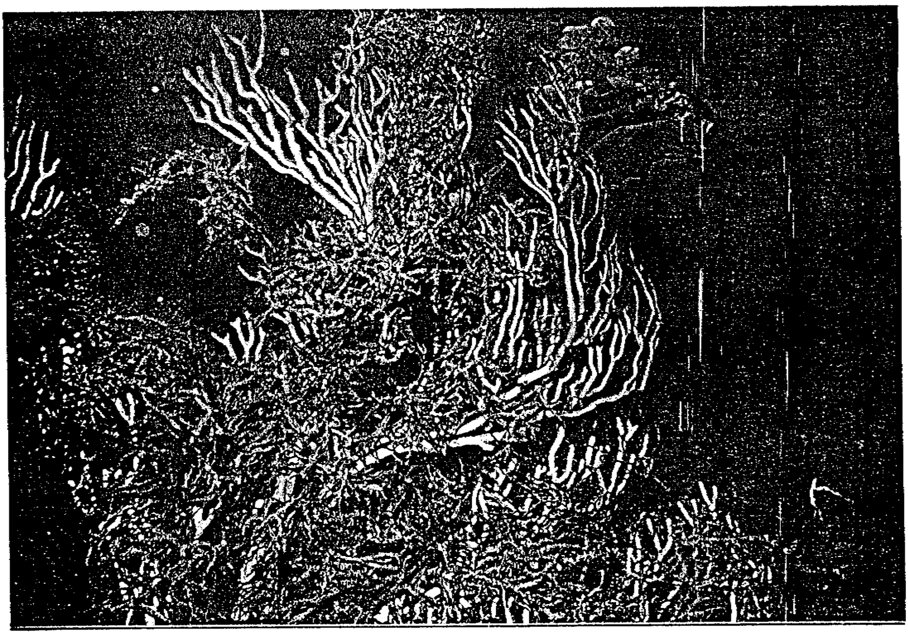

Fig. 2. Sponges and corals overgrown by the seaweed Codium isthmocladum in Southeast Florida. (Credit: B. LaPointe.)

Economic and Societal Impacts

HABs have a wide array of economic impacts, including the costs of conducting routine monitoring programs for shellfish and other affected resources, short-term and permanent closure of harvestable shellfish and fish stocks, reductions in seafood sales (including the avoidance of "safe" seafoods as a result of over-reaction to health advisories), mortalities of wild and farmed fish, shellfish, submerged aquatic vegetation and coral reefs, impacts on tourism and tourism-related businesses, and medical treatment of exposed populations. Estimates of actual impacts are few, in part because these economic losses are difficult to approximate. A conservative estimate of the average annual economic impact resulting from HABs in the U.S. was approximately $49 million over the period 1987 to 1992 (Anderson et al.;[9] Hoagland et al.[10]). Individual blooms, however, can easily exceed this annual average, as occurred for example in 1976 when a massive bloom of the dinoflagellate *Ceratium tripos* led to extensive oxygen depletion in the New York Bight, affecting surf clams, ocean quahogs, sea scallops, and some finfish and lobster. Total lost sales in all sectors combined were estimated to be $1.33 billion in 2000 dollars (Figley et al.[11]).

Losses have been significant in other countries as well. In Japan, for example, fish mortalities due to red tides in the Seto Inland Sea cost fishermen tens of millions of dollars per year, especially during the early 1970s. Even now, after pollution control efforts have decreased bloom incidence (see below), blooms of raphidophytes and dinoflagellates still kill cultured finfish and shellfish, resulting in significant losses (GEOHAB[12]). In China, a widespread red tide in 1989 along the coast of Hebei Province affected 1.5 x 10^4 hectares of shrimp ponds, resulting in a loss of 10^4 tons of shrimp valued at up to 300 million yuan or $U.S. 40 million (Xu et al.;[13] Wang and Li[14]). This is but one of many similar HAB outbreaks that continue to plague the aquaculture industry along the Chinese coast.

RECENT TRENDS

The nature of the HAB problem has changed considerably over the last three decades throughout the world. Simply judging by participation in international HAB conferences, more than 50 countries are threatened by harmful or toxic algal species, whereas 30 years ago, only a handful of countries were involved. A more definitive view of the expanding global problem is given in Figure 3, which shows the cumulative increase in the recorded distribution of the causative organisms and the confirmed appearance of PSP toxins in shellfish. Clearly, a dramatic expansion in the areas affected by PSP outbreaks has occurred in recent years, and a similar pattern applies to many of the other HAB types. Few would argue that 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 throughout the world (Anderson;[2] Smayda;[15] Hallegraeff[3]). Disagreement only arises with respect to the reasons for this expansion.

The first thought of many is that pollution or other human activities are involved. On close inspection, however, many of the "new" or expanded HAB problems have occurred in waters where pollution is not an obvious factor. The organisms responsible for HABs have been on earth for thousands or even millions of years, during which time they had ample opportunities to disperse, assisted by changing climate, movement of tectonic plates, and other global changes. Some new bloom events may thus simply reflect indigenous populations that are discovered because of better detection methods and more observers rather than new species introductions or dispersal events (Anderson[2]). The appearance of ASP along the United States west coast is a good example of this, as the diatom species that are now known to be responsible for that toxin had been observed in those waters many years before the 1991 outbreak (Work et al.[16]). The discovery of ASP toxins in California in 1991 was a direct result of communication with Canadian scientists who had discovered the same toxin four years earlier and developed new chemical detection methods exclusively for domoic acid (see below).

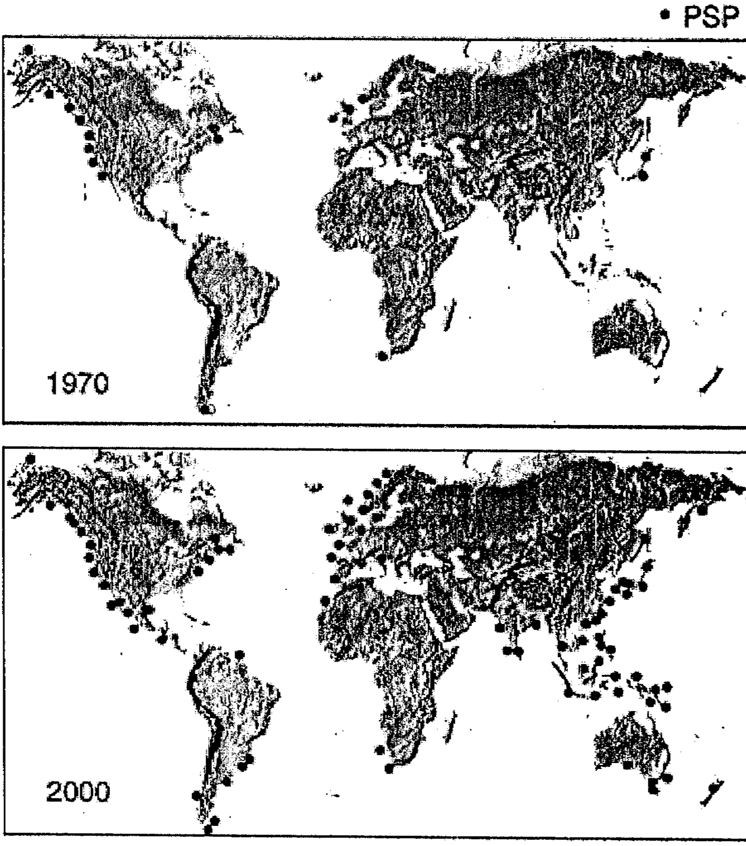

Fig. 3. Expansion of the PSP problem over the past 30 years. Sites with proven records of PSP-causing organisms are noted in 1970, and again in 2000. (Modified from Hallegraeff[3].)

Several other "spreading events" are most easily attributed to natural dispersal via currents, rather than as a result of pollution enhancement or other human activities. The first NSP event ever to occur in North Carolina (Tester et al.[17]) was shown to be a Florida bloom transported over 1500 km by the Gulf Stream to North Carolina waters—a totally natural phenomenon with no linkage to humans. Likewise, a massive

1972 red tide caused by favorable weather problems (including a hurricane) was responsible for transporting dormant cysts of the PSP-producing species *Alexandrium tamarense* to southern New England waters, where it has persisted to this day (Anderson and Wall;[18] Anderson[2]).

It is also clear that man may have contributed to the global HAB expansion by transporting toxic species in ship ballast water (Hallegraeff and Bolch;[19] McMinn et al.;[20] Lilly et al.[21]). In the past, proof of such accidental introductions has relied on inspection of historical plankton records or analysis of sediment cores for the resting stages of certain harmful or toxic species (e.g., McMinn et al.[20]), but the advent of molecular techniques and extensive sequence databases for HAB species now allows researchers to undertake genetic comparisons that provide forensic documentation of introduction events. One example of such a study was for Thau Lagoon in the Mediterranean, where PSP toxicity was first detected in 1998. A variety of chemical and genetic techniques were used to demonstrate that the closest relatives of the Thau Lagoon populations of the toxic dinoflagellate *Alexandrium catenella* were from temperate Asian waters. Thau Lagoon cells show no sequence homology to strains of this organism from western European waters, including the Mediterranean. The most likely scenario is that *A. catenella* was introduced into Thau Lagoon in the ballast water of a ship docked at Sete, France, a shipping port in direct communication with the lagoon. Spreading events of this type have occurred elsewhere and will continue to occur until ballast waters are more carefully regulated and treated.

Another factor underlying the global increase in HABs is the dramatic increase in aquaculture activities that has occurred in many countries. This leads to increased monitoring of product quality and safety, revealing indigenous toxic algae that were probably always there (Anderson[2]). The construction of aquaculture facilities places fish or shellfish resources in areas where toxic algal species occur but were previously unknown, leading to mortality events or toxicity outbreaks that would not have been noticed had the aquaculture facility not been placed there. Another potential linkage is that the pollution from aquaculture facilities may stimulate HABs. A single fish farm can contribute fish feces, urine, and uneaten food equivalent to the pollution loading from a small city.

Of considerable concern, particularly for coastal resource managers, is the potential relationship between the apparent increase in HABs and the accelerated eutrophication of coastal waters due to human activities (Anderson et al.[22]). As mentioned above, some HAB outbreaks occur in pristine waters with no influence from pollution or other anthropogenic effects, but linkages between HABs and eutrophication have been frequently noted within the past several decades (e.g., Lam and Ho;[23] Smayda;[4,15] Riegman;[24] Richardson and Jorgensen;[25] Richardson[26]). Coastal waters are receiving massive and increasing quantities of industrial, agricultural and sewage effluents through a variety of pathways (Vitousek et al.[27]). In many urbanized coastal regions, these anthropogenic inputs have altered the size and composition of the nutrient pool which may, in turn, create a more favorable nutrient environment for certain HAB species. Just as the application of fertilizer to lawns can enhance grass growth, marine algae can grow in response to various types of 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 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 (Okaichi[28]). Effluent controls were instituted in the mid-1970's, resulting in a 70% reduction in the number of red tides, and that reduction 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 (Bodeanu and Ruta[29]). That decrease, which also has continued to this day, has been linked to reductions in the use of fertilizer in upstream watersheds by former Soviet Union countries that are no longer able to afford large, state-subsidized fertilizer applications to agricultural land (Anderson et al.[22]).

In retrospect, it is now clear that the worldwide expansion of HAB phenomena is in part a reflection of our ability to better define the boundaries of the problem: the nature and extent of toxic or harmful species and their impacts. Those boundaries are, however, also expanding due to natural species dispersal via storms or currents, as well as to enhanced HAB population growth as a result of pollution or other anthropogenic influences. The fact that part of the expansion is simply a result of increased awareness should not temper our concern. The global problem of HABs is serious and large—much larger than we thought.

MANAGEMENT ISSUES

Management options for dealing with the impacts of HABs include reducing their incidence and extent (prevention), minimizing impacts (mitigation), and stopping or containing blooms (control). Where possible, it is preferable to prevent HABs rather than to treat their symptoms, but this is a significant challenge.

Prevention

Since increased pollution and nutrient loading may cause an increased incidence of outbreaks of some HAB species (Smayda;[4] Anderson et al.[22]), these events may be prevented by reducing pollution inputs to coastal waters, particularly industrial, agricultural, and domestic effluents high in plant nutrients. As discussed above, there is evidence from several areas (the Seto Inland Sea and the Black Sea, for example) that major changes in sewage treatment or agricultural fertilization can improve water quality and reduce the number of red tides and algal blooms, but the time-frame for achieving HAB reduction by pollution control policies is long (years to decades) and there is no guarantee that those actions will actually reduce harmful bloom incidence in other areas. These policy considerations are especially relevant to coastal areas where human activities are having a significant impact on the cycling of nutrients through the input of large quantities of agricultural runoff and domestic sewage. The trends in this regard are indeed alarming. The flux of P to the coastal oceans has increased 3-fold compared to pre-industrial, pre-agricultural levels, and N has increased even more dramatically, especially over the last 4 decades (Caraco;[30] Smil[31]). During that time, the flux of N increased 4-fold into the Mississippi River and more than 10-fold into the rivers entering the North Sea (National Research Council;[32] Smil[31]). Human activity is estimated to have increased N inputs to the coastal waters of the northeastern United States generally, and to Chesapeake Bay specifically, by 6-8-fold (Boynton et al.;[33] Howarth[34]).

These numbers are alarming, but non-point sources of nutrients from agricultural activities, fossil-fuel combustion, and animal feeding operations can be of greater concern than point sources (e.g., sewage treatment plants) because the former are larger and more difficult to control. Fertilizer application on land remains a major contributor to non-point nutrient pollution, and this source is increasing dramatically in many regions (Vitousek[27]). Both industrial and developing nations are using significantly higher loadings of fertilizer in agriculture, with global N and P fertilizer usage increasing 8-fold and 3-fold, respectively, since the early 1960s (Smil[31]). When these nutrient supplies reach rivers, estuaries and coastal waters, they are available for phytoplankton uptake and growth. The nitrate component of fertilizers, in particular, can travel long distances. The "dead zone" of hypoxia in the Gulf of Mexico is a particularly striking example of the long-distance transport of fertilizer nitrogen (Rabalais[35]). Seventeen different states in the Mississippi watershed contribute nutrients that are implicated in enhanced algal growth and the persistent oxygen depletion problem in the Gulf.

Other management 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).

Mitigation

The most effective mitigation tools are monitoring programs that detect toxins in shellfish and/or monitor the environment for evidence of HAB events (e.g., Shumway et al.;[36] Anderson et al.[5]). Numerous monitoring programs have been established worldwide in coastal waters to provide advance warning of outbreaks or to delineate areas that require harvest restrictions. This monitoring is conducted for both HAB species and for their toxins. The latter has become quite expensive in recent times due to the proliferation of toxins and potentially affected resources. The costs of such monitoring programs are significant and growing in parallel with the proliferation of HAB toxins. Molluscs, bivalves and gastropods are typically the primary vectors of algal biotoxins to human consumers, although crustaceans (e.g. crabs and lobsters) can also transfer algal biotoxins through the food chain (reviewed by Shumway[37]). Clearly, the optimum, safest and most commonly used practice involves sampling and testing of wild or cultured product directly from the natural environment, as this allows unequivocal tracking of toxins to their site of origin and targeted regulatory action. A useful review of selected shellfish monitoring programs is given by Anderson et al.[5]

A common mitigation strategy used by fish farms is the towing of fish net pens away from the sites of intense HABs. Though expensive and occasionally costly with respect to lost or damaged fish, this remains one of the primary tools used by fish farmers to combat HABs (Rensel and Whyte[38]). A strategy to mitigate the impact of HAB toxins in shellfish is to process those shellfish in such a way as to reduce toxicity to an acceptable level. A clear example is the removal of scallop viscera and the marketing of only the adductor muscle, which generally contains little or no HAB toxins.

Remote sensing has great potential as a tool to assist in monitoring the development and movement of HAB phenomena over larger spatial and shorter time scales than those accessible through ship- or land-based sampling. This technology has, however, only recently lived up to part of this potential. Although multi-spectral scanners (e.g. Coastal Zone Color Scanner; CZCS) can be used to detect the reflectance of chlorophyll-*a* and other pigments, these efforts have been constrained by the inability of the sensors to discriminate phytoplankton populations at the species level. This is, of course, a fundamental requirement of HAB programs. Instead, progress has been made by first linking specific water masses to HAB organisms and then identifying and tracking that water mass with an appropriate remote sensing technique (e.g., Keafer and Anderson[39]). In particular, remotely-sensed sea surface temperatures (SST) have been used to follow the movement of fronts, water masses, or other physical features where HAB species accumulate. A coastal current that dominates PSP dynamics in the southwestern Gulf of Maine is easily identified by its temperature signature (Franks and Anderson[40]). Likewise, the long-distance advection of *Karenia brevis* from Florida into the nearshore waters of North Carolina via the Gulf Stream in 1987 was documented with this approach (Tester et al.[17]).

Applying a related type of remote sensing technology, scientists and engineers are now developing automated instruments that can be moored along the coast to detect HAB cells or their toxins while simultaneously measuring the physical and optical characteristics of the water column to provide the complementary information needed to make "algal forecasts" of impending toxicity. These instruments are taking advantage of new molecular and analytical methodologies that allow chemicals (such as HAB toxins) and cells to be detected with great sensitivity and specificity.

A long-term goal of HAB monitoring programs is to develop the ability to forecast bloom development and movement, but predictive models for HABs are only in their infancy (Franks[41]). Prediction of HAB outbreaks requires physical/biological coupled models which account for both the growth and behavior of the toxic algal species, as well as the movement and dynamics of the surrounding water (McGillicuddy et al.[42]). Numerical models of coastal circulation are advancing rapidly, but difficulties arise in incorporating biological and chemical processes into the physics. The growth and accumulation of individual harmful algal species in a mixed planktonic assemblage are exceedingly complex processes involving an array of chemical, physical, and biological interactions. Our level of knowledge about each of the many HAB species varies significantly, and even the best-studied remain poorly characterized with respect to bloom or population dynamics. Resolution of various rate processes integral to the population dynamics (e.g., input and losses due to growth, grazing, encystment, excystment, and physical advection) has not been accomplished, but is fundamental to model formulation. Many of these processes are difficult to quantify in the field because HAB species are often only a small fraction of the planktonic biomass in natural samples. The end result is that despite the proven utility of numerical models in many oceanographic disciplines, there are no predictive models of population development, transport, and toxin accumulation for any of the major harmful algal species. Several are under development, but there is a clear need to increase efforts to formulate realistic physical models for regions subject to HAB events, and to incorporate biological behavior and population dynamics into those simulations.

Control

Human efforts to control insects, diseases, and fungi are common agricultural practices on land, but similar attempts to control unwanted plants or animals in the ocean are rare. The significant public health, economic, and ecosystem impacts of HABs would seem to make these phenomena legitimate targets for control efforts, but research on this topic has been minimal because of concerns about costs, effectiveness, and environmental impacts (Anderson[1]). Potential approaches to controlling HABs are similar to those used to control pests on land – e.g., biological, physical, or chemical treatments that directly target the bloom cells.

Control methodologies can be categorized as either "direct" or "indirect" depending on whether the effort targets the bloom organisms specifically, or strives to reduce impacts, such as through bloom prediction or through alteration of pollution inputs that might stimulate blooms (Boesch et al.;[43] Anderson et al.[5]). General approaches to direct control include: 1) chemicals that kill or disrupt HAB cells during blooms; 2) clays or other materials that flocculate (precipitate) and scavenge cells and other particles from the water column, transporting them to the ocean floor; and 3) biological agents such as viruses, bacteria, or parasites which are lethal pathogens to HAB species.

Despite the significant impacts of HABs on coastal regions, direct intervention efforts to control bloom populations have not been attempted to any significant extent in natural waters, other than large-scale clay treatments used to flocculate and sediment red tide cells in Korea (Na et al.[44]). Research programs on promising control methodologies are needed, concurrent with continued field and laboratory studies to better understand the ecological mechanisms underlying the HABs (Anderson[1]). This would represent a departure from the status quo, as past research activities have been largely focused on monitoring and understanding HAB phenomena, not controlling them.

HABS AS NATURAL DISASTERS

HAB phenomena have all the characteristics of natural disasters, yet they have not typically been viewed as such by government agencies. Disaster assistance is often not provided to areas hit by destructive red tides or HABs, yet the economic and social impacts can be equivalent to, or more severe than storms or other natural phenomena. The following sections provide brief examples of the scales and types of impacts that can be caused by HABs that affect human health, ecosystem health, aquaculture operations, and even the public confidence in seafood quality. These examples are offered to highlight the diverse and significant impacts that can strike a country or region without warning, as is the case with other natural disasters. There are many more outbreaks that could be highlighted under each category of impact, but these few are selected because they best illustrate the nature of the events and their outcomes.

Aquacultural disaster – the Hong Kong red tide of 1998

In March/April 1998, a red tide occurred that caused the most serious fish kill in Hong Kong history, affecting most of the region's aquaculture zones. The alga involved is now known as *Karenia digitata* (Yang et al.[45]), but was previously unknown to science. Most of Hong Kong's mariculture farms (estimates are 1,000 out of 1,500) were affected by the bloom, which appeared first in northeast waters and then proceeded south and then west and northwest through time.

Warnings were given by government officials, but in several locations fish farmers were unaware of the seriousness of the outbreak until fish began to die. The major concern of government departments was to provide warnings of the movement of the red tide, to collect and dispose of the dead fish, and to protect public health. Statements were issued to the public concerning safety aspects of consuming fish and shellfish and about swimming in red tide-affected waters.

The main mitigation measures against the red tide were taken by some of the fish culturists themselves. There are reports of cages being moved and aeration being used, and some culturists managed to harvest their fish before mortalities occurred. The most novel approach involved a group of fishermen who used their boats, outboard motors and water jets to "repel" the incoming algae.

The 1998 Hong Kong red tide was massive in its scale and the extent of its impacts. Estimates of the losses from this event vary dramatically depending on the source of information. The Federation of Hong Kong Aquaculture Association estimated a total, direct loss of U.S. $32 million (Yang and Hodgkiss[46]), though government officials argue that the true loss was lower. The cost of collecting and disposing of dead fish was estimated by the Hong Kong Marine Department to be U.S. $130,000. This does not include staff salaries. Another aspect of the economic impact is that the Hong Kong Government provided low-interest loans for mariculturists. Approximately U.S. $20 million was loaned to farmers at a low interest rate.

Although no direct losses to the capture fisheries can be assumed, monetary losses were incurred due to a phenomenon known as the "halo" effect. This refers to the tendency of consumers to switch to substitute foods or activities because of their concern about the possible toxicity of seafood due to a specific HAB event. Even though only one type of product might be potentially affected, consumers avoid a much broader range of goods and services, over-reacting to the risk and exaggerating the dangers. In Hong Kong, the uncertainty and conflict regarding toxicity of the killed fish contributed to a major decline in the value of all fish sales. The Joint Committee on Hong Kong Fishermen's Organizations estimated that captured fish sold for approximately half their former value during the red tide. Assuming that the average daily earnings for each of 4,000 trawlers decreased by half over a one-month period, the Organization estimates the total loss to be U.S. $77 million. This estimate seems high, however, given that the total annual capture fishery production in 1997 was HK $1.57 billion. Nevertheless, the impact on capture fisheries was significant, and may have exceeded the loss to the mariculturists.

While it would be expected that fish sales and prices would decline during a large-scale HAB event, lack of a consistent or coordinated government response may have exacerbated the problem. As fish and fish products were moved freely about Hong Kong without detailed source and shipping records, wholesale buyers, sellers and consumers were unsure of the source of fish in the markets. There was considerable confusion about whether the dead and dying aquaculture fish should be sold for consumption, since policies on that topic were not well-established in Hong Kong, or worldwide, and government agencies sometimes contradicted each other in their statements. Evidence from other regions of the world suggested that toxin transfer to fish tissues during harmful *Gymnodinium* and *Karenia* blooms does not occur, and thus that freshly killed fish might be safe to eat. Indeed, numerous chemical and bioassay tests of the Hong Kong fish flesh showed no signs of toxin. The government eventually recommended that consumers only buy fish that showed no gill damage or signs of hemorrhage.

The 1998 red tide in Hong Kong was a media event, with reports and speculations filling the newspapers, television, and radio. In many cases, the information that was released was contradictory, incorrect, or misinterpreted, leading to widespread over-reaction to the nature of the problem. One example was cited above as the "halo effect" that halved the price of captured fish even though they were safe to eat. Other impacts include frustrated, angry fishermen and confused retailers and consumers. As a result, a team of HAB experts was hired to evaluate the existing red tide monitoring and management system in Hong Kong, and recommend sweeping modifications in organizational structure and policy (Anderson et al.[47]).

Ecosystem disaster – the Chrysochromulina bloom in Scandinavia – 1988

In May/June 1988, the Kattegat and Skagerrak waters that mark the transition area between the North Sea and the Baltic were the site of an unprecedented bloom of the flagellate *Chrysochromulina polylepis*. This bloom is unusual and noteworthy because of its extent (covering in excess of 75,000 km^2) and the tremendous ecosystem damage it caused (Rosenberg et al.[48]). The toxin produced by *Chrysochromulina* had drastic effects on the marine ecosystem, indiscriminantly killing large numbers of macroalgae, invertebrates, and fish. SCUBA divers reported mass mortalities of invertebrates down to 20 meters, including gastropods, polychaetes, tunicates, anthozoans, and sponges. Many species of fish were killed as well, and those that were not killed were lethargic and easily caught by divers (Rosenberg et al.[48]). Even seaweeds were affected. The red seaweed *Delesseria* was killed or affected in such a way that its color turned from red to orange and finally green, indicating the breakdown of pigments. The brown seaweed *Laminaria* was similarly affected. Caged fish were killed at several sites along the west coast of Sweden (100 tons) and along the southern coast of Norway (500 tons). The economic loss to these fish farming industries was about U.S. $10 million.

As was the case in Hong Kong, this *Chrysochromulina* bloom attracted considerable public and political attention throughout Europe. Many linked the outbreak to the pollution of the Kattegat and Skagerrak, and subsequent work has confirmed that *Chrysochromulina* toxicity is enhanced by phosphorus limitation (Granéli et al.[49]), as would occur in waters with excess nitrogen inputs. Although other types of HABs were recurrent in that region (including blooms that kill fish and cause shellfish toxicity), this single event precipitated a significant governmental response in the form of research and monitoring funds for HABs that lasted for many years. This *Chrysochromulina* bloom can be credited with opening the eyes of administrators and program managers in Europe to the sudden and devastating impacts of HABs, and the need for research on the factors that stimulate their growth, especially those linked to human activities.

Human health disaster – the 1987 ASP outbreak in Canada

In November 1987, a human poisoning episode took place that highlights the many issues that arise when a new, unknown toxin appears in seafood. This event occurred in northeastern Canada, a region with a long history of PSP outbreaks, and thus with a health system that was familiar with some aspects of marine biotoxin exposure. The outbreak began when hospital emergency rooms began admitting individuals complaining of vomiting, diarrhea and confusion. As the number of patients grew, medical personnel began to search for a source, and soon the illnesses were correlated with consumption of mussels originating in the Prince Edward Island region, the site of a major aquaculture operation (Smith et al.[50]). When the mussels were tested using the standard PSP mouse bioassay, however, the mice did not show typical PSP symptoms, but nevertheless demonstrated that a neurotoxin was present. The entire Canadian Atlantic shellfishery was closed pending investigation, causing millions of dollars in losses, not only in Canada, but in the U.S. as well, especially in Maine and areas immediately to the south of the affected region. There are even reports that seafood sales dropped on the west coast of the U.S. – thousands of miles away. This latter impact is yet another example of the "halo effect" whereby safe fisheries products lose value because of the avoidance of all seafood by wary and poorly informed consumers.

A team of 40 Canadian government scientists was assembled, including chemists and biologists and personnel experienced with PSP issues. In a matter of days, a new toxin (domoic acid) was identified as the causative agent. Ultimately, 150 people had become seriously ill, with 30 hospitalizations and 3 deaths. This caused considerable alarm and fear among the general public, and was a frequent issue in the national news for several weeks (Fig. 4). One important symptom that persisted with those who were poisoned but survived was permanent short-term memory loss. As a result, the new poisoning syndrome was named Amnesic Shellfish Poisoning, or ASP (Todd[51]). The causative organism was subsequently found to be the diatom *Pseudo-nitzschia multiseries,* an organism well-known to science, but previously thought to be non-toxic. As a result of continued monitoring by the Canadian Food Inspection Agency (CFIA) there have been no human poisonings with ASP in Canada since that event.

In response to the 1987 ASP event, Canada's Department of Fisheries and Oceans (DFO) set up new research and monitoring programs and established a Phycotoxin Working Group that continues to coordinate national biotoxin research and monitoring activities. Many other countries have since documented the occurrence of domoic acid in their seafood (typically shellfish and crabs), and there are numerous ASP closures on an annual basis throughout the world. It is clear that this toxin was present in seafood long before the 1987 outbreak. The Canadian episode simply alerted government officials and scientists to the existence of the toxin, which is now regulated in seafood globally.


Mystery toxin taints mussels from P.E.I.

SHELLFISH ALERT WIDENS

Naked man taken for ride

Warning covers area from Newfoundland to Florida

Fishery clobbered by inept Epp

Isolated case escalated into industry crisis

Fatal P.E.I. mussel toxin found

Acid from rare seaweed ID'd by fed scientists

Fig. 4. Newspaper headlines regarding 1987 ASP incident in Canada. (Credit: S. Bates.

<u>Economic disaster – the 1997 Pfiesteria outbreak in Maryland</u>

In 1997, a relatively minor HAB event resulted in a significant economic crisis in the Chesapeake Bay region of the U.S., demonstrating the large impact that even small outbreaks can sometimes have. During that summer, several fish kills occurred in small tributaries along Maryland's eastern shore. Approximately 30,000 fish died - a relatively low number to those experienced with fish mortality events, but sufficient to be alarming to the local population and politicians. Many fish had open lesions on them (Fig. 5), a disturbing image to many, and at the time, thought to be indicative of the involvement of the predator dinoflagellate *Pfiesteria piscicida*. (Burkholder and Glasgow[7,52]). *Pfiesteria* outbreaks had previously been documented in the Albermarle-Pamlico estuary of North Carolina (Burkholder et al.[53]), but had never been reported in the Chesapeake. There the blooms were confined to small areas such as the Pocomoke River, and only a few commercially and recreationally important fish species were affected. The situation attracted considerable attention when a few commercial fishermen complained of health effects – in particular confusion and memory problems. These claims, although initially discounted by some officials, were ultimately confirmed to be medically reliable (Grattan et al.[54]). The state of Maryland closed several Chesapeake tributaries for all recreation and for commercial fishing as a precautionary measure.

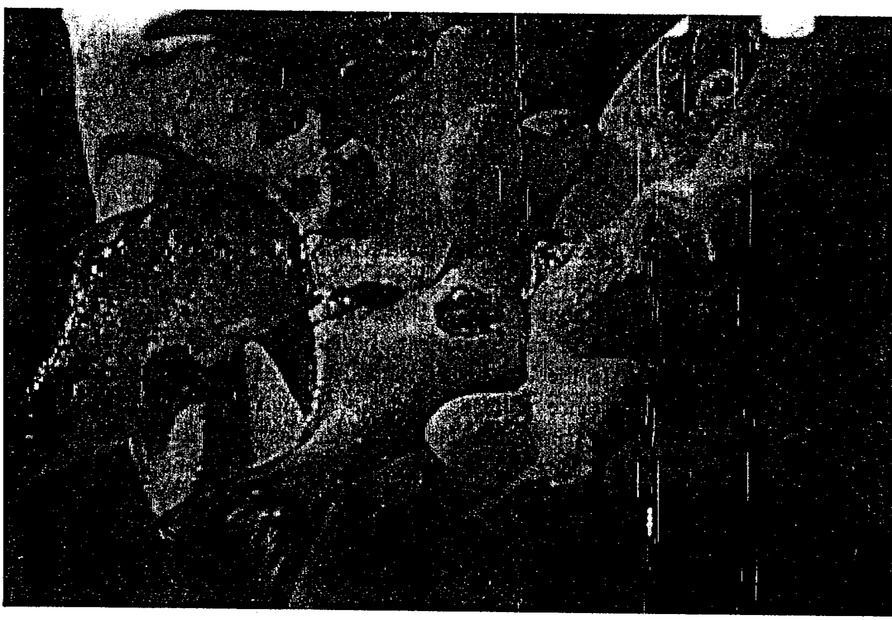

Fig. 5. Lesions on menhaden thought to have been caused by the dinoflagellate Pfiesteria. (Credit: J. Burkholder.)

Tests by the U.S. Food and Drug Administration and other officials consistently failed to demonstrate any toxicity in fish or shellfish from the affected areas. Nevertheless, the high level of attention given the *Pfiesteria* outbreak by the press and the recreation restrictions and fisheries closures by the government contributed to an over-reaction by the general public. Consumers stopped buying seafood of all types and avoided exposure to the water. Some grocery chains went so far as to post signs that they were selling "no Chesapeake Bay seafood products" – greatly exacerbating the concern by consumers, and worsening the economic losses for that regional industry. Based on the differences between the 1996 and 1997 seafood sales, economic losses are estimated at U.S. $43 million to the Chesapeake seafood industry. Further losses were experienced by the recreational fishing industry, bringing the total loss from the event to U.S. $50 million (Lipton[55]).

The consumer panic caused by the *P. piscicida* outbreak in Maryland is yet another striking example of the "halo effect" discussed above. The fish kills were small in size, and only a handful of people were affected medically, yet the economic losses were massive. In general, the halo effect typically affects producers of seafood or providers of recreation and tourist services. Because consumers can switch to other foods or to other recreational activities, the halo effect is not generally serious for consumers, but it can be disastrous for producers.

Another outcome of the *Pfiesteria* outbreak was a substantial inflow of research and monitoring funds. Millions of dollars were committed to these activities, and much of those funds continue to be provided to this day, even though there have not been any major fish kills or human health problems in the Chesapeake region since 1997.

DEFINITION OF 'NATURAL DISASTER'

The foregoing sections highlight the many different types of impacts that can occur with HABs. These were just a few selected examples from a long list of outbreaks and negative consequences. The scale of these impacts can be significant, and many of those who have been impacted have argued that HABs should be considered natural disasters, and thus that they should be given government assistance, as is done after storms or earthquakes, for example. In the U.S., when natural disasters such as hurricanes and floods occur, financial assistance is made available through the Federal Emergency Management Agency (FEMA) provided that the region has been declared a 'disaster area' by the federal government. This allows low-interest loans and other financial assistance to those in need. Following a particularly severe red tide in North Carolina in 1987 (Tester[17]), legislation was enacted that has placed red tides and HABs into the natural disaster category. The definition of 'disaster' has now been modified to be: "a sudden event which causes severe damage including, but not limited to, floods, hurricanes, tornadoes, earthquakes, fires,..., **ocean conditions resulting in the closure of customary fishing waters**, riots,..." (Conference Report on H.R. 4174, SBA Reauthorization and Amendment Act of 1988). This change in the law means that should a red tide or HAB affect fisheries, the federal disaster loan program will be available to aid those who are harmed. Other countries might want to consider enacting similar legislation to provide a much-needed level of protection to fishermen, the tourist industry, and others who are subject to the unpredictable and often devastating effects of an HAB.

CONCLUSION

HABs are increasingly common along the coasts of countries throughout the world. The impacts from these aquatic disasters are substantial, affecting public health, fisheries resources (both wild and farmed), local economies, tourism, ecosystem health, and coastal aesthetics. One alarming aspect of HAB phenomena is that they have been increasing in frequency, areal coverage, and diversity over the past several decades. There are now more algal toxins, more toxic algal species, more fisheries resources affected, larger areas affected, and higher economic losses. Reasons for this expansion are many, and include natural species dispersal via storms or currents, human assisted dispersal (e.g., via ballast water discharge), better detection as a result of increased monitoring and better analytical techniques for toxins, and enhancement of the bloom populations due to nutrients supplied by sewage, agricultural runoff, and other pollution sources. HAB phenomena have all the characteristics of natural disasters, yet they have not typically been viewed as such by government agencies. Disaster assistance has often not been provided to areas hit by destructive red tides or HABs, yet the economic and social impacts can be equivalent to, or more severe than storms or other natural phenomena. Expanded research on management, mitigation, and control of HABs is easily justified in this context, as is legislation to protect fishermen, tourist industries, and others who are subject to their unpredictable and often devastating effects.

Acknowledgments
Work on this paper was supported by NOAA Cooperative Agreement NA17RJ1223 and NOAA National Ocean Service Purchase Order 674-W10197. Contribution No. 10806 from the Woods Hole Oceanographic Institution.

REFERENCES

1. Anderson, D. 1994. "Red tides." *Scientific American*: 271: 52-58.
2. Anderson, D.M. 1989. "Toxic algal blooms and red tides: a global perspective." pp. 11-16, in: *Red tides: biology, environmental science, and toxicology*, Okaichi, T., D.M. Anderson, and T. Nemoto (eds.), Elsevier Science Publishing Co., Inc., New York.
3. Hallegraeff, G.M. 1993. "A review of harmful algal blooms and their apparent global increase." *Phycologia* 32: 79-99.
4. Smayda, T.J. 1989. "Primary production and the global epidemic of phytoplankton blooms in the sea: A linkage?" pp. 449-484, in: *Novel Phytoplankton Blooms*, Cosper, E.M., V.M. Bricelj, and E.J. Carpenter (eds.), Coastal and Estuarine Studies No. 35, Springer-Verlag, New York.
5. Anderson, D.M., P. Andersen, V.M. Bricelj, J.J. Cullen and J.E. Rensel. 2001. *Monitoring and Management Strategies for Harmful Algal Blooms in Coastal*

Waters. Singapore and Paris, Asia Pacific Economic Program and Intergovernmental Oceanographic Commission, 268 pp.

6. Anderson, D.M. and P.S. Lobel. 1987. "The continuing enigma of ciguatera." *Biol. Bull.* 172: 89-107.
7. Burkholder, J.M. and H.B. Glasgow, Jr. 1997. "*Pfiesteria piscicida* and other *Pfiesteria*-l ke dinoflagellates: behavior, impacts, and environmental controls." *Limnol. Oceanogr.* 42(5, Pt. 2): 1052-1075.
8. Burkholder, J.M. 1998. "Implications of harmful microalgae and heterotrophic dinoflagellates in management of sustainable marine fisheries. Ecological Applications." *Ecol. Appl.* 8(1, suppl.): S37-S62.
9. Anderson, D.M., P. Hoagland, Y. Kaoru and A.W. White. 2000. *Estimated annual economic impacts from harmful algal blooms (HABs) in the United States*, Woods Hole Oceanographic Institution, 97 pp.
10. Hoagland, P., D.M. Anderson, Y. Kaoru and A.W. White. (in press). "Average annual economic impacts of harmful algal blooms in the United States: some preliminary estimates." *Estuaries*.
11. Figley, W., B. Pyle and B. Halgren. 1979. "Socioeconomic impacts." Chapter 14, in: *Oxygen Depletion and Associated Benthic Mortalities in New York Bight, 1976.* Swanson, R.L. and C.J. Sindermann (eds.), Professional Paper 11, December, NOAA, U.S. Department of Commerce.
12. GEOHAB. 2001. *Global Ecology and Oceanography of Harmful Algal Blooms, Science Plan.* Baltimore and Paris, SCOR and IOC.
13. Xu, Z., X. Gu, Y. Wang, H. Yuan and Y. Chen. 1993. "Analyses of ecological eigenvalue of plankton during red tide occurrence in the Xiangshan Sound, Zhejiang in 1988." Collected oceanic works/Haiyang Wenji. Tianjin 16(1): 83-89.
14. Wang, L. and X. Li. 1998. "Management of shellfish safety in China." *J. Shellfish Res.* 17(5): 1609-1611.
15. Smayda, T. 1990. "Novel and nuisance phytoplankton blooms in the sea: Evidence for a global epidemic." pp. 29-40, in: *Toxic Marine Phytoplankton*, Granéli, E., B. Sundstrom, L. Edler, and D.M. Anderson (eds.), New York, Elsevier.
16. Work, T.M., A.M. Beale, L. Fritz, M.A. Quilliam, M. Silver, K. Buck and J.L.C. Wright. 1993. "Domoic acid intoxication of brown pelicans and cormorants in Santa Cruz, California." pp. 643-650, in: *Toxic Phytoplankton Blooms in the Sea*, Smayda, T. J. and Y. Shimizu (eds.), Elsevier, Amsterdam.
17. Tester, P.A., R.P. Stumpf, F.M. Vukovich, P.K. Folwer and J.T. Turner. 1991. "An expatriate red tide bloom: Transport, distribution, and persistence." *Limnol. Oceanogr.* 36(5): 1053-1061.
18. Anderson, D.M. and D. Wall. 1978. "Potential importance of benthic cysts of *Gonyaulax tamarensis* and *G. excavata* in initiating toxin dinoflagellate blooms." *J. Phycol.* 14(2): 224-234.

19. Hallegraeff, G.M. and C.J. Bolch. 1992. "Transport of diatom and dinoflagellate resting spores in ships' ballast water: Implications for plankton biogeography and aquaculture." *J. Plankt. Res.* 14(8): 1067-1084.
20. McMinn, A., G.M. Hallegraeff, P. Thomson, A.V. Jenkinson and H. Heijnis. 1997. "Cyst and radionucleotide evidence for the recent introduction of the toxic dinoflagellate *Gymnodinium catenatum* into Tasmanian waters." *Mar. Ecol. Prog. Ser.* 161: 165-172.
21. Lilly, E.L., D.M. Kulis, P. Gentien and D.M. Anderson. 2002. "Paralytic shellfish poisoning toxins in France linked to a human-introduced strain of *Alexandrium catenella* from the Western Pacific: Evidence from DNA and toxin analysis." *J. Plankt. Res.* 24(5): 443-452.
22. Anderson, D.M., P.M. Glibert and J.M. Burkholder. (in press). "Harmful algal blooms and eutrophication: nutrient sources, composition, and consequences." *Estuaries.*
23. Lam, C.W.Y. and K.C. Ho. 1989. "Red tides in Tolo Harbour, Hong Kong." pp. 49-52, in: *Red Tides: Biology, Environmental Science and Toxicology,* Okaichi, T., D. M. Anderson, and T. Nemoto (eds.), Elsevier.
24. Riegman, R. 1995. "Nutrient-related selection mechanisms in marine phytoplankton communities and the impact of eutrophication on the planktonic food web." *Water Science and Technology* 32(4): 63-75.
25. Richardson, K. and B.B. Jorgensen. 1996. "Eutrophication: Definition, history and effects." pp. 1-19, in: *Eutrophication in Coastal Marine Ecosystems. Coastal and Estuarine Studies*, Jorgensen, B. and K. Richardson, (eds.), American Geophysical Union, Washington, DC.
26. Richardson, K. 1997. "Harmful or exceptional phytoplankton blooms in the marine ecosystem." *Adv. Mar. Biol.* 31: 301-385.
27. Vitousek, P.M., J.D. Aber, R.W. Howarth, G.E. Likens, P.A. Matson, D.W. Schindler, W.H. Schlesinger and D.G. Tilman. 1997. "Human alteration of the global nitrogen cycle: Sources and consequences." *Ecol. Appl.* 7(3): 737-750.
28. Okaichi, T. 1997. "Red tides in the Seto Inland Sea." pp. 251-304, in: *Sustainable Development in the Seto Inland Sea, Japan - From the Viewpoint of Fisheries*, Okaichi, T. and Y. Yanagi (eds.), Terra Scientific Publishing Company, Tokyo, Japan.
29. Bodeanu, N. and G. Ruta. 1998. "Development of the planktonic algae in the Romanian Black Sea sector in 1981-1996." pp. 188-191 in: *Harmful Algae*, Reguera, B., J. Blanco, M.L. Fernández, and T. Wyatt (eds.), Paris, France, Xunta de Galicia and Intergovernmental Oceanographic Commission of UNESCO.
30. Caraco, N.F. 1995. "Influence of human populations on P transfers to aquatic systems: A regional scale study using large rivers." pp. 235-247 in: *Phosphorus in the Global Environment*, H. Tiessen (ed.), New York, John Wiley & Sons Ltd.
31. Smil, V. 2001. *Enriching the Earth: Fritz Haber, Carl Bosch, and the Transformation of World Food.* MIT Press, Cambridge, MA.

32. National Research Council. 2000. *Clean Coastal Waters: Understanding and Reducing the Effects of Nutrient Pollution*. Washington, DC, National Academy Press.
33. Boynton, W.R., J.H. Garber, R. Summers and W.M. Kemp. 1995. "Inputs, transformations, and transport to nitrogen and phosphorus in Chesapeake Bay and selected tributaries." *Estuaries* 18: 285-314.
34. Howarth, R.W. 1998. "An assessment of human influences on inputs of nitrogen to the estuaries and continental shelves of the North Atlantic Ocean." *Nutrient Cycling in Agroecosystems* 52: 213-223.
35. Rabalais, N.N. 2000. "Hypoxia in the Gulf of Mexico." *J. Environ. Qual.* 30(2): 320-329.
36. Shumway, S.E., S. "Sherman-Caswell and J.W. Hurst. 1988. Paralytic shellfish poisoning in Maine: monitoring a monster." *J. Shellfish Res.* 7: 643-652.
37. [s]Shumway, S.E. 1990. "A review of the effects of algal blooms on shellfish and aquaculture." *J. World Aquacult.* Soc. 21: 65-104.
38. Rensel, J.E. and J.N.C. Whyte. (in press). "Finfish mariculture and harmful algal blooms." in: *Manual on Harmful Marine Microalgae - Revised Edition*, Hallegraeff, G.M., D.M. Anderson and A.D. Cembella,(eds.), IOC, UNESCO.
39. Keafer, B.A. and D.M. Anderson. 1993. "Use of remotely-sensed sea surface temperatures in studies of *Alexandrium ta*marense bloom dynamics." pp. 763-768 in: *Toxic Phytoplankton Blooms in the Sea*, Smayda, T. and Y. Shimizu (eds.), Elsevier, Amsterdam.
40. Franks, P.J.S. and D.M. Anderson. 1992. "Alongshore transport of a toxic phytoplankton bloom in a buoyancy current: *Alexandrium tamarensis* in the Gulf of Maine." *Marine Biology* 112: 153-164.
41. Franks, P.J.S. 1997. "Models of harmful algal blooms." *Limnol. Oceanogr.*42(5, Pt. 2): 1273-1282.
42. McGillicuddy, Jr., D.J., C.A. Stock, D.M. "Anderson and R.P. Signell. (in press). Hindcasting blooms of the toxic dinoflagellate Alexandrium spp. in the western Gulf of Maine." *Ecological Forecasting Report.*
43. Boesch, D.F., Anderson, D.M., Horner, R.A., Shumway, S.A., Tester, P.A. and Whitledge, T.E. 1997. *Harmful Algal Blooms in Coastal Waters: Options for Prevention, Control and Mitigation.* Silver Springs, MD, NOAA Coastal Ocean Program Decision Analysis Series No. 10, NOAA Coastal Ocean Office, 46 pp.
44. Na, G., Choi, W., and Chun, Y. 1996. A study on red tide control with Loess suspension. J. Aquacult. 9(3): 239-245.
45. Yang, Z.B., H. Takayama, K. Matsuoka and I.J. Hodgkiss. 2000. "*Karenia digitata* sp. nov. (Gymnodiniales, Dinophyceae), a new harmful algal bloom species from the coastal waters of west Japan and Hong Kong." *Phycologia* 39(6): 463-470.
46. Yang, Z.B. and I.J. Hodgkiss. 1999. "Massive fish killing by *Gyrodinium* sp." *Harmful Algae News*. 18: 4-5.

47. Anderson, D.M., P. Andersen, V.M. Bricelj, J.J. Cullen, I.J. Hodgkiss, K.C. Ho, J.E. Rensel, J.T.Y. Wong and R. Wu. 1999. *Red Tide and HAB Monitoring and Management in Hong Kong.* Final Report prepared for the Hong Kong, Agriculture and Fisheries Department, 254 pp.

48. Rosenberg, R., O. Lindahl and H. Blanck. 1988. "Silent spring in the sea." *Ambio.* 17(4): 289-290.

49. Granéli, E., N. Johansson and R. Panosso. 1998. "Cellular toxin contents in relation to nutrient conditions for different groups of phycotoxins". pp. 321-324 in: *Harmful Algae*, Reguera. B., J. Blanco, M.L. Fernández and Wyatt T. (eds.), Xunta de Galicia and Intergovern. Oceanographic Comm. of UNESCO.

50. Smith, J.C., R. Cormier, J. Worms, C.J. Bird, M.A. Quilliam, R. Pocklington, R. "Angus and et al. 1990. Toxic blooms of the domoic acid containing diatom *Nitzschia* pungens in the Cardigan River, Prince Edward Island, in 1988." pp. 227-232, in: *Toxic marine phytoplankton*, Granéli, E., B. Sundström, L. Edler, and D.M. Anderson (eds.), Elsevier Sci. Publ. Co., Inc., New York.

51. Todd, E.C.D. 1990. "Amnesic shellfish poisoning -- a new seafood toxin syndrome." pp. 504-508, in: *Toxic Marine Phytoplankton*, Granéli, E., B. Sundstrom, L. Edler, and D.M. Anderson (eds.), New York, Elsevier.

52. Burkholder, J.M. and H.B. Glasgow, Jr. 1997. "Trophic controls on stage transformations of a toxic ambush-predator dinoflagellate." *J. Eukaryot. Microbiol.* 44(3): 200-205.

53. Burkholder, J.M., E.J. Noga, C.H. Hobbs, and H.B. Glasgow, Jr. 1992. "New "phantom" dinoflagellate is the causative agent of major estuarine fish kills." *Nature*: 407-410.

54. Grattan, L.M., D. Oldach, T.M. Perl, M H. Lowitt, D.L. Matuszak, C. Dickson, C. Parrott, R.C. Shoemaker, C.L. Kauffman, M.P. Wasserman, et al. 1998. "Learning and memory difficulties after environmental exposure to waterways containing toxin-producing *Pfiesteria* or *Pfiesteria*-like dinoflagellates." *Lancet* 352: 532-539.

55. Lipton, D. 1999. *Pfiesteria*'s Impact on Seafood Industry Sales, Sea Grant Extension, University of Maryland.