Marine Biology 112, 165–174 (1992)

Marine Biology
International Journal on Life in Oceans and Coastal Waters

Toxic phytoplankton blooms in the southwestern Gulf of Maine: testing hypotheses of physical control using historical data

P. J. S. Franks* and D. M. Anderson

Woods Hole Oceanographic Institution, Woods Hole, Massachusetts 02543, USA

Date of final manuscript acceptance: September 17, 1991. Communicated by J. Grassle, New Brunswick

Abstract. Blooms of the toxic dinoflagellate *Alexandrium tamarense* Lebour have been nearly annual features along the coasts of southern Maine, New Hampshire and Massachusetts, USA, since 1972. In 1990 two hypotheses which have been used to explain the initiation of these blooms in the southwestern portion of the Gulf of Maine were tested using historical records of shellfish toxicity, wind, and river flow. The first hypothesis states that the blooms were initiated or advected to shore by wind-driven coastal upwelling. The second states that established blooms were advected from north to south alongshore in a coastally trapped buoyant plume of water. Of the eleven years examined (1979 to 1989), we found seven cases inconsistent with the wind-driven upwelling hypothesis, and only one case (1985) which contradicts the plume-advection hypothesis. 1985 was an unusual year in many respects, and we suggest that some other mechanism was responsible for the toxic outbreaks. In addition, the wind-driven upwelling hypothesis could not explain the observed north-to-south temporal progression of toxicity each year. The plume-advection hypothesis was found to best explain the datails of the timing and spread of shellfish toxicity in Gulf of Maine waters to the south of Penobscot Bay, Maine. These include the variable north-to-south progression with time, the presence of a toxin-free zone south of Cape Ann, Massachusetts, the sporadic nature of toxic outbreaks south of Massachusetts Bay, and the apparently rare occurrence of high toxicity levels well offshore on Nantucket Shoals and Georges Bank.

Introduction

Since 1972, blooms of the toxic dinoflagellate *Alexandrium tamarense*[1] have caused nearly annual shellfish bed closures along the coasts of southern Maine, New Hampshire and Massachusetts, USA. Shellfish that ingest this dinoflagellate can accumulate its potent neurotoxins, becoming agents of paralytic shellfish poisoning (PSP). Monitoring programs set up by state agencies regularly sample coastal shellfish and test for PSP toxins using the mouse assay (e.g. Hurst 1979, Hurst et al. 1985, Shumway et al. 1988). Such programs, combined with laboratory experiments, have demonstrated a clear relationship between the toxicity of the mussel *Mytilus edulis* and the presence of *A. tamarense* cells. Mussels accumulate measurable toxin in as little as 24 h after exposure to *A. tamarense* cells (Gainey and Shumway 1988, Bricelj et al. 1990). In the analyses below, we use the toxicity of *M. edulis*, and the clam, *Mya arenaria*, as surrogates for the presence or absence of *A. tamarense* in the field.

Between southwestern Maine and Massachusetts, blooms of *Alexandrium tamarense* generally begin in April or early May, causing PSP outbreaks which appear to progress from Maine southward to Massachusetts. This spatial and temporal progression of toxic events, and presumably *A. tamarense* blooms, could be due to either local growth of the cells, or advection of cells along the coast. The former type of bloom was first discussed by Mulligan (1973, 1975), Hartwell (1975), and Seliger et al. (1979) who hypothesized that the blooms were initiated by wind-driven upwelling in the southern Gulf of Maine. They suggested that populations of *A. tamarense*, growing at mid-depth in offshore waters were advected onshore into the coastal zone where they formed local blooms. This hypothesis, which presumes a population of *A. tamarense* growing just offshore, below the pycnocline, predicts that toxic outbreaks in intertidal shellfish should be preceded by wind-driven upwelling and that the *A. tamarense* cells are of nearby, offshore origin.

* Current address: College of Oceanography, Oregon State University, Corvallis, Oregon 97331-5503, USA

[1] *Alexandrium tamarense* and *Alexandrium fundyense* were formerly included in the genera *Protogonyaulax* or *Gonyaulax* but are now accepted as *Alexandrium* species (Steidinger and Moestrup 1990). Both species bloom in the Gulf of Maine (Anderson unpublished data), but since discrimination between them is impossible for large-scale field programs or when referring to shellfish toxicity, only the more familiar name *Alexandrium tamarense* will be used here

Martin and Main (1981) performed the first study of the spatial and temporal distribution of *Alexandrium tamarense* in the embayments to the north of Cape Ann. They concluded that *A. tamarense* moved into Massachusetts coastal waters as a direct result of the southward movement of prevailing currents, and that occasional reports of minimal increases in shellfish toxicity or the presence of *A. tamarense* in Gloucester and Essex as early as March represented totally independent occurrences that could not have been precursors for the toxicity outbreaks in late June and early July. These authors thus make a clear distinction between local populations of *A. tamarense* and the exogenous populations which are thought to cause more widespread toxicity outbreaks.

Franks and Anderson (1992) hypothesized that the annual southward progression of toxicity was a result of alongshore advection of an *Alexandrium tamarense* population in a coastally trapped buoyant plume. The plumes were suggested to form by enhanced outflow of the Androscoggin and Kennebec Rivers in Maine (Fig. 1), following heavy spring rainfalls in late April or early May. The plumes carry *A. tamarense* populations for hundreds of kilometers along the coast, delivering them to shellfish beds along the way. Alongshore winds were hypothesized to influence the motion of the plume: upwelling-favorable (northeastward) winds would slow the plume and possibly move it offshore, while downwelling-favorable (southwestward) winds would hold the plume to the coast and speed it alongshore to the southwest. This hypothesis predicts that spring PSP outbreaks are likely to follow a peak in the regional river flow rate, and that downwelling-favorable winds should speed the southward progression of PSP toxicity. Furthermore, in years of high river discharge, the timing of toxic outbreaks should be relatively independent of the wind, while in years of low discharge, the wind should have greater control over the toxicity patterns, due to the dependence of plume velocities on volume of river discharge. The present study tested the wind-driven and plume-advection hypotheses, using historical records of shellfish toxicity, river flow rates, and wind records. The data and conclusions are site-specific and only apply to the areas examined, namely Gulf of Maine coastal waters south of Penobscot Bay.

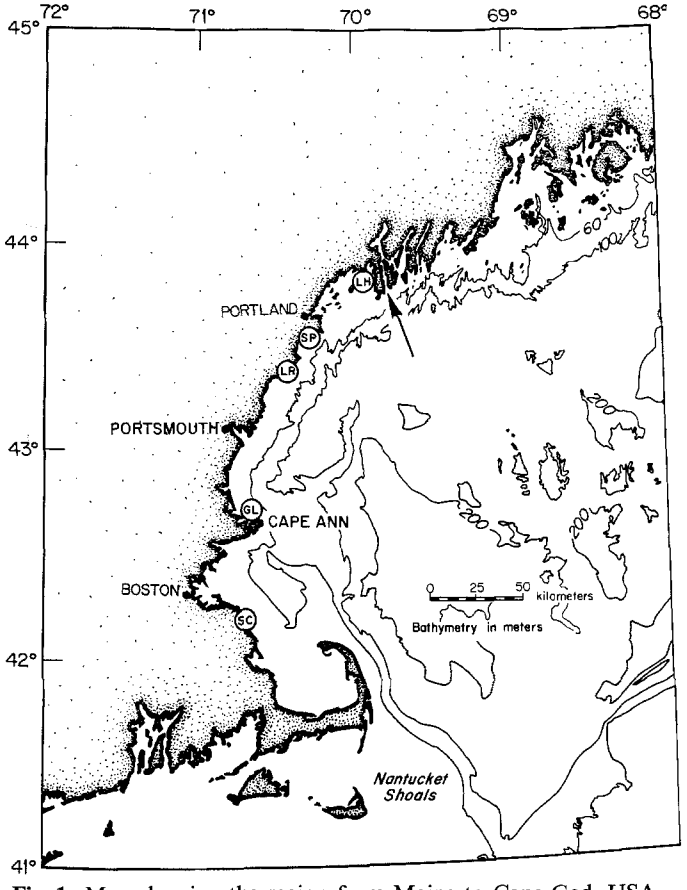

Fig. 1. Map showing the region from Maine to Cape Cod, USA, with the five sampling stations indicated: Lumbos Hole (LH), Spurwink River (SP), Little River (LR), Gloucester (GL), and Scituate (SC). Estuary into which the Androscoggin and Kennebec Rivers empty is indicated by the arrow

Description of the data sets

Shellfish toxicity data were obtained through the Department of Marine Resources, Maine, and the Department of Environmental Quality Engineering, Massachusetts. Five stations were selected from the many which are regularly sampled. These stations were Lumbos Hole (LH), Spurwink River (SP) and Little River Kennebunkport (LR), all in Maine, and Gloucester (Annisquam Yacht Club; GL) and Scituate (Yacht Club; SC), Massachusetts (Fig. 1). These stations were chosen for the completeness of their historical records, their geographic distribution along the coast, their exposure to coastal waters, and their regular manifestation of shellfish toxicity.

Shellfish toxicity caused by *Alexandrium tamarense* Lebour was measured using the mouse assay (Association of Official Agricultural Chemists 1975). The different strains of mice used in Maine and Massachusetts showed different sensitivities to the dinoflagellate neurotoxins, giving different threshold responses for the two data sets (S. Sherman-Caswell personal communication). In addition, different species of shellfish show different intoxication and depuration rates (Shumway et al. 1988). The mussel *Mytilus edulis* L. becomes toxified very rapidly (Gainey and Shumway 1988, Bricelj et al. 1990), and thus can be used as an indicator for early exposure to toxic cells, and the onset of shellfish toxicity. The clam *Mya arenaria* L., which depurates the toxins fairly slowly, can be used to indicate the disappearance of the cells and toxicity. Data from Maine have continuous records of toxicity in *M. edulis*, whereas the Massachusetts data reflect the mid-season switch from monitoring *M. edulis* to *M. arenaria*, creating a discontinuity in the toxicity records. Thus the data for the initiation of toxicity are internally consistent (all *M. edulis*), while the data for the duration of toxicity are not (*M. edulis* or *M. arenaria*). For these reasons, toxicity records were converted to a binary form: 1 = measurable toxicity [>58 μg 100 g^{-1} shellfish meat (Maine); >40 μg 100 g^{-1} shellfish meat (Massachusetts)], 0 = no measurable toxicity. Thus the magnitude of the toxicity is not considered in the present analyses.

The freshwater flow statistics for the Androscoggin River (indicated in Fig. 1) were obtained through the United States Geological Survey. These data give the mean daily flow of the river, measured well inland of any tidal influence. The Androscoggin and Kennebec Rivers empty into the same estuary, and show strongly correlated flow patterns and volumes. For April to October 1987 to 1989, the correlation between the two river flows was $r^2 = 0.90$ (Kennebec $= 33\ m^3 s^{-1} + 1.16 \times$ Androscoggin). Only the Androscoggin River flow rates are used here since the Kennebec River data con-

tained gaps, but the importance of the Kennebec River should be recognized. Using only the Androscoggin River data has no effect on the arguments presented below. The drainage basin of the Androscoggin River is $\sim 8500\ km^2$, and of the Kennebec is $\sim 14\,000\ km^2$. The combined drainage areas are equivalent to 25% of the area of Maine.

Wind data were obtained from the National Climatic Data Center (National Oceanographic and Atmospheric Administration). These hourly observations of wind speed and direction at Logan Airport, Boston, Massachusetts were rotated into the coordinate system of the coast by subtracting 30°. The data were resolved into across- and alongshore wind stresses (Pa), using the algorithm of Large and Pond (1981). The wind stresses were smoothed and gridded to points every 3 h using the objective mapping routine of Levy and Brown (1986). This process smooths the spikes in the data, making them easier to visualize, and reduces the number of points to be manipulated. The weather patterns show the Boston data to be representative of a region from Cape Cod to southern Maine. For the present analyses, the slight lag of the Maine winds relative to the Boston winds is not important, so the wind patterns were considered to be identical throughout the study region.

Toxicity records were available for the years 1973 to 1989 from Massachusetts. However, regular sampling of stations did not become commonplace in both Maine and Massachusetts until 1979. For this reason the years to be analyzed extend only from 1979 to 1989. No toxicity data were found for Scituate for 1982.

Observations

Toxicity

Figs. 2 to 5 show the binary toxicity time series and flow rate of the Androscoggin River for the years 1979 to 1989. An average trend of north-to-south movement of toxicity can be seen for all years (Fig. 6a). For years such as 1979, the first samples taken in Maine were toxic, so the first occurrence of toxicity cannot be specified. In other years, the time between samples at a given station is large enough that the north-to-south trend could be an artifact of the sampling (e.g. 1979). However, the mean dates of initiation of toxicity at Lumbos Hole, Spurwink River, and Little River, Maine were significantly earlier ($P<0.05$) than the initiation of toxicity at Gloucester or Scituate, Massachusetts. Since the date of initiation of toxicity varied from year to year, the data for the first occurrence of toxicity at the five stations, T_i, were scaled by the time interval between the first occurrence of toxicity at Lumbos Hole and Gloucester, as follows:

$$T_i = \frac{T_i - T_{LH}}{T_{GL} - T_{LH}}. \quad (1)$$

Here T_i is the time of initiation of toxicity at Stn i (i = LH, SP, LR, GL or SC). Thus Lumbos Hole scales to zero, and Gloucester to unity for each year, with the other stations distributed around zero and one. When the data are plotted in this way (Fig. 6b), the north-to-south trend is enhanced. Even Spurwink River is found to show initiation of toxicity significantly later than Lumbos Hole, which is 50 km to the north ($P<0.05$). Toxicity at Gloucester and Scituate occurs significantly later than at the more northerly stations ($P<0.05$). The large confidence intervals on the Scituate data are due to the low number of data points; no toxicity was detected at Scituate in 1981, 1983 or 1987, and no data were available for 1982.

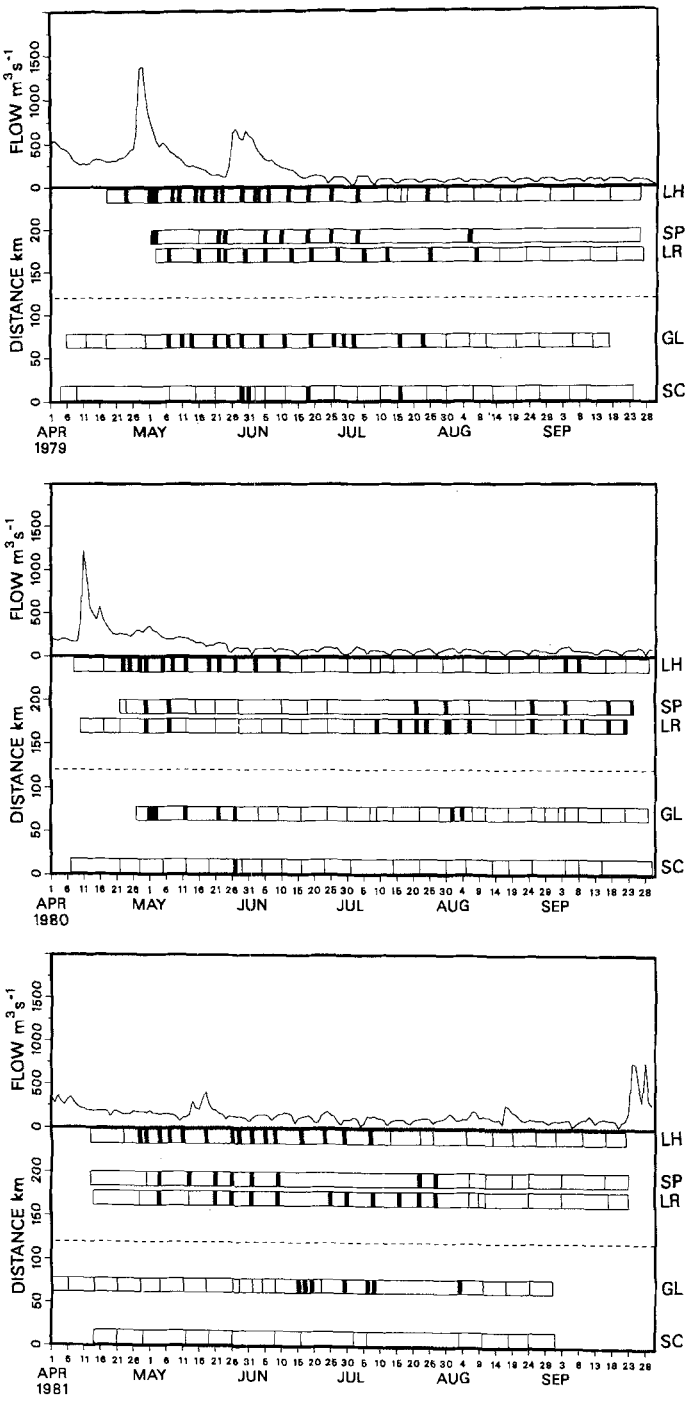

Fig. 2. River flow and toxicity data for the southwest coast of the Gulf of Maine 1979 to 1981. Lower section of each panel: shellfish toxicity vs time. Heavy vertical lines indicate toxic samples, while light vertical lines indicate non-toxic samples. The alongshore distance between stations is indicated on the vertical axis. Stn abbreviations as in Fig. 1. Top section of each panel: flow rate ($m^3\ s^{-1}$) of the Androscoggin River, for the dates indicated on the horizontal axis

The data from 1985 weaken the apparent north-to-south trend since Little River was the first station to show toxicity in that year, giving a negative scaled time of PSP initiation.

The first measurable PSP toxin is usually detected in southern Maine between mid-April and mid-May. The

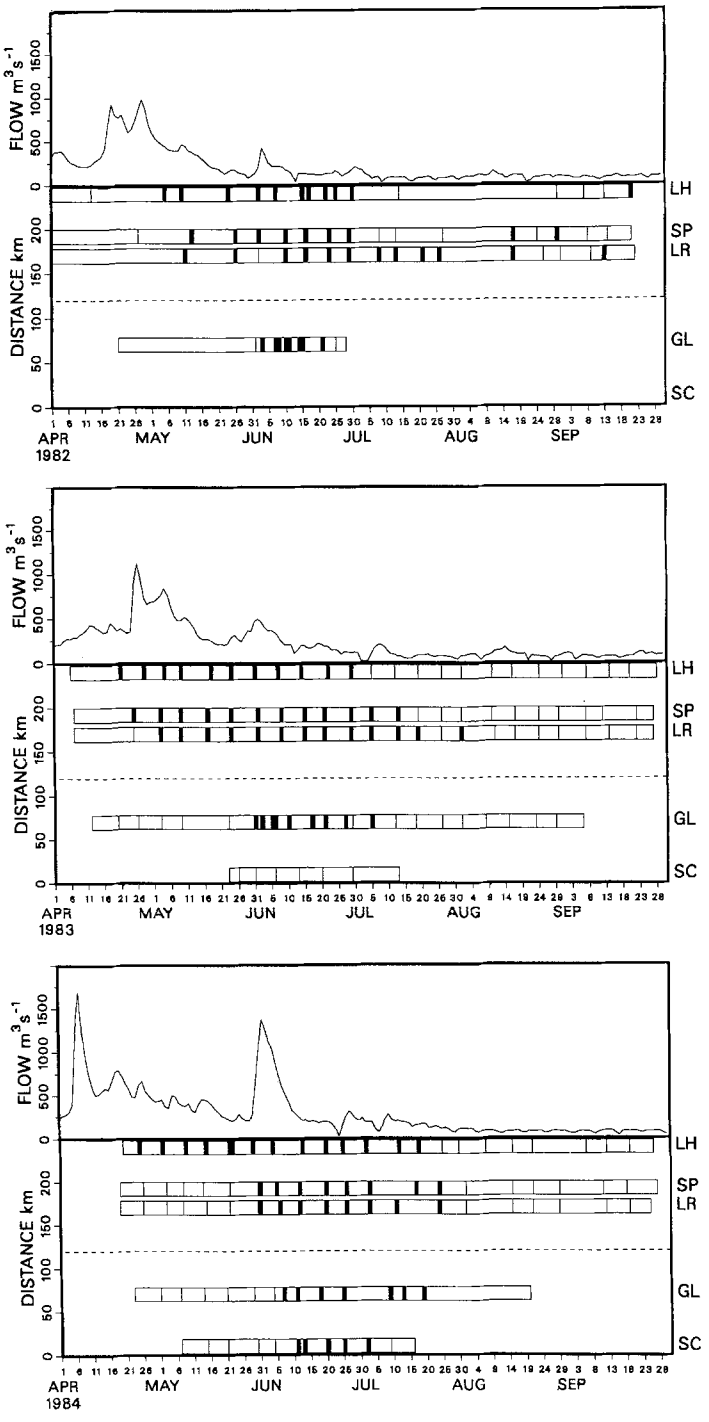

Fig. 3. River flow and toxicity date for the southwest coast of the Gulf of Maine 1982 to 1984. Legend as in Fig. 2. No data available for Scituate, 1982

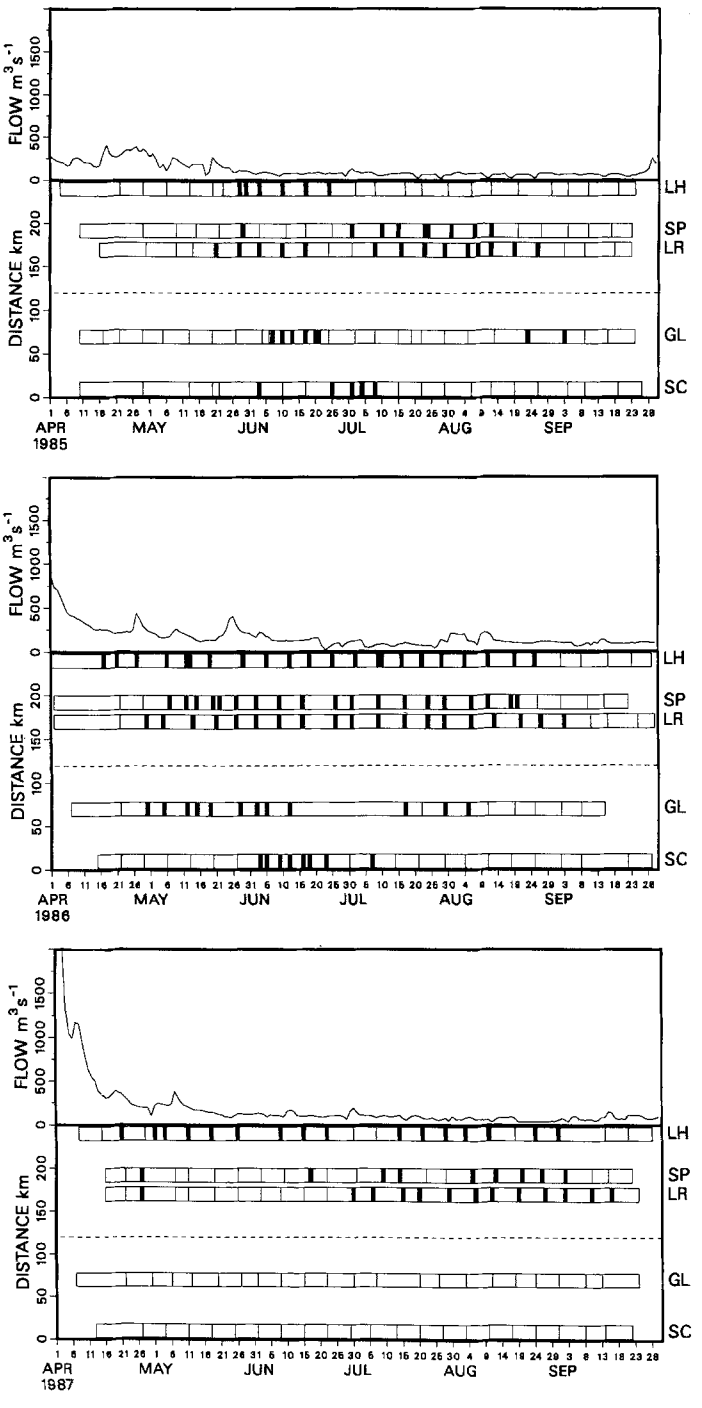

Fig. 4. River flow and toxicity for the southwest coast of the Gulf of Maine 1985 to 1987. Legend as in Fig. 2

one exception to this was the year 1985, when the first toxicity was seen in the last ten days of May (Fig. 3). As we shall see below, 1985 presents the only case contradicting the plume-advection hypothesis.

Two temporal patterns of toxicity can be seen from Fig. 2 to 5. In years such as 1980, 1985 and 1987, toxicity showed a patchy temporal distribution, with several outbreaks of short duration at each station. In most other years, the toxicity persisted over several months. These patterns appear to be related to the volume of freshwater outflow. Years with low flow during the summer show patchy toxicity, whereas years with high flow, particularly in early May, show sustained toxicity.

Freshwater flow

The outflow of the Androscoggin river generally shows two distinct peaks: the first in early April, driven by meltwater runoff, and the second in late April to early May,

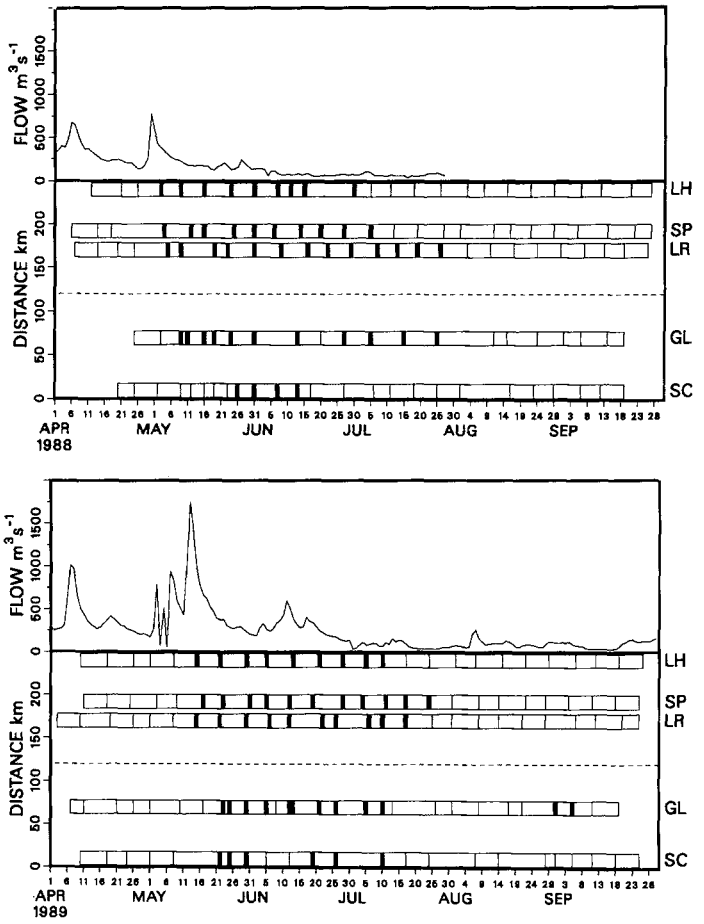

Fig. 5. River flow and toxicity data for the southwest coast of the Gulf of Maine 1988 to 1989. Legend as in Fig. 2

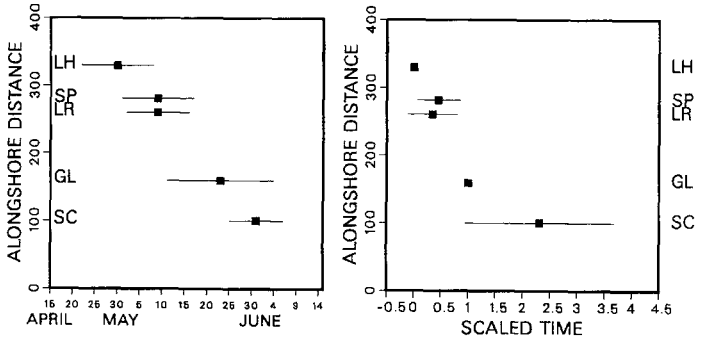

Fig. 6. Toxicity data. (a) Average time of initiation of toxicity at Lumbos Hole (LH), Spurwink River (SP), Little River (LR), Gloucester (GL), and Scituate (SC). (b) Scaled time lag of initiation of toxicity relative to Lumbos Hole. The error bars are 95% confidence intervals

in response to rainfall. It is the second flow peak (late April to early May) which is hypothesized to control the toxicity pattens. In every year, the first toxicity at Gloucester is preceded by a peak in the flow rate of the Androscoggin River (Fig. 2 to 5). In years such as 1981, 1985 and 1986 this flow peak was low; it was absent in May of 1987, the only year since 1972 that shellfish toxicity was not detected in northern Massachusetts.

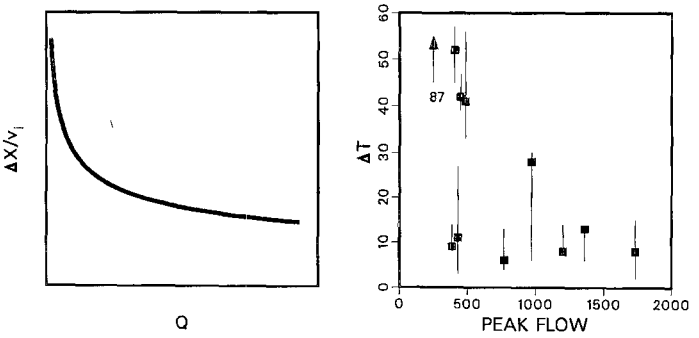

Fig. 7. Timing of toxicity vs river flow rate. (a) Predicted relationship of transit time of toxicity alongshore ($\Delta x/v_i$) to flow rate of the river (Q). (b) Time interval between initiation of toxicity at Lumbos Hole and Gloucester (Δt, days), plotted vs the peak flow volume of the Androscoggin River (peak flow, $m^3\ s^{-1}$). The error bars indicate the uncertainty of the estimate of Δt due to the interval between the last non-toxic sample and the first toxic sample. 1987 did not show toxicity at Gloucester, so Δt is effectively infinite

The plume-advection hypothesis predicts that years with high flow rates should show reduced transit times of toxicity along the coast. That is, the interval between the first occurrence of toxicity in Maine, and the initiation of toxicity at Gloucester should be smaller in years of large flow. Chao (1988) recasts some work of Kao et al. (1978) to give an equation relating the speed of a river plume along a coast (intrusion speed, v_i) with the volume of flow of the river:

$$v_i = (g' Q)^{1/3} \tag{2}$$

where Q is the flow volume per unit width of the channel, and g' is the reduced gravity,

$$g' = g \frac{\varrho' - \varrho}{\varrho'}. \tag{3}$$

Here g is the acceleration due to gravity, ϱ is the density of the freshwater, and ϱ' is the density of the ambient offshore water. We can rearrange (2) to give the transit time, Δt, between points on the coast separated by a distance Δx:

$$\Delta t = \frac{\Delta x}{v_i} = \frac{\Delta x}{(g' Q)^{1/3}}. \tag{4}$$

The parameter g' will tend to decrease over time as mixing reduces the density contrast of the plume. Using a constant g' and Δx in Eq. 4 will thus give estimates of Δt which are likely to be low. Nevertheless, the qualitative argument should hold: a plot of Δt versus Q should yield a shape similar to that of Fig. 7a. To test this prediction, we plotted the peak flow volume of the Androscoggin River in the interval preceding the first occurrence of toxicity ($\equiv Q$), vs the time interval between the initiation of toxicity at Lumbos Hole, Maine, and at Gloucester, Massachusetts ($\equiv \Delta t$) (Fig. 7b). The points of Fig. 7b show the same basic pattern as the curve of Fig. 7a: large Δt for low flows ($< 500\ m^3\ s^{-1}$), and low Δt for high peak flow volumes ($> 1000\ m^3\ s^{-1}$). There is a great deal of scatter of points about the predicted curve, however. Much of this scatter is explained by the wind forcing, as discussed below.

The shape of the curve in Fig. 7a suggests that for low river flows ($<500\ m^3\ s^{-1}$), the time interval between toxic events along the coast, Δt may vary considerably. For higher flows, Δt should be almost constant, varying little with flow volume. This trend is reflected in the data of Fig. 7b: for low flows Δt varied from 9 to 53 d. For flows $>1000\ m^3\ s^{-1}$, Δt was between 8 and 13 d. The distance between Lumbos Hole and Gloucester is ~ 170 km. Assuming $\Delta t = 10$ d (high flow volume), then v_i is calculated to be $v_i \sim 0.2\ m\ s^{-1}$. This is the same as the plume velocity calculated independently in Franks and Anderson (1992), using hydrographic data and Margule's equation. For low flow rates, we might expect plume velocities of $v_i \sim 0.02\ m\ s^{-1}$. At this rate, the plume would take ~ 85 d to cover the distance from Lumbos Hole to Gloucester. Note that in 1987, a low-flow year, shellfish toxicity was not measurable at Gloucester or Scituate, despite its occurrence at stations to the north in Maine. From Fig. 6a, the average progression rate of toxic events along the coast, or plume velocity, is given by the slope of the regression line fit to the mean values. This slope is 8.23 km d^{-1}, ($r^2 = 0.98$), or $\sim 0.1\ m\ s^{-1}$ to the south, in good agreement with the values calculated above. Note that these calculations do not depend on our choice of the Androscoggin River as a flow indicator.

The river flow data thus support the plume-advection hypothesis: every toxicity event in the south of the study area was preceded by an increase in the flow rate of the Androscoggin River. Years of high flow tended to show small transit times, Δt, between the first occurrence of toxicity at Lumbos Hole, and at Gloucester. The advective speed of the plume for high flow rates, calculated from $\Delta x/\Delta t$, was the same as that calculated independently using the observed density of water masses from hydrographic surveys in the study area.

Wind effects

As discussed in Franks and Anderson (1992) and by Mulligan (1973, 1975), Hartwell (1975) and Seliger et al. (1979), the wind is likely to have an influence on the timing and location of toxic shellfish outbreaks. The last-named three authors predicted that toxic events should be preceded by upwelling-favorable winds, whereas the first predicted that downwelling-favorable winds should have the greater effect by holding the buoyant plume (and thus the toxic cells) to the coast, and accelerating it southward. The plume-advection hypothesis also predicts that sustained upwelling-favorable winds in years of low flow may slow or halt the progression of the plume, and potentially halt the alongshore spread of toxicity. This would occur because winds from the south would force a weak plume northward and offshore.

To obtain more quantitative information on the effects of the wind, the model of Janowitz and Pietrafesa (1980) was employed to calculate the wind-forced alongshore velocities. This very simple time-dependent model gives a solution for the wind-induced quasi-geostrophic motion of interior fluid (independent of surface and bottom boundary layers), given rigid lid, Boussinesq and hydrostatic approximations. The rigid lid approximation assumes that vertical motions of the sea surface are negligible. The Boussinesq approximation allows the use of a constant density over a region, except for buoyancy effects. The hydrostatic approximation assumes that the vertical gradient of pressure is dependent only on the vertical gradient of density (i.e., vertical accelerations, vertical Coriolis accelerations and vertical stress gradients are neglected). Alongshore variation is assumed negligible.

The alongshore momentum equation was integrated vertically, so that changes in the alongshore flux are a balance of the surface alongshore wind stress, $\tau^y(t)$, and the bottom frictional stress. If a linear bottom stress is used, an equation for the time-dependent alongshore velocity, $v(t)$, is obtained:

$$v(t) = \frac{1}{\varrho h} \int_0^t \tau^y(t') \exp\left[-fd(t-t')/h\right] dt'. \tag{5}$$

The linear bottom stress, τ^B, was parameterized as

$$\tau^B = \varrho c_1 v(-h, t) \tag{6a}$$

$$d = c_1/f \tag{6b}$$

with $c_1 = 2.5 \times 10^{-4}\ m\ s^{-1}$, and the Coriolis frequency, $f = 1 \times 10^{-4}\ s^{-1}$. The water depth, h, was set at a constant 20 m, and 1024 kg m^{-3} was used for the water density, ϱ. The model described by Eq. 5 indicates that the alongshore velocity of the interior fluid is the result of an exponential weighting of the present and past alongshore wind stresses.

The alongshore current is driven by the across-shelf pressure gadient created by the alongshore wind stress. The criteria stipulated by Janowitz and Pietrafesa (1980) for applicability of the model to this region are met (Franks 1990), although baroclinic effects may be important. However, the calculations of Clarke and Brink (1985) indicate that waters respond barotropically on wide, gently-sloping shelves, and that even in baroclinic cases, flow over the shelf tends to be barotropic.

In applying model Eqs. 5 and 6, any alongshore advection due to a plume will be ignored. If a plume were present, it would contribute a southward component to the velocities plotted in the figures below. Thus, the calculated alongshore velocities of the coastal waters are inaccurate by some unknown factor. We recognize this limitation, and will use the results of this model mainly to indicate directional trends, rather than as a predictive tool for the alongshore velocity structure. The alongshore direction of motion of a particle as predicted by this model is probably accurate for upwelling and downwelling-favorable wind stresses.

The model was applied to the data by calculating the wind-driven alongshore velocities of water at the coast ($y = 0$). The results of these calculations can be seen in Figs. 8 and 9 for the years 1979 to 1989. The dates of the first measured toxicity at each of the five stations along the coast are also plotted. The upwelling hypothesis predicts that toxicity at each station should be preceded by an upwelling-favorable wind event. A sustained up-

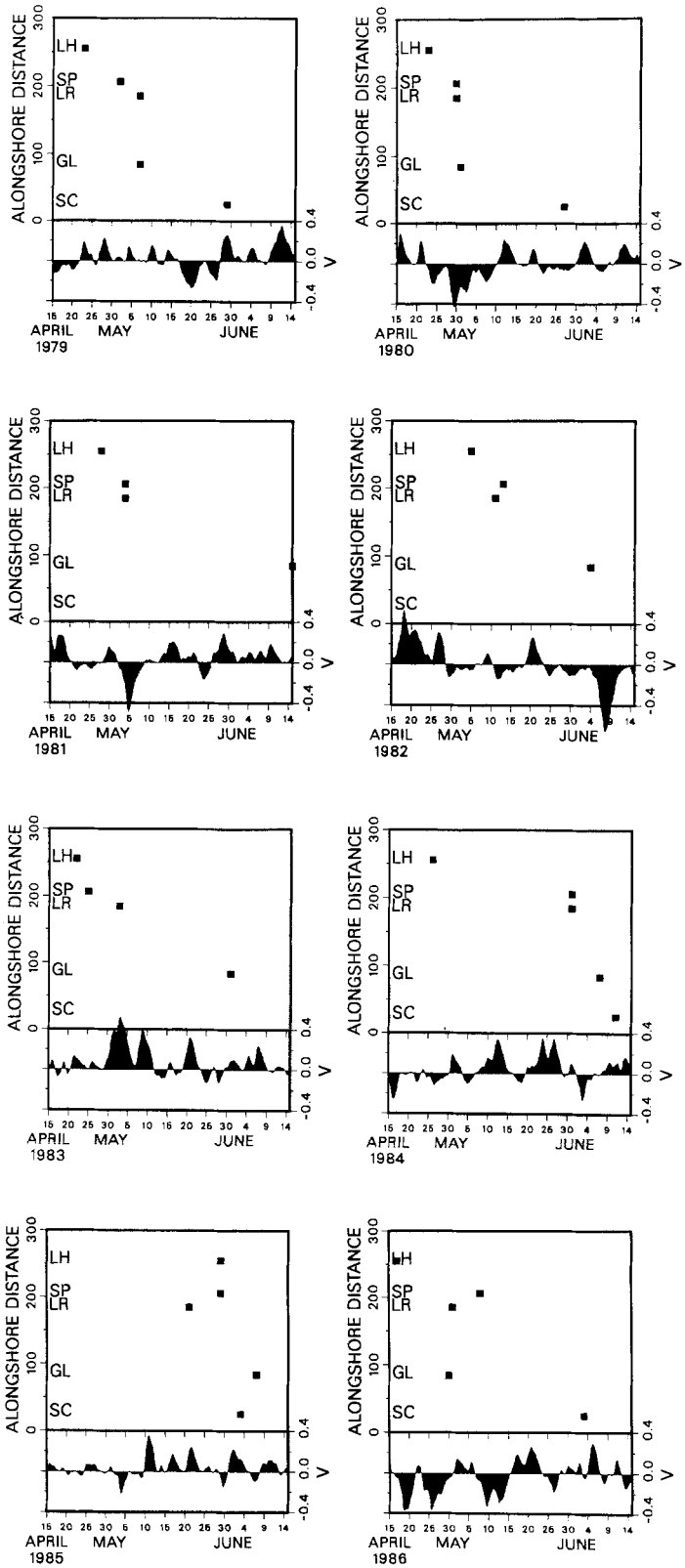

Fig. 8. Time of initiation of toxicity and wind-driven alongshore velocities 1979 to 1986. Data points indicate the date of initiation of toxicity at stn indicated to the left (LH, SP, LR, GL and SC). Stn abbreviations as in Fig. 1. The lower section of each panel shows the wind-driven alongshore velocity, v (m s^{-1}) calculated from Eqs. 5 and 6. Positive velocities are toward the northeast, driven by an upwelling-favorable wind. The alongshore distance (in km) between the stations is shown on the vertical axis. Year is shown at the lower left of each plot

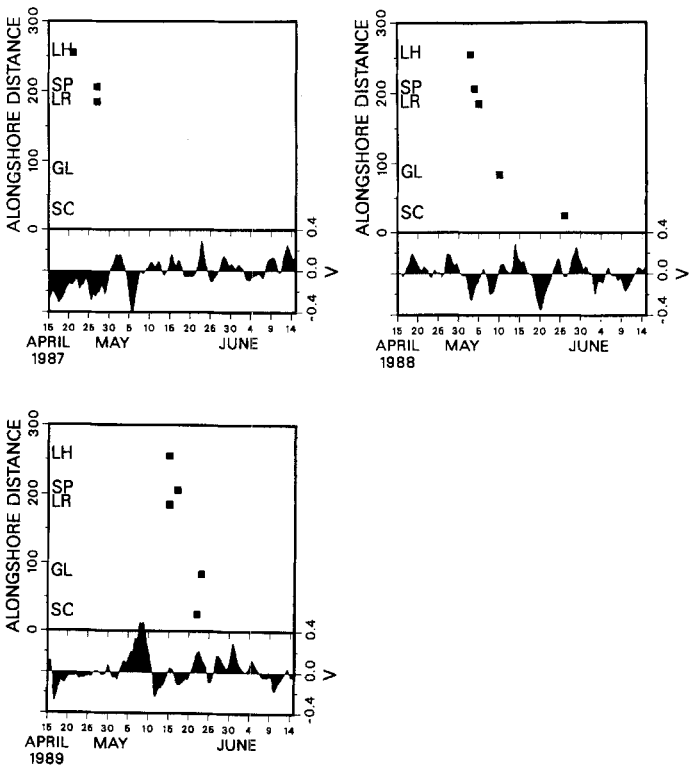

Fig. 9. Time of initiation of toxicity and wind-driven alongshore velocities 1987 to 1989. Legend as in Fig. 8

welling event would appear as northward velocities of the water, i.e., the velocity vectors would be positive as was observed for 1979, 1983, 1984 and 1985. However, the data from 1980, 1981, 1982, 1986, 1987, 1988 and 1989 all show downwelling prior to the occurrence of toxicity. In contrast, the plume-advection hypothesis predicts that southern-most toxicity events are likely to follow downwelling events (negative velocity vectors). This prediction is supported by the data from 1979, 1980, 1982, 1983, 1984, 1988 and 1989. In 1983 and 1984, the winds were predominantly upwelling-favorable, yet the initiation of toxicity at the southern stations followed a period of downwelling-favorable winds. Seliger et al. (1979) suggested that the *Alexandrium tamarense* cells were distributed alongshore in offshore waters. Thus we might expect upwelling-favorable winds to cause PSP outbreaks simultaneously over large regions of coastline. This appears to be the case in only one year, 1985.

The plume-advection hypothesis predicts that in years of high river runoff, the alongshore advection of the plume should largely control the spread of toxicity, with only slight moderation by wind. From Figs. 2 to 5, we see that 1979, 1980, 1982, 1983, 1984, 1988 and 1989 were all years of high peak river flow, and in all cases the toxicity spread from north to south, even though in many cases the wind stresses were northward, opposing the southward flow (e.g. 1979, 1983, 1984, 1988 and 1989). Thus the dominance of river flow over wind effects is evident in years of high flow.

The plume-advection hypothesis also predicts that in years of low flow, winds should have more impact on the

spread of toxicity, since the plume velocities would be weak. 1981, 1985, 1986 and 1987 were years of low river flow, and except for 1985, toxicity was either delayed or absent from the southern stations. In 1987 neither Gloucester nor Scituate showed measurable toxicity; in 1981, Scituate was free from toxin; in 1986, toxicity at Scituate lagged Gloucester by over a month. These low-flow years all had weak or predominantly upwelling-favorable winds, in support of this hypothesis.

Discussion

The wind-driven upwelling hypothesis of Mulligan (1973, 1975), Hartwell (1975), and Seliger et al. (1979), and the plume-advection hypothesis of Franks and Anderson (1992) were tested using historical records of shellfish toxicity, river flow, and wind stress for the southwestern Gulf of Maine. A north-to-south pattern of toxicity was seen for every case examined (1979 to 1989). Every toxic outbreak in Massachusetts was preceded by an outbreak in Maine, and by an increase in the flow rate of the Androscoggin River, as predicted by the plume-advection hypothesis. Seven years were inconsistent with the hypothesis of wind-driven upwelling as the factor initiating a toxic outbreak, and only one year, 1985, suggests rejection of the plume-advection hypothesis.

The north-to-south trend of PSP toxicity documented in Fig. 6 cannot be explained by the wind-driven upwelling hypothesis, but is entirely consistent with the plume-advection hypothesis. Furthermore, transit times of toxicity along the coast were found to agree with the plume-advection model, and it was shown that sustained winds opposing the direction of motion of the plume had little effect in years of high peak river flow. Thus the wind-mediated motion of a coastally trapped buoyant plume is a consistent and sufficient explanation for the spatial and temporal patterns of spring coastal shellfish toxicity outbreaks in the study area. The wind-driven upwelling hypothesis and the plume-advection hypothesis are, however, not mutually exclusive. It is likely that at different times of the year, different mechanisms will dominate in controlling toxic phytoplankton blooms.

The 1985 data appear to be the only exception to the dominance of plume-advection effects. That year was unusual in the lateness of the onset of toxicity, and it is possible that some mechanism other than advection within a buoyant plume was operating. The near-simultaneous occurrence of toxicity over a wide region of coastline, following several upwelling-favorable wind events, lends support to the wind-driven upwelling hypothesis. It is possible that late-blooming local populations of *Alexandrium tamarense* were advected from offshore waters to the shellfish beds by upwelling-favorable winds.

Martin and Main (1981) proposed a dual mechanism for PSP outbreaks at Ipswich, Massachusetts, a tidally-flushed estuary to the north of Cape Ann. The first mechanism was a restricted bloom of cells from local populations which occasionally cause a slight increase in shellfish toxicity. The second was alongshore advection of a population from Maine which could be swept into the estuary, causing sudden increases in the toxicity levels of the bivalves during early summer. This scenario agrees with the hypothesis of Franks and Anderson (1992), in which the alongshore transport mechanism was identified as a coastally trapped buoyant plume.

The alongshore advection of toxic *Alexandrium tamarense* cells in a buoyant plume can explain many details of the spread of PSP toxicity along the coasts of Maine and Massachusetts. The north-to-south motion is a result of the balance of forces which control the plume: the Coriolis force deflects the plume to its right as it exits the estuary, and keeps it coastally trapped. Wind effects have less influence over the plume dynamics in years of high river flow. This was particularly apparent during 1989, when the winds were predominantly upwelling-favorable (to the north), while the huge runoff during May forced the cells alongshore to the south through the study area in a matter of days. The upwelling-favorable winds appear to have been too weak to stop the alongshore advection of the plume. However, they did separate the low-salinity water and its associated cells from the coast through the formation of an offshore lens (Franks and Anderson 1992).

A coastally trapped buoyant plume should also show predictable behavior at sharp topographic features such as Cape Ann. Butman (1976) describes the dynamics and hydrographic signature of a buoyant plume crossing Massachusetts Bay, south of Cape Ann, during May 1973. In all likelihood, the plume he documented consisted of the one we have studied, formed by the Androscoggin and Kennebec Rivers, supplemented by the outflow of the Merrimack River. A feature of particular interest here is that the plume separated from the coast south of Cape Ann, crossed the bay in open water, and rejoined the coast south of Boston, near Scituate (Fig. 10). Had there been toxic cells in this plume, they would have bypassed the Boston Harbor area, leaving a PSP-free zone. This appears to be the general case, as only three occurrences of toxic mussels have ever been recorded from this zone: July 1979, June 1986, and May 1988. These events were each preceded by weak winds from the southeast which would theoretically have forced the plume into the coast south of Cape Ann. However, it is surprising, given this scenario and the frequent occurrence of toxicity at Scituate, that toxic outbreaks do not occur more frequently between Cape Ann and Scituate.

Bormans and Garrett (1989) suggested that a criterion for the separation of a current from a coast at a sharp topographic feature is given by the Rossby number, R_o:

$$R_o = \frac{v}{fr_c}. \tag{7}$$

The alongshore velocity, v, is compared to the product of the Coriolis frequency, f, and the radius of curvature of the coast, r_c. If R_o is less than unity, the Coriolis force should dominate, and the plume should remain coastally trapped. When R_o is greater than unity, the inertial forces dominate, and the current should separate from the coast. For Cape Ann, $r_c \sim 2.5$ km. Using a surface velocity for the plume suggested in Franks and Anderson

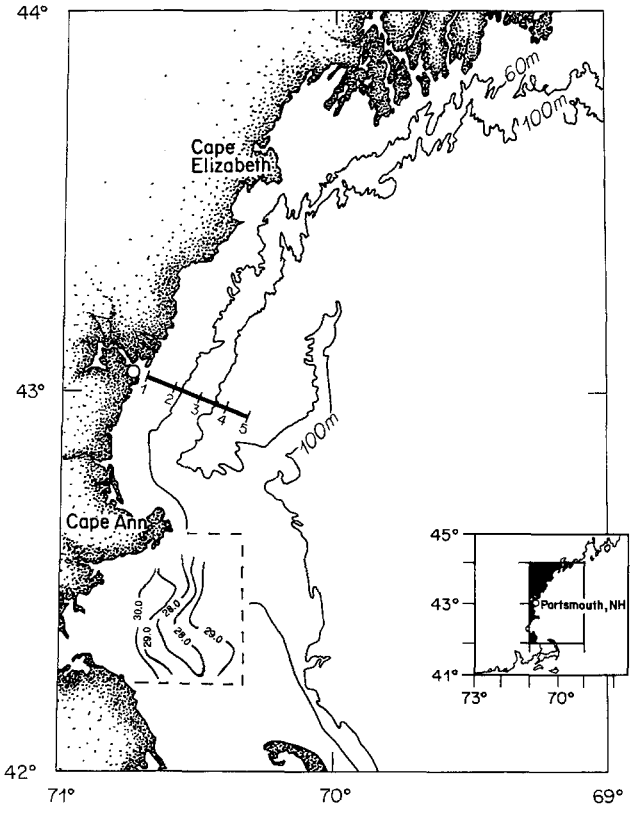

Fig. 10. A plume of low salinity water crossing Massachusetts Bay, recorded 5 to 6 May 1973 by Butman (1976). Redrawn from Butman (1976). Dashed box indicates sampling region for Butman's (1976) study. Stns 1 to 5 are sampling locations of Franks and Anderson (1991)

(1992), $v \sim 0.2$ m s^{-1}, and $f = 10^{-4}$ s^{-1}, we find $R_o \sim 0.8$. This suggests that separation of the plume from the coast may occur during situations in which the velocity is slightly increased, e.g. high volume of flow, or a southward wind stress. Note that these calculations of the plume velocity do not include the contribution of the Merrimack River, which lies just to the north of Cape Ann. This extra freshwater input would accelerate the plume, increasing the likelihood of separation from Cape Ann. This is consistent with the absence of PSP toxicity along the south shore of Cape Ann and Boston Harbor.

The separation of the plume from the coast at Cape Ann may also explain the sporadic nature of PSP outbreaks along the south shore of Massachusetts, from Scituate to Plymouth. Once the plume is in open water, it is easily influenced by the wind. A wind that is upwelling-favorable north of Cape Ann would tend to force a plume in Massachusetts Bay towards the east, or offshore. Thus we would expect that in years of persistent upwelling wind stress during the spread of toxicity, PSP outbreaks along the shore south of Cape Ann would be less likely. From Fig. 8 and 9, we find 1981, 1983, 1984, 1985, and 1987 to have been years with persistent upwelling-favorable winds. Of these 1981, 1983 and 1987 were toxin-free years for Scituate. Toxicity occurred at Scituate during 1984, a year in which the exceptionally high river flow may have dominated the surface wind stress. Thus, except for 1985 the data support this hypothesis.

If the plume were forced offshore by upwelling-favorable winds as it crossed Massachusetts Bay, it would tend to flow around the tip of Cape Cod, and southward along its east coast. In doing so, it would advect toxic *Alexandrium tamarense* cells toward Nantucket, and possibly onto Georges Bank. In this context, it is of note that in 1988 moderate levels of PSP toxins were detected in blue mussels from Nantucket Shoals and in 1989 and 1990 high levels of toxins were detected in surf clams from Georges Bank. Unfortunately, the discovery of this toxicity coincided with the first efforts to assay these shellfish for PSP toxins, so it is not known whether the toxin outbreaks are new or are common occurrences. In either case, the buoyant coastal plume provides a possible mechanism for the delivery of toxic dinoflagellates to these distant offshore shellfish. The movement of lower salinity water from Massachusetts Bay onto Georges Bank has been documented by Hopkins and Garfield (1981) and Limeburner and Beardsley (1982). Our work suggests a source for this water, and documents how this water enters Massachusetts Bay.

Thus the predictions of the plume-advection hypothesis of Franks and Anderson (1992) are supported, both in general and in detail, by the historical records of river flow, wind, and PSP toxicity in the southwestern Gulf of Maine. The predicted relationship of river flow rate to transport time of the toxicity alongshore was shown to hold. The predicted influence of the wind stress on plumes of varying strengths was supported for all years but 1985. The generally toxin-free region between Cape Ann and Scituate can be explained through the separation of the plume from the coast at Cape Ann, an occurrence which has been independently documented. Finally, the occurrence of PSP toxins in shellfish of Nantucket Shoals and Georges Bank might be linked to the same buoyant plume which moves across Massachusetts Bay and into offshore waters.

Acknowledgements. We are pleased to acknowledge the helpful comments and criticisms of K. Brink, W. Brown, J. Cullen, C. Davis, G. Flierl and D. Kelley. J. Hurst and S. Sherman-Caswell of the Maine Department of Marine Resources and F. Germano and M. Hickey of the Massachusetts Department of Marine Fisheries kindly made their toxicity data available to us. Thanks also go to D. McCartney and G. Abend of the United States Geological Survey for supplying us with the river flow statistics. This work was supported by Office of Naval Research contract N00014-87-0007 and grant N00014-89-J-111, by the National Sea Grant College Program Office, Department of Commerce under Grant Nos. NA86AA-D-SG090 and NA90-AA-D-SG480 (WHOI Sea Grant Projects R/B-92 and R/B-100), and the Florence and John Schumann Foundation. This is WHOI contribution number 7523.

Literature cited

Association of Official Agricultural Chemists (1965). Paralytic shellfish toxin, biological methods. (18). In: Official methods analysis, 10th edn. A.O.A.C., Washington, D. C., p. 282–284

Bormans, M., Garrett, C. (1989). A simple criterion for gyre formation by the surface outflow from a strait, with application to the Alboran Sea. J. geophys. Res. 94: 12637–12644

Bricelj, M., Lee, J. H., Cembella, A. D., Anderson, D. M. (1990). Uptake of *Alexandrium fundyense* by *Mytilus edulis* and *Merce-*

naria mercenaria under controlled conditions. In: Graneli E., Sundstrom B., Edler, L., Anderson, D. (eds.) Toxic marine phytoplankton. Elsevier, New York, p. 269–274

Butman, B. (1976). Hydrography and low-frequency currents associated with the spring runoff in Massachusetts Bay. Mém. Soc. r. Sci. Liège 6(X): 247–275

Chao, S.-Y. (1988). River-forced estuarine plumes. J. phys. Oceanogr. 18: 72–88

Clarke, A. J., Brink, K. H. (1985). The response of a stratified, frictional flow of shelf and slope waters to fluctuating large-scale, low-frequency wind forcing. J. phys. Oceanogr. 15: 439–453

Franks, P. J. S. (1990). Dinoflagellate blooms and physical systems in the Gulf of Maine. Ph. D. thesis, MIT/WHOI Joint Program in Oceanography, Woods Hole, Massachusetts

Franks, P. J. S., Anderson, D. M. (1992). Alongshore transport of a toxic phytoplankton bloom in a buoyancy current: *Alexandrium tamarense* in the Gulf of Maine. Mar. Biol. 112: 153–164

Gainey, L. F., Shumway, S. E. (1988). A compendium of the responses of vivalve molluscs to toxic dinoflagellates. J. Shellfish Res. 7: 623–628

Hartwell, A. D. (1975). Hydrographic factors affecting the distribution and movement of toxic dinoflagellates in the western Gulf of Maine. In: LoCicero, V. R. (ed.) Proceedings of the first international conference on toxic dinoflagellate blooms. Massachusetts Science and Technology Foundation. Wakefield, Massachusetts, p. 47–68

Hopkins, T. S., Garfield, N. (1981). Physical origins of Georges Bank water. J. mar. Res. 39: 465–500

Hurst, J. W. (1979). Shellfish monitoring in Maine. In: LoCicero, V. R. (ed.) Proceedings of the first international conference on toxic dinoflagellate blooms. Massachusetts Science and Technology Foundation, Wakefield, Massachusetts, p. 23–40

Hurst, J. W., Selvin, R., Sullivan, J. J., Yentsch, C. M., Guillard, R. L. (1985). Intercomparison of various assay methods for the detection of shellfish toxins. In: Anderson, D., White, A., Baden, D. (eds.) Toxic dinoflagellates. Elsevier, New York, p. 427–432

Janowitz, G. S., Pietrafesa, L. J. (1980). A model and observations of time-dependent upwelling over the mid-shelf and slope. J. phys. Oceanogr. 10: 1573–1583

Kao, T., Pao, H.-P., Park, C. (1978). Surface intrusions, fronts, and internal waves: a numerical study. J. geophys. Res. 83: 4641–4650

Large, W. S., Pond, S. (1981). Open ocean momentum flux measurments in moderate to strong winds. J. phys. Oceanogr. 11: 324–336

Levy, G., Brown, R. A. (1986). A simple, objective analysis scheme for scatterometer data. J. geophys. Res 91: 5153–5158

Limeburner, R., Beardsley, R. C. (1982). The seasonal hydrography and circulation over Nantucket Shoals. J. mar. Res. 40 (suppl.): 371–405

Martin, C., Main, J. M. (1981). Toxic dinoflagellate blooms (red tides) and shellfish resources in Plum Island Sound and adjacent Massachusetts waters. Final report to the Town of Ipswich, Massachusetts, under UMass/Amherst OGCA Contract No. 80A613. Office of Coastal Zone Management, NOAA, Washington D.C.

Mulligan, H. (1973). Probable causes for the 1972 red tide in the Cape Ann region of the Gulf of Maine. J. Fish. Res. Bd. Can. 30: 1363–1366

Mulligan, H. (1975). Oceanographic factors associated with New England red tide blooms. In: Lo Cicero, V. R. (ed.) Proceedings of the first international conference on toxic dinoflagellate blooms. Massachusetts Science and Technology Foundation, Wakefield, Massachusetts, p. 23–40

Seliger, H., Tyler, M. A., McKinley, K. R. (1979). Phytoplankton distributions and red tides resulting from frontal circulation patterns. In: Taylor, D. L., Seliger, H. H. (eds.) Toxic dinoflagellate blooms. Elsevier, New York, p. 239–248

Shumway, S., Sherman-Caswell, S., Hurst, J. W. (1988). Paralytic shellfish poisoning in Maine: monitoring a monster. J. Shellfish Res. 7: 643–652

Steidinger, K. A., Moestrup, Ø. (1990). The taxonomy of *Gonyaulax*, *Pyrodinium*, *Alexandrium*, *Gessnerium*, *Protogonyaulax* and *Goniiodoma*. In: Graneli, E., Sundstrom, B., Edler, L., Anderson, D. (eds.) Toxic marine phytoplankton. Elsevier, New York, p. 522–523