

Toxic red tides and harmful algal blooms (HABs): threats to the recreational boater

Donald M. Anderson
Judith L. Kleindinst
Woods Hole Oceanographic Institution

INTRODUCTION

Over the last several decades, countries throughout the world have experienced an escalating and worrisome trend in the incidence of problems termed “harmful algal blooms” (HABs). HAB events are characterized by the proliferation and occasional dominance of particular species of toxic or harmful algae. In some cases, these microscopic cells increase in abundance until their pigments discolor the water - hence the commonly used term "red tide" (Fig. 1). There are, however, “blooms” of species which do not have high cell concentrations and thus do not discolor the water, but which still cause harm, typically because of potent toxins produced by those algae. The term “harmful algal bloom” is very broad and covers blooms of many types, but HABs all 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.

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



Figure 1. A “red tide bloom” (non-toxic) of *Noctiluca scintillans* in New Zealand. (Photo credit: M. Godfrey)

harmful or toxic species (Anderson, 1989; Hallegraeff, 1993). The causes behind this expansion are debated, with possible explanations ranging from natural mechanisms of species dispersal and enhancement (e.g., climate change) to a host of human-related phenomena such as pollution, climatic shifts, or transport of algal species via ship ballast water (Anderson, 1989; Smayda, 1989; Hallegraeff, 1993). Whatever the mechanisms, 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, larger areas affected, more fisheries resources impacted, and higher economic losses.

One group that is particularly at risk from marine biotoxins and harmful algae are yachtsmen and small boat operators. These individuals are often not informed about the biotoxin risks that are present in the waters in which they operate their vessels. A common occurrence in the tropics, for example, is for individuals who have chartered a boat or sailboat to consume fish that they have caught, only to become ill from a HAB syndrome called ciguatera fish poisoning. Local residents know that they should not eat certain species of fish from ciguatera-prone areas, but this information is not usually communicated by tourist agencies, charter companies and others with a financial interest in such activities. Likewise, individuals cruising in small boats along the coast of many countries are at risk should they harvest mussels or other shellfish and consume them, thinking they are having a healthy and natural meal, but instead finding sudden and debilitating toxicity that can be life threatening, especially when medical care is not in close proximity. Kayakers and canoeists are also at risk in this regard, particularly those who are on extended trips where they camp and collect food from coastal waters. A common fallacy is that red tide problems are associated with polluted waters, and therefore, that shellfish or fish from pristine areas are safe. Likewise, many believe that shellfish or fish are dangerous to consume only when the water is reddish and discolored. These misconceptions are often the cause of serious poisoning among the misinformed.

Yet another threat to small boat operators comes from certain species of algae which can cause skin irritation upon contact during swimming, or other species which produce toxins that are aerosolized in sea spray, and which can cause respiratory irritation, coughing, wheezing and great difficulty in breathing. These latter symptoms can be frightening for those who have respiratory problems already, or those who simply do not understand what is happening to them.

The following text summarizes the nature of the HAB threat to small boat operators in coastal waters, and provides some guidance on what to be concerned about, how to respond to possible poisoning events, and how to prevent those poisonings or other negative impacts from HABs in the first place. The focus will be predominantly in the US, but a global perspective will be provided whenever possible. It is hoped that this information will lead to a better-informed boating community, and thus reduce the risks to this vulnerable population.

HAB Impacts. When toxic algae are filtered from the water as food by shellfish, their toxins accumulate in those shellfish to levels that can be dangerous or 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). 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 toxins produced by dinoflagellates that live on surfaces in many coral reef communities. Ciguatoxins are transferred through the food chain from herbivorous reef fishes to larger carnivorous, commercially valuable finfish.

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 have increased considerably at aquaculture sites 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. Impacts of this type are often of no direct threat to boat operators, and thus will not be discussed further here.

The global distribution of each of the major HAB shellfish poisoning syndromes, as well as those for ciguatera and fish mortalities linked to HABs are available at the following website - <http://www.whoi.edu/redtide/HABdistribution/HABmap.html#World%20HAB%20toxin%20maps>. In some cases, the relatively recent discovery of a toxin (e.g., the ASP toxin domoic acid in 1987; Bates et al., 1989) is reflected in a global distribution that is sparse at present but which is expected to expand as the pace of discovery of that new toxin accelerates. In other cases where the syndrome has been known for many years (e.g., PSP, DSP, CFP), the distribution is truly

global and widespread in scale.

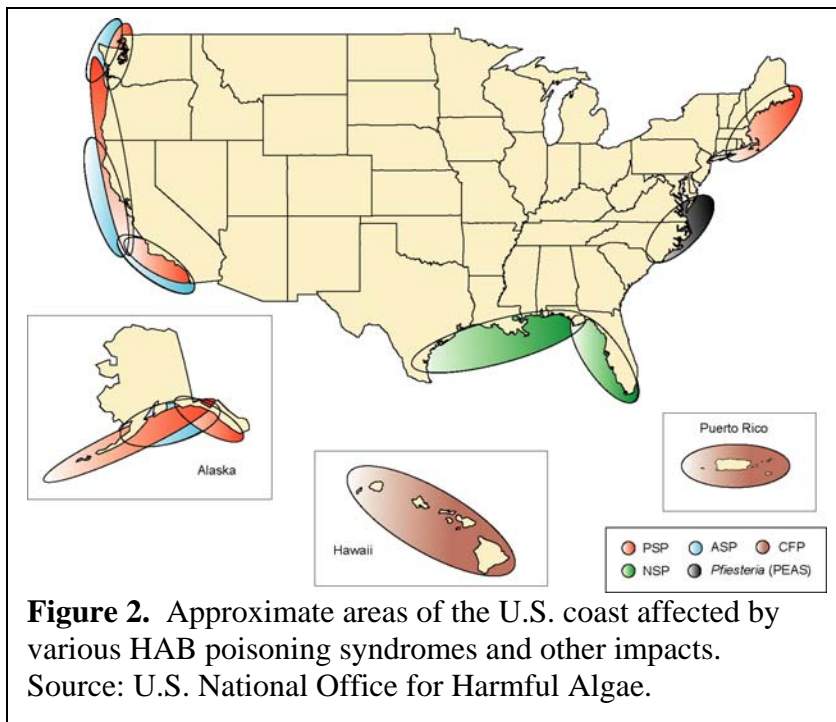


Figure 2. Approximate areas of the U.S. coast affected by various HAB poisoning syndromes and other impacts. Source: U.S. National Office for Harmful Algae.

Figure 2 shows the approximate areas of the US coast that are affected by these and other HAB syndromes and impacts.

Monitoring programs.

Molluscs, bivalves and gastropods (i.e., clams, mussels, oysters, scallops, carnivorous whelks) are typically the primary vectors of algal biotoxins to human consumers, although crustaceans (e.g. crabs and lobsters) and some filter-feeding fish (e.g., herring, anchovies, menhaden) can

also accumulate and transfer biotoxins through the food chain (reviewed by Shumway, 1990). The safest and most commonly used monitoring practice is that used by states in the U.S. who sample wild or cultured shellfish directly from the natural environment and issuing harvesting restrictions when toxins exceed threshold values. These tests are often conducted using a mouse bioassay, which gives an indication of the potency of the various toxins that might accumulate in the shellfish tissues. New techniques for toxin detection are emerging, ranging from highly complex instrument-based analyses to portable kits that resemble pregnancy test kits. These simple kits could be of great value to yachtsmen, as they would allow shellfish to be tested onboard in remote locations where information is lacking on the risk from algal toxins.

Currently, test kits of this type are available for the PSP, ASP, and CFP toxin families. In all of these cases, the user is cautioned to check carefully before relying on these kits to guide personal

consumption. Some have received mixed reviews from scientists (e.g., Lewis 199[?]). Note also that kits for ASP and DSP toxins are available from Biosense Laboratories (<http://www.biosense.com/index.asp>), but these are relatively expensive kits (~\$500) for multiple, concurrent analyses and require considerable technical skills to use, and therefore are not listed in Table 1 with the other, simpler kits.

Table 1. Portable test kits for HAB toxins

Toxin family	Manufacturer	Approx. cost per test	Web reference
PSP	Jellett Rapid Testing Ltd	\$24	www.jellett.ca/
ASP	Jellett Rapid Testing Ltd	\$24	www.jellett.ca/
CFP	Cigua-Check	\$28	http://cigua.oceanit.com/

Typically, state- or federally run biotoxin monitoring programs are highly effective in safeguarding public health, but the results of testing need to be communicated to all potential consumers of shellfish. The amount of information available to the general public on the risks in a particular area varies considerably throughout the world, however. Some countries operate extensive monitoring programs and disseminate closure information widely. Others are less open about the dangers, do not effectively monitor local waters, or do not provide appropriate warnings. Again, those traveling to a given area on a small boat will often not have access to the newspapers and other communication outlets that might provide information on a current or developing threat from a toxic bloom or other such event.

RISKS TO BOATERS

Recreational boaters can encounter HABs in a number of ways, and these interactions can lead to illness, discomfort, and even death. It is therefore imperative for operators of small vessels to be aware of the risks, as well as the steps that should be taken to avoid HABs and their toxins, and to respond to a poisoning event if it occurs. The following sections address the major HAB poisoning syndromes in marine waters. There are a number of problems associated with toxic and harmful algae in fresh waters, but many of those are beyond the scope of this review. The threats from freshwater HAB toxins are associated with either ingestion of water containing those toxins, or contact with water containing certain types of freshwater algae (discussed below), leading to rashes and skin irritation (Stone and Bress, 2007). Exposure to freshwater toxins via the consumption of fish or shellfish is not considered a significant risk to humans at this time.

Marine HAB Syndromes

The most significant threat to recreational boaters from marine HAB species is through the consumption of toxic fish or shellfish, but it is also possible to be affected by inhalation of aerosolized toxins, as well as to encounter skin irritants during swimming. Table 2 lists each of these syndromes, as well as the causative organisms, the approximate areas affected, the major toxin or chemical involved, route of poisoning, a list of common symptoms, and types of medical treatment that are recommended to victims. Each of these is discussed in more detail in the sections below.

Toxins Known to Accumulate in Fish Tissues: Ciguatera toxins are well known for their ability to accumulate in fish tissues (e.g., Anderson and Lobel, 1987), but PSP and ASP toxins can do the same, but to a lesser extent. Typically, fish are sensitive to PSP toxins and will die before they accumulate doses that represent a health risk to humans (White, 1981). Whales and other marine mammals have died from consuming PSP toxin-contaminated fish (e.g., Geraci et al., 1989), but the quantity of fish consumed is typically enormous. Filter feeding fish such as sardines and herring can accumulate PSP and ASP toxins (or more appropriately can carry large amounts of undigested algae in their stomachs), and this has resulted in poisonings in human populations who consume whole fish. Another group of toxins (brevetoxins produced by *Gymnodinium* spp.) may contaminate fish flesh as well, but so far the evidence is limited and only shows low levels not thought to represent a threat to humans.

Unfortunately, the group of toxins that kill the most mariculture fish (those from *Heterosigma*, *Chattonella*, and the *Karlodinium/Karenia* complex) remain poorly or only partially characterized chemically and poorly studied with respect to accumulation in fish. Although we believe these toxins do not accumulate in fish to any significant extent, and thus do not pose a risk to humans, dead fish found floating in the water should not be consumed under any circumstances.

Paralytic Shellfish Poisoning (PSP)

Paralytic shellfish poisoning (PSP) is arguably the most widespread of the human poisoning syndromes linked to HABs. PSP results when shellfish are consumed that have accumulated toxins produced by microscopic algae. In the United States, the dinoflagellates *Alexandrium tamarense*, *A. fundyense*, and *A. catenella* are the predominant causative organisms, though recently *Pyrodinium bahamense* has been linked to this syndrome in eastern Florida. These organisms produce a family of potent neurotoxins called the saxitoxins.

States or countries at risk from PSP typically conduct monitoring programs to regulate the harvest of shellfish so as to protect human health (Anderson et al., 2001). This involves the collection of shellfish samples during the “bloom” season of the causative organism, and testing of these organisms for saxitoxins. When toxins are detected above an action level (80 micrograms of saxitoxin “equivalents”/100 grams shellfish meat) the area is closed to harvesting. Table 3 lists the state web sites and hotlines that can be contacted to determine areas that are open or closed due to PSP toxins at any particular time.

The predominant threat to recreational boaters from PSP would be through the consumption of contaminated shellfish. Filter-feeding shellfish of many different types accumulate PSP toxins. This includes soft shell clams, mussels, oysters, scallops, and hard clams. These shellfish can be intertidal or subtidal. There is no visible evidence that a particular bivalve is or is not toxic - only extraction and analysis will provide that information. Carnivorous whelks can also accumulate PSP toxins since they eat shellfish. Likewise crabs and lobsters can accumulate PSP toxins, but only in the viscera. There are no state regulations restricting consumption of lobster or crab viscera, but it is wise to restrict the consumption of the hepatopancreas or “tomalley” material from these crustaceans as it can accumulate PSP toxins. Unless one consumes a large quantity of this material, however, there is no danger. For shellfish, the danger is much more severe - in some cases, even one or two clams can be fatal to a human, so extreme caution must be exercised when shellfish are harvested and consumed from areas known to experience PSP outbreaks.

Fish can accumulate PSP toxins, but these are typically at dangerous levels only in the viscera, and thus are of danger only to those who consume entire fish, as is sometimes the case for some ethnic communities. There is no danger from aerosolized PSP toxins, nor is there generally any danger associated with swimming in water that has resulted in the closure of shellfish due to PSP. The toxins that might be ingested in small amounts of seawater that are swallowed are insignificant.

One risk that is unique to boaters is that they might enter an area from the water, and thus not see signs that are posted on shore to warn the public that the shellfish in that area are toxic. Indeed, human fatalities from PSP occurred on the coast of California when recreational divers unknowingly collected and consumed whole scallops from an area that was closed to harvesting.

The symptoms of PSP include: tingling, numbness, and burning of the lips and area around the mouth, loss of coordination, giddiness, drowsiness, fever, rash, and staggering. The most severe cases result in respiratory arrest within 24 hours of consumption of the toxic shellfish. There is no antidote for PSP. Onset of the symptoms is generally very short – 0.5 – 2 hours after consuming the contaminated shellfish, depending on the amount of shellfish eaten. In general, supportive measures are the basis of treatment, and artificial respiration may be required. Without supportive treatment, up to 75% of severely affected persons die within 12 hours. When such support is applied within 12 hours of exposure, recovery usually is complete, with no lasting side effects. In unusual cases, because of the weak hypotensive action of the toxin, death may occur from cardiovascular collapse despite respiratory support.

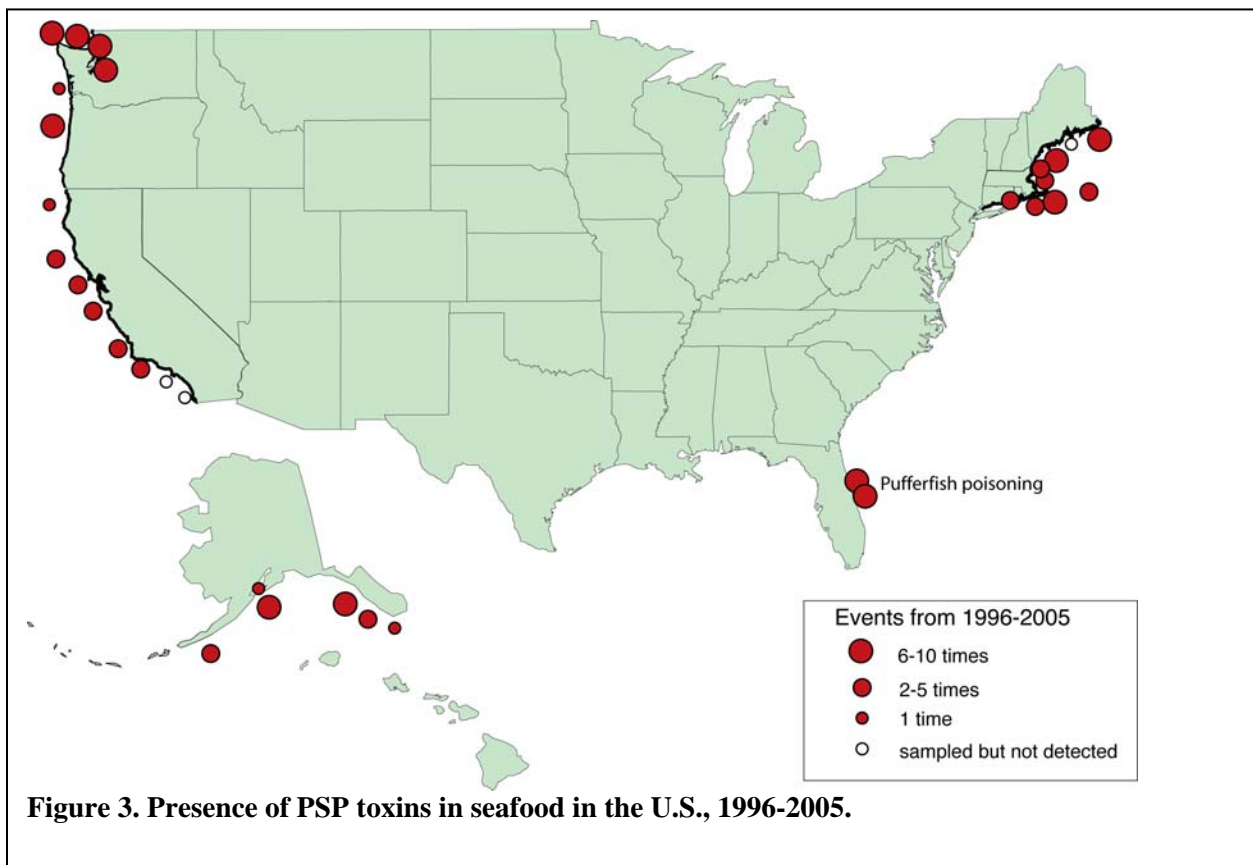
If the consumption of contaminated shellfish is recent, gut decontamination by gastric lavage and administration of activated charcoal or dilute bicarbonate solution is recommended. Care must be taken concerning aspiration with the neurologically compromised patient.

Many endemic areas have traditionally used local treatments with variable success. In the Philippines, a drink of coconut and brown sugar is administered; demonstrations in mice show that these ingredients may have active detoxification substances (Viviani, 1992).

Precautions. When in areas subject to PSP outbreaks, the boater is advised to obtain up-to-date information about shellfish closures in the area. Table 3 lists state websites in the U.S., but local shellfish officers should also be contacted for current information. Travelers in other countries should endeavor to find information from health or marine resource agencies. The advent of test kits similar to home pregnancy tests (Table 1) makes it possible to test shellfish directly, but this should only be done when information from state monitoring programs is unavailable, and then only with a recognition that toxicity can be highly variable in a given area and that the testing may not reveal the actual risk. Cooking does not destroy PSP toxins.

Note that scallops can typically be consumed from areas with PSP outbreaks but only if the viscera and other tissues are discarded and only the adductor muscle is eaten.

Affected Areas. Figure 3 shows the areas of the US that have been affected by PSP, and the frequency at which these types of events have occurred over the last ten years. On the east coast, the problem is most significant in the Gulf of Maine region, but also occurs in southern New England waters, including in isolated embayments on Cape Cod, in Connecticut, and Long Island. The causative organism has been reported in New Jersey, but no PSP outbreaks have occurred in that state. On the west coast the problem extends from southern California to Alaska. PSP is thus the most widespread of all of the HAB syndromes in the United States. PSP is also widespread globally. A map showing the worldwide distribution of PSP events is given at http://www.whoi.edu/redtide/HABdistribution/PSP_worldmap_2005.gif



Neurotoxic Shellfish Poisoning (NSP)

There are two types of human illness associated with the brevetoxins that cause neurotoxic shellfish poisoning – one results when shellfish are consumed that have accumulated toxins; the second (and more common) is associated with inhalation of the aerosolized toxins from sea spray exposure. The dinoflagellate *Karenia brevis*, the predominant causative organism, produces a family of neurotoxins known as brevetoxins.

As with PSP, states that are at risk from NSP are required to conduct monitoring programs to protect human health. Typically, this involves the collection of plankton samples during the “bloom” season of the causative organism, to calculate the number of cells. When cell concentrations exceed 5000 cells/L, harvesting closures are initiated, and additional monitoring is implemented (ISSC, 1997). Website addresses providing information on NSP are given in Table 3. In addition, Florida uses satellite remote sensing to detect *K. brevis* blooms and to predict their magnitude and movement along the coast. A “HAB bulletin” containing this information and satellite images is e-mailed to subscribers (Stumpf et al., 2003) and is available on the web as well <<http://www.csc.noaa.gov/crs/habf/>>.

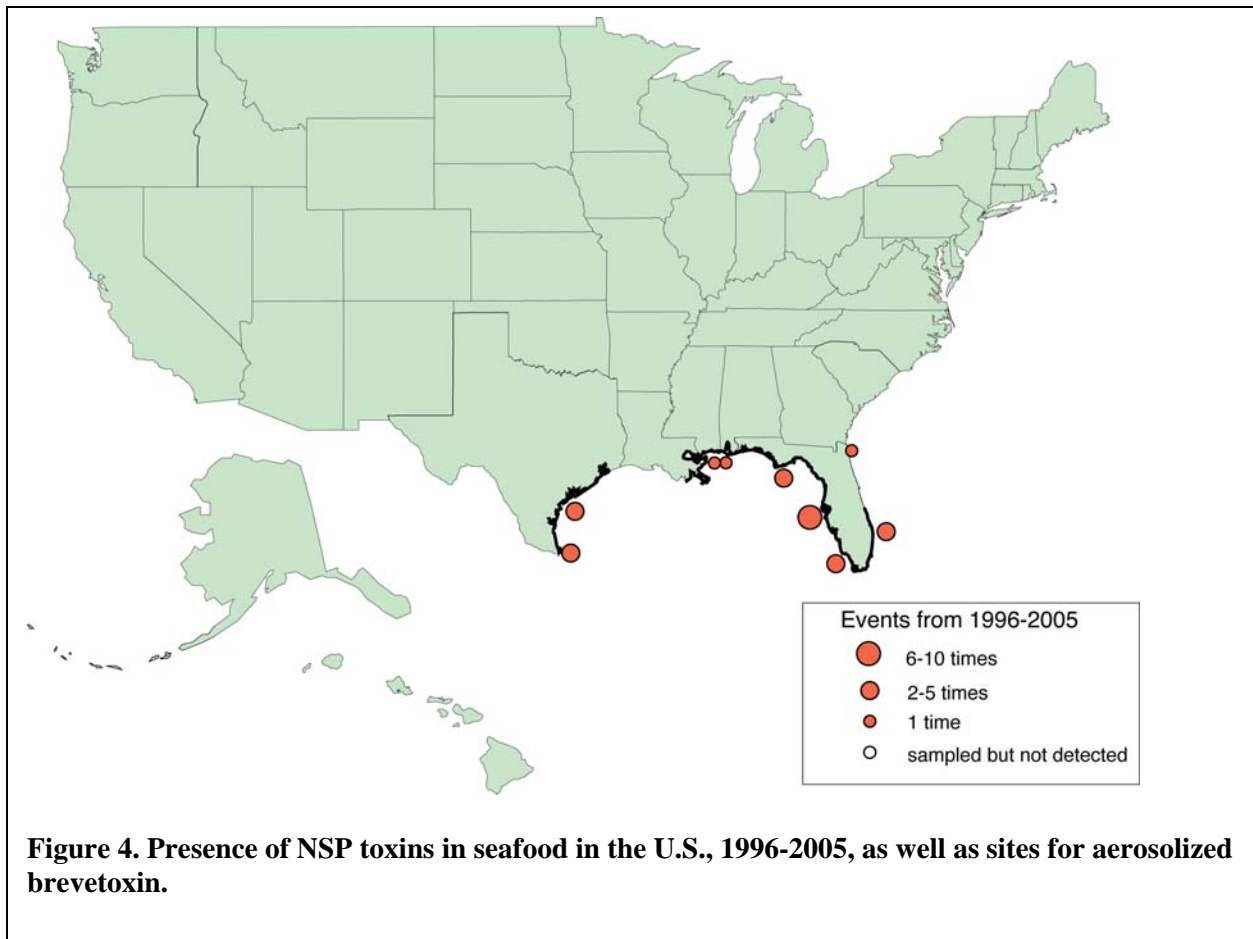
NSP typically results from ingestion of shellfish, such as oysters, clams and other suspension-feeders contaminated with brevetoxins. Onset of symptoms can be from a few minutes to a few hours of ingestion, and include abdominal pain, nausea, vomiting, diarrhea, chills, headache, dilated pupils, dizziness, numbness and tingling of the face, hands and feet. With NSP caused by ingestion of contaminated shellfish, treatment is supportive and patients usually recover completely within a few days. Recovery is complete with few after effects; no fatalities have been reported.

Humans can also be exposed to brevetoxins by inhalation of a sea spray aerosol containing fragments of algal cells and/or toxins released into the surf by lysed algae (Fleming et al., 2005). This is typically associated with major blooms with significant fish kills. Respiratory problems include severe irritation of conjunctivae and mucus membranes (particularly of the nose) followed by persistent coughing and sneezing and tingling of the lips. The use of particle filter masks or retreat to an air-conditioned environment is reported to provide relief, as does moving to an area further inland where the aerosol is not present.

Precautions. As with PSP, boaters should be aware of shellfish harvesting closures and only consume shellfish from open areas. Minimize prolonged exposure to the aerosolized toxins during significant blooms by avoiding beaches and swimming. If a boater encounters the aerosolized toxin, particle filter masks or some other means of filtering air (i.e., breathing through a towel) should be used while efforts are made to leave the affected area. Moving upwind of the red tide patch will be beneficial, but recognize that some of these outbreaks can cover many square miles. Individuals with respiratory problems (asthma, etc.) are particularly susceptible and should take special precautions, such as staying informed about the potential for *Karenia* blooms and carrying masks and respiratory medications.

Affected Areas. In the U.S., NSP is predominantly a problem along the Gulf of Mexico coast from Florida to Texas, with the west coast of Florida being the most affected (Figure 4). It has

also occurred on the east coast of Florida, and once as far north as North Carolina, when carried northward by currents. The syndrome is not widespread globally, with documented cases only in New Zealand (see http://www.whoi.edu/redtide/HABdistribution/NSP_worldmap_2005.gif).



Amnesic Shellfish Poisoning (ASP)

Amnesic shellfish poisoning (ASP) results when fish or shellfish are consumed that have accumulated toxins produced by diatoms in the genus *Pseudo-nitzschia*. The species associated with this syndrome produce a toxin known as domoic acid. ASP first came to the attention of public health authorities in 1987 when 156 cases of acute intoxication occurred as a result of ingestion of cultured blue mussels (*Mytilus edulis*) harvested off Prince Edward Island, in eastern Canada; 22 individuals were hospitalized and three elderly patients eventually died (Bates et al 1988).

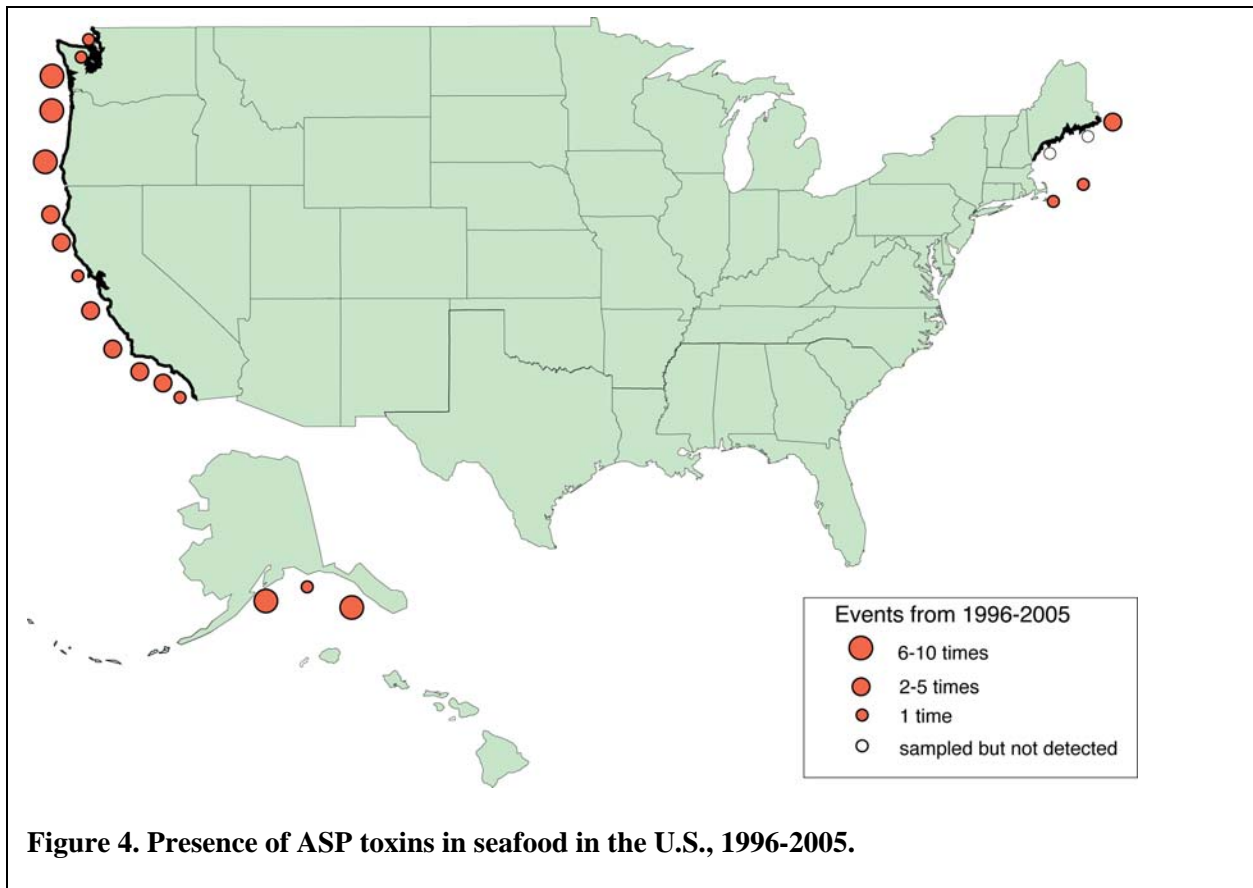
States and countries at risks from ASP are required to conduct monitoring programs or to regulate the harvest of shellfish so as to protect human health. Typically, this involves the collection of shellfish samples during the “bloom” season of the causative organism, and testing of these organisms for domoic acid. When these toxins are detected above an action level (20 micrograms DA/100 grams shellfish meat) the area is closed to harvesting. In some areas, phytoplankton sampling is conducted to determine the cell concentration of the causative

organism and to guide shellfish flesh testing. Table 3 lists the U.S. state web sites and hotlines that can be contacted to determine areas that are opened or closed due to ASP at any particular time.

ASP toxicosis is characterized by the onset of gastrointestinal symptoms within 24 hours; neurological symptoms occur within 48 hours. Symptoms include gastrointestinal effects such as nausea, vomiting, gastric distress, gastric bleeding and diarrhea, followed by neurological symptoms including incapacitating headache, dizziness, confusion, weakness, lethargy, somnolence, coma, seizures, and permanent short-term memory loss. Infants, elderly individuals and individuals with compromised renal function are considered high-risk groups for death or memory impairment (reminiscent of Alzheimer's disease) by ASP. All fatalities to date have involved elderly patients.

Treatment of ASP is symptomatic and supportive. It has been reported that seizures respond to IV diazepam and phenobarbital.

Precautions. As with PSP and NSP, individuals are advised to consult with local officials if they are in an area with ASP risk. Table 3 lists state websites in the U.S. A relatively inexpensive test kit for ASP is available (Table 1), making it possible to test shellfish directly, but this should only be done when information from state monitoring programs is unavailable, and then only with a recognition that toxicity can be highly variable in a given area and that the testing may not reveal the actual risk. Cooking does not destroy ASP toxins.



Affected Areas. ASP occurs along the entire west coast of the U.S., including Alaska (Figure 5) and is believed to be implicated in the deaths of humpback whales along Georges Bank on the east coast. No east coast shellfish in US waters have shown domoic acid levels above quarantine, but toxin has been detected at lower levels. Globally, the problem is fairly widespread (see http://www.whoi.edu/redtide/HABdistribution/ASP_worldmap_2005.gif), occurring in eastern and western Canada, western Europe, New Zealand, and Chile. This distribution will likely expand in time, as the toxin is fairly new to science.

Diarrhetic Shellfish Poisoning (DSP)

DSP is a gastrointestinal illness without neurological manifestations. It is caused by the consumption of contaminated shellfish that have accumulated toxins known as okadaic acid and dinophysistoxins (DTXs). The causative organisms are from two unrelated dinoflagellate genera - *Dinophysis* and *Prorocentrum*.

Many countries test shellfish samples for the presence of okadaic acid. When these toxins are detected above an action level (typically 20 micrograms /100 grams shellfish meat) the area is closed to harvesting. In the U.S., DSP is not considered a major threat, so no state currently monitors for it directly. Some states utilize plankton sampling to alert them of the presence of potentially toxic algae, after which confirmatory shellfish flesh tests are conducted.

DSP produces severe gastrointestinal symptoms, usually beginning within 30 min to a few hours after consumption of toxic shellfish. The illness, which is not fatal, is characterized by incapacitating diarrhea, nausea, vomiting, abdominal cramps, and chills lasting as long as 2-3 days. Recovery occurs within three days, with or without medical treatment. Recovery is complete with no after effects; the disease is generally not life threatening, but is very unpleasant.

Treatment is symptomatic and supportive with regards to short-term diarrhea and accompanying fluid and electrolyte losses. In general, hospitalization is not necessary; fluid and electrolytes can usually be replaced orally.

Affected Areas. DSP has a widespread global distribution, occurring in western Europe, the Mediterranean, southeast Asia, New Zealand, Australia, and Chile (see http://www.whoi.edu/redtide/HABdistribution/DSP_worldmap_2005.gif). The unique aspect of this distribution is that there has been virtually no DSP reported in North America or much of Central and South America, even though the causative species are found in those locations.

Precautions. The same precautions used in areas subject to PSP and ASP should be followed. There are, however, no inexpensive kits for DSP toxin analysis as yet.

Azaspiracid shellfish poisoning (AZP)

Several years ago, a new type of shellfish poisoning was discovered when European consumers of Irish mussels became very ill. The resulting investigations characterized a toxin family called the azaspiracids, and the poisoning syndrome was thus named azaspiracid shellfish poisoning or AZP. Most countries (including the U.S.) are not yet monitoring for this toxin family, so we know very little of its distribution.

The symptoms associated with AZP are similar to that of DSP (i.e., nausea, vomiting, severe diarrhea, and stomach cramps) but also include headaches and cramps.

Affected areas. AZP has been found in the United Kingdom, northwest Spain, France, Morocco and Norway (see http://www.whoi.edu/redtide/HABdistribution/AZP_worldmap_2005.gif).

Ciguatera Fish Poisoning (CFP)

The most commonly reported marine toxin disease in the world is ciguatera fish poisoning (CFP), associated with consumption of contaminated reef fish such as barracuda, grouper, and snapper. CFP is caused by biotoxins produced by dinoflagellates attached to surfaces in many coral reef communities. The primary organism associated with CFP is *Gambierdiscus toxicus* although other benthic dinoflagellates may be associated with this syndrome. Ciguatera toxins, known as ciguatoxins and maitotoxins, are transferred through the food chain from herbivorous reef fishes to larger carnivorous, commercially valuable finfish.

Unfortunately, routine monitoring of fish at the market or at the harvest site for ciguatera is presently not conducted in any country. Instead, restrictions are placed on the sale or consumption of certain species of fish that are known to be frequently contaminated with ciguatera toxins. For example, in Queensland, Australia, fish species such as red bass, “chinaman fish”, and paddletail are not commercially exploited due to ciguatera (Lewis, 1994). On islands like St. Barthelemy in the Caribbean, fish are not harvested from areas of the island where history has shown the fish to be frequently contaminated with ciguatera toxins (Lobel et al., 1988). Likewise, fishermen are often advised to not eat barracuda that weigh more than 10 pounds. This precaution stems from the fact that the ciguatera toxins biomagnify as they move through the food web, and accumulate in the higher predators, especially the largest ones.

The Cigua-Check™ kit is available directly from Oceanit Test Systems (about \$28). Each kit can test five fish in less than an hour without any special equipment. The kit is reported to detect ciguatoxin at levels down to 0.1 ppb, which is comparable to the threshold for human symptomology of 0.05-0.1 ppb. The immunoassay consists of a membrane laminated on a plastic stick which is pushed into a rice grain-sized piece of fish tissue. After some simple processing steps, the color intensity of the membrane is related to the concentration of toxin bound to the membrane, yielding negative, borderline or positive results.

The method can be used in the field, and has been used to survey areas in Hawaii where CFP is endemic, and for analysis of fish implicated in clinically documented cases of CFP. The main problems associated with the application of this kit are: 1) the difficulty in obtaining pure CTX1 for calibration; 2) the false positives obtained due to cross-reactivity of the CTX1 antibodies with other polyether compounds, including less potent ciguatoxins, palytoxin, maitotoxins, and

brevetoxins and okadaic acid (causative agents of NSP and DSP) that are structurally related to CTX; 3) difficulties reading test results, as many samples show some color, whereas non-toxic fish should be colorless; and 4) the structural differences between ciguatoxins from other regions of the world, potentially leading to false negatives. Cases have been reported of fishermen in the Caribbean who used this kit and then consumed supposedly safe fish, only to develop severe CFP symptoms shortly thereafter. The antibody used in the kit was developed against Pacific ciguatoxins, and it has been determined that there are differences in toxin composition between ciguatoxic fish in the Pacific, Indian Ocean and Caribbean region.

The precursor compounds to ciguatera toxins (gambiertoxins) originate in benthic dinoflagellates that are grazed by herbivorous reef fish. The gambiertoxins are then metabolized to ciguatoxins as they move through the food chain via carnivorous fish. Marine finfish most commonly implicated include the groupers, barracudas, snappers, jacks, king mackerel, and triggerfish. Many other species of warm-water fishes harbor ciguatera toxins as well. The occurrence of toxic fish is sporadic, and not all fish of a given species or from a given locality will be toxic.

The most distinguishing symptom of CFP is temperature reversal (cold things taste or feel hot and vice versa) (Fleming et al., 2001). Other symptoms of ciguatera vary geographically and between individuals, but generally include early onset (2-6 hr) of gastrointestinal disturbance – nausea, vomiting, and diarrhea – and may be followed by a later onset (18 hr) of neurological symptoms such as numbness of the lips, mouth and tongue, reversal of temperature sensation, muscle and joint aches, headache, blurred vision and paralysis. Ciguatera on rare occasions can be fatal. Gastrointestinal symptoms generally persist only a few days, whereas neurologic symptoms may persist for weeks, months or even years. People who do not receive treatment within 72 hours may experience tingling, numbness and other neurologic symptoms for weeks to months (Blythe et al., 1994). Ciguatera symptoms in the Caribbean differ somewhat from those in the Pacific; gastrointestinal symptoms dominate in the former, whereas in the latter neurological symptoms tend to dominate.

Medical treatment for CFP has been to a large extent symptomatic; a variety of agents, including vitamins, antihistamines, anticholinesterases, steroids and tricyclic antidepressants, have been tried with limited results. Gut emptying and decontamination with charcoal is recommended acutely although often the severe ongoing vomiting and diarrhea prevents this. Atropine is indicated for bradycardia, and dopamine or calcium gluconate for shock.

With considerable success, at least acutely, mannitol infusions have been used. Reports show that mannitol appears to be most effective in completely relieving symptoms when given within the first 48-72 hours from ingestion of contaminated fish.

Affected Areas. Ciguatera is found in subtropical and tropical areas throughout the world (between 35°N and 34°S), including Hawaii, the Florida Keys, Puerto Rico, the Caribbean, Australia and many Pacific islands (see http://www.whoi.edu/redtide/HABdistribution/CFP_worldmap_2005.gif).

Precautions. Among all HAB poisoning syndromes, CFP is perhaps the largest risk to operators of small boats. Many an idyllic vacation to a tropical island has ended in disaster due to this toxin syndrome. In an area where CFP is known, individuals should not eat barracuda or moray eel, and should be cautious with certain types of grouper and snapper. , as well as enquiring about local fish associated with ciguatera. Since there is no reliable way to "decontaminate" or even to distinguish contaminated fish by smell or appearance, at a minimum, people are advised to avoid the viscera of any reef fish as well as avoiding consuming unusually large predatory reef fish especially during the reproductive season. Note that these fish, when caught, seem healthy and have a normal taste and appearance. In addition, the toxicity (or lack thereof) of one fish cannot be used to predict the health of other fish in the region. In the Pacific, the CiguaCheck kit may be used to test individual fish, but the risk of false positives and negatives is significant.

Possible estuary-associated syndrome (PEAS)

In the 1990's some fish kills from the Chesapeake Bay to North Carolina were linked to a toxic dinoflagellate called *Pfiesteria piscicida*. There is considerable controversy about this organism and its toxicity. Some scientists believe that *Pfiesteria* becomes toxic in the presence of fish, particularly schooling fish like Atlantic menhaden, triggered by their secretions or excrement in the water. Others attribute some of the fish kills to other, closely related organisms such as *Karlodinium micrum*. Moeller et al. (2007) recently reported the isolation and preliminary characterization of a toxin from *P. piscicida*, but considerable work is needed before we fully understand the nature and extent of the toxin threat to humans.

The organism is known to occur in brackish coastal waters along the mid-Atlantic coast of the U.S., and has been reported in a few scattered locations worldwide as well. There are many organisms that superficially resemble *Pfiesteria*, so identification is problematic. One common co-occurring species is *Karlodinium micrum*, which produces a toxin called karlotoxin. It may be that some of the impacts attributed to *Pfiesteria* are from this or other species.

Preliminary evidence suggests that exposure to *Pfiesteria* toxins in the air, water, or fish at the site of an outbreak can cause skin and eye irritation as well as short-term memory loss, confusion, and other cognitive impairments in people (Grattan et al., 1998). No toxic activity has been detected in shellfish harvested from sites of *Pfiesteria* blooms.

Precautions. As with other possible HAB syndromes, the boater is advised to obtain up-to-date information regarding bloom events, paying particular attention to fish kills. Avoid handling or consuming fish that are dead or dying, that exhibit sores or other signs of disease or were acting abnormally when caught. It is also advisable to move away from the site of a fish kill, as some human illnesses have been reported from relatively minor aerosol or water contact.

Lyngbya

Lyngbya is a toxic marine cyanobacterium (blue-green algae) which forms clumps or mats of fine strands that attach to seaweed and rocks. Through the accumulation of gas bubbles mats can

rise to the surface to form large floating mats and these mats can wash up on beaches often mixed with seagrass.

Exposure to *Lyngbya* can cause skin, eye, and respiratory irritation (Osborne, et al., 2001). People who have come into contact with *Lyngbya* may complain of a stinging, burning or itching sensation within minutes to hours after exposure. Affected areas may appear red and swollen, and small blisters may form. In severe cases, affected skin may peel off. Reddening and swelling of the conjunctiva of the eye and the mucous membrane of the nose may also occur if those parts of the body have direct contact with *Lyngbya* (Queensland Environmental Protection Agency website)

Precautions. Because of the potential for severe irritation, boaters should avoid swimming or wading in areas where *Lyngbya* is growing or floating in the water and should not have direct contact with material washed onto the beach.

SUMMARY

Harmful algal blooms are a significant and growing problem in coastal waters worldwide. These events tend to be sporadic and dispersed, and thus the risk at any one time or place is not large. The consequences from the different poisoning syndromes or from direct contact with species like *Lyngbya* can, however, be severe and even life threatening. Armed with a knowledge of the fundamental nature of HAB toxins and their effects and symptoms, and a willingness to assess possible risks by contacting local authorities or staying alert via hotlines and other information sources, boaters can minimize their exposure to dangers while continuing to enjoy the fish, shellfish, and recreational resources around them.

ACKNOWLEDGEMENTS

We thank T. Brainard and J. Hain for their support and encouragement in the preparation of this review. We also acknowledge the many state, federal, and university websites and other information sources for their efforts to communicate all aspects of HAB and their impacts. Support provided by: the Bonnell Cove foundation; the NOAA National Center for Sponsored Coastal Research through Cooperative Agreement NA17RJ1223; NIEHS Grant 1 P50 ES012742; and NSF Grants OCE-0430724 and OCE-0402707.

Table 2. Human illnesses associated with harmful algal blooms. (Modified from Anderson et al. – 2001).

Syndrome	Areas Affected	Causative organisms	Toxins produced	Route of acquisition	Common symptoms ¹	Types of medical treatment
Ciguatera fish poisoning (CFP)	Hawaii, Florida Keys, Puerto Rico, the Caribbean, Australia, and many Pacific islands	<i>Gambierdiscus toxicus</i> (benthic) and others	Ciguatoxins	Toxin transfer up the marine food chain; illness generally results from consumption of large, reef fish	Acute gastroenteritis, paresthesia and other neurological symptoms	Rapid treatment (within 48-72 hours of ingestion) with mannitol is reported to relieve some symptoms; Atropine is indicated for bradycardia, and dopamine or calcium gluconate for shock
Paralytic shellfish poisoning (PSP)	US west coast, incl. Alaska, New England coastal states, Canada, Chile, Brazil, Europe, South Africa, Asia, Australia, New	<i>Alexandrium</i> spp, <i>Gymnodinium catenatum</i> , <i>Pyrodinium bahamense</i> var. <i>compressum</i> and others	Saxitoxin family	Consumption of shellfish harvested from affected areas; filter-feeding fish can also vector toxins to humans	Acute paresthesias and other neurological manifestations; may progress rapidly to respiratory distress, muscular paralysis and death	no antidote, supportive therapy is the rule and survivors recover fully. Ventilatory support is required in severe cases
Neurotoxic shellfish poisoning (NSP)	US Gulf coast, and New Zealand	<i>Karenia brevis</i> , <i>Karenia brevisulcatum</i> and others	Brevetoxins	Consumption of shellfish harvested from affected areas; toxins may be aerosolized by wave action, causing respiratory problems and eye irritation	Gastrointestinal and neurological symptoms; respiratory and eye irritation with aerosols	No antidote for shellfish poisoning – provide supportive therapy; for aerosolized toxins, use particle filter masks or retreat to air conditioned environment
Diarrhetic shellfish poisoning (DSP)	Europe and Japan, Atlantic Canada, South Africa, Chile, Thailand, New Zealand, and Australia	<i>Dinophysis</i> spp., <i>Prorocentrum lima</i>	Okadaic acid and dinophysistoxins (DTXs)	Consumption of shellfish harvested from affected areas	Acute gastroenteritis	Treatment is symptomatic and supportive; replace fluid and electrolytes; recovery generally occurs within 3 days with or without treatment

Azspiracid shellfish poisoning (AZP)	England, Scotland, Ireland, France, Spain, Morocco, Norway	<i>Protoperidinium crassipes</i>	Azspiracids	Consumption of shellfish harvested from affected areas	Neurotoxic effects with severe damage to the intestine, spleen, and liver tissues in animal tests; possible carcinogen	Treatment is symptomatic and supportive; replace fluid and electrolytes;
Amnesic shellfish poisoning (ASP)	US west coast, incl. Alaska, Atlantic Canada, Georges Bank, Chile, Australia, New Zealand, the United Kingdom	<i>Pseudo-nitzschia</i> spp.	Domoic acid and isomers	Consumption of shellfish (or, possibly, fish) harvested from affected areas	Gastroenteritis, neurological manifestations, leading in severe cases to amnesia (permanent short-term memory loss), coma, and death	Treatment is symptomatic and supportive; IV diazepam and phenobarbital for seizures
Possible estuary-associated syndrome (PEAS)	North Carolina, Maryland, Delaware??	<i>Pfiesteria piscicida</i> and other <i>Pfiesteria</i> spp.	<i>Pfiesteria</i> toxins	Exposure to water or aerosols containing toxins	Deficiencies in learning and memory; acute respiratory and eye irritation, acute confusional syndrome	no known management and treatment
Swimmer's itch & dermatitis	Australia, Florida, worldwide throughout the tropics and subtropics	<i>Lyngbya</i>	Lyngbyatoxin A and debromoaplysiat oxin	Exposure to water or aerosols containing toxins; swimming	"swimmer's itch"; Skin irritation, dermatitis, eye and nose irritations. Inhalation of dried <i>Lyngbya</i> may exacerbate respiratory problems like asthma	Wash exposed skin with soap and water; wash clothes before rewearing; flush eyes with water if irritated. Seek medical treatment if respiratory symptoms continue, particularly if suffering from asthma

¹Definitions of Symptoms:

Paresthesia – *An abnormal sensation of the skin, such as numbness, tingling, pricking, burning, or creeping on the skin*

Dermatitis – *Inflammation of the skin*

Gastroenteritis - *Inflammation of the stomach and the intestines. Can cause nausea and vomiting and/or diarrhea.*

Neurotoxic – *Poisonous to nerves or nerve tissue.*

Table 3. Websites and hotlines for shellfish harvesting closures and related information in the United States

State	Syndrome	Agency	Website	Hotline
Alaska	ASP, PSP	Department of Environmental Conservation	www.dec.state.ak.us/eh/fss/recalls/recallsalerts.htm	(907) 269-7596
California	ASP, PSP	Department of Health Services	www.dhs.ca.gov/ps/ddwem/environmental/shellfish/default.htm	(800) 553-4133
Florida	CFP, NSP, PSP, SPFP	Florida Department of Agriculture and Consumer Services; Florida Fish and Wildlife Conservation Commission; Florida Department of Health	www.floridaaquaculture.com/SEAS/SEAS_mngmt.htm www.floridamarine.org/features/default.asp?id=1018 www.doh.state.fl.us/environment/community/aquatic/index.html	Updates - (866) 300-9399 (within Florida) (727) 552-2448 (elsewhere) Panama City: (850) 236-2200 Apalachicola: (850) 653-8317 Cedar Key: (352) 543-5181 Murdock: (941) 255-7405 Palm Bay: (407) 984-4890 <u>Toxin hotline</u> - (888) 232-8635
Hawaii	CFP	Hawaii Department of Health		Oahu (808) 586-4586 Maui (808) 984-8213 Hawaii (808) 933-0912 Kona (808) 322-4877 Kauai (808) 241-3563 After business hours: Oahu (808) 566-5049 Neighbor Island 1-800-360-2575
Maine	PSP	Department of Marine Resources	www.state.me.us/dmr/rm/public_health/closures/pspclosures.htm	(800) 232-4733
Maryland	Pfiesteria; fishkills	Department of Natural Resources and Department of the Environment	www.dnr.state.md.us/bay/hab/index.html www.mde.state.md.us/ContactUs/emerNumbers/index.asp www.dnr.state.md.us/hotline.asp	(877) 224-7229
Massachusetts	PSP	Division of Marine Fisheries	www.mass.gov/dfwele/dmf/programsandprojects/psp_notice.htm#shelsani	(617) 626-15207
New Hampshire	PSP	Fish and Game Department	www.wildlife.state.nh.us/Fishing/clam_flat_status.htm	(800) 43-CLAMS
Oregon	ASP, PSP	Department of Agriculture, Food Safety Division	egov.oregon.gov/ODA/FSD/shellfish_status.shtml	(800) 448-2474
Texas	NSP	Parks and Wildlife	www.tpwd.state.tx.us/landwater/water/envirnoncerns/hab/redtide/status.phtml	(800) 792-1112 select fishing, then select red tide

Washington	ASP, PSP	Department of Health	ww4.doh.wa.gov/gis/mogifs/biotoxin.htm	(800) 562-5632
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ASP = Amnesic Shellfish Poisoning

DSP = Diarrhetic Shellfish Poisoning

NSP = Neurotoxic Shellfish Poisoning

PSP = Paralytic Shellfish Poisoning

SPFP = Saxitoxin Puffer Fish Poisoning

REFERENCES:

- Anderson, D.M. 1989. Toxic algal blooms and red tides: A global perspective. *In*: T. Okaichi, D.M. Anderson, and T. Nemoto (Eds.), *Red Tides: Biology, Environmental Science and Toxicology*, Elsevier, pp. 11-16.
- Anderson, D.M. and P.S. Lobel. 1987. The continuing enigma of ciguatera. *Biol. Bull.* 172: 89-107.
- 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*. Asia Pacific Economic Program, Singapore, and Intergovernmental Oceanographic Commission, Paris. 268 pp.
- Bates, S.S., C.J. Bird, A.S.W. de Freitas, R. Foxall, M. Gilgan, L.A. Hanic, G.R. Johnson, A.W. McCulloch, P. Odense, R. Pocklington, M.A. Quilliam, P.G. Sim, J.C. Smith, D.V. Subba Rao, E.C.D. Todd, J.A. Walter, and J.L.C. Wright. 1989. Pennate diatom *Nitzschia pungens* as the primary source of domoic acid, a toxin in shellfish from eastern Prince Edward Island, Canada. *Can. J. Fish. Aquat. Sci.* 46: 1203-1215.
- Blythe, D., L.E. Fleming, D.R. Ayyar, D. Baden, D. De Sylva, and K. Shrank. 1994. Mannitol Treatment for Acute and Chronic Ciguatera fish Poisoning. *Memoirs Queensland Museum* 34: 465-470.
- Fleming, LE, J.A. Bean, D. Katz, and R. Hammond. 2001. The epidemiology of seafood poisoning. *In*: Hui, Y.H., D. Kits, and P.S. Stanfield (Eds.), *Foodborne Disease Handbook, vol. 4, Seafood and Environmental Toxins*, Dekker, New York, pp. 287-310.
- Fleming, L.E., L. Backer, and A. Rowan. 2002. The epidemiology of human illnesses associated with harmful algal blooms. *In*: Adams, D.J., D. Baden, J. Bloomquist, M. Ehrich, T. Guilarte, and A. Harvey (Eds.), *Neurotoxicology Handbook, Volume 1: Natural Toxins of Marine Origins*, Humana Pres, Totowa, NJ, pp. 363-381.
- Fleming, L.E., L.C. Backer, and D.G. Baden. 2005. Overview of aerosolized Florida red tide toxins: Exposures and Effects. *Environ Health Perspect.* 113(5): 618-620.
- Geraci, J.A., D.M. Anderson, R.J. Timperi, D.J. St. Aubin, G.A. Early, J.A. Prescott and C.A. Mayo. 1989. Humpback whales (*Megaptera novaeangliae*) fatally poisoned by dinoflagellate toxin. *Can. J. Fish. and Aquatic Sci.* 46: 1895-1898.
- 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.
- Hallegraef, G.M. 1993. A review of harmful algal blooms and their apparent global increase. *Phycologia* 32: 79-99.
- Interstate Shellfish Sanitation Conference (ISSC). 1997. *National Shellfish Sanitation Program. Guide for the Control of Molluscan Shellfish*. U.S. Department of Health and Human Services, Public Health Service, Food and Drug Administration, 406 pp.
- Lewis, 199?
- Moeller, P.R., K.R. Beauchesne, K.M. Huncik, W. Claydavis, S.J. Christopher, P. Riggs-Gelasco, and A.K. Gelasco. 2007. Metal complexes and free radical toxins produced by *Pfiesteria piscicida*. *Environ. Sci. and Tech.* 41(4): 1166-1172.
- Osborne, N.J., P.M. Webb, and G.R. Shaw. 2001. The toxins of *Lyngbya majuscula* and their human and ecological health effects. *Environ. Int.* 27(5): 381-392.
- Queensland Environmental Protection Agency website.
http://www.epa.qld.gov.au/environmental_management/coast_and_oceans/marine_habitats/lyngbya_management_strategy/
- Shumway, S.E. 1990. A review of the effects of algal blooms on shellfish aquaculture. *J. World Aquacult. Soc.* 21: 65-104.
- Smayda, T.J. 1989. Primary production and the global epidemic of phytoplankton blooms in the sea: a linkage? *In: Coper, E.M., E.J. Carpenter and M. Bricelj (Eds.), Novel Phytoplankton Blooms: Causes and Impacts of Recurrent Brown Tide and Other Unusual Blooms*, Springer-Verlag, New York, pp. 213-228.
- Stone, D. and W. Bress. 2007. Addressing public health risks for Cyanobacteria in recreational freshwaters: The Oregon and Vermont Framework. *Integrated Environ. Assess. and Management* 3(1): 137-143.
- Stumpf, R.P., M.E. Culver, P.A. Tester, M. Tomlinson, G.J. Kirkpatrick, B.A. Pederson, E. Truby, V. Ransibrahmanakul, and M. Soracco. 2003. Monitoring *Karenia brevis* blooms in the Gulf of Mexico using satellite ocean color imagery and other data. *Harmful Algae* 2 (2): 147-160.
- Viviani, R. 1992. Eutrophication, marine biotoxins, human health. *Sci. Total Environ. Supplement*, 631-62.

White, A. W. 1981. Marine zooplankton can accumulate and retain dinoflagellate toxins and cause fish kills. *Limnol. Oceanogr.* 26: 103-109.