

RECENT OCCURRENCE OF PARALYTIC SHELLFISH TOXINS IN OFFSHORE SHELLFISH IN THE NORTHEASTERN UNITED STATES

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ABSTRACT

Since 1989, paralytic shellfish toxins have been detected in shellfish from the American sector of Georges Bank at levels exceeding the public health safety threshold, necessitating closure of shellfisheries in these offshore waters for the first time. The toxins reached maximum levels in offshore shellfish during the summer of 1990, but have persisted at lower levels, still exceeding the safety threshold, in some species. Maximum toxin levels in whole animals, determined by mouse bioassay, were 6400 μg STX equiv./100 g tissue for Atlantic surfclams (*Spisula solidissima*), 3900 μg STX equiv./100 g tissue for sea scallops (*Placopecten magellanicus*), 3600 μg STX equiv./100 g tissue for northern horse mussels (*Modiolus modiolus*), and 1200 μg STX equiv./100 g tissue for ocean quahogs (*Arctica islandica*). High toxin levels have also been detected in carnivorous molluscs such as northern moon snails (*Euspira heros*) and waved whelks (*Buccinum undatum*). In May and June 1990, eight fishermen were poisoned after eating blue mussels (*Mytilus edulis*) taken as bycatch from Georges Bank; in the June incident the mussels contained more than 24000 μg STX equiv./100 g tissue before cooking. The source of the offshore toxins is presumed to be the dinoflagellate *Alexandrium tamarense*, which causes shellfish toxicity annually in the inshore waters of New England and eastern Canada, although blooms of *A. tamarense* have not been confirmed in the Georges Bank area. The occurrence of high levels of toxins in offshore shellfish is apparently a new event and raises scientific questions as well as fisheries and public health management issues.

HISTORY

Blooms of the toxic dinoflagellate *Alexandrium tamarense* and the resulting contamination of shellfish with paralytic shellfish toxins have been nearly annual events in coastal waters of eastern Canada and New England for many years. Little attention has been given to the extent to which this organism blooms in the offshore waters of the Gulf of Maine.

In 1960 and 1961 Bourne [1] found low levels of paralytic shellfish toxins, 62-66 μg STX equiv./100 g tissue, in the digestive glands of sea scallops (*Placopecten magellanicus*) from the Northern Edge region of Georges Bank (Fig. 1). A re-examination of sea scallops in the same area by Jamieson and Chandler [2] in May 1980 and June 1981 also found low levels of the toxins in digestive glands, with maximum concentration of 140 μg STX equiv./100 g tissue.

These first studies of paralytic shellfish toxins on Georges Bank indicated the presence of toxins as much as 30 years ago; however, the source of the toxins was not identified. The low levels of toxins detected in scallop digestive glands, an organ known to accumulate extremely high levels of the toxins [2], suggests that the contamination of Georges Bank shellfish was not pronounced during those periods. That there were no reports of paralytic shellfish poisoning (PSP) from consumption of offshore shellfish before 1990 is circumstantial evidence that paralytic shellfish toxins did not occur at high levels in offshore shellfish before the present events.

APPARENT NEW EVENTS

The first indication of elevated toxin levels in offshore shellfish came in the summer of 1988. As part of the monitoring for a newly developed Canadian fishery for roe-on sea scallops, the toxins were detected at 2000 μg STX equiv./100 g tissue in digestive glands of scallops from the Canadian sector of Georges Bank (W. Watson-Wright, pers. comm.). The toxins were also detected in whole, blue mussels (*Mytilus edulis*) from Nantucket Shoals at levels up to 100 μg STX equiv./100 g tissue (J. Hurst, pers. comm.).

A harbinger of these offshore events in 1988 may have been the occurrence of a mass mortality of humpback whales off the tip of Cape Cod in the fall and early winter of 1987. Implicated in this kill were paralytic shellfish toxins [3,4], apparently acquired by the whales through ingestion of Atlantic mackerel which contained toxins in their viscera. Subsequent study has shown the widespread occurrence of the toxins in the viscera of Atlantic mackerel throughout the Northeast [C. Martin, pers. comm.], raising the possibility of an offshore origin of the toxins. In July 1978, the toxins were implicated in a kill of sand lance -- which in turn killed carnivorous fish and birds -- along the outer reaches of Cape Cod [5] at a time when there was no evidence of shellfish toxicity in inshore areas, suggesting the possible involvement of an unnoticed offshore bloom of *Alexandrium*.

By mid-July 1989 toxin levels in the digestive glands of scallops from the Canadian sector of Georges Bank reached 7500 μg STX equiv./100 g tissue and exceeded the public health safety standard of 80 μg STX equiv./100 g tissue in scallop roe, causing the closure of the Canadian roe-on fishery in that area. In mid-August 1989, a routine market survey conducted by the Massachusetts Department of Public Health detected the toxins in whole surfclams (*Spisula solidissima*) from southern Georges Bank at 300 to 500 μg STX equiv./100 g tissue. Samples of sea scallop viscera from Georges Bank at that time contained up to 4200 μg STX equiv./100 g tissue (C. Martin, pers. comm.)

CLOSURE OF GEORGES BANK

On August 11, 1989, a 90-day emergency closure of the American sector of Georges Bank to the harvesting of surfclams was enacted by the National Marine Fisheries Service. The closure pertained to American shellfisheries east of 69° W longitude and south of 42° 20' N latitude (Fig. 1). In November, the persistence of the toxins in offshore shellfish necessitated an extension of the closure for another 90-day period.

After a brief reopening in the spring of 1990, the offshore fishery was closed again in May when toxin levels in surfclams were found to range from 600 to 1530 μg STX equiv./100 g tissue. The closure was extended to include not only surfclams, but ocean quahogs, mussels and any part of sea scallops other than the adductor muscle. As of this writing (December 1991) the closure continues indefinitely because toxin levels in some shellfish species from the area remain above the public health safety threshold of 80 μg STX equiv./100 g tissue.

POISONINGS

On May 22, 1990, a fisherman and his wife suffered PSP after eating blue mussels taken as bycatch from Georges Bank. On June 5, 1990, six more fishermen were poisoned, also after eating blue mussels taken as bycatch from the west central part of Georges Bank south of Cultivator Shoal and Georges Shoal (Fig. 1). In this second instance, two of the victims became seriously ill; one nearly died [6]. During this episode the toxin levels in raw, blue mussels from the area involved reached 24417 μg STX equiv./100 g tissue; the toxin level in the cooked mussels (which is how the mussels were consumed), was 4280 μg STX equiv./100 g tissue [6].

In response to this situation, radio announcements and press releases were issued to warn fishermen of the danger of eating shellfish taken as bycatch from the Georges Bank area.

MONITORING

Toxin levels in shellfish from the Georges Bank area have been monitored since May 1990 at six stations on Georges Bank through a cooperative effort of the National Marine Fisheries Service and the U.S. Food and Drug Administration (Fig. 1). Determinations of toxin levels in surfclams at these stations from May 1990 until September 1991 (Fig. 2) indicate a rapid accumulation of the toxins in May and June 1990, followed by a fairly rapid, then gradual elimination of the toxins through 1990 and 1991. The pulse in offshore toxicity in May and June 1990 occurred at nearly the same time as an *Alexandrium* bloom was present along the New England coast north of Cape Cod, suggesting that the two events may have been connected.

There was no evidence of an *Alexandrium* bloom occurring offshore in 1991 (and only a very limited bloom occurred in some coastal areas). Nevertheless, the toxins have persisted in offshore surfclams at several hundred μg STX equiv./100 g tissue until the time of this writing (December 1991). Data on toxin levels in various tissues of surfclams indicate that initially the toxins were present primarily in the digestive gland, but over the sampling period the gill, siphon, and mantle became the primary storage sites, as has been described for other shellfish [7,8,9]. The toxins have been detected in several species of molluscs from Georges Bank, including filter-feeding bivalves (blue mussel, horse mussel, surfclam, ocean quahog, sea scallop), carnivorous gastropods (moon snails and waved whelks), and the common Atlantic slipper snail, *Crepidula fornicata* (Table I). Highest levels were recorded for blue mussels and sea scallops. Blue mussels and ocean quahogs eliminated the toxins quickly so that by the fall of 1990, toxins were below the limit of detection ($< 40 \mu\text{g}$ STX equiv./100 g tissue) in these animals. As mentioned above, surfclams have retained the toxins for more than a year. Carnivorous moon snails, which, no doubt, acquired the toxins from ingestion of filter-feeding shellfish, maintained high toxin levels through the summer of 1991.

Substantial variation was found in toxin levels among individual shellfish from the same area (see White et al., this volume).

IMPLICATIONS

This recent episode of offshore shellfish toxicity raises many scientific questions. For example, what is the actual source of the toxins? Is the offshore contamination caused by *in situ* blooms of *Alexandrium tamarense*, or is it linked to the mass transport of inshore blooms which occur along the coast of New England? Will the offshore contamination recur with some regularity, or is this an isolated or occasional event? Could these new events signal a shift in the phytoplankton community structure in the Gulf of Maine? Are these new events related to changes in nutrient levels or nutrient ratios in the Gulf of Maine?

Perhaps more importantly, these recent offshore events also raise fisheries and public health concerns. The present closure of the Georges Bank surfclam fishery represents a potential loss of \$4-5 million per year in landed value. Although the traditional sea scallop fishery in this region is not affected because the toxins do not accumulate in the scallop adductor muscle, possible expansion of this fishery to include whole or roe-on scallops is compromised. Options for optimal management of this new offshore toxicity problem are now being developed by the National Marine Fisheries Service, in conjunction with the U.S. Food and Drug Administration.

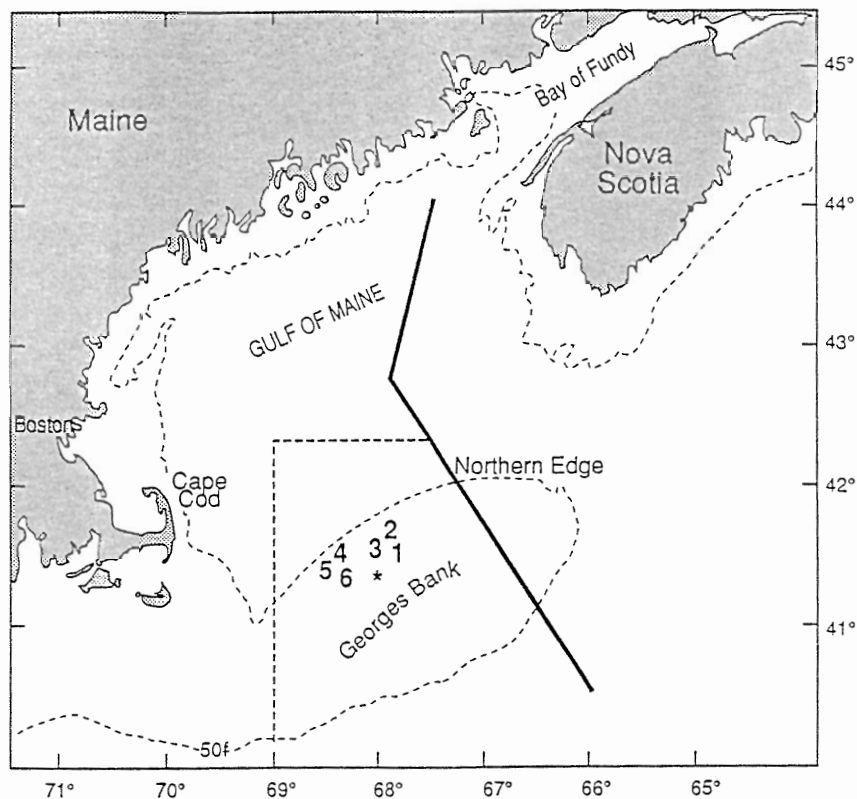


FIG. 1. The Georges Bank region of the Gulf of Maine in the northwestern Atlantic. Solid line is the Hagué Line, demarcating American and Canadian waters. Heavy dashed line bounds the American sector of Georges Bank closed to harvesting of shellfish because of the presence of paralytic shellfish toxins. Numbers represent the sampling stations specified in Table I. Star shows area where bycatch mussels caused cases of PSP among fishermen in June 1990.

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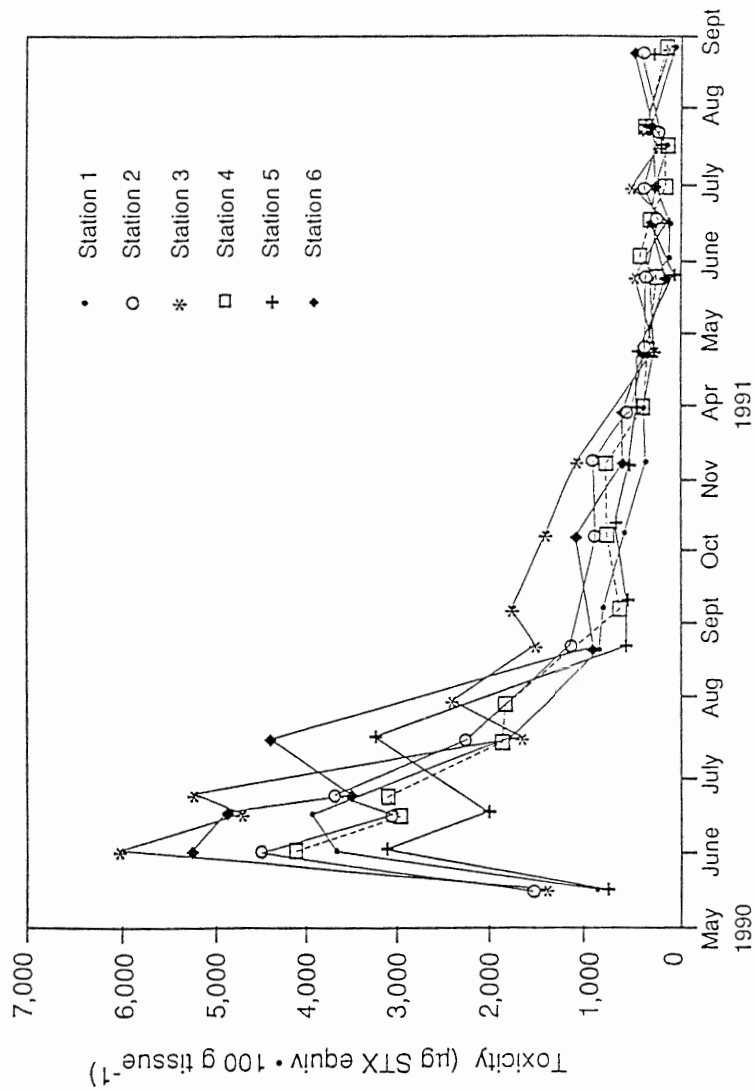


FIG. 2. Levels of paralytic shellfish toxins (μg STX equivalents/100 g tissue) in whole surfclams (*Spisula solidissima*) at the six stations on Georges Bank shown in Figure 1. Results are based on standard mouse bioassays (3-mouse test) conducted on composite samples of 12 animals for each sample.

TABLE I. Highest levels of paralytic shellfish toxins detected in shellfish from Georges Bank.

Animal	Date	Location (Loran C)*	Toxin Level** (μg STX equiv/100 g)
<i>Mytilus edulis</i>	6/04/90	13275-43710***	24417
<i>Modiolus modiolus</i>	6/28/90	13110-43830	5016
	6/28/90	13175-43825	3648
	8/03/90	13364-43778 (6)	2142
<i>Spisula solidissima</i>	6/08/90	13193-43799 (3)	6017
	6/09/90	13367-43753	6423
<i>Arctica islandica</i>	6/08/90	13364-43778 (6)	1218
<i>Placopecten magellanicus</i>	6/28/90	13364-43778 (6)	14775****
	8/03/90	13200-43804	3888
<i>Euspira heros</i>	6/28/90	13110-43830	2764
	6/28/90	13153-43808 (2)	2112
	4/04/91	13360-43803 (4)	2454
	7/16/91	13372-43801 (5)	2922
<i>Buccinum undatum</i>	6/28/90	13850-43580	3337
<i>Crepidula fornicata</i>	8/03/90	13200-43804	58
	9/13/90	13193-43799 (3)	46

*Sampling station number in parentheses (see Fig. 1).

**Standard, 3-mouse bioassay tests were conducted on whole animals. Results for *Mytilus* and *Modiolus* are based on composite samples of 4-6 animals each. Results for *Spisula* and *Arctica* are based on composite samples of 12 animals each. Results for *Placopecten*, *Euspira*, *Buccinum* and *Crepidula* are based on individuals.

***Sample actually collected somewhere between 13225-43700 and 13325-43700.

****Adductor muscle not included.

REFERENCES

1. N. Bourne, J. Fish. Res. Bd. Can. 22, 1137-1149 (1965).
2. G.S. Jamieson and R.A. Chandler, Can. J. Fish. Aquat. Sci. 40, 313-318 (1983).
3. D.M. Anderson and A.W. White (eds.), Toxic Dinoflagellates and Marine Mammal Mortalities (Woods Hole Oceanogr. Inst. Tech. Rept., WHOI-89-36, 1989).
4. J.R. Geraci, D.M. Anderson, R.J. Timperi, D.J. St.Aubin, G.A. Early, J.H. Prescott and C.A. Mayo, Can. J. Fish. Aquat. Sci. 46, 1895-1898 (1989).
5. I.C.T. Nisbet, Condor 85, 338-345 (1983).
6. K. Sharifzadeh, N. Ridley, R. Waskiewicz, P. Luongo, G.F. Grady, A. DeMaria, R.J. Timperi, J. Nassif, M. Sugita, V. Gehrman, P. Peterson, A. Alexander, R. Barrett, K. Ballentine, J.P. Middaugh, and I. Somerset, Mortality and Morbidity Weekly Report 40, 157-161 (1991).
7. S.E. Shumway, S. Sherman-Caswell and J. Hurst, J. Shellfish Res. 7, 643-652 (1988).
8. J.L. Martin, A.W. White and J.J. Sullivan in: Toxic Marine Phytoplankton, E. Graneli et al., eds. (Elsevier, Amsterdam 1989) pp. 379-384.
9. J.C. Medcof, A.H. Leim, A.B. Needler, A.W.H. Needler, J. Gibbard and J. Naubert, Bull. Fish. Res. Board Can. 75, 32 pp.