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Harmful Algal Blooms: At the Interface Between Coastal Oceanography and Human Health

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The Nature of Harmful Algal Blooms

Algal blooms are a common occurrence in aquatic environments. A subset of these blooms poses environmental or public-health threats, and is therefore referred to as “harmful algal blooms,” or HABs. Some HABs are harmful by virtue of their sheer biomass, whereas others are associated with algal blooms capable of producing toxins. During a HAB event, algal toxins can accumulate in predators and organisms higher up the food web. Toxins may also be present in ambient waters, where wave action or human activities can create aerosols containing toxins and cellular debris. Animals, including humans, can thus be exposed to HAB-related toxins when they eat contaminated seafood, have contact with contaminated water, or inhale contaminated aerosols.

We have known for decades about many of the illnesses associated with marine HABs. We know that the toxins causing these illnesses are very stable molecules and are not destroyed by any method of food preservation or preparation. However, many unanswered questions remain about diagnosis, treatment, chronic effects, and other clinical and epidemiologic characteristics of these illnesses. Given that HAB events are becoming more frequent in the world’s waters (Glibert et al., 2005), a pressing need exists to understand, predict, and eventually mitigate the public-health effects from these blooms.

The nature of a HAB event, including physical bloom characteristics and its ultimate effects on public health and the environment, is inextricably related to coastal oceanography. Many algal blooms originate well offshore, with the ambient physical, chemical, and biological environments playing a major role in bloom evolution. As such, the threat to human health is a nearshore manifestation of regional-scale plankton dynamics. In other cases, anthropogenic, point-source pollution is clearly responsible for triggering both the bloom and the subsequent adverse effects. Regardless of whether the problem begins offshore as part of a natural cycle or is triggered by a manmade nearshore environmental disturbance, HAB effects are ultimately regulated by hydrodynamic transport of the harmful organisms into and out of coastal areas that provides a conduit for human exposure to the organisms and their associated toxins.

HABs and Human Health

Probably the best-known human health effects caused by HAB-related organisms are the shellfish poisonings: amnesiac shellfish poisoning (ASP), azaspiridic shellfish poisoning (AZP), diarrhetic shellfish poisoning (DSP), neurotoxic shellfish poisoning (NSP), and paralytic shellfish poisoning (PSP). A specific illness associated with eating contaminated fish is ciguatera fish poisoning, and another group of illnesses may be associated with water exposure to the cyanobacteria or blue green algae. The following provides brief descriptions of some key HAB-related health effects, the culprit organisms, their toxins, and some illustrative oceanographic examples (see reviews by Fleming et al., 2002; Backer et al., 2003; and Backer et al., 2005).

Amnesic Shellfish Poisoning (ASP)

In 1987, a new type of human illness, later termed amnesiac shellfish poisoning (ASP), was diagnosed in people who had eaten mussels from Prince Edward Island (Perl et al., 1990). ASP is caused by domoic acid, which can act as an excitatory neurotransmitter. The source of the domoic acid in this 1987 outbreak was apparently a diatom, *Pseudo-nitzschia multiseries* (= *Nitzschia pungens*) (Figure 1), found in the estuaries where mussels were cultivated. ASP victims reported gastrointestinal symptoms (e.g., vomiting, abdominal cramps, diarrhea) and neurologic symptoms (e.g., incapacitating headache and short-term memory loss) (Perl et al., 1990).

ASP outbreaks along the Pacific coast between Washington state and British Columbia are primarily modulated by the activity of an eddy that typically resides offshore of the Straits of Juan de Fuca (Figure 2; also see results from the Ecology and Oceanography of Harmful Algal Blooms-Pacific Northwest program at http://www.ecohabpnw.org/). The nutrient supply in this eddy is quasi-steady, leading to enhanced biomass (including *Pseudo-nitzschia* in varying numbers) in this region, particularly on the perimeter of the eddy (MacFadyen et al., in preparation). Surface currents can transport materials from the eddy to adjacent shelf waters (MacFadyen et al., 2005), providing a direct pathway for intoxication of coastal razor clam populations, and creating a periodic public-health risk for people harvesting the clams.

The Olympic Region Harmful Algal Blooms partnership (Trainer and Suddleson, 2005; see http://www.orhab.org/) monitors seawater at several coastal sites for a rapid increase in the numbers of *Pseudo-nitzschia* and for the toxins that may originate from the Juan de Fuca eddy. The combination of microscopic monitoring of algae and the assessment of cellular toxicity using commercially available test strips gives resource managers an early warning of dangerous levels of toxins in razor clams.

Diarrhetic Shellfish Poisoning (DSP)

The first cases of human illness from eating mussels contaminated with algal toxins occurred in the 1970s in the Netherlands (Kat, 1979) and Japan (Yasumoto et al., 1978). The toxin syndrome, called diarhetic shellfish poisoning (DSP), is found primarily in Europe and Japan, and outbreaks can involve hundreds of people (Aune and Yndestad, 1993).

The toxins associated with DSP, including okadaic acid and several congeners of dinophysistoxins (DTX), are produced by dinoflagellates (e.g., *Dinophysis* spp. or *Prorocentrum* spp.). Mussels and scallops feeding on these microalgae accumulate the toxins (Aune and Yndestad, 1993). DSP produces severe gastrointestinal (e.g., diarrhea, nausea, vomiting, abdominal pain) symptoms within 30 minutes to 3 hours of eating contaminated shellfish. Victims reportedly recover within a few days (Kat, 1979; Yasumoto et al., 1978).

Algae of the genus *Dinophysis* typically inhabit the transition region between coastal and offshore waters (Smayda and Reynolds, 2001), and are therefore subject to a wide variety of physical-oceanographic forcing mechanisms. For example, studies along the Iberian coast have revealed intricate relationships between *D. acuta* populations and wind-driven coastal upwelling phenomena (Reguera et al., 1995). Peak concentrations of *D. acuta* generally occur in the subsurface stratified waters of the pycnocline in association with relaxation of a prior upwelling event, or even during downwelling conditions. These blooms are thought to result from *in situ* growth and physical aggregation that takes place when upward-swimming organisms encounter regions of convergence (e.g., Franks, 1997).

Neurotoxic shellfish poisoning and respiratory irritation

Neurotoxic shellfish poisoning (NSP) has been reported along the Gulf Coast in the south-eastern United States and eastern Mexico since the 1890s (Steidinger, 1993) and NSP-like symptoms have been reported by people eating shellfish from New Zealand (Ishida et al., 1996). Outbreaks of NSP have involved toxic oysters, clams, and other suspension-feeders that accumulate toxins during red tide HAB events.

The toxins associated with NSP are polyether compounds called brevetoxins (Baden, 1989; Schulman et al., 1990) produced by the dinoflagellate *Gymnodinium breve* (formerly *Ptychodiscus brevis* and recently renamed *Karenia brevis* [Daugbjerg et al., 2000]) (Figure 3). The acute symptoms of NSP are similar to those reported with ciguatera fish poisoning, and include abdominal pain, nausea, diarrhea, burning pain in the rectum, headache, bradycardia, and dilated pupils. NSP victims have also reported temperature sensation reversals, myalgia, vertigo, and ataxia (Hughes and Merson, 1976: Sakamoto et al., 1987; McFarren et al., 1965; Baden, 1983).

In addition to NSP, brevetoxins can cause respiratory distress and eye irritation when individuals inhale sea spray contaminated with these toxins (Music et al., 1973). Recent studies have found that healthy lifeguards periodically exposed to aerosolized brevetoxins during Florida red tide events experience acute respiratory symptoms, but do not appear to have chronic effects (Backer et al., 2005). By contrast, people with asthma show small changes in lung function when they spend even short periods of time on the beach during Florida red tides if onshore winds cause aerosol exposures (Fleming et al., 2005) (see Case Study by Abraham and Baden, this issue).

Blooms of *K. brevis* on the west Florida shelf typically originate in offshore waters, 20 km–70 km from the coast (Steidinger and Haddad, 1981). The organism can form extraordinarily dense blooms, clearly visible to the naked eye (Figure 3, lower panel) and to satellite-based ocean color sensors (Figure 4). Although the underlying ecologic dynamics of these blooms

and the ultimate source of nutrients needed to produce such biomass are still under debate (Walsh and Steidinger, 2001; Walsh et al., 2003), their visibility from space has led to satellite-based methods for monitoring and prediction (Stumpf et al., 2003). The Gulf of Mexico HAB Bulletin uses satellite imagery, field observations, and buoy data to provide information on the location, extent, and potential for development or movement of *K. brevis* blooms (http://www.csc.noaa.gov/crs/habf/). Satellite imagery is used to identify areas that have undergone rapid changes in chlorophyll concentrations, usually due to high growth, aggregation, or resuspension.

Because such temporal changes can also be caused by blooms of non-toxic phytoplankton species, suspected areas of *K. brevis* red tide must be confirmed with *in situ* measurements. Following this confirmation, short-term predictions of bloom transport and landfall can be computed using meteorologic forecasts to compute estimates of wind-driven surface currents. The NOAA “HAB Bulletin” (http://coastwatch.noaa.gov/hab/bulletins_ms.htm) and the Florida Fish and Wildlife Research Institute Bulletin (http://research.myfwc.com/) include up-to-date analyses of the current situation, predictions, and supporting data to help managers decide where to obtain their next samples and how to prepare for these blooms.

Paralytic shellfish poisoning (PSP)

Paralytic shellfish poisoning (PSP) has been known in the Pacific Northwest of the United States for centuries (Kao, 1993). Human poisonings have been recorded primarily in North America and Europe (Prakash et al., 1971), but outbreaks have been reported elsewhere (Anderson et al., 1996; Kao, 1993; Popkiss et al., 1979). PSP occurs in people who have eaten bivalve shellfish (i.e., clams, mussels, scallops, etc.) contaminated with one or more of a group of structurally related congeners of saxitoxin (Halstead and Shantz, 1984).

The PSP toxins are produced by dinoflagellates of the genera *Gymnodinium* (Anderson et al., 1989), *Alexandrium* (Anderson et al., 1996), and *Pyrodinium* (Halstead and Shantz, 1984). These toxins act selectively to block the voltage-gated sodium channel of excitable membranes, thus blocking the generation and propagation of action potentials in nerve axons and skeletal muscle fibers (Kao, 1993). Mammals, birds, and fish can be affected by PSP toxins; however, humans are the most sensitive—the fatal oral dose of saxitoxin is 1–4 mg (Baden et al., 1995).

The onset of PSP symptoms usually occurs within 30 minutes to 3 hours. The initial symptoms of PSP are paresthesias and numbness around the lips and mouth (see Kao, 1993). These sensations then spread to the face and neck. Victims may also experience nausea and vomiting. In moderately severe poisonings, the paresthesias progresses to the arms and legs. Victims may experience giddiness, incoherent speech, and light-headedness. In severe poisonings, death can result from respiratory failure and hypoxia. The fatality rate from PSP varies from no deaths in recent outbreaks in the United States or Europe to rates of 2–14 percent in other parts of the world (Kao, 1993). The frequency of mortality is primarily related to the availability of emergency hospital care and past experience with PSP outbreaks as well as possibly the age of the victim.

The causative organism in New England PSP outbreaks is *Alexandrium fundyense* (Figure 5), whose complex life cycle includes a resting cyst, a phase of vegetative growth, sexual reproduction, and re-encystment (Figure 6, panel A). Observations indicate several salient characteristics of the vegetative cell distributions: patterns of abundance are gulf-wide in geographic scope; the distributions are associated with the Maine Coastal Current; and the center of mass of the distribution is from west to east during the April-to-August growing season (Townsend et al., 2001). This latter aspect is particularly notable given that the coastal current flows in the opposite direction (Figure 6, panel B). A model based on the seasonal mean flow that includes germination, growth, mortality, and nutrient limitation can produce simulations that are qualitatively consistent with the observations (Figure 6, panel C) (McGillicuddy et al., 2005). In general, cells germinated from the major cyst beds in the Bay of Fundy and near Penobscot and Casco Bays (Figure 6, panel D) are advected parallel to the shoreline from east to west in the coastal current. Growth of the vegetative cells is limited primarily by temperature from April through June throughout the gulf, whereas nutrient limitation occurs in July and August in the western gulf. Thus, the seasonal shift in the center of mass of cells from west to east can be explained by changing growth conditions: growth is more rapid in the western gulf early in the season because of warmer temperatures, whereas growth is more rapid in the eastern gulf later in the season because of severe nutrient limitation in the western gulf during that time period. Hydrodynamic transport of these offshore populations to inshore shellfish beds is a key aspect regulating the PSP threat to human health (Luerssen et al., 2005).

Ciguatera Fish Poisoning (CFP)

Ciguatera fish poisoning (CFP) outbreaks typically occur within a circumglobal belt extending in latitude approximately from 35°N to 34°S (Hessel et al., 1960), and most reported U.S. cases occur in Hawaii, southern Florida, or after travel to the Caribbean (Glaziou and Legrand, 1994). The unicellular marine dinoflagellate, *Gambierdiscus toxicus*, found associated with coral reefs (Legrand et al., 1992), is the origin of ciguatoxins, a group of lipid-soluble polyether compounds. The CFP toxins are passed through the coral reef food web to accumulate in large herbivorous and carnivorous fish (Lange, 1987). When caught, the fish appear to be healthy and have a normal taste and appearance. In addition, the toxicity of one fish does not predict the toxicity of other fish caught in the same geographic area.

CFP is clinically characterized by gastrointestinal effects (appearing a few hours after eating the fish) accompanied or followed by neurologic, and occasionally by cardiovascular, symptoms (e.g., bradycardia, hypotension) (Glaziou and Legrand, 1994). Sensory disturbances or paresthesias (such as numbness of the mouth and extremities and reversal of temperature sensation) and sometimes a generalized rash are distinctive features of CFP. The gastrointestinal symptoms usually persist for only a few days, whereas the neurologic symptoms may persist for up to several months (Quod and Turquet, 1995; Blythe et al., 1994; Glaziou and Legrand, 1994).

All the symptoms of CFP are reportedly more common in people suffering from a second or subsequent poisoning (Bagnis et al., 1979; Glaziou and Martin, 1993), and symptoms may

recur when victims eat fish or nuts or drink alcohol or caffeinated beverages (see Baden et al., 1995 for review). CFP is pleiomorphic with subjective symptoms without easily available objective measures of health effects, making this disease one of the most challenging to diagnose (Pearn, 1994).

G. toxicus and other ciguatera-associated dinoflagellates generally occupy either benthic or epiphytic niches (Tindall and Morton, 1998). As such, they are not as strongly affected by the oceanographic processes that planktonic forms encounter. However, their growth rates depend on temperature and salinity, thus making their population dynamics sensitive to the ambient fluid environment. Hales et al. (1999) observed strong positive correlations between the annual incidence of CFP and local warming of the sea surface in a group of Pacific Islands that experienced heating during El Niño Southern Oscillation events. At another group of islands that experienced cooling of the sea surface during El Niño events, the opposite was observed. Several mechanisms were offered as explanations for this observed relationship between elevated temperatures and increased CFP incidence, including coral bleaching and disease.

As pointed out by Yasumoto et al. (1980), dead coral surfaces are rapidly colonized by filamentous or calcareous macroalgae, the preferred substrate of CFP-associated dinoflagellates. Physical disturbances of coral reefs (e.g., harbor construction) has also been associated with increased *G. toxicus* abundance (Lewis, 1986) and outbreaks of CFP (Ruff, 1989; de Sylva, 1994). Several other natural and anthropogenic influences can lead to degradation of the reef environment as well, including tourism, eutrophication, sewage and freshwater runoff, sedimentation due to erosion or dredging, and ship groundings (Lehane and Lewis, 2000). As a result, some have argued that CFP may be one of our most sensitive indicators of environmental disturbance in tropical marine ecosystems (Hales et al., 1999), although at present determining whether environmental degradation and change in tropical regions are affecting the incidence or severity of CFP is not possible.

Illnesses from Cyanobacterial Toxins

In many areas of the world (e.g., Australia, United States), the HABs with potentially the greatest public health impact are cyanobacteria blooms in drinking-water sources and recreational waters (both marine and freshwater) (see review by Backer, 2002). The primary toxin-producing cyanobacteria genera include: *Anabena, Aphanizomenon, Cylindrospermopsis*, *Nodularia, Planktothrix* (*Oscillatoria*), and *Microcystis* (Figure 7).

The neurotoxic cyanobacterial toxins include alkaloids (anatoxin-a and saxitoxins and neo-saxitoxin) and an organophosphate (anatoxin-a[s]) (Falconer, 1993). Anatoxin-a (from *Anabena* spp.) is a post-synaptic depolarizing neuromuscular blocker (Carmichael et al., 1990). Anatoxin-a(s) (also from *Anabena* spp.) is a potent anticholinesterase inhibitor similar in function to synthetic organophosphate insecticides. Saxitoxins (produced by some strains of *Anabena* and *Aphanizomenon*) are also produced by some marine dinoflagellates and can cause PSP.

The hepatotoxic cyanobacterial toxins are cyclic or ringed peptides (Carmichael and Falconer, 1993). Those with seven amino acids are microcystins (produced by some species

and strains of *Anabena*, *Microcystis*, and *Planktothrix*). The peptides with five amino acids are nodularins (produced by *Nodularin spumigena*). These toxins inhibit specific protein phosphatase enzymes that are common to all eukaryotic cells (Falconer, 1993). Laboratory animal studies indicate that microcystins are tumor-promotors (Nishiwaki-Matshushima et al., 1992) and teratogens (Falconer et al., 1988). An additional hepatotoxin, cylindrospermopsin, has been isolated from *Cylindrospermopsis raciborskii* (Hawkins et al., 1997). Cylindrospermopsin is a potent protein synthesis inhibitor and causes chromosome breakage and loss *in vitro* (Humpage et al., 2000).

Animals are more frequently and more seriously poisoned than humans because animals are more likely to drink or swim in water that humans avoid because of foul taste or smell (Senior, 1960). Also, dogs have died from nodularin or anatoxin-a poisoning after licking blue-green scum from their coats (Codd et al., 1992).

Humans may be exposed to cyanobacterial toxins through drinking and recreational water (Carmichael and Falconer, 1993), either by direct contact or accidental uptake by swallowing water or inhaling aerosols during recreational and occupational activities. The use of untreated water sources for irrigation and lawn watering may put humans at risk for exposure through aerosols containing cells or toxins. The Florida Department of Health has reported both respiratory (e.g., irritation and shortness of breath) and dermatologic (e.g., itchy skin, rashes) symptoms in people occupationally exposed to an extensive bloom of *Microcystis aeruginosa* on the St. Johns River during the summer of 2005 (Andrew Reich, Florida Department of Health, personal communication, September 2005).

Extraordinary blooms of *Microcystis* have taken place in the Great Lakes recently (Figure 8). In August 2003, a massive bloom of the cyanobacterium *M. aeruginosa* formed in western Lake Erie, and persisted for nearly a month. Surface scums of *Microcystis* containing high concentrations of the toxin microcystin washed ashore in Michigan and Ohio, resulting in foul-smelling, rotting algal mats. Beaches and recreational boating areas were rendered unusable, and sport fishing was adversely affected. The *Microcystis* bloom of 2003 was perhaps the most severe in Lake Erie's recent history, but it was only the latest in a trend towards increasing frequency of *Microcystis* blooms in the last decade (Bridgeman, 2005; see http://www.lakeerie.utoledo.edu/html/tomres1.htm). Understanding the dynamics of these blooms is a focus of the National Oceanic and Atmospheric Administration (NOAA) Center of Excellence for Great Lakes and Human Health (http://www.glerl.noaa.gov/res/Centers/HumanHealth/). Given the clear evidence for substantial transport of these blooms via coastal currents, numerical models are being developed in which cyanobacterial dynamics are coupled to hydrodynamics with the aim of eventually transitioning such models to ecologic forecasting applications.

Summary

HABs represent one component of the inexorable connection between oceans and human health. As more people choose to make their permanent homes along marine and freshwater coastlines, and the popularity of water-based recreational activities increases, threats from HAB-related organisms and their toxins will increase. Monitoring programs have

successfully limited human exposure to, and illness from, brevetoxins and saxitoxins in seafood. However, new threats appear to be emerging, including increases in CFP in the wake of coral reef degradation, the occurrence of organisms in geographic areas where they had not been found before, and increases in the intensity and frequency of blooms. Partnership between oceanographers and public health practitioners is critical for a more complete understanding of how HABs affect human health and for addressing these emerging threats.

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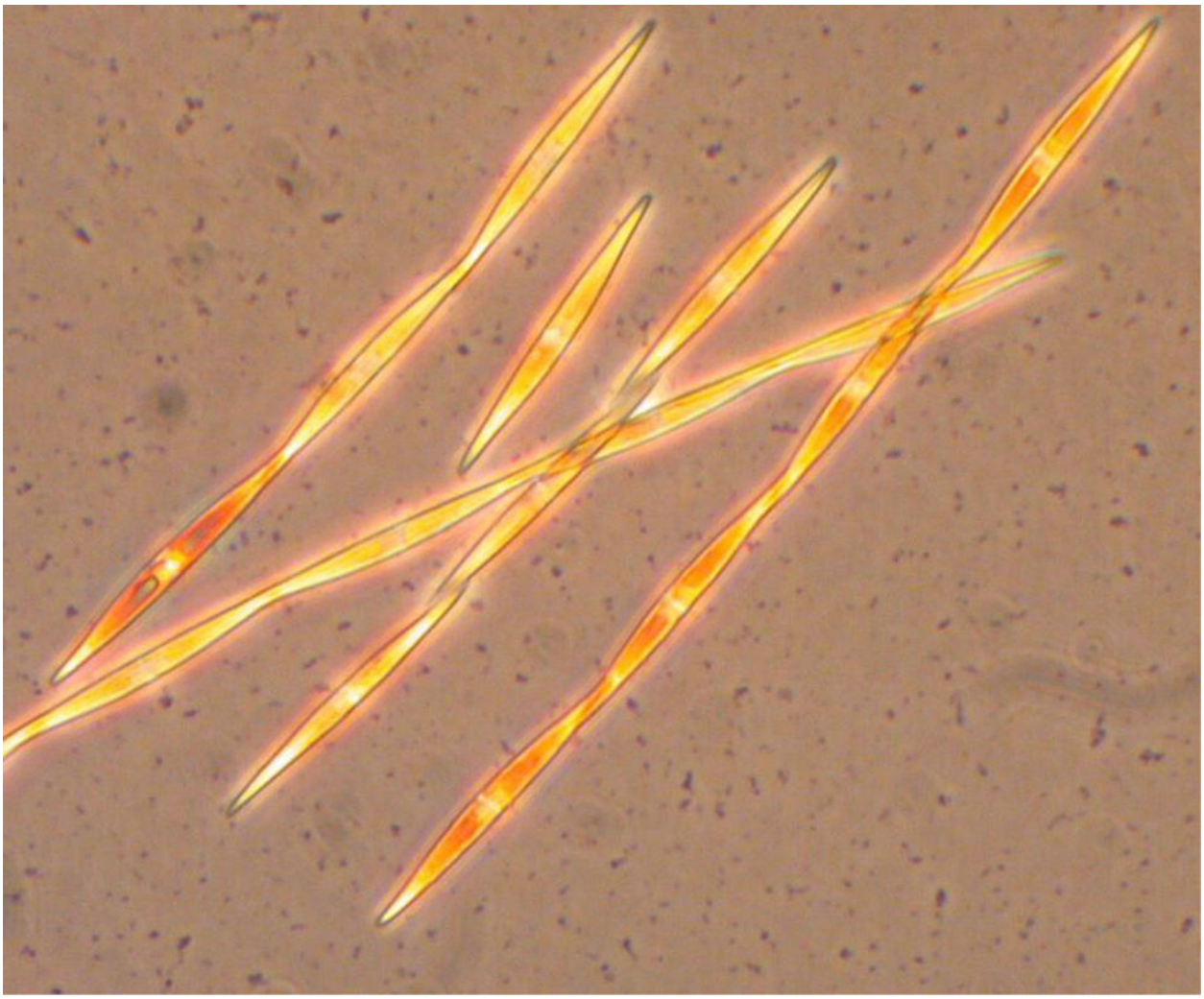

Figure 1.
Pseudo-nitzschia sp., a diatom, is the source of domoic acid, which causes amnesiac shellfish poisoning. Image provided by Brian Bill (Northwest Fisheries Science Center, Seattle, WA).

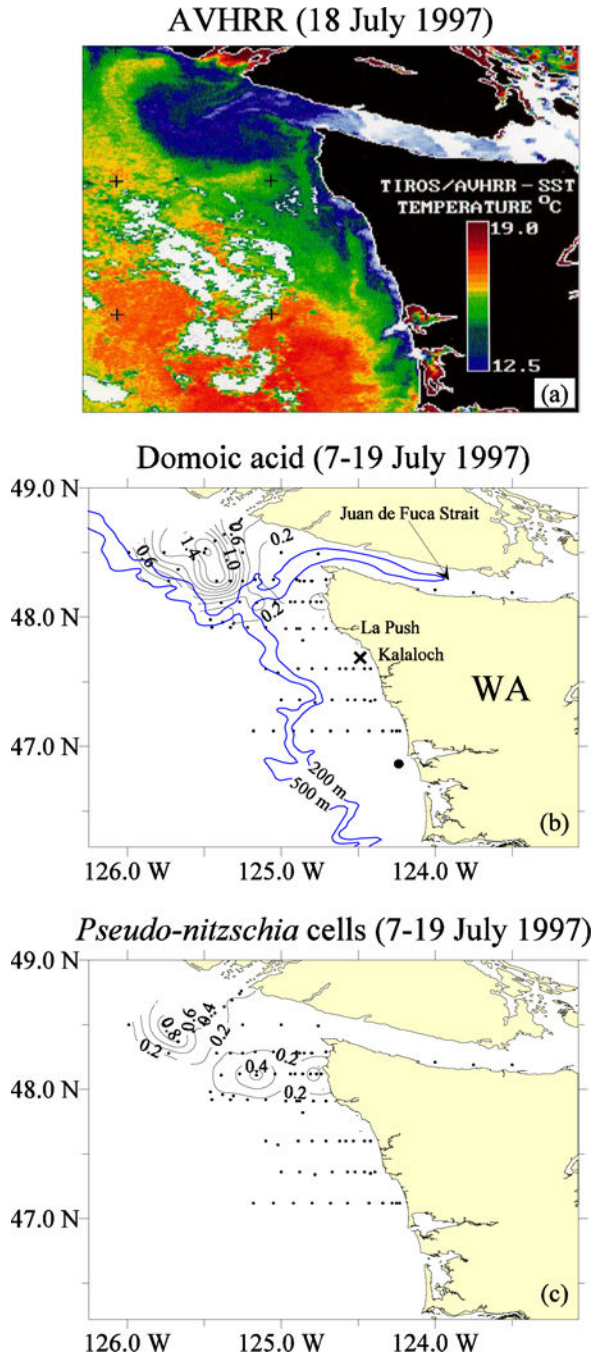

Figure 2.

Satellite-derived sea surface temperature (SST), particulate domoic acid (μg/L) and total *Pseudo-nitzschia* cell numbers (10^6 cells/L) in surface seawater July 1997 (modified from Trainer et al., 2002). Spatial patterns show a coincidence of colder temperature (dark blue in upper panel), higher domoic acid, and greater numbers of *Pseudo-nitzschia* cells offshore of the Juan de Fuca Strait. The colder offshore water is indicative of the Juan de Fuca eddy. Colder water along the Washington coast is indicative of local wind-driven upwelling. Source: http://www.ecohabpnw.org/overview.htm

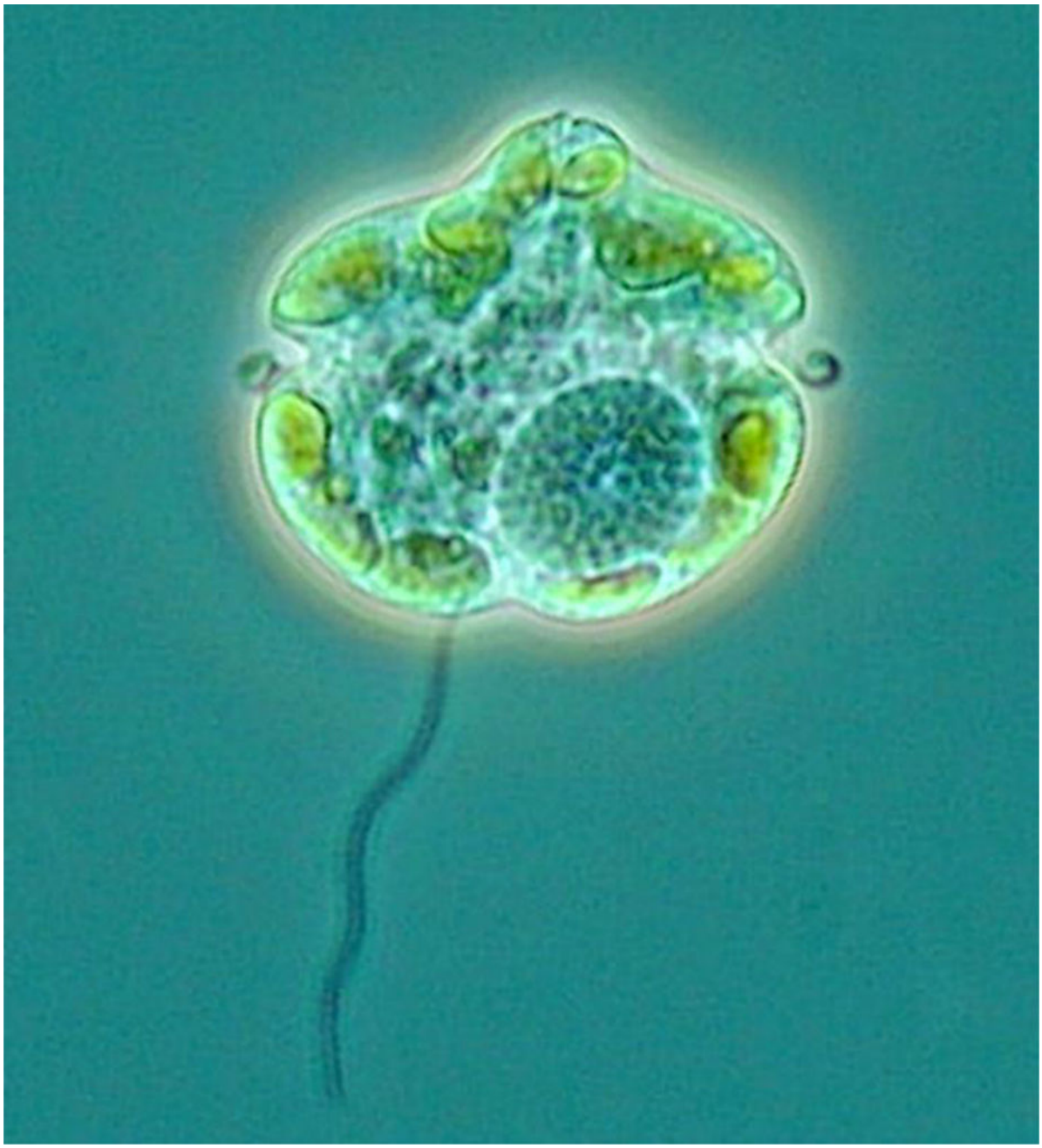

Figure 3.

Upper panel: image of *K. brevis* taken by David Patterson (Marine Biological Laboratory, Woods Hole, MA) and provided by micro*scope (http://microscope.mbl.edu). Lower panel: Aerial view of a *K. brevis* bloom along a Florida beach, photo by Paul Schmidt, *Charlotte Sun*.

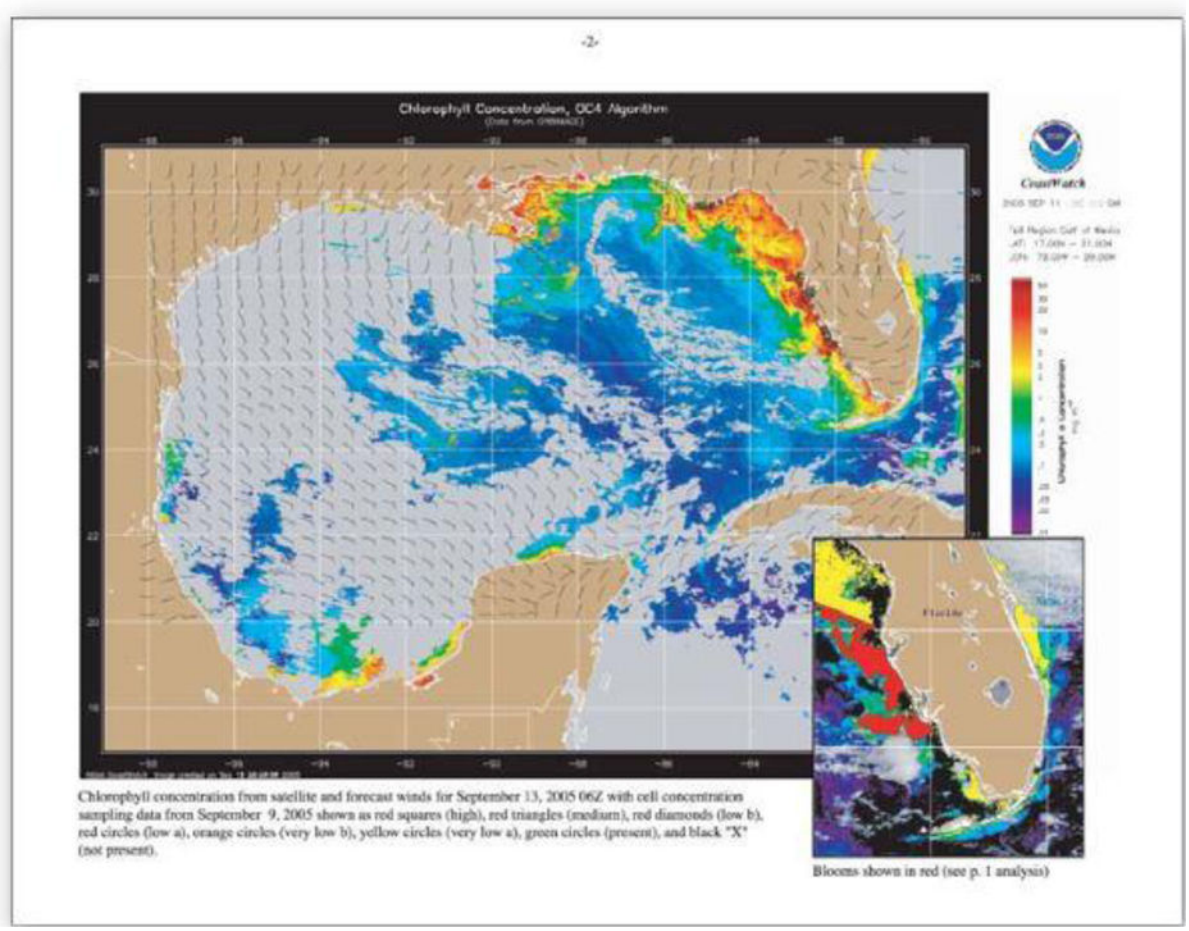

Figure 4.

Sample issue of NOAA's Gulf of Mexico HAB bulletin, illustrating identification and tracking of *K. brevis* blooms using ocean color imagery (see http://www.csc.noaa.gov/crs/habf/).

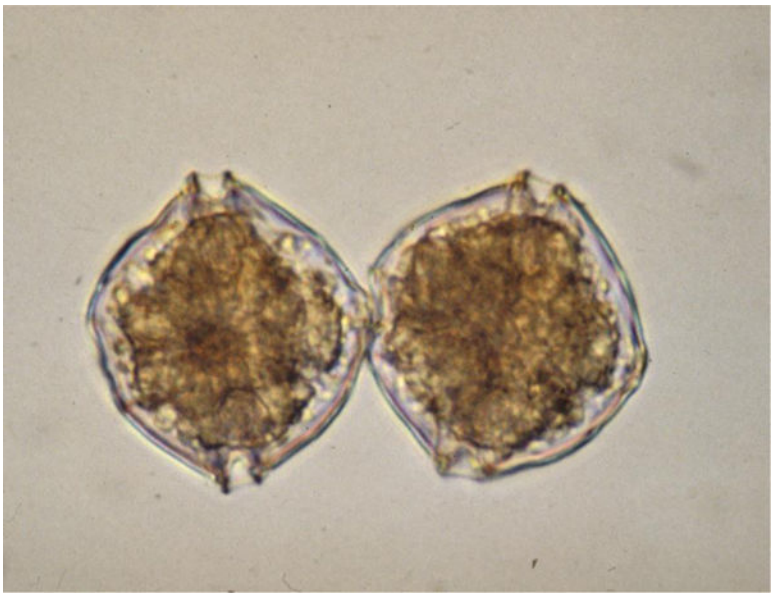

Figure 5.
Photograph of two *A. fundyense* cells side-by-side. Image from D. Wall (ret.) provided courtesy of D. Anderson (Woods Hole Oceanographic Institution).

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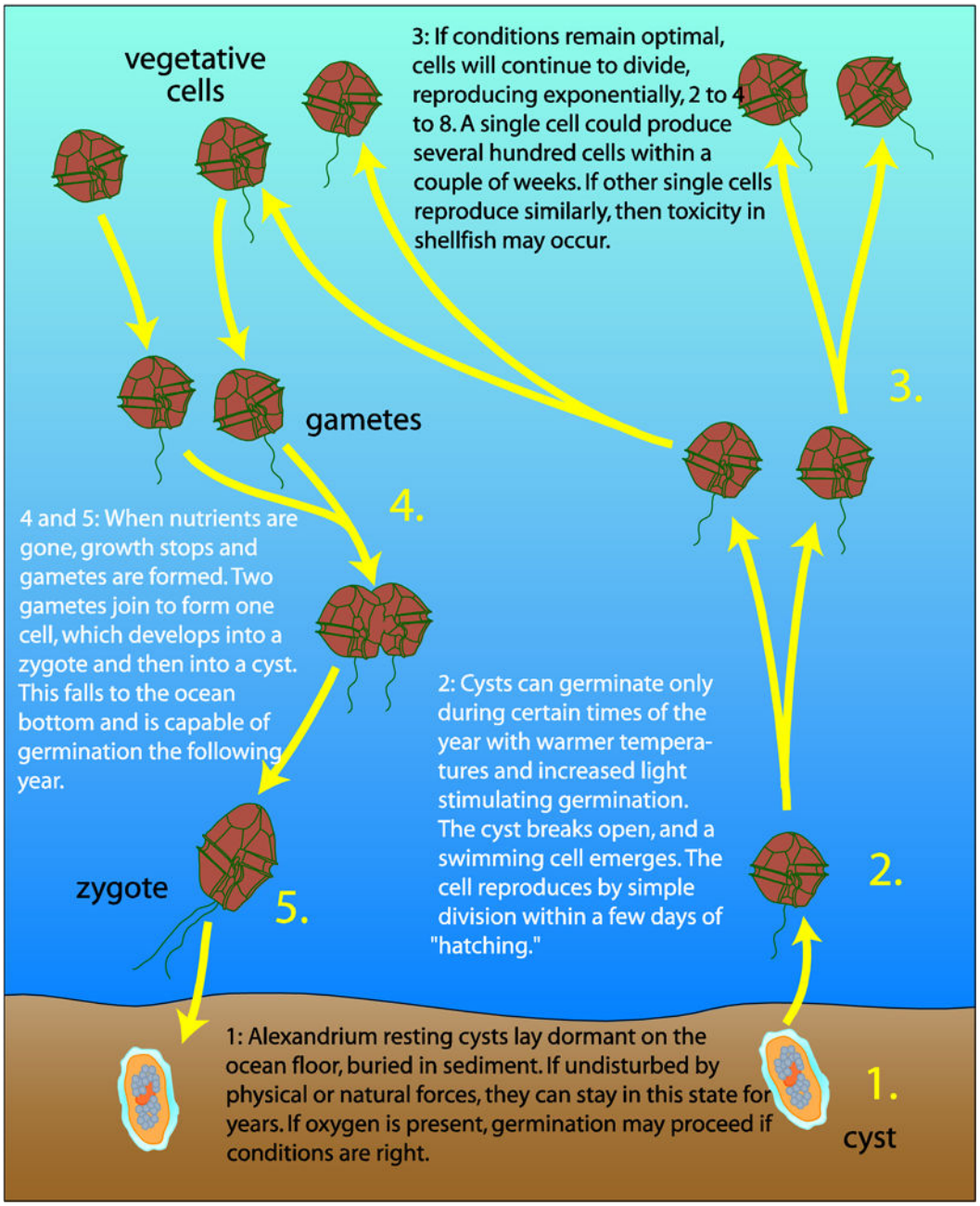

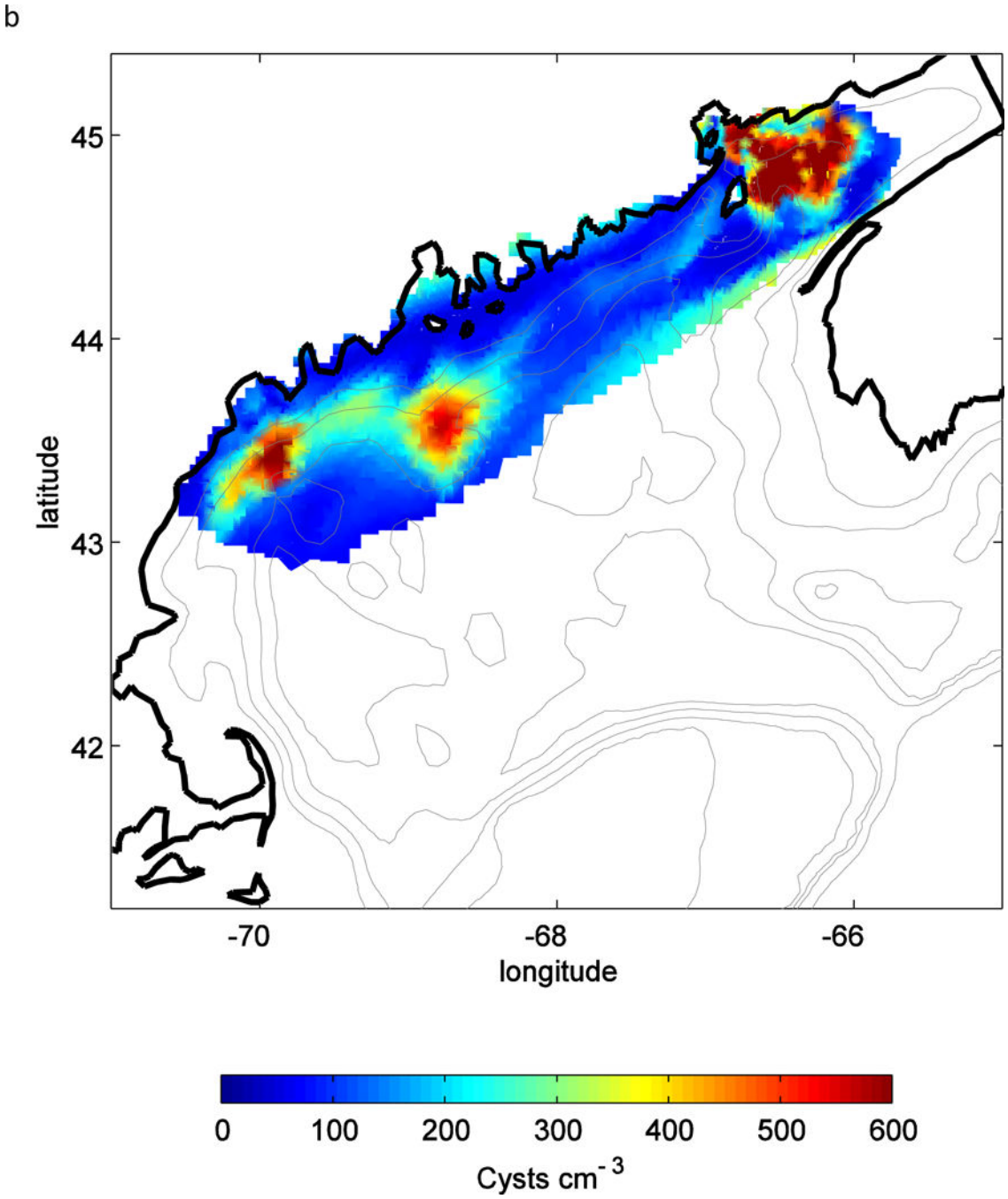

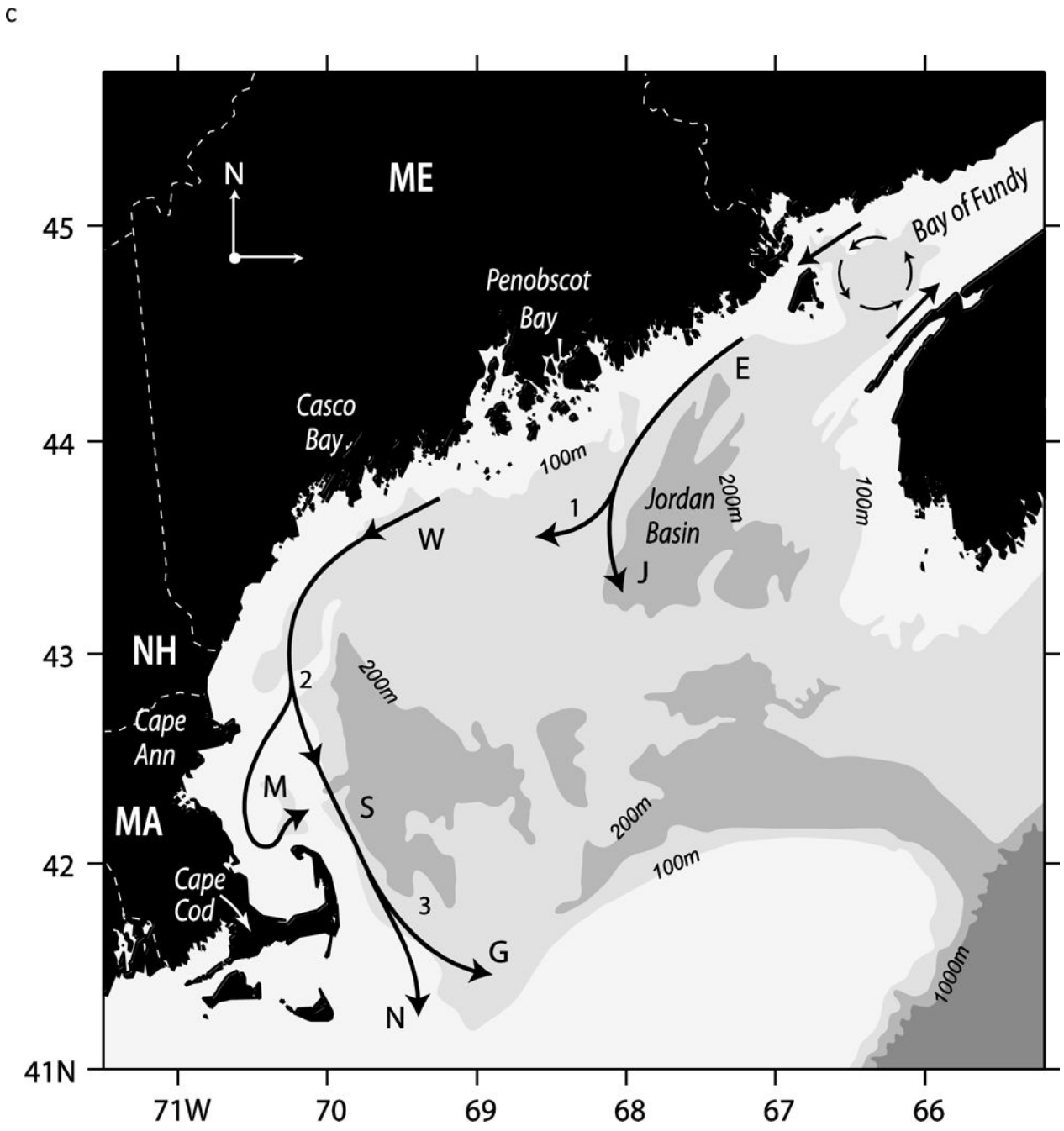

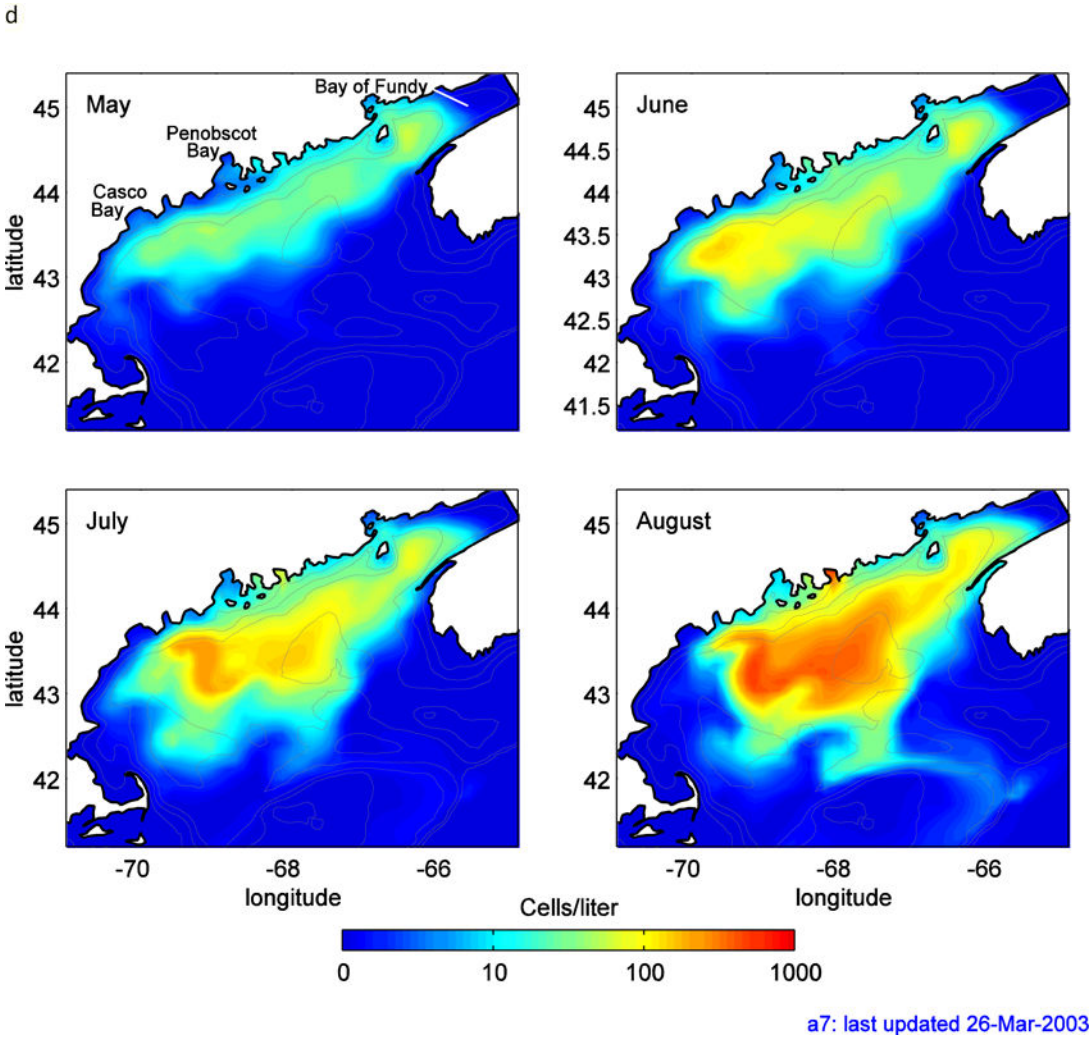

Figure 6.

Upper left: life cycle of *A. fundyense*. Upper middle: Distribution of cysts (number of cysts cm^{-3}) in the upper 3 cm of sediment derived from a 1997 survey of the Gulf of Maine (Anderson et al., 2005) and surveys of the Bay of Fundy in 1981 (White and Lewis, 1982), 1982 and 1983 (data provided by Jennifer Martin, Fisheries and Oceans Canada). Upper right: Schematic of the Maine Coastal Current, reprinted from McGillicuddy et al. (2005). Branch points are located at (1) Penobscot Bay, (2) Cape Ann, and (3) Great South Channel. Seven segments of the current are indicated: (E)astern, (J)ordan, (W)estern, (M)assachusetts, (S)tellwagen, (N)antucket, and (G)eorges Bank. Dashed white lines denote state boundaries of Maine (ME), New Hampshire (NH), and Massachusetts (MA). Lower panel: simulated *A. fundyense* distribution under climatological forcing conditions.

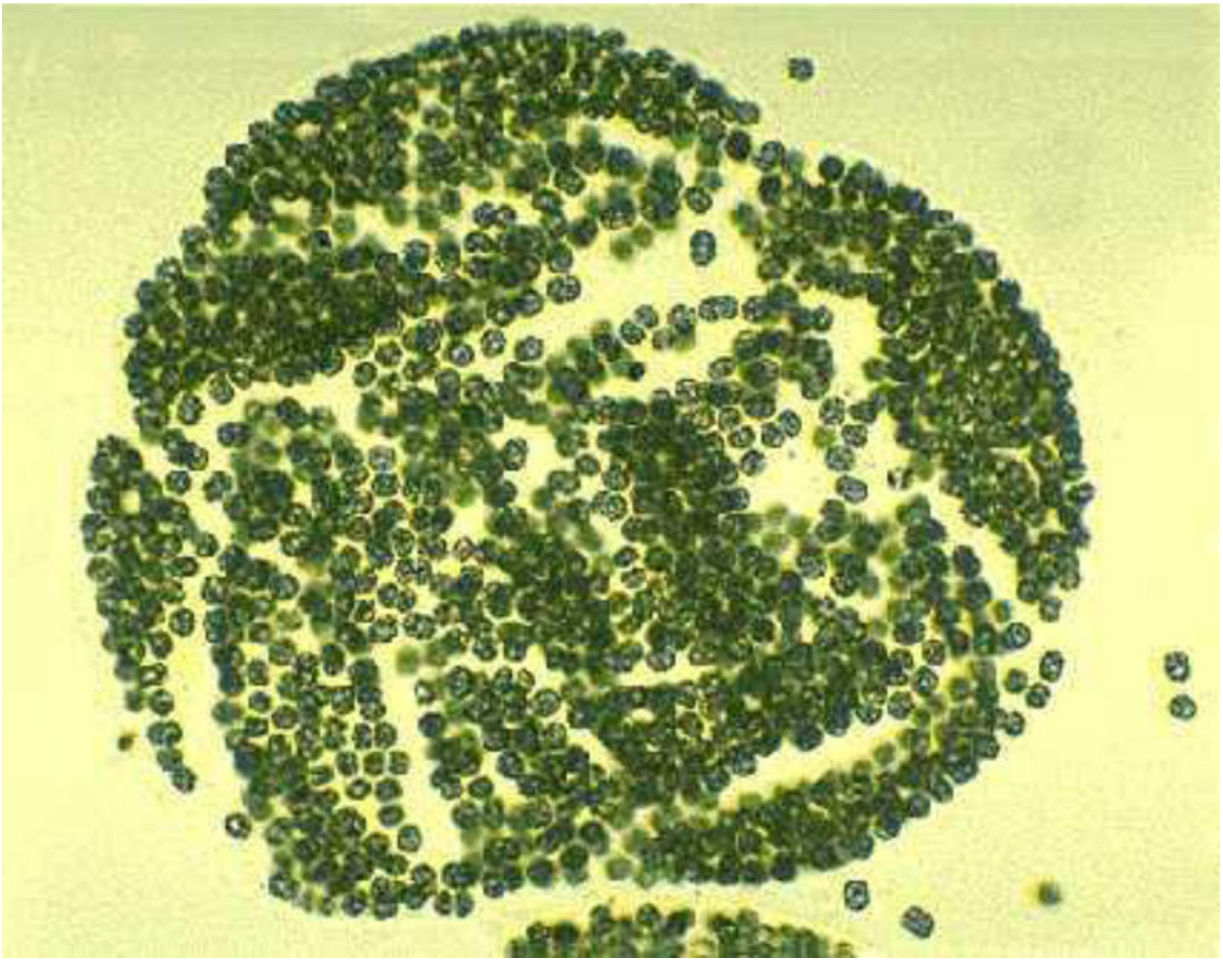

Figure 7.
Microcystis sp. image courtesy of Isao Inouye (University of Tsukuba), Mark Schneegurt (Wichita State University), and Cyanosite (www-cyanosite.bio.purdue.edu).

Figure 8.
Microcystis bloom August 2003. Top: Landsat 7 photo of western Lake Erie provided by OhioView/T. Bridgeman. Bottom: *Microcystis* “shake” from a surface sample, photo courtesy of T. Bridgeman.