tivity reduces to L. This happens for wavelengths shorter than 480 nm. For wavelengths longer than 480 nm, the two equations are identical. The theoretical curves are plotted in Fig. 2B. The cancellation ($M - S$) equation is linear while the matching equation is not, which implies that the $r - g$ opponent spectral sensitivities are linear combinations of the cone pigments if measured under conditions for which the Θ fields do not become zero—that is, where the M centers are more responsive than the S surrounds.

This theory shows that adding green light to a violet field has opposite effects. The green light cancels the redness in the usual opponent sense. But green light also activates the silent S surrounds, which by opposing the M cone necessarily signal redness.

When the L and M cone sensitivities are normalized at the long-wavelength neutral point (Fig. 2B), L can never signal r at $\lambda < 580$. The M cone is always more sensitive than the L cone, so unless the S cone inhibits the M cone enough to reduce its sensitivity to less than that of the L cone, the L cone cannot produce the $+r$ signal on opponent spectral sensitivity curves at short wavelengths. Although for the Θ S curves the $+r$ sensitivity arises from the L cone, it is the inhibition of M by S that allows this $+r$ to appear at short wavelengths (*12*). This fact also explains why long-wave adaptations may decrease short-wavelength $+r$. From the viewpoint of opponent theory, any cones that inhibit M signal $+r$ regardless of their spectral sensitivity.

The transformation equations from cone to channel spectral sensitivities for the $r - g$ channel require two differencing mechanisms within the channel. One of these mechanisms opposes the L cone to the M cone, the other the S cone to the M cone. Comparing the results of a hue-cancellation experiment with a hue-matching experiment suggests that the two mechanisms have different properties. The difference computed by the $M - L$ mechanism is a true $r - g$ signal in the sense that the difference signifies redness or greenness, depending upon the polarity of the difference. The M Θ S mechanism apparently signals only redness; the difference is zero unless the output of the M cones is greater than that of the S cone.

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8. Our red primary was not unique in that some yellow is present in a deep red 680-nm light; however, this is immaterial to the arguments.
9. To obtain greater accuracy, instead of using a reference white surround or matching field, the other side of the bipartite field was a mixture of the yellow (580 nm) and blue (480 nm) primaries. These primaries were adjusted to match the canceled side. Thus, to get a cancellation setting for the red or green primary required making a complete trichromatic color match. In a separate experiment, we verified that a mixture of the 480- and 580-nm primaries defines the locus in the chromaticity chart of hues that are neither reddish nor greenish. Therefore, making the color match ensures that the exact amount of red or green primary required to make the test side of the field neither reddish nor greenish has been added. This method is greatly superior to the usual method in reliability and precision.
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1 March 1978; revised 14 June 1978

Toxicity in Resting Cysts of the Red-Tide Dinoflagellate *Gonyaulax excavata* from Deeper Water Coastal Sediments

Abstract. *For the first time,* Gonyaulax excavata *cysts have been shown to be toxic. Bottom sediments from a water depth of 90 meters off the Maine coast were extremely rich in cysts, which were approximately ten times more toxic than the corresponding motile stages. Cysts are probably ingested by shellfish, thereby causing shellfish toxicity in deeper waters offshore and contributing to shellfish toxicity in shallower coastal waters. A new approach to the problem of paralytic shellfish poisoning is therefore needed, one that takes into account benthic cysts and sedimentary factors affecting their distribution. The possible dangers of spreading poisoning through human activities must be considered.*

Paralytic shellfish poisoning (PSP) is a well-documented food poisoning (*1*, *2*) in which toxins produced by microscopic phytoplankters (dinoflagellates) are accumulated in shellfish and passed on to humans who eat the shellfish. At least 300 human fatalities are known to have been caused by PSP worldwide (*3*), and there is disturbing evidence that outbreaks are increasing in intensity and spreading to new areas (*3*, *4*). These outbreaks focus attention on the hazards to both public health and the fisheries industry and emphasize the need for extended monitoring and better forecasting programs. Earlier suggestions for developing forecasting programs have relied solely on the monitoring of dinoflagellate motile stages in the water column and on environmental factors contributing to their growth and distribution. We report here large concentrations of highly toxic dinoflagellate resting cysts in bottom sediments. The relationship between toxic dinoflagellate blooms and shellfish toxicity may therefore be more complicated than has been thought, and toxicity forecasting will need to include consideration of benthic cysts and sedimentary factors affecting their distribution.

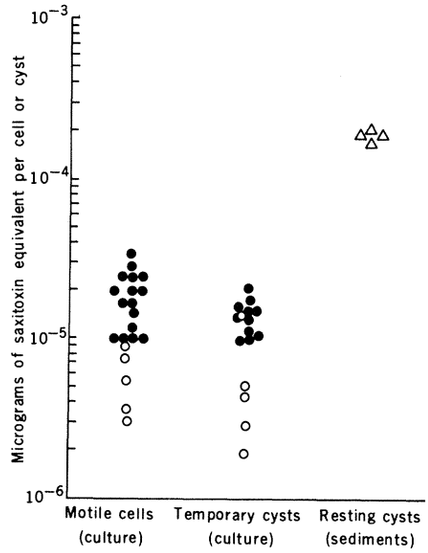

Fig. 1. Saxitoxin equivalent per cell or cyst for (○) motile cells or temporary cysts induced from logarithmic-phase motile cells in batch culture, (●) motile cells or temporary cysts induced from stationary-phase cells in batch culture, and (△) resting cysts estimated from sieved sediments.

Recently, temporary cysts and benthic resting cysts were described for the New England red-tide dinoflagellate *Gonyaulax excavata* (*5*). Prior to this work, benthic cysts were implicated as a likely cause of shellfish toxicity in deeper waters, even though no such cysts had been identified from either plankton or sediments (*6*). This implication was based on

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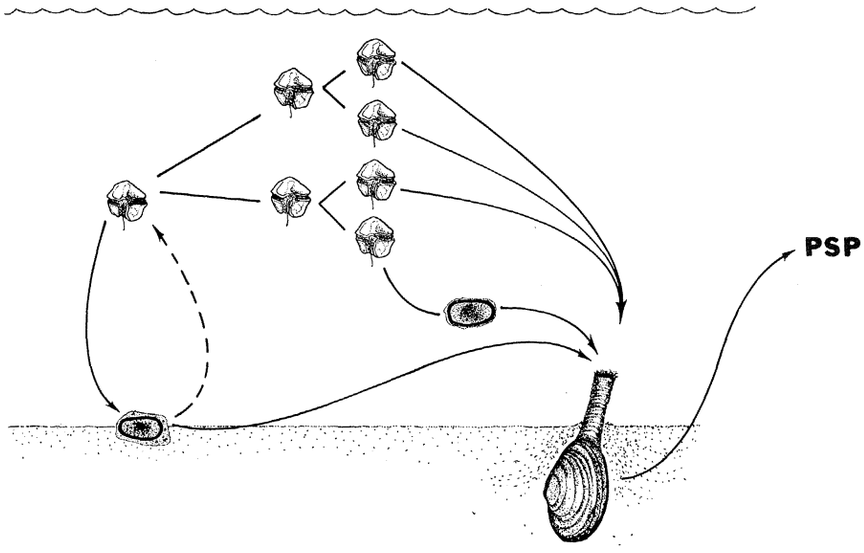

Fig. 2. Schematic representation of cyst reseeding bloom (----) and two mechanisms of accumulation of shellfish toxins: (i) motile cells and (ii) resting cysts in sediments; *PSP*, paralytic shellfish poisoning.

two facts: (i) motile stages are not known to occur at the low temperatures (4°C) that occur in deeper waters (*6*) and (ii) deeper water shellfish toxicity in the Bay of Fundy peaked in midwinter, a time when motile cells were not found in the water column (*6*). Since then, two additional facts supporting this implication have been noted: (i) there have been reports of PSP with little or no visible evidence of motile dinoflagellates (*1*), and (ii) toxification rates of shellfish at times have exceeded the maximum published rates attained by shellfish feeding on motile cells (*7*).

The work reported here is part of a survey of *G. excavata* cyst distribution along the Maine coast. To date, we have sampled for the most part sediments in shallower coastal water (5 to 20 m), but sediments for this study were from a locality (depth, 90 m) that is 27 km from the mainland and 2 km east off Monhegan Island (43°46′N, 69°19′W). We sampled bottom sediments by using a standard gravity corer 7 cm in diameter or a 0.04-m^3 grab. The recovered sediment was sticky, gray-brown mud topped by a flocculent layer 2 cm thick, which was suctioned off and used for cyst studies. Samples were disaggregated by gentle sonication and sieved with a stainless steel wire sieve (37-μm mesh). Sediment retained on the sieve was washed off with filtered seawater and examined microscopically (*8*). This procedure revealed rich concentrations of dinoflagellate cysts (*9*); *G. excavata* made up 95 percent of the cyst assemblage in a total count of 252 (*10*). Cysts collected on 17 December 1977 were tested for toxicity on 29 January 1978 (*11*).

Resting cysts of *G. excavata* proved toxic (Fig. 1)—approximately 0.0002 μg of saxitoxin equivalent per cyst. This is more toxic by at least one order of magnitude than per motile cell (*12*). Shellfish toxicity is generally presumed to be caused by shellfish feeding on toxic dinoflagellate motile stages. However, in light of the evidence presented here and the apparent likelihood that *G. excavata* resting cysts may break down in shellfish digestion (*13*), we believe that resting cysts probably cause toxicity in deeper water shellfish and contribute significantly to toxicity in shallower water (Fig. 2).

Cysts behave as fine silt particles in the sedimentary regime and are thus concentrated by sedimentary processes (*14*); some areas form "sinks" for cysts, whereas others are relatively cyst-free. At one extreme, coastal bays with restricted water circulation may have a regularly repeated cycle whereby cysts in bottom sediments form "seedbeds" (*15*), producing local toxic plankton blooms which in turn produce a fresh crop of cysts (Fig. 2). At the other extreme, especially in deeper waters, cysts may collect in areas far removed from their plankton source as a result of transport and concentration by hydrographic and sedimentary processes.

An important question here is: Do the deeper water cysts merely accumulate at depths from which they cannot reestablish planktonic stages? Or are the deeper water cysts also seedbeds which, as a result of upwelling or storm activity, are carried back up to the photic zone to reestablish offshore plankton blooms?

Our observations to date support the seedbed hypothesis. We have witnessed a progressive change in the morphological development of the cysts over the 4-month period from October 1977 to February 1978. This change suggests maturation of cysts (*16*) and argues against accumulation of older cysts, no evidence of which was seen in the earlier samples. In addition, viability tests (*17*) were not successful until the collection of January 1978; this result seems to suggest that until that time the cysts had not completed a mandatory resting period (*5*). We believe the cysts were local in origin and recently formed prior to the collection of 4 October.

The possible effects of cysts in bottom sediments has implications far beyond toxicity forecasting. These effects may be manifested in a wide range of marine activities. In particular, those engaged in projects such as the artificial seeding of shellfish beds, shellfish culture, and marine dredging operations should be alerted to possible dangers. Dredged-up sediment should be presumed to contain toxic cysts, which may be carried far from the source after dumping at sea (*18*). Caution should be exercised when shellfish are being transferred from one area to another; microscopic cysts could easily be carried with them. Once introduced into a new area, cysts may directly contaminate shellfish and they may establish more permanent local toxic dinoflagellate populations (*5*).

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4. Severe new outbreaks occurred: (i) in 1972, along parts of the New England coast where no previous history of PSP was known; lower Maine, New Hampshire, and the entire 3200-km Massachusetts coastline were closed to shellfishing; (ii) in 1974, off the coast of New Guinea; and (iii) in 1976, off the northern coast of Spain.
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frequently found in sediments. Work presented here does not disprove the earlier hypothesis that temporary cysts may cause shellfish toxicity. However, temporary cysts were not seen in the sediments and we believe that toxicity was caused by the large numbers of toxic resting cysts present.
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9. Concentrations of cysts were often in excess of 100 per slide as compared with 5 to 20 per slide in samples from other areas.
10. Cysts with intact cell contents included four species of *Gonyaulax*, three species of *Protoperidinium*, and a species of *Scrippsiella*.
11. We used a micropipette to pick off cysts and cleaned them by several transfers through drops of distilled water. Clean cysts were concentrated by centrifugation and rinsed three times with distilled water with centifugation. Then 509 cysts were sonicated vigorously, acidified, and injected into mice (Jackson strain) with the standard PSP bioassay. Isolated cysts produced classical signs of toxicity, yet the death time was not within the standard limits. Sieved sediments containing 14,700 cysts (cyst numbers were estimated on the basis of counting 0.1 ml of the 15-ml total sample) were then analyzed according to the same procedure, divided into four parts, and injected into four mice; the death times (267 sec/19.0 g, 261 sec/19.0 g, 250 sec/19.5 g, and 239 sec/19.5 g) were well within the standard limits. Since isolated cysts proved to be toxic, we assume that toxicity in sieved sediment was caused by the included cysts, but the theoretical possibility that other particles in the sediment were significantly toxic cannot be discounted.
12. We induced temporary cysts (*5*) from motile cells by subjecting cultures growing at 18° to 5°C. Both temporary cysts and motile cells were filtered onto Gelman glass-fiber filters and ground with 0.1*N* HCl with a tissue homogenizer. We then refiltered this solution and injected it into mice (Jackson strain), using the standard PSP bioassay.
13. If cysts break down to release toxins in human digestion, even intact cysts in the stomach contents of ingested shellfish could cause PSP; moreover, it seems likely that cysts may break down in shellfish digestion since *G. excavata* cyst walls are less resistant to chemical and biological degradation than those of most other dinoflagellates. Chemical treatment of sediments for palynological preparation destroy *G. excavata* cysts; in naturally occurring sediments *G. excavata* cysts apparently break down and are lost whereas most other cysts persist into the fossil record.
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16. Cysts collected on 4 October 1977 lacked conspicuous red-pigmented cell inclusions previously described for mature *G. excavata* resting cysts (*5*), whereas most had developed this by 21 November 1977 and virtually all cysts had developed this by 17 December 1977. By the 30 January 1978 collection, cyst contents darkened and showed Brownian-like motion.
17. We periodically isolated resting cysts from the sediments and tested for viability by placing cysts at conditions of light and temperature known to induce excystment (*5*). Several excysted from sediment collected on 30 January 1978 and tested viable on 6 February 1978, suggesting a mandatory resting period of at least 4 months.
18. It should not be presumed that possible dangers would be avoided because dumping dredged-up sediment at sea would bury or dilute any cysts present. Cysts would sediment out with the fine silt fraction after dumping and thus would usually settle at or near the sediment surface.
19. We thank M. L. Brann, R. Bryer, C. Lewis, F. Mague, and C. Mickelson for field and laboratory assistance. We thank Drs. T. Braarud, R. C. Dugdale, J. Gray, G. Hasle, I. Morris, E. Paasche, T. T. Packard, and C. S. Yentsch for constructive criticisms of the manuscript. This work was supported in part by Food and Drug Administration contract 223-76-2311, National Institute of Environmental Health Sciences contract ESO-1329-01A1, National Science Foundation contract OCE-76-10518, Norwegian Research Council for Science and Humanities contract D40.31-15, and Maine Department of Marine Resources contract 4-77. Bigelow Laboratory contribution No. 77023.

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Gulf Stream Deflection by a Bottom Feature off Charleston, South Carolina

Abstract. *A topographic feature on the continental slope off Charleston at 32°N persistently deflects the Gulf Stream seaward, with the inshore surface thermal front deflected east or south of east in 27 of the 39 cases examined. Meanders often form downstream of the deflection, suggesting that the "Charleston bump" induces Gulf Stream fluctuations.*

A frequent seaward deflection of the Gulf Stream occurs over a ridge and trough bottom irregularity in the continental slope at about 32°N off Charleston, South Carolina. Satellite observations of sea-surface thermal patterns show a considerable difference in the magnitude of the Gulf Stream fluctuations upstream and downstream of the "Charleston bump," with greater fluctuations occurring downstream. The winter example in Fig. 1 shows large undulations most evident in the shoreward thermal front between the warm Gulf Stream surface waters and the relatively cold shelf waters (*1*). The existence of Gulf Stream meanders in the South Atlantic Bight has been recognized for several decades (*2*), and several theories have been advanced, with varying degrees of success, to explain their generation, propagation, and decay (*3*). However, persistence of the deflection off Charleston (*4*), its association with a bottom irregularity (*5*), and its potential significance in modifying the downstream character of meanders (*6*) have only recently been appreciated.

Surface isotherms are known to be reasonably well correlated with the deeper hydrographic and velocity structure of the Gulf Stream in the South Atlantic Bight (*7*), and the inshore surface thermal front in Fig. 1 may be associated with the shoreward edge of subsurface Gulf Stream flow. The Stream extends essentially to the bottom in this area (*8*) and thus must make dynamical adjustments as it flows over bottom topography. The simplest application of the vorticity conservation theorem predicts a seaward curvature of the Stream when it flows over a bump on an otherwise featureless continental slope.

The detailed bottom topography including the region enclosed in a box in Fig. 1 is shown in Fig. 2 (*9*). The crest of the ridge runs approximately transversely to the Stream as it enters the boxed area, and turns generally eastward farther offshore, trending toward the Blake Outer Ridge. A narrow trough or channel traceable to the base of the Blake Plateau at a depth of about 2 km runs eastward on the downstream side of the ridge. Ewing *et al.* (*10*) suggested that the ridge and trough system off Charleston may be a result of erosion along the southwestern flank of a geologic structure known as the Cape Fear Arch, which extends seaward toward the Blake Outer Ridge. Uchupi (*11*) sug-

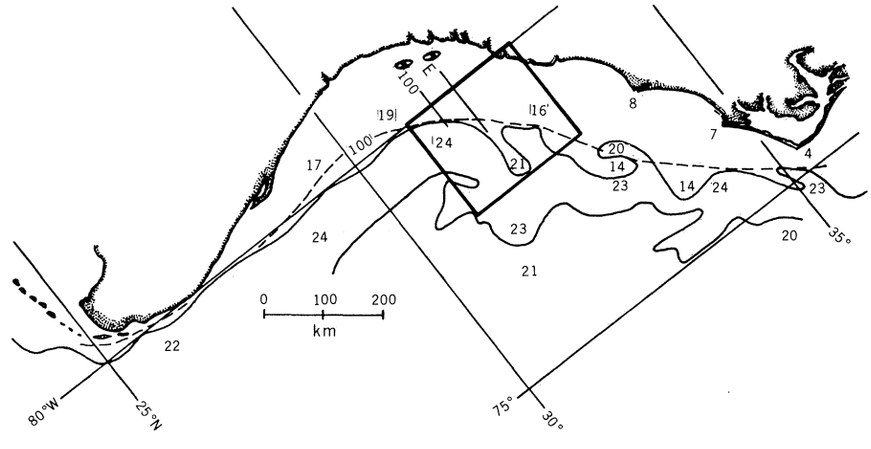

Fig. 1. Composite map of Gulf Stream surface thermal features in the South Atlantic Bight for 15 February 1978 prepared by the Naval Oceanographic Office (*1*). The NOAA-5 satellite infrared images from 12 to 14 February were used to locate shoreward and seaward surface fronts, shown by the solid lines. Ground-truth sea surface temperatures (degrees Celsius) are shown for comparison with the frontal structure. Symbols: θ_{100} is the latitude at which the inshore front turns seaward of the 100-fathom contour shown by the dashed line, and θ_E is the latitude at which the front reaches an eastwardly deflection. The detailed bottom topography in the region in the box is shown in Fig. 2.

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