Limnol. Oceanogr., 42(5, part 2), 1997, 1009–1022

Bloom dynamics of toxic *Alexandrium* species in the northeastern U.S.

Donald M. Anderson
Department of Biology, Woods Hole Oceanographic Institution, Woods Hole, Massachusetts 02543

Abstract

Coastal waters of the northeastern U.S. are subject to recurrent outbreaks of paralytic shellfish poisoning (PSP) caused by toxic dinoflagellates in the genus *Alexandrium*. PSP is not uniform across the large region, but instead reflects *Alexandrium* growth and toxin accumulation in five separate habitats or zones defined by circulation patterns and the discontinuous distribution of the dinoflagellates. Each of these habitats has a unique set of environmental and oceanographic forcings that determine the timing and extent of bloom development and transport and that regulate the extent of genetic exchange with adjacent populations. Several habitats (e.g. the southwestern Gulf of Maine, Massachusetts Bay, and Georges Bank) are linked hydrographically and may share the same *Alexandrium* population via large-scale transport in a coastal current, whereas the other two habitats (eastern Maine and southern salt ponds-embayments) seem to be isolated and have little or no hydrographic or genetic linkage to adjacent regions during bloom seasons. My paper provides an overview of the regional ecology and oceanography of *Alexandrium* through a focus on these five subpopulations. Issues that relate to PSP and *Alexandrium* dynamics throughout the world are highlighted, including species dispersal, the role of cysts and "initiation zones" in bloom development, and the influence of large- and small-scale hydrography on population development and transport. The ability of *Alexandrium* to colonize multiple habitats and to persist over a large region is emphasized in recognition of the adaptability and resilience of this important organism.

In many parts of the world, paralytic shellfish poisoning (PSP) is a recurrent and serious problem associated with blooms of toxic dinoflagellates in the genus *Alexandrium*. The potent neurotoxins produced by these organisms are accumulated by filter-feeding shellfish and other grazers and are passed on to humans and other animals at higher trophic levels, leading to illness, incapacitation, and even death. *Alexandrium* causes toxicity in many different hydrographic and climatic regimes, from temperate to tropical (e.g. Cembella et al. 1988; La Barbera-Sanchez et al. 1993). This ecological distribution reflects a genetic diversity that is far more extensive than simple morphology might imply. Scholin et al. (1994, 1995) clearly demonstrated that even though a single morphospecies such as *Alexandrium tamarense* can be found in widely different geographic zones, isolates from those different regions have unique genotypes based on ribosomal RNA sequences.

Acknowledgments

Special thanks to D. M. Kulis and B. A. Keafer for many years of field and laboratory assistance underlying much of the work presented here. Thanks also to J. Hurst and D. Whitaker for providing shellfish toxicity data.

This research was supported by grants from the National Science Foundation (OCE 94-15536), the National Oceanic and Atmospheric Administration (NA36RM0190) through the Gulf of Maine Regional Marine Research Program, the National Sea Grant College Program Office (NA90-AA-D-SG480; WHOI Sea Grant Project R/B-121), and the Massachusetts Water Resources Authority.

Contribution 9389 from the Woods Hole Oceanographic Institution.

Another reason for growth success across such a variety of habitats is that many *Alexandrium* species have a cyst stage in their life histories. This allows the organism to remain dormant in bottom sediments through temperature extremes, with seasonal germination inoculating vegetative cells into the water column only during intervals where temperature and light are suitable for growth. Population development is thus possible in more locations than would otherwise be the case if year-round persistence in the plankton were the only means for survival.

PSP is a relatively new phenomenon in the northeastern United States (*see below*), but is now recurrent and widespread, affecting vast expanses of the Gulf of Maine coastline (Anderson et al. 1982). PSP outbreaks and the causative *Alexandrium* blooms are not uniform throughout the region, however. Rather, there are several discrete hydrographic regimes or habitats that support *Alexandrium* blooms, each with a unique set of environmental and oceanographic forcings. Genetic exchange across these boundaries varies—some habitats are virtually isolated, whereas others exchange cells frequently. In all of these habitats, *Alexandrium* blooms are seasonal and recurrent in nature. Given the temperate climate of the region and the general absence of *Alexandrium* cells and toxins during winter (e.g. Anderson and Morel 1979; Shumway et al. 1988), blooms are heavily dependant on benthic cysts for re-inoculation. The mechanisms of bloom initiation and the quantitative importance of cyst seedbeds differ among habitats, however, and toxicity episodes are variable with respect to magnitude, frequency, duration, and type (Anderson et al. 1994).

Given this diversity and complexity and more than two decades of research and observations, a comprehensive overview of the biogeography and bloom dynamics of toxic *Alexandrium* spp. in New England is overdue. This paper attempts to provide that synthesis, focusing on the western Gulf of Maine and waters to the immediate south. Though restricted in geographic coverage, this overview highlights many issues involved in PSP and *Alexandrium* dynamics throughout the world. Topics such as species dispersal, historical increases in bloom impacts, and the effect of human activities on the blooms and patterns of toxicity are relevant to many areas worldwide. Furthermore, the segregation of *Alexandrium* populations into several different habitats or hydrographic systems provides insights into the manner in which this versatile organism adapts to different environmental and oceanographic systems. Of special importance are differences in the roles that cysts can play in bloom dynamics, the mechanisms controlling bloom initiation, and the impact of large- and small-scale hydrography on population dynamics.

History and geographic extent of PSP

PSP was reported in northeastern Canada more than 100 yr ago (Ganong 1889), but in the New England region, toxicity was restricted to the far eastern sections of Maine near the Canadian border, with the first documented PSP in 1958 (Hurst 1975; Shumway et al. 1988). In 1972, a massive, visible red tide of *A. tamarense* stretched from southern Maine through New Hampshire and into Massachusetts, causing shellfish toxicity in southern areas for the first time. Low levels of toxicity had occasionally been reported in southwestern Maine west of Penobscot Bay prior to 1972 (Shumway et al. 1988), but present-day outbreaks in that area are far more numerous and involve much higher levels of toxicity. Virtually every year since the 1972 outbreak, Maine, New Hampshire, and Massachusetts have experienced PSP outbreaks—a direct result of *Alexandrium* cysts being retained in southern waters once introduced there by the massive bloom (Anderson and Wall 1978). In 1979, mussel beds were closed in Narragansett Bay, Rhode Island, when PSP was detected, but no causative organism was ever identified. In subsequent years, PSP toxicity and *A. tamarense* cells and cysts were documented in small embayments in Connecticut and Long Island (Anderson et al. 1982; Schrey et al. 1984). Cysts and motile cells of *A. tamarense* have also been reported as far south as New Jersey (Cohn et al. 1988), but no PSP toxicity has ever been detected that far south. Recent history is thus suggestive of a gradual, southward dispersal of toxic *Alexandrium* species in the northeastern U.S. over the last several decades. This is certainly true with respect to the effects of the 1972 bloom, but some of the spreading can also be attributed to the detection of indigenous, low-toxicity populations in southern waters (Anderson et al. 1982, 1994).

Gulf of Maine circulation

The patterns of PSP within the region must be viewed in the context of the large- and small-scale hydrographic characteristics of the Gulf of Maine and adjacent waters. Circulation in the gulf (Fig. 1) tends to be counterclockwise (Bigelow 1927; Brooks 1985), with southwestward flow along the coast of Maine toward Massachusetts Bay. The regional circulation is principally driven by the buoyancy of freshwater entering from the Scotian shelf, overlying deep, salty slope water entering the gulf through the Northeast Channel. These inputs establish the gradients necessary to drive the large-scale circulation. Superimposed on this southwestward flow are episodic pulses of freshwater from the rivers entering the gulf, producing plumes that extend southwestward along the coast and sometimes into Massachusetts Bay (Butman 1975; Franks and Anderson 1992*a*; Geyer et al. 1992). Freshwater input, Coriolis acceleration, wind stress, and advection by the ambient coastal flow are all important factors influencing the structure and advection speed of the plumes. Of these factors, wind stress seems to be particularly important in determining plume variability (Franks and Anderson 1992*a*).

Regional subpopulations

Toxic *Alexandrium* cells are distributed from the northern waters of the study area (Fig. 1) south to New Jersey. Thus far, only two morphospecies have been linked to the production of PSP toxins within this large region—*A. tamarense* (which possesses a ventral pore on its theca) and *A. fundyense* (which has no pore; Anderson et al. 1994). *Alexandrium ostenfeldii* has been observed (unpubl. data) but never cultured or tested for toxicity. Within the Gulf of Maine, all species examined to date have been *A. fundyense,* whereas *A. tamarense* and *A. fundyense* are both found in southern waters. For convenience, the genus name *Alexandrium* will often be used hereafter to refer to toxic cells from areas where the species designation is unknown or where the two species are interspersed.

The *Alexandrium* distribution is far from uniform across the area depicted in Fig. 1. Cells are abundant and broadly dispersed in the coastal and estuarine waters along the northern shore of the gulf from the Bay of Fundy south and west to Massachusetts Bay; PSP is correspondingly widespread (Shumway et al. 1988; Franks and Anderson 1992*b*). Toxic *Alexandrium* species occur farther to the north in the Bay of Fundy and in the Gulf of St. Lawrence area (e.g. Cembella et al. 1988), but those regions lie outside the geographic domain of this review. Proceeding south from Massachusetts Bay, populations become patchy and isolated between Cape Cod, Connecticut, and Long Island (Anderson et al. 1982; Schrey et al. 1984) and are even more rare in New Jersey (Cohn et al. 1988). New Jersey thus represents the southern biogeographic limit of *A. tamarense* on the east coast of North America.

Within this large area, five different habitats or regions can be identified in which toxic *Alexandrium* blooms occur (Fig. 1, Table 1). These are defined as eastern Maine, western Maine, Massachusetts and Cape Cod Bays, Georges Bank, and southern salt ponds and embayments. Our knowledge of *Alexandrium* dynamics in each of these regions varies greatly as a result of unequal research emphasis and dif-

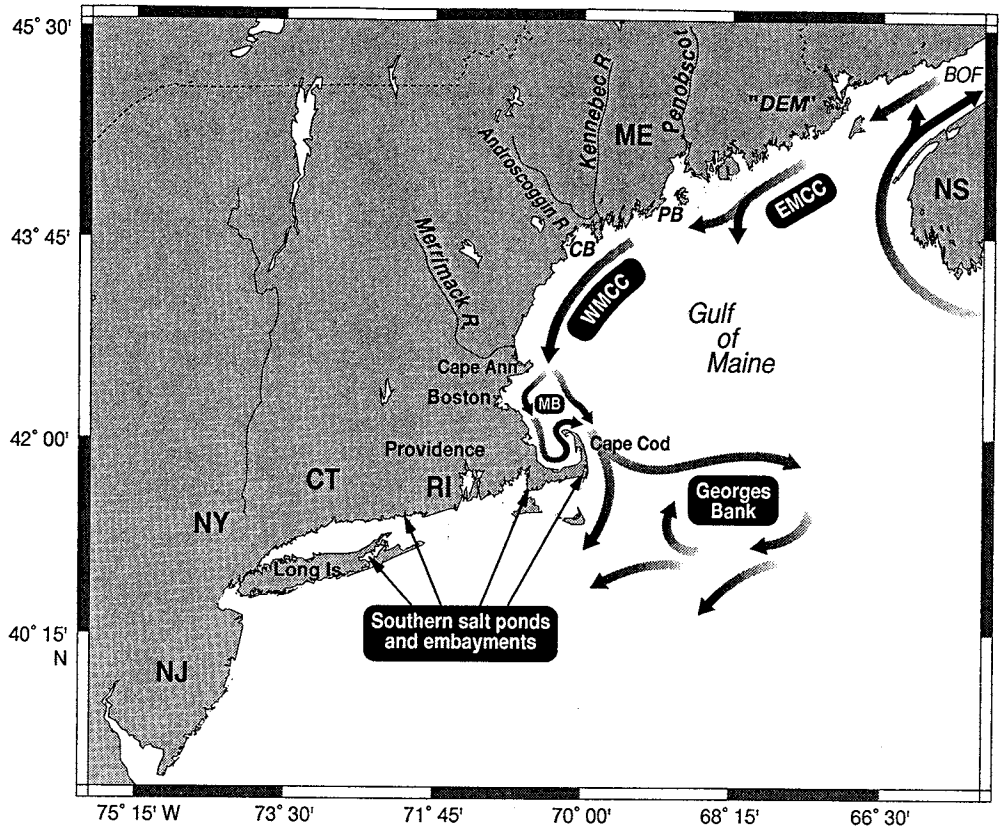

Fig. 1. *Alexandrium* subpopulations or habitats in the northeastern U.S. Five regional populations (black boxes) are identified, defined by circulation patterns and the discontinuous distribution of the dinoflagellate: EMCC—eastern Maine coastal current; WMCC—western Maine coastal current; MB—Massachusetts Bay (includes Cape Cod Bay); Georges Bank; and southern salt ponds and embayments. Qualitative surface circulation patterns are indicated by arrows. Other abbreviations: BOF—Bay of Fundy; DEM—downeast Maine; PB—Penobscot Bay; CB—Casco Bay.

ferent levels of shellfish monitoring. It is nevertheless clear that despite the geographic proximity of these areas, the mechanisms of bloom formation and toxicity development for each vary significantly. One example of the different timing of shellfish toxicity at selected sites in the region is given in Fig. 2. These differences (as well at certain similarities and linkages) are highlighted below, providing an overview of the oceanographic, environmental, and behavioral mechanisms that control *Alexandrium* population dynamics in each system.

Eastern Maine—From the Canadian border south to Penobscot Bay in Maine (traditionally termed "downeast" Maine), PSP is a recurrent problem. Monitoring for PSP toxins was initiated in eastern Maine in 1957 as a result of shellfish toxicity across the border in Canada, and harvesting quarantines due to excessive PSP levels have occurred from that time through to the present day. The timing of the toxicity outbreaks is typically later than for stations in western Maine and other areas to the south, with toxin levels in shellfish often peaking in mid-late summer (Shumway et al. 1988). Figure 2 shows this pattern for 1988, with peak toxicity at Gleason Cove (eastern Maine) in mid-July—a time when toxicity had already peaked and dropped below detection limits at Lumbo's Hole (a station in the western Maine habitat).

Alexandrium cysts have been found at numerous locations along the eastern Maine coast (Thayer et al. 1983), but only nearshore stations were sampled and cyst abundance has never been quantified. No field investigations of *Alexandrium* motile cells have been conducted in the region, and therefore much of what follows with respect to bloom dynamics should be considered speculative.

A dominant hydrographic feature of the eastern Maine region is a nearshore plume of cold surface water that extends along the coast from Grand Manan Island at the mouth of the Bay of Fundy to Mt. Desert Island, just north of Penobscot Bay (Brooks and Townsend 1989). Termed the eastern Maine coastal current, this water mass is important in part because it is a significant transport pathway, but also because it carries nutrient-rich waters from the tidally mixed eastern gulf into the warm and stratified western and central regions. No linkage between this coastal current and *Alexandrium* blooms has yet been established, but it seems likely that this water mass plays a significant role in population growth and transport, and thus in the patterns of shellfish toxicity. One aspect of this linkage is a feature called the "PSP sandwich," a term used by Shumway et al. (1988) to

Table 1. Summary of toxicity patterns and ecological factors controlling *Alexandrium* blooms and PSP outbreaks in five habitats in the northeastern U.S.

Habitat	PSP pattern	Linkage to other habitats	Dominant environmental forcings
Eastern Maine	Recurrent, widespread, often mid–late summer	Limited exchange with western Maine coastal current and other habitats to the south; source populations in Bay of Fundy?	Poorly understood: eastern Maine coastal current likely important; possibly tidal fronts, outflow from St. Johns river as well
Western Maine	Annually recurrent late spring blooms, with summer and fall outbreaks also possible; N–S progression in timing of toxicity	Input of cells from eastern Maine limited; supply of cells to Massachusetts Bay confirmed; linkage to Georges Bank likely, but not confirmed	River discharge, buoyant coastal current, and local wind stress influence plume behavior; underlying Gulf of Maine circulation also influences plume behavior
Massachusetts and Cape Cod Bays	Late spring, early-summer blooms, but outbreaks less frequent than along coast to north	Supply of cells in established blooms from the western Maine habitat confirmed; in situ germination of cysts less likely as significant inoculum; localized growth possible if water residence time in bays increases	Delivery of cells via western Maine coastal current affected by timing of NE winds which push plume into Mass. Bay; residence time of delivered cells within bays determined by volume of runoff and wind stress
Georges Bank	Low level, historical toxicity with occasional high toxicity event	Unknown; low salinity water in western Maine coastal current reaches Georges Bank, but it is unknown whether *Alexandrium* blooms in that current remain intact; localized in situ growth possible; delivery of cysts as possible toxin source	Unknown
Southern salt ponds and embayments	Recurrent, localized outbreaks; point-source pattern to toxicity; generally low levels of toxicity	No linkage; self-sustaining, localized populations of *Alexandrium* with no input from adjacent coastal waters	Limited flushing; topography, hydrography, and cell swimming behavior conducive to motile cell and cyst retention

describe a toxin-free zone at the downstream end of the eastern Maine coastal current near Penobscot Bay. Areas to the immediate east and west have recurrent PSP, but shellfish from the midcoast region are typically nontoxic. Lewis et al. (1979) also found no *Alexandrium* cysts in the Penobscot Bay region. This lack of toxicity at times when adjacent waters are highly toxic is seen in Fig. 2. The most likely explanation for this toxin-free anomaly is that the eastern Maine coastal current deflects away from shore near Penobscot Bay (Brooks and Townsend 1989), which would carry *Alexandrium* cells in that current into offshore waters and away from intertidal shellfish. The western boundary of the eastern Maine *Alexandrium* habitat is thus defined by the point at which the eastern Maine coastal current alters its alongshore path and moves away from shore. Brooks and Townsend (1989) observed that the location of this deflection varies from year to year as a result of the quantity of dense, deep water in nearby Jordan basin. As discussed below, the region to the west of Penobscot Bay seems to function independently with respect to *Alexandrium* populations, having its own source region for cells and a separate coastal current transport pathway.

Tidal mixing is another important oceanographic feature to be considered in the eastern Maine region. Tidal ranges increase from west to east along the northern coast of the gulf, from 2–3 m in the west to 6 m or more in downeast Maine (Townsend et al. 1987). These large tides in the east give rise to high tidal current velocities and thus significant turbulent mixing (Garrett et al. 1978). This mixing may be important with respect to cyst resuspension and bloom initiation, but also because the transition from tidally mixed areas to adjacent thermally stratified waters is often marked by sharp surface fronts (Yentsch and Garfield 1981). Examples of the importance of fronts in dinoflagellate bloom dynamics are many (*see* Franks 1992), so it is reasonable to expect that *Alexandrium* blooms in the region may have a close linkage to the formation, persistence, movement, and breakdown of fronts. Tidally generated fronts in the North Sea are important sites for the accumulation of the toxic dinoflagellate *Gyrodinium aureolum* (Pingree et al. 1975; Simpson et al. 1979). This organism accumulates at tidal fronts and is transported toward shore where contact with fish and other susceptible resources results in massive mortalities (Holligan 1979). Similar frontal accumulation and onshore delivery mechanisms may be responsible for PSP toxicity from *Alexandrium* in the eastern Maine region.

Summarizing, a population of toxic *Alexandrium* cells exists in the eastern Maine region which may have only limited exchange with other populations to the south and west (Table 1). The geographic separation of this population results from

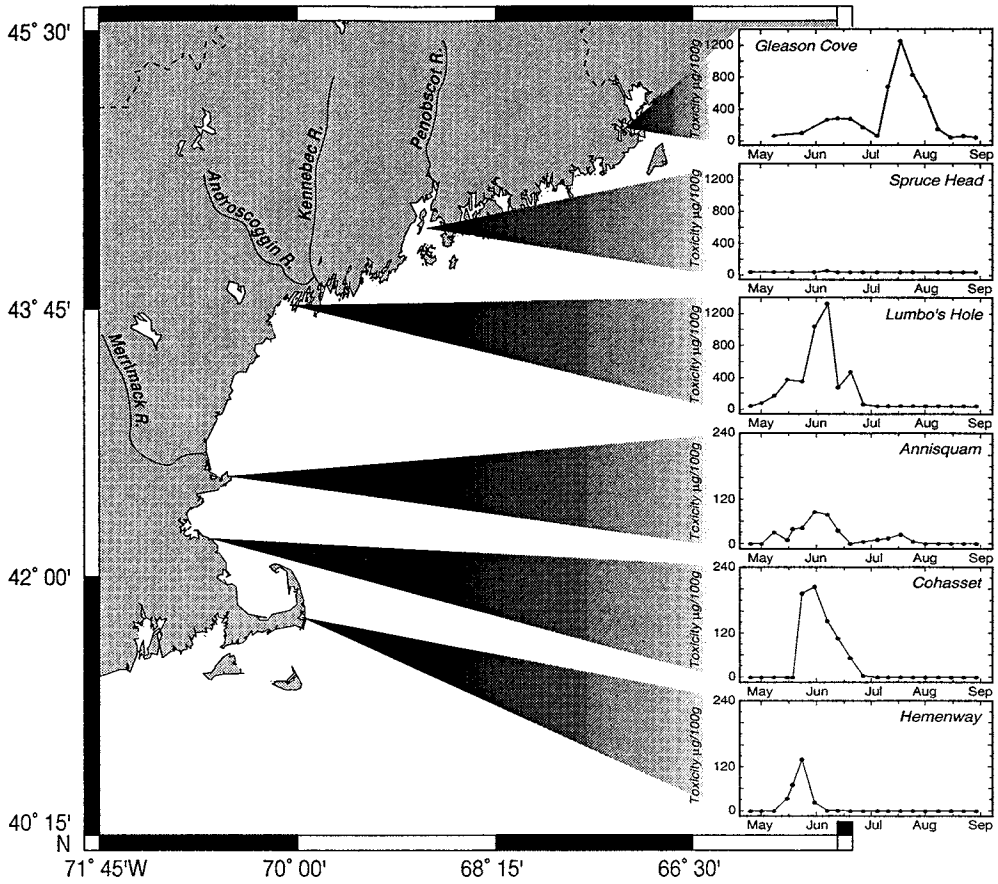

Fig. 2. Shellfish toxicities in mussels (*Mytilus edulis*) from representative PSP stations of the *Alexandrium* habitats in the Gulf of Maine in 1988. Toxicity is often first reported either in southern salt ponds and embayments (e.g. Hemenway) or in western Maine (Lumbo's Hole), followed immediately by outbreaks ~1 week later in northern Massachusetts (Annisquam) and 2 weeks later in Massachusetts Bay (Cohasset). The rapid increase in toxicity at Cohasset suggests delivery of established *Alexandrium* populations from the western Maine coastal current, rather than localized, in situ growth. Later in the bloom season, toxicity peaks in the EMCC areas of downeast Maine (e.g. Gleason Cove), with no subsequent toxicity observed downstream to the west, either in the middle of the PSP sandwich in Penobscot Bay (e.g. Spruce Head) or at western Maine stations. EMCC populations are apparently isolated from habitats to the south and west. (Note: scale changes on several figures, and a switch from mussels to clams on 15 May and then from clams back to mussels on 15 June at the Annisquam station only.) (Modified from Franks 1990.)

the behavior of a strong coastal current which deflects from shore at the western boundary of the region. Some cells carried into the offshore waters by this deflection may travel back toward shore and join the western Maine coastal current and its *Alexandrium* population (*see below*), but most cells are likely to remain in the offshore waters or to be entrained into a counterclockwise gyre over Jordan basin that would bring them back toward the coast in downeast Maine. The source or inoculum for bloom populations could be cells or cysts in the coastal waters, estuaries, and sounds of eastern Maine, or established bloom populations in the Bay of Fundy that are entrained into the coastal current and carried to the southwest. Areas also exist where *Alexandrium* cells would accumulate at tidal fronts at the boundary between the well-mixed nearshore waters and stratified offshore waters, with delivery of those cells to shore, possibly as a result of upwelling (e.g. Seliger et al. 1979). The coastal current and tidal fronts could both be important physical features in *Alexandrium* population dynamics in the region, but considerable field effort is needed to confirm or reject these hypothesized mechanisms.

Western Maine—Within this region, which extends from the western edge of Penobscot Bay to Massachusetts Bay (Fig. 1), a coupling has been inferred between the abundance and distribution of *A. fundyense* and a buoyant coastal current that travels from northeast to southwest. A conceptual model of this linkage has been proposed, termed the "plume advection hypothesis" (Franks and Anderson 1992*a*). Critical features of this model include a source population of cells located to the north of Massachusetts Bay, possibly near the Androscoggin-Kennebec estuary; freshwater outflow from the Androscoggin-Kennebec estuary, resulting in a coastally trapped, buoyant plume (the western Maine coastal current)

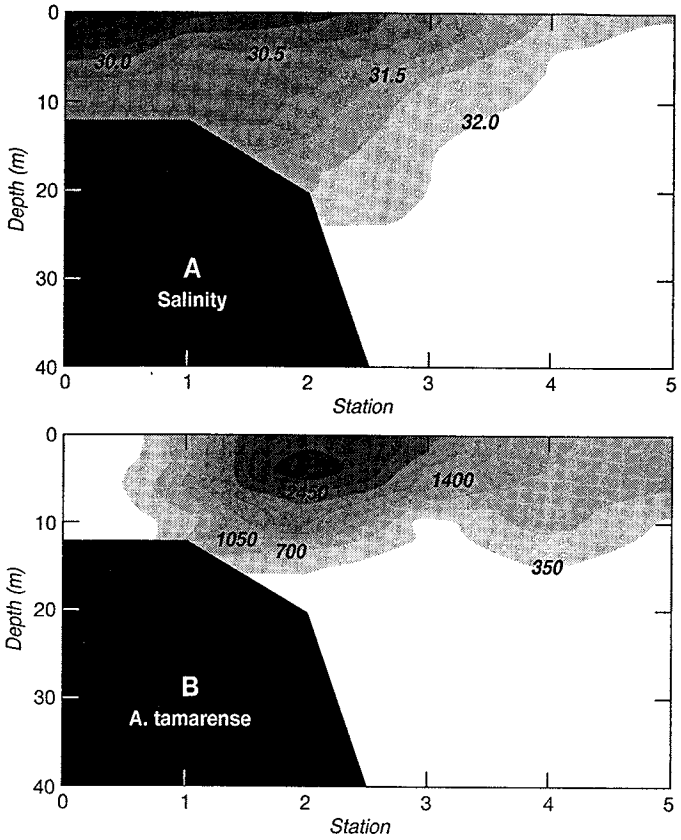

Fig. 3. Hydrographic sections from transect off Portsmouth, New Hampshire, on 27 May 1988, extending 35 km into the Gulf of Maine. A. Section of salinity (psu) showing a cross-section of the less-saline plume or coastal current. B. Section of *Alexandrium tamarense* cell concentrations (cells liter^{-1}), with the bulk of the cells contained within the low salinity plume. (Adapted from Franks and Anderson 1992*a*.)

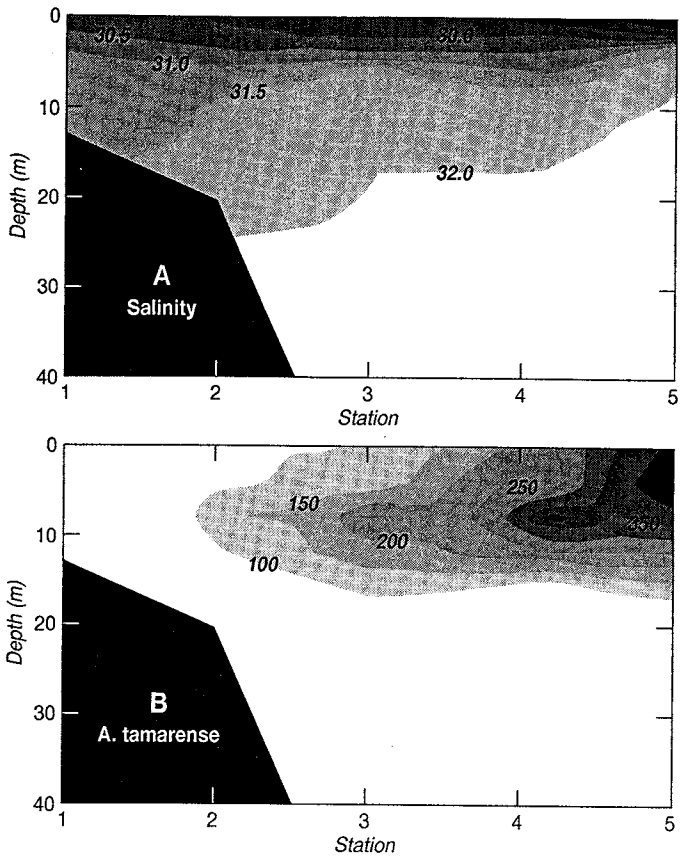

Fig. 4. Hydrographic sections along same transect shown in Fig. 3, but following upwelling-favorable winds (23 May 1989). A. Section of salinity (psu) showing how the plume has been spread offshore in a thin layer, with a lens of low salinity water more than 35 km from shore, and higher salinity water upwelling nearshore. B. Section of *Alexandrium tamarense* cell concentrations (cells liter^{-1}), with the bulk of the cells transported offshore with the low salinity water. This upwelling event resulted in a decrease in coastal shellfish toxicity. (Adapted from Franks and Anderson 1992*a*.)

which supports the growth of *A. fundyense* cells and transports them to the south and west; and plume behavior, as influenced by the volume of freshwater outflow, the local wind stress, and underlying gulf circulation, all of which combine to regulate the alongshore and cross-shore location of the plume, its associated cells, and PSP toxicity.

The dynamics of this plume and its *Alexandrium* cells have been investigated in a series of small-scale hydrographic surveys (Franks and Anderson 1992*a*) and more recently in two major field investigations covering the entire western gulf region as well as Massachusetts Bay (unpubl. data). From these studies, it is now clear that downwelling-favorable winds (i.e. those from the north and east) accelerate the plume toward the west and trap it against the shore, resulting in a rapid progression of toxicity from east to west. *Alexandrium* cells are most abundant in the plume (Fig. 3), often in subsurface patches near its offshore edge. Upwelling-favorable winds move the plume offshore, spreading it out to a thin, broad layer (Fig. 4; Franks and Anderson 1992*a*; Keafer and Anderson 1993). Persistent upwelling has been typically associated with a decrease in toxicity and often in the termination of the bloom. If downwelling-favorable winds recur, the plume can be transported back to shore where a new surge in toxicity can develop. Some of these relationships have been inferred from large-scale field surveys in 1993 and 1994 (unpubl. data), but remote sensing has also proved useful. The warmer temperatures of the plume waters in the spring and early summer allow this feature to be tracked using remote sensing of sea surface temperature (Keafer and Anderson 1993).

The cells that populate this coastal current presumably originate from germinated cysts, but the location of specific cyst seedbeds and the mechanisms underlying cyst germination at the onset of the blooms remain uncertain. Several cyst surveys have been conducted in the western Maine region (Lewis et al. 1979; Thayer et al. 1983; Anderson and Keafer 1985), documenting a widespread distribution both nearshore and offshore (everywhere except in the PSP sandwich zone near Penobscot Bay; Lewis et al. 1979). The highest cyst concentrations were typically found offshore in the deeper basins that accumulate clay and fine particles (up to 60×10^6 cysts m^{-2}; Anderson and Keafer 1985). Based on the development of chlorophyll fluorescence as functional chloroplasts are produced (Anderson and Keafer 1985), cysts

in these deep-water sediments do germinate, despite darkness and low and relatively invariant temperatures. This germination is regulated by an endogenous annual clock that restricts germination to a seasonal window irrespective of the external environment (Anderson and Keafer 1987). Clock-controlled germination leads to the release of motile cells into deep waters at times when temperature and light at the surface are suitable for growth.

Despite the existence of this timing mechanism to provide motile cells from deep-water cyst deposits, it is not known whether those cysts are a major factor in the *Alexandrium* blooms associated with the coastal current. Cysts in shallow coastal waters or in bays and estuaries could provide a larger and more synchronized inoculum, especially if their germination is controlled by seasonal temperature changes and not by an endogenous annual clock (Anderson and Keafer 1987). Offshore cyst deposits might only be sinks where cysts accumulate through time and germinate too slowly to have a major impact on blooms. Offshore cyst deposits do represent a potential source of toxin, especially to scallops and other deep-water filter-feeding animals, however (Shumway et al. 1988).

The fate of deep-water cysts may be unknown, but evidence is accumulating that there is a source region or initiation zone in nearshore waters where motile cells accumulate at the start of the bloom season. As discussed above, seeding of the western Maine coastal current from *Alexandrium* populations originating in eastern Maine is unlikely due to the offshore deflection of the eastern Maine coastal current, the toxin-free zone immediately "upstream" near Penobscot Bay, and the late-season nature of downeast toxicity (Fig. 2). Franks and Anderson (1992*a*) implicated the area near the common mouth of the Kennebec and Androscoggin Rivers as a source region, but follow-up work (Anderson and Keafer 1992) did not find cells in the Kennebec River itself. Field studies in 1993 and 1994 have since implicated the eastern Casco Bay region just "downstream" from the Kennebec River as the most likely area for bloom initiation. In two successive years (1993 and 1994), cells were generally absent from water samples at all stations except those near the Casco Bay region in mid-April (unpubl. data). Concentrations were low near Casco Bay (~50–100 cells liter^{-1}) and quickly increased in the low salinity coastal current.

The early-season bloom dynamics of the putative source region are not the only issue, however. The persistent southwesterly flow of the coastal current during the bloom season raises a significant question. If *Alexandrium* cells are transported to the west and south each year with no return flow, how does the system restart itself from cysts each subsequent spring? With no resupply of cells to this source region, the red-tide problem downstream should gradually diminish and disappear, yet PSP in western Maine has been recurrent nearly every year since 1972 and shows no signs of abatement. A reseeding mechanism thus seems necessary. Unpublished field surveys from 1993 and 1994 suggest how this may be occurring. A late-season population of *Alexandrium* was observed in the waters of Casco Bay at the end of May 1993, long after an initial pulse of cells had been carried toward Massachusetts by the coastal current. With much-reduced rainfall and no snowmelt to drive the coastal current in May and early June, transport out of the region was limited. Cells from that source region not only might represent the initial populations that are transported rapidly to the south early in the bloom season, but they also may provide the late-season cysts that are deposited locally to initiate blooms the following year.

Mechanisms to explain this apparent population retention and localized blooming are under investigation. Recent observations (N. Pettigrew pers. comm.) indicate that freshwater lenses originating from the Kennebec plume are trapped in eastern Casco Bay, providing a physical mechanism for the retention and localization of dinoflagellates. Alternatively, estuarine fronts have been shown to be sites of dinoflagellate accumulation (Tyler and Seliger 1978; Tyler et al. 1982), and these do exist in the Casco Bay area. Incze and Yentsch (1981) found concentrated populations of *Alexandrium* in a frontal zone in the nearby Damariscotta estuary, for example. The importance of retention of dinoflagellate populations at an estuarine front is highlighted by a study of *Gyrodinium uncatenum* in the Potomac River (Tyler et al. 1982) where the dinoflagellate remained highly concentrated in the pycnocline at an estuarine front for a month or more, oscillating upstream and downstream with the tides and runoff and persisting as a localized red tide in the convergence zone. This retention and accumulation was a result of interactions between the vertical swimming behavior of the dinoflagellate and the flow regime at the front. *G. uncatenum* cyst deposition was defined by the upstream and downstream extent of the estuarine front's oscillations, forming a concentrated seedbed to inoculate future blooms in a narrowly defined area. In the context of *Alexandrium* blooms in the Casco Bay region, heavy runoff or changing wind patterns could transport blooms accumulated at a front or in a freshwater lens into the path of the alongshore coastal current early in the bloom season, entraining *Alexandrium* cells and transporting them to the south and west. This would result in pulsed or nonhomogeneous *Alexandrium* populations in the coastal current. *Alexandrium* cells are not uniformly distributed throughout the low salinity plume, but rather are concentrated in patches that seem to travel downstream relatively intact. A pulsed delivery or entrainment of cells into the coastal current near its origin is consistent with this type of population distribution. These same fronts or lenses could persist in the source region in later months as well, with delivery into the coastal current less likely to occur due to seasonal changes in rainfall, runoff, and winds. The result would be localized blooms and cyst deposition.

Summarizing, evidence is strong that the western Maine *Alexandrium* populations originate near the mouth of the Androscoggin-Kennebec estuary, with little or no input of cells from eastern Maine. Large offshore cyst seedbeds exist, and mechanisms have been identified which allow cysts to germinate at times appropriate for bloom initiation, but the quantitative importance of deep-water cysts remains uncertain. A shallow water source region near the origin of the coastal current has been identified, but the mechanisms for motile cell accumulation there remain unknown, and the ultimate origin of the cysts that germinate to produce the inoculum cells is unknown as well. Cells are apparently entrained into the coastal current in an episodic or pulsed

fashion, and transport to the south and west toward Massachusetts Bay can be strong and rapid given the appropriate winds and heavy freshwater runoff. Alternatively, the downstream progression of the plume and PSP toxicity can slow or even cease entirely due to a change in wind patterns (Franks and Anderson 1992*a*). Localized, late-season blooms in or near Casco Bay may well provide the cyst deposits that initiate blooms in subsequent years. Overall, the western Maine *Alexandrium* dynamics have been well studied, and an excellent conceptual understanding of the system exists. Considerable work remains to refine the details of this model and to fully understand the extent to which management and even mitigation efforts focused on this region can affect blooms and shellfish toxicity over a much larger region downstream. Issues such as the importance of nutrients in bloom dynamics, the magnitude of losses due to grazing, and the nature of the biological-physical coupling that leads to population accmulation need further study if we are to explain how an intact population of cells can be maintained in a coastal current for weeks and even months.

Massachusetts and Cape Cod Bays—Massachusetts Bay (which here refers to Cape Cod Bay as well) is a semienclosed basin bounded on the east by the relatively shallow waters of Stellwagen Bank, which rises to within 20 m of the surface (Fig. 1). There are two silled channels or passages on either side of Stellwagen Bank. North Passage (Geyer et al. 1992) is 60 m deep and runs between Cape Ann and the northern flank of Stellwagen; South Passage (50 m deep) runs between the tip of Cape Cod and the southern flank of the bank. The deepest portion of the bay is Stellwagen basin at 80 m.

The dominant circulation regime in the bay is a counterclockwise flow that enters via North Passage, travels south through most of Massachusetts Bay, and exits through South Passage, heading offshore toward Georges Bank (Geyer et al. 1992). Superimposed on this mesoscale pattern are episodic intrusions of low salinity water from the western Maine coastal current which can enter the bay through North Passage (Butman 1975; Franks and Anderson 1992*a*). The entrance to North Passage represents a fork or bifurcation point. Depending on local wind stress, water from the coastal current can either enter the bay there or bypass it entirely, traveling instead along the eastern flank of Stellwagen Bank toward Georges Bank. Within the bay, the mesoscale circulation and the location of the plume can be modified by southwest or northeast winds that cause significant upwelling or downwelling. Outbreaks of PSP in Massachusetts Bay are more sporadic than those in the western Maine region, occurring every few years rather than annually (Franks and Anderson 1992*b*). Because the western Maine coastal current can enter the bay, cells transported in that current enter as well. The residence time of the low salinity water mass within the bay can be several weeks or longer (Geyer et al. 1992), so the introduced population can increase in abundance, causing toxin to accumulate in shellfish along the transport pathway. In years when rainfall is low in late spring, the residence time of water in the bay can increase 2-fold or more (Geyer pers. comm.), allowing the introduced populations additional time to grow before mesoscale circulation carries them out of the bay. This occurred in 1993, for example, a year in which toxicity was very high in the bay and extended to stations on Cape Cod where PSP had never before been detected (unpubl. data).

The foregoing argues that the same *Alexandrium* blooms that cause toxicity in western Maine are responsible for toxicity in Massachusetts Bay and that the low frequency of PSP outbreaks in the bay reflects the frequency at which the coastal current enters the bay at times when it contains toxic *Alexandrium.* Failure of the coastal current and its pulses of cells to enter the bay can result in a year with little or no toxicity in the bay, even though PSP scores are high in western Maine. In other years, delivery of larger, established populations can cause rapid increases in toxicity in the bay that cannot be explained by in situ growth alone. An example of this is seen in Fig. 2, which documents a sudden increase in shellfish toxicity in Cohasset which we now know was associated with the delivery of low salinity water carrying *Alexandrium* cells.

An alternative scenario for bloom development in Massachusetts Bay does not require transport and introduction of cells from the coastal current, but instead invokes localized *Alexandrium* blooms in the bay, initiated from cysts deposited there during previous blooms. The only cyst survey conducted for Massachusetts Bay reported cysts in central portions, with highest concentrations an order of magnitude lower than in deposits in the basins of western Maine (Anderson and Keafer 1985). The existence of these cysts implies that germination does occur in the bay and that introduction of cells from the north is not absolutely necessary for bloom formation. However, the relatively low abundance of cysts and the relatively rapid flow of water through the bay during the bloom season argue that cyst germination and in situ growth in the bay are not likely to be significant except in years when the residence time of water is long. Our working hypothesis at this stage is that blooms of *Alexandrium* in Massachusetts Bay are predominantly linked to cells delivered there by the western Maine coastal current—that the input of cells from germinated cysts from bay sediments is too small to produce significant blooms given the typical residence time of water in the bay.

Summarizing, *Alexandrium* bloom dynamics in Massachusetts Bay are not well known, as thus far relatively few field surveys have been conducted at the scale needed to verify either of two possible scenarios. The most likely scenario is that toxic *Alexandrium* cells are delivered to the bay as established populations via the western Maine coastal current. This does not occur every year because the buoyant plume does not always enter the bay sufficiently intact and with sufficient *Alexandrium* cells to cause nearshore shellfish to accumulate toxins. Upwelling-favorable winds can quickly spread the western Maine coastal current out into a thin layer and move it offshore. If this occurs before the cells reach the bay, very few cells would enter and toxicity would then be low or nonexistent due to lack of an inoculum. The second possibility is that localized, in situ growth occurs in the bay following germination from cysts in bottom sediments. These in situ blooms could be completely independent of the advected populations from the coastal current, or

they could augment the delivered cells. The extent to which in situ cyst germination in the bay results in major blooms depends on the water residence time, which is typically too short for a small cyst inoculum to reach bloom proportions. To determine which of these options is the best representation of the system, we need detailed field surveys in the bay as well as across the transport pathway into the bay via North Passage. An update is also needed on *Alexandrium* cyst abundance, distribution, and germination dynamics as the last cyst map was produced more than 10 yr ago.

Georges Bank—In 1989, PSP toxins were detected in shellfish from the American sector of Georges Bank, a shallow, well-mixed fishing ground located several hundred kilometers east of Cape Cod (White et al. 1993). PSP toxins have occasionally been found in scallop digestive glands from Georges Bank since the mid-1960s (Bourne 1965; Jamieson and Chandler 1983), but levels were low and were not cause for concern given the removal of that organ with viscera during normal processing. Because the digestive gland is known to accumulate extremely high levels of the toxins, and because there were no reports of PSP from consumers of Georges Bank shellfish prior to 1990, the assumption has been that toxin levels were historically low before the outbreak that year (White et al. 1993).

The first indications of elevated toxins in offshore shellfish came in summer 1988 when scallop digestive glands were shown to contain >2,000 μg saxitoxin equiv per 100 g of meat (White et al. 1993). These animals were from the Canadian sector of the bank. Although sampling was infrequent thereafter, a general pattern of increasing toxicity occurred, culminating in the closure of large areas of the American sector of Georges Bank for the harvesting of surfclams, ocean quahogs, mussels, and scallops. High levels of PSP in mussels in late May 1990 (24,000 μg saxitoxin equiv per 100 g of meat), sufficient to seriously poison eight fishermen, clearly indicates that a significant source of toxicity was present in those offshore waters. Toxin levels remained high for several years, especially in surfclams which still were dangerously toxic 3 yr after the initial outbreak. Surfclams seem to be particularly slow in depurating PSP toxins. Levels in other shellfish showed persistent but declining levels of PSP toxins after the initial outbreak. This trend, combined with changes in the relative composition of the different saxitoxins in those shellfish through time (Nassif and Timperi 1993; Shumway et al. 1994), suggests that there were probably one or two high-toxicity events in the 1989–1990 interval that contaminated the shellfish or that added to low levels of toxin accumulated in preceding years. Thereafter, no new toxin was ingested and retained toxins were slowly depurated and bioconverted within the shellfish. The source of the Georges Bank toxin remains unknown to this day.

Three alternative hypotheses have been advanced to explain these outbreaks (Anderson and Keafer 1992): the dinoflagellate *A. tamarense* grows in the waters on or near Georges Bank, possibly accumulating at frontal features (in situ growth); dormant resting cysts of *A. tamarense,* produced by blooms at distant locations, are advected onto Georges Bank where they are consumed by shellfish which retain the PSP toxins; and large, nearshore blooms of *A. tamarense* in western Maine are advected through or by Massachusetts Bay and onto Georges Bank. This latter option could either deliver large, previously established populations to Georges Bank or could deliver cells in small numbers that serve as the inoculum for in situ growth. Logistical constraints and an apparent lack of subsequent toxicity episodes since 1990 have made it difficult to determine which of these possibilities is correct. Sediment samples from the Georges Bank region contained very few *Alexandrium* cysts (unpubl. data), but delivery could be episodic and thus easily missed. Nevertheless, the low abundance of cysts in the immediate vicinity of Georges Bank makes the advection of resuspended cysts onto the bank seem unlikely as a major source of toxicity.

The final possibility listed above is an extension of the plume advection hypothesis proposed for the western Maine habitat (Franks and Anderson 1992*a*). Field investigations of this transport pathway in western Maine and in Massachusetts Bay in 1990 document the initiation of a bloom population of *A. tamarense* in southern Maine waters in mid-April, the development and entrainment of the bloom in the low salinity coastal current, movement of the plume past Cape Ann into Massachusetts Bay and possibly offshore toward Georges Bank as well, and high levels of PSP toxicity along the north shore of Massachusetts and in Massachusetts Bay in late May (Anderson and Keafer 1992). Extensive shellfish toxicity along the western Maine coast, in Massachusetts Bay, and on Georges Bank—all in May and early June 1990—suggest that these episodes may be linked, presumably through the presence and behavior of the buoyant coastal current.

Supporting evidence for the movement of a plume of less-saline water across Massachusetts Bay and offshore toward Georges Bank during this interval comes from a mooring in Stellwagen basin which documented a significant decrease in the salinity of the surface waters (2 psu) from 27 May through the early part of June. These dates should be viewed in the context of the onset of PSP toxicity along the coast, first detected on the north shore of Massachusetts on 21 May and then within the bay on 29 May (Anderson and Keafer 1992). A serious poisoning episode involving two fishermen who had consumed mussel bycatch from Georges Bank on 22 May and another on 5 June is strong coincidental evidence that these blooms may all be related to the same population of *Alexandrium* in the coastal current. Therefore, we believe that a population of *Alexandrium* developed in the western Maine coastal current and traveled along the north shore of Massachusetts. Some cells clearly entered Massachusetts Bay via North Passage, but others may have bypassed the bay and headed directly to Georges Bank. The populations that accumulated in the bay would eventually have been advected through South Passage and toward Georges Bank as well.

Logistical constraints have made it difficult to adequately document the fate of the buoyant plume and its associated toxic dinoflagellate population at the entrance and exit to Massachusetts Bay, but previous workers (Bigelow 1927; Butman 1975; Bumpus 1976) and more recent studies (Chen et al. 1992) have documented a circulation pattern of surface

water movement past Cape Ann, Cape Cod, and farther offshore to Georges Bank. Lagrangian pathways are thus documented linking nearshore riverine outflow to the distant, offshore waters of Georges Bank. The timing of shellfish toxicity along the coast and the movement of less saline water across Massachusetts Bay are strong supporting evidence for the hypothesis that toxicity on Georges Bank was not due to in situ growth of dinoflagellates but rather to the advection of nearshore populations to the offshore waters.

A series of cruises was conducted from 1991 to 1993 to sample shellfish and to examine the plankton (Nassif and Timperi 1993), but *Alexandrium* cells were virtually nonexistent in the many samples examined. Those were also years on which there was also little or no toxicity in Massachusetts Bay or western Maine. This again supports the linkage between Georges Bank toxicity and blooms in the western Maine coastal current, but it is not conclusive.

In summary, the high levels of PSP toxicity detected on Georges Bank in 1989 and 1990 seem to reflect a linkage to the western Maine coastal current (Fig. 1). All data collected in 1990 are consistent with the long distance, relatively rapid advection of a nearshore *A. tamarense* bloom population to Massachusetts Bay and onto Georges Bank. A transport pathway exists to carry low salinity water from the western Maine coastal current to the bank, but it remains unknown whether *Alexandrium* populations can remain sufficiently intact during the long transit to represent a significant source of toxicity. The infrequent nature of high toxicity on Georges Bank suggests that a rare combination of events leads to the delivery of large *Alexandrium* populations to offshore shellfish. Although the coastal current linkage is compelling, the hypotheses of cyst resuspension and advection or of in situ localized blooms of *A. tamarense* on or near Georges Bank cannot be rejected due to lack of data.

Southern salt ponds and embayments—Throughout Cape Cod, Connecticut, and Long Island (Fig. 1), PSP outbreaks occur, but those episodes are sporadic and highly independent of each other or of the large-scale coastal blooms described above (Anderson and Wall 1978; Anderson et al. 1982; Schrey et al. 1984). The coastal bloom populations of eastern and western Maine and Massachusetts Bay presumably form cysts that fall from the water column or are dispersed and diluted in the deep offshore waters of the gulf before they have any opportunity to impact the coastlines of Cape Cod, Rhode Island, Connecticut, and New York. Instead, isolated and localized blooms occur in those areas, with very tight linkage in time and space to benthic cyst populations.

Several studies in Perch Pond, a salt pond on Cape Cod, provide a perspective on the dynamics of localized blooms that is probably applicable to many of the southern salt ponds and embayments. These locations can be viewed as point sources of toxicity, in that *Alexandrium* populations originate within the embayments or estuaries from isolated cyst seedbeds, with no input of cells from coastal waters. In Perch Pond, for example, *Alexandrium* cysts are present at high concentrations in the muddy bottom sediments, but virtually none can be found 1–2 km away in adjacent coastal waters (Anderson et al. 1982). The cysts are clearly responsible for in situ overwintering of the species, with germination prevented by temperatures of 4°C or less (Anderson and Morel 1979; Anderson 1980). As bottom waters warm in early spring, the cysts germinate to provide the vegetative cell inoculum that initiates the bloom. In contrast to the highly seasonal, endogenous clock-controlled cyst germination of *Alexandrium* cysts from the deeper waters of the Gulf of Maine (Anderson and Keafer 1987), mature cysts from Perch Pond can germinate at any time as long as the temperature is favorable. Germination is thus tied to the external temperature, so that a long winter or an early spring will directly affect the timing of the resulting *Alexandrium* bloom. Due to the shallowness of the waters and the rapid rate of warming, germination occurs in a discrete pulse lasting ~1 month (Anderson and Keafer 1985). This pulsed circulation is again in contrast to the rather protracted and gradual clock-controlled germination of *Alexandrium* cysts in deep, cold waters of the gulf. The timing of shellfish toxicity in these embayments is typically earlier and of shorter duration than in Massachusetts Bay or western Maine (Fig. 2; Anderson et al. 1983).

A long-standing question has been whether the size of the cyst germination inoculum determines the magnitude of the resulting dinoflagellate bloom. The isolated nature of the salt ponds, with little or no input of cells from adjacent waters, makes these locations ideal for addressing this question. To this end, the distinctive large cells produced by germination (planomeiocytes) were enumerated during studies of *Alexandrium* blooms in two Cape Cod salt ponds over two successive years (Anderson et al. 1983). At the onset of the bloom, the number of germling cells was as high as 30% of the total motile population, but these cells were quickly outnumbered by dividing cells. Cysts were clearly critical to the timing of bloom initiation, but the magnitude of the bloom seemed to depend on factors regulating the rate of growth and accumulation of the vegetative population.

The decline of the blooms in the salt ponds can be rapid, with cyst formation a significant loss factor. Thirty percent or more of the motile cell population in a 1980 Perch Pond bloom were planozygotes—the large and distinctive cells that are the precursors to cysts (Anderson et al. 1983). This number does not represent the total percentage of cells that became cysts, because it cannot account for the dynamic nature of the zygote pool. Each day, some planozygotes fall to the sediments as cysts, but new planozygotes are also formed following gamete fusion. It does, however, provide an indication of the large fraction of the population that is deposited to initiate future blooms. The eventual role of those cysts in bloom dynamics is determined not only by seasonal warming, as discussed above, but by the survival of the cysts in the benthos and the extent to which they remain in the oxygenated surface sediments where germination is possible (Anderson et al. 1987).

The onset of sexuality leading to dinoflagellate cyst formation has generally been attributed to nutrient limitation (*see* Pfiester and Anderson 1987). The localized blooms in the salt ponds suggest that the situation may be more complex than is implied by culture experiments. In particular, cyst formation occurred in several salt ponds when nutrient concentrations were as high or higher than levels at the start

of the bloom (Anderson et al. 1983). In other words, planozygotes and cysts appeared in large numbers, but there had been no dramatic decrease in ambient nutrients. One explanation is that the absolute nutrient concentration of the waters is not the controlling factor, but rather the internal nutrient pools of the dinoflagellate cells. Those are affected by the balance between nutrient availability and the rate of growth of the cells. As waters warm during the bloom, growth rates increase and thus the nutrient uptake demands increase as well. Concentrations that were sufficient for uptake and balanced growth at the start of the bloom when temperatures and growth rates were lower may not be high enough to satisfy the demands of high growth rates at warmer temperatures, leading to depletion of internal pools and initiation of sexuality.

The localization of *Alexandrium* populations to discrete sites such as Perch Pond on Cape Cod or Palmer Cove in Connecticut (Anderson et al. 1982) is perhaps surprising, because a gradual dispersal to adjacent estuaries and embayments would seem likely given two decades of recurrent blooms. Furthermore, a density-driven exchange mechanism has been documented that flushes water very efficiently from these salt ponds (Anderson and Stolzenbach 1985). Nevertheless, the blooms persist and are localized, indicating that the residence time of the *Alexandrium* cells is much longer than that of the water. One explanation for this apparent discrepancy may lie in a unique linkage between the vertical migration behavior of the dinoflagellates and the flushing characteristics of some of these embayments (Anderson and Stolzenbach 1985; Garcon et al. 1986). Several of the Cape Cod salt ponds where PSP is recurrent have shallow entrance sills that restrict outflowing water to the low density surface layer. The diel vertical migration behavior of *Alexandrium* keeps cells below that depth at night, and even when the cells are closest to the surface during the day, they remain deep enough to avoid transport out of the embayment. This retention mechanism not only restricts the extent to which cells and cysts can colonize adjacent waters, but it allows *Alexandrium* populations to accumulate to levels where toxicity becomes dangerous. This is a particularly important feature, as the toxicity of the *Alexandrium* populations in the southern salt ponds and embayments is lower than the coastal populations to the north (Maranda et al. 1985; Anderson et al. 1994).

Summarizing, *Alexandrium* populations in southern waters of the study region are found in a few scattered embayments which are the sites of localized, recurrent blooms. Cyst dynamics are regulated by the external environment rather than by an internal clock, and blooms are closely linked to the location of cyst seedbeds and the timing of excystment and encystment. Even though the *Alexandrium* populations that inhabit the region are generally low potency strains, PSP closures still occur due to the accumulation of high cell concentrations. This can result from physical-biological coupling in which diel vertical migration behavior restricts advective losses. Localized blooms such as those in the southern salt ponds and embayments probably also occur along the coast of the Gulf of Maine, and perhaps in Massachusetts Bay as well, but the widespread coastal blooms seem to dominate with respect to the PSP outbreaks in those regions. The scattered nature of PSP in southern waters also suggests that the geographic limit of the regional population has been reached. Warmer temperatures and different habitats and ecosystems apparently do not favor the further proliferation and expansion of this toxic dinoflagellate.

General biogeographic considerations

The historical absence of PSP everywhere in the region except in downeast Maine before 1972 and the extensive toxicity that now occurs in many locations suggest that *Alexandrium* expanded its geographic range in the recent past. Much of this dispersal resulted from the 1972 New England red tide, which exposed large expanses of the coast to cell populations sufficiently dense to discolor the water (Mulligan 1975). Cyst deposition into previously unaffected areas then provided the species with a widely dispersed inoculum for future blooms (Anderson and Wall 1978). The extent of dispersal during this single bloom event remains unknown, however.

If the bloom covered the entire region, the *Alexandrium* population should be genetically uniform—derived from a single common population. This is not the case, however. Maranda et al. (1985) compared the toxicity of the regional *Alexandrium* populations originating between Nova Scotia and New York using the mouse bioassay and documented a systematic decrease in toxin content from north to south. Because the differences were genetic, the data are not consistent with recent dispersal from a common source during the 1972 bloom. A subsequent comparison conducted with allozyme electrophoresis (Hayhome et al. 1989) revealed a high degree of similarity among regional isolates, but it is now clear that the allozymes chosen could not resolve fine-scale differences that later were documented in other characters. The most useful comparisons thus far have been provided by a detailed toxin analysis in which the suite of different saxitoxins produced by each isolate was determined with HPLC (Anderson et al. 1994). These "toxin composition" comparisons identified at least two and perhaps three regional populations (Anderson et al. 1994). Differences were apparent between GOM isolates and those from the southern salt ponds and embayments, and there was a suggestion that the southern populations could be further subdivided into a Cape Cod cluster and a Connecticut-Long Island cluster. The differences in toxin composition explain the north-to-south trend of declining PSP toxicity described by Maranda et al. (1985) because northern isolates contain the most potent of the saxitoxin derivatives, whereas southern isolates contain predominantly sulfamate derivatives, which are an order of magnitude less potent. It remains unknown, however, why southern isolates would have that particular suite of toxins and if, for example, the southern habitat somehow favors cells with sulfamate vs. carbamate saxitoxins.

Distinct subpopulations cannot be separated on the basis of morphology, as only two morphospecies are responsible for the production of PSP toxins within the region—*A. tamarense* and *A. fundyense*—and they are interspersed geographically, especially in southern waters (Anderson et al.

1994). On the basis of careful examination of cultures established from cysts collected throughout the region, the general taxonomic distribution can be described as a pure population of *A. fundyense* in western Maine (eastern Maine was not sampled) and a mixture of the two species from Cape Cod southward.

Sexual compatibility between *A. tamarense* and *A. fundyense,* demonstrated by Anderson et al. (1994), allows gene flow between subpopulations, but the degree to which this has occurred depends directly on circulation pathways. Several of the regional subpopulations described here (e.g. western Maine, Massachusetts Bay, and Georges Bank) probably share the same *Alexandrium* seed population via large-scale transport in the coastal current, whereas the other two habitats (eastern Maine and southern salt ponds-embayments) seem to be more isolated and may have little or no genetic exchange with adjacent regions (Table 1).

Where did the southern *Alexandrium* populations originate? Anderson et al. (1994) proposed two scenarios to explain their origin. The first invokes recent and continuing dispersal of *Alexandrium* to the south from a center of origin in the Gulf of Maine, with recombination and strong selection for southern genotypes. One argument against this hypothesis is that mating of different toxin composition genotypes within *Alexandrium* is now known to produce progeny with the toxin characteristics of the parent isolates (Sako et al. 1992; Anderson unpubl. data). If intermixing and genetic exchange were occurring between western Maine population and those in southern salt ponds, for example, the crosses should be yielding some low toxicity strains and some high toxicity strains in the south. The latter, however, are rare (Maranda et al. 1985; Anderson et al. 1994), so some type of environmental selection for southern waters must be invoked which favors strains producing sulfamate toxins and is ultimately lethal to strains producing mostly carbamates. This is a theoretical possibility, but seems unlikely.

The second hypothesis is that northern and southern populations diverged from a common ancestor (vicariance) but now represent localized populations that have evolved independently since their initial separation. The evidence in favor of this hypothesis is strong. Cape Cod and Georges Bank act as hydrographic barriers to force the predominant north–south flow offshore, away from the estuaries and embayments where southern *Alexandrium* cells and cysts are localized. Most cells are probably lost to the deeper offshore waters, with a low probability that any are carried back to shore on a regular basis. Furthermore, germination of Cape Cod *Alexandrium* cysts is apparently regulated by a different mechanism than the internal clock that controls cyst germination in the Gulf of Maine (Anderson and Keafer 1987). In shallow waters where seasonal variability is large, a direct coupling between cyst germination and the external environment (such as temperature) is a more logical strategy than one in which germination is tied to an internal clock.

Overview

When toxic organisms are detected in a previously unaffected area, several explanations are possible (Anderson 1989). Species dispersal via storms or natural currents is a distinct possibility, but human activities can also be implicated. Bloom biomass and extent can be enhanced through anthropogenic nutrient enrichment, and shellfish seeding efforts or ballast water discharge can disperse cysts of an organism within a region on a small scale. The growth of the PSP problem in the northeastern U.S. over the last several decades is often cited as an example of a broader expansion of harmful algal bloom problems throughout the world (e.g. Anderson 1989). We can now say with confidence that the present-day species distribution is most easily explained through natural dispersal via existing circulation patterns. Species dispersal via natural currents occurred within the region as a result of the massive 1972 New England red tide, which took place shortly after a major hurricane hit the coast (Mulligan 1975). However, the detection of toxicity in other areas such as Connecticut or Long Island (Anderson et al. 1982; Schrey et al. 1984) is not an indication that the 1972 event reached that far south or that further spreading occurred in subsequent years due to accidental introductions through human activities. Instead, a better understanding of *Alexandrium* taxonomy and life history details led to the identification of sites where blooms were likely (Anderson et al. 1982), and expanded monitoring at those sites resulted in the detection of PSP toxins in areas where low-level PSP toxicity has probably been occurring sporadically for decades.

It has taken a great deal of work to resolve *Alexandrium* subpopulations in the northeastern U.S., and more effort is needed to better characterize the bloom dynamics and population genetics of these groups. What first appears to be a homogeneous regional population of *Alexandrium* is clearly heterogeneous with respect to both genetics and bloom dynamics. Natural dispersal via established, long-distance transport pathways such as coastal currents gives a species the opportunity to colonize new areas, and life history strategies and strong adaptive abilities then allow it to persist in a wide range of habitats.

References

ANDERSON, D. M. 1980. Effects of temperature conditioning on development and germination of *Gonyaulax tamarensis* (Dinophyceae) hypnozygotes. J. Phycol. **16:** 166–172.

———. 1989. Toxic algal blooms and red tides: A global perspective, p. 11–16. *In* Red tides: Biology, environmental science, and toxicology. Proc. 1st Int. Symp. on Red Tides. Elsevier.

———, S. W. CHISHOLM, AND C. J. WATRAS. 1983. The importance of life cycle events in the population dynamics of *Gonyaulax tamarensis.* Mar. Biol. **76:** 179–190.

———, AND B. A. KEAFER. 1985. Dinoflagellate cyst dynamics in coastal and estuarine waters, p. 219–224. *In* Toxic dinoflagellates: Proc. 3rd Int. Conf. Elsevier.

———, AND ———. 1987. An endogenous annual clock in the toxic marine dinoflagellate *Gonyaulax tamarensis.* Nature **325:** 616–617.

———, AND ———. 1992. Paralytic shellfish poisoning on Georges Bank: In situ growth or advection of established dinoflagellate populations?, p. 217–224. *In* Proc. Gulf of Maine Sci. Workshop. Urban Harbors Inst.

———, D. M. KULIS, G. J. DOUCETTE, J. C. GALLAGHER, AND E.

BALECH. 1994. Biogeography of toxic dinoflagellates in the genus *Alexandrium* from the northeastern United States and Canada. Mar. Biol. **120:** 467–478.

———, ———, J. A. ORPHANOS, AND A. R. CEURVELS. 1982. Distribution of the toxic red tide dinoflagellate *Gonyaulax tamarensis* in the southern New England region. Estuarine Coastal Shelf Sci. **14:** 447–458.

———, AND F. M. M. MOREL. 1979. The seeding of two red tide blooms by the germination of benthic *Gonyaulax tamarensis* hypnocysts. Estuarine Coastal Mar. Sci. **8:** 279–293.

———, AND K. D. STOLZENBACH. 1985. Selective retention of two dinoflagellates in a well-mixed estuarine embayment: The importance of diel vertical migration and surface avoidance. Mar. Ecol. Prog. Ser. **25:** 39–50.

———, C. D. TAYLOR, AND E. V. ARMBRUST. 1987. The effects of darkness and anaerobiosis on dinoflagellate cyst germination. Limnol. Oceanogr. **32:** 340–351.

———, AND D. WALL. 1978. The potential importance of benthic cysts of *Gonyaulax tamarensis* and *Gonyaulax excavata* in initiating toxic dinoflagellate blooms. J. Phycol. **14:** 224–234.

BIGELOW, H. B. 1927. Physical oceanography of the Gulf of Maine. Bull. U.S. Bur. Fish. **40**(Part 2)**:** 511–1027.

BOURNE, N. 1965. Paralytic shellfish poisoning in sea scallops (*Plecopecten magellanicus,* Gmelin). J. Fish. Res. Bd. Can. **22:** 1137–1149.

BROOKS, D. A. 1985. Vernal circulation in the Gulf of Maine. J. Geophys. Res. **90:** 4687–4705.

———, AND D. W. TOWNSEND. 1989. Variability of the coastal current and nutrient pathways in the eastern Gulf of Maine. J. Mar. Res. **47:** 303–321.

BUMPUS, D. F. 1976. A review of the physical oceanography of Georges Bank. Int. Comm. NW Atl. Fish. Res. Bull. **12:** 119–134.

BUTMAN, B. 1975. On the dynamics of shallow water currents in Massachusetts Bay and on the New England continental shelf. Woods Hole Oceanogr. Inst. Tech. Rep. 77-15.

CEMBELLA, A. D., J. C. THERRIAULT, AND P. BELAND. 1988. Toxicity of cultured isolates and natural populations of *Protogonyaulax tamarensis* (Lebour) Taylor from the St. Lawrence estuary. J. Shellfish Res. **7:** 611–622.

CHEN, C., R. C. BEARDSLEY, AND R. LIMEBURNER. 1992. Lagrangian circulation in the Great South Channel and on Georges Bank during summer, p. 236. *In* Proc. Gulf of Maine Sci. Workshop. Urban Harbors Inst.

COHN, M. S., P. OLSEN, J. P. B. MAHONEY, AND E. FEERST. 1988. Occurrence of the dinoflagellate *Gonyaulax tamarensis* in New Jersey. Bull. N.J. Acad. Sci. **33:** 45–49.

FRANKS, P. J. S. 1990. Dinoflagellate blooms and physical systems in the Gulf of Maine. Ph.D. thesis, Mass. Inst. Technol. and Woods Hole Oceanogr. Inst. 253 p.

———. 1992. Phytoplankton blooms at fronts: Patterns, scales, and physical forcing mechanisms. Rev. Aquat. Sci. **6:** 121–137.

———, AND D. M. ANDERSON. 1992*a*. Alongshore transport of a toxic phytoplankton bloom in a buoyancy current: *Alexandrium tamarense* in the Gulf of Maine. Mar. Biol. **112:** 153–164.

———, AND ———. 1992*b*. Toxic phytoplankton blooms in the southwestern Gulf of Maine: Testing hypotheses of physical control using historical data. Mar. Biol. **112:** 165–174.

GANONG, W. F. 1889. The economic mollusca of Acadia. Bull. Nat. Hist. Soc. N.B. 8. 116 p.

GARCON, V. C., K. D. STOLZENBACH, AND D. M. ANDERSON. 1986. Tidal flushing of an estuarine embayment subject to recurrent dinoflagellate blooms. Estuaries **9:** 179–187.

GARRETT, C. J. R., J. R. KEELEY, AND D. A. GREENBERG. 1978. Tidal mixing versus thermal stratification in the Bay of Fundy and Gulf of Maine. Atmosphere Ocean **16:** 403–423.

GEYER, W. R., AND OTHERS. 1992. Physical oceanographic investigation of Massachusetts and Cape Cod Bays. Mass. Bay Program Final Rep. 250 p.

HAYHOME, B. A., D. M. ANDERSON, D. M. KULIS, AND D. J. WHITTEN. 1989. Variation among congeneric dinoflagellates from the northeastern United States and Canada. 1. Enzyme electrophoresis. Mar. Biol. **101:** 427–435.

HOLLIGAN, P. M. 1979. Dinoflagellate blooms associated with tidal fronts around the British Isles, p. 249–256. *In* Toxic dinoflagellate blooms: Proc. 2nd Int. Conf. Elsevier.

HURST, J. W., JR. 1975. History of paralytic shellfish poisoning on the Maine coast, p. 525–528. *In* Proc. 1st Int. Conf. on Toxic Dinoflagellate Blooms. Mass. Sci. Technol. Found.

INCZE, L., AND C. M. YENTSCH. 1981. Stable density fronts and dinoflagellate patches in a tidal estuary. Estuarine Coastal Mar. Sci. **13:** 547–556.

JAMIESON, G. S., AND R. A. CHANDLER. 1983. Paralytic shellfish poison in sea scallops (*Placopecten magellanicus*) in the West Atlantic. Can. J. Fish. Aquat. Sci. **40:** 313–318.

KEAFER, B. A., AND D. M. ANDERSON. 1993. Use of remotely-sensed sea surface temperatures in studies of *Alexandrium tamarense* bloom dynamics, p. 763–768. *In* Toxic phytoplankton blooms in the sea. Proc. 5th Int. Conf. on Toxic Marine Phytoplankton. Elsevier.

LA BARBERA-SANCHEZ, A., S. HALL, AND E. FERRAZ-REYES. 1993. *Alexandrium* sp., *Gymnodinium catenatum* and PSP in Venezuela, p. 763–768. *In* Toxic phytoplankton blooms in the sea. Proc. 5th Int. Conf. on Toxic Marine Phytoplankton. Elsevier.

LEWIS, C. M., C. M. YENTSCH, AND B. DALE. 1979. Distribution of *Gonyaulax excavata* resting cysts in the sediments of Gulf of Maine, p. 235–238. *In* Toxic dinoflagellate blooms: Proc. 2nd. Int. Conf. Elsevier.

MARANDA, L., D. M. ANDERSON, AND Y. SHIMIZU. 1985. Comparison of toxicity between populations of *Gonyaulax tamarensis* of eastern North American waters. Estuarine. Coastal Shelf Sci. **21:** 401–410.

MULLIGAN, H. F. 1975. Oceanographic factors associated with New England red-tide blooms, p. 23–40. *In* Proc. 1st Int. Conf. on Toxic Dinoflagellate Blooms. Mass. Sci. Technol. Found.

NASSIF, J., AND R. J. TIMPERI. 1993. Survey of molluscan shellfish for certain marine biotoxins. Mass. Dep. Public Health. Final Rep. 140 p.

PFIESTER, L. A., AND D. M. ANDERSON. 1987. Dinoflagellate life cycles and their environmental control, p. 611–648. *In* F. J. R. Taylor [ed.], The biology of dinoflagellates. Blackwell.

PINGREE, R., P. PUGH, P. HOLLIGAN, AND G. FORSTER. 1975. Summer phytoplankton blooms and red tides along tidal fronts in the approaches to the English Channel. Nature **258:** 672–677.

SAKO, Y., C. H. KIM, AND Y. ISHIDA. 1992. Mendelian inheritance of paralytic shellfish poisoning toxin in the marine dinoflagellate *Alexandrium catenella.* Biosci. Biotechnol. Biochem. **56:** 692–694.

SCHOLIN, C. A., G. M. HALLEGRAEFF, AND D. M. ANDERSON. 1995. Molecular evolution of the *Alexandrium tamarense* "species complex" (Dinophyceae): Dispersal in the North American and West Pacific regions. Phycologia **34:** 472–85.

———, M. HERZOG, M. SOGIN, AND D. M. ANDERSON. 1994. Identification of group- and strain-specific genetic markers for globally distributed *Alexandrium* (Dinophyceae) species: 2. Sequence analysis of a fragment of the LSU rRNA gene. J. Phycol. **30:** 999–1011.

SCHREY, S. E., E. J. CARPENTER, AND D. M. ANDERSON. 1984. The abundance and distribution of the toxic dinoflagellate, *Gon-*

yaulax tamarensis, in Long Island estuaries. Estuaries **7:** 472–477.

SELIGER, H. H., M. A. TYLER, AND K. R. MCKINLEY. 1979. Phytoplankton distributions and red tides resulting from frontal circulation patterns, p. 239–248. *In* Toxic dinoflagellate blooms: Proc. 2nd Int. Conf. Elsevier.

SHUMWAY, S. E., S. A. SHERMAN, AND A. E. CEMBELLA. 1994. Accumulation of paralytic shellfish toxins by surfclams, *Spisula solidissima* (Dillwyn, 1897) in the Gulf of Maine: Seasonal changes, distribution between tissues, and notes on feeding habits. Nat. Toxins **2:** 236–251.

———, S. SHERMAN-CASWELL, AND J. W. HURST. 1988. Paralytic shellfish poisoning in Maine: Monitoring a monster. J. Shellfish Res. **7:** 643–652.

SIMPSON, J. H., D. J. EDELSTEIN, A. EDWARDS, N. C. G. MORRIS, AND P. B. TETT. 1979. The Islay Front: Physical structure and phytoplankton distribution. Estuarine Coastal Mar. Sci. **9:** 713–726.

THAYER, P. E., J. W. HURST, C. M. LEWIS, R. SELVIN, AND C. M. YENTSCH. 1983. Distribution of resting cysts of *Gonyaulax tamarensis* var. *excavata* and shellfish toxicity. Can. J. Fish. Aquat. Sci. **40:** 1308–1314.

TOWNSEND, D. W., J. P. CHRISTENSEN, D. K. STEVENSON, J. J. GRAHAM, AND S. B. CHENOWETH. 1987. The importance of a plume of tidally mixed water to the biological oceanography of the Gulf of Maine. J. Mar. Res. **45:** 699–728.

TYLER, M. A., D. W. COATS, AND D. M. ANDERSON. 1982. Encystment in a dynamic environment: Deposition of dinoflagellate cysts by a frontal convergence. Mar. Ecol. Prog. Ser. **7:** 163–178.

———, AND H. H. SELIGER. 1978. Annual subsurface transport of a red tide dinoflagellate to its bloom area: Water circulation patterns and organism distributions in the Chesapeake Bay. Limnol. Oceanogr. **23:** 227–246.

WHITE, A. W., J. NASSIF, S. E. SHUMWAY, AND D. K. WHITTAKER. 1993. Recent occurrence of paralytic shellfish toxins in offshore shellfish in the northeastern United States, p. 435–440. *In* Toxic phytoplankton blooms in the sea. Proc. 5th Int. Conf. on Toxic Marine Phytoplankton. Elsevier.

YENTSCH, C. S., AND N. GARFIELD. 1981. Principal areas of vertical mixing in the waters of the Gulf of Maine, with reference to the total productivity of the area, p. 303–312. *In* J. F. R. Gowen [ed.], Oceanography from space. Plenum.