

PARALYTIC SHELLFISH POISONING IN MAINE: MONITORING A MONSTER

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INTRODUCTION

Blooms of the toxic dinoflagellate, *Protogonyaulax tamaricensis*, are a common, seasonal occurrence in the Gulf of Maine. Shellfish (e.g., mussels, clams, oysters, scallops) can accumulate the toxins produced by *P. tamaricensis* rendering them vectors of paralytic shellfish poisoning (PSP).

Outbreaks of PSP present a problem with respect to optimal utilization of the shellfish resource as well as public safety. Magnitude of the economic losses is large, ranging from catastrophes (e.g., New England, 1972) and an estimated loss in excess of \$7,000,000 in Maine in 1980 to the recurrent costs associated with the preventative shellfish monitoring programs.

Maine has established a comprehensive sampling program that has expanded over the years to accommodate the rising value of the resource (see Figure 1; Table 1) and the expansion of the species of shellfish utilized. Maine has the largest PSP monitoring/testing program in the country with approximately 3500 samples being run annually. This number, too, increases with the increasing number of areas affected.

In this paper we describe the monitoring program from its initial efforts to determine toxicity levels of shellfish to current efforts to effectively manage a resource in the presence of a potentially lethal phenomena. In addition, data are presented on detoxification studies in *Placopecten magellanicus* and their implications discussed.

HISTORY

The monitoring of shellfish as potential vectors for paralytic shellfish poisoning (PSP) and closure of shellfishing areas began in Maine in 1958. Following a serious outbreak of PSP in nearby Canada in 1957, five monitoring sites were established in this eastern Maine area. Closures were made in portions of this area in 1958, 1959, 1961, 1964, 1969, 1970 and 1972 whenever scores exceeded 80 μg of toxin. This limited monitoring plan, coupled with state of the art knowledge of the monitoring results in nearby Canada, provided adequate public health protection in eastern Maine. Prior to 1972, only occasional testing of the entire coast was conducted. This expanded sampling in 1961 resulted in two permanently closed areas around Martinicus and Monhegan Islands. Until 1972, no other areas

were closed, although occasional low toxin scores were noted. In 1972, there was a closure in early August in eastern Maine.

In mid-September of 1972, it became evident that there were extremely toxic shellfish from Cape Ann, Massachusetts into western Maine. The aftermath of this discovery was the closure on September 15, 1972 from Cape Elizabeth to New Hampshire followed by closure of the entire coast on September 17th. Most of the coast, with the exception of Cape Elizabeth to New Hampshire, was reopened on September 30, 1972. Much of the area remained closed into 1973.

1973 did not require any new closures other than the historical area in eastern Maine; however, 1974, was a year of high toxicity. In the absence at that time of a precise sampling plan, it was a year of multiple crises. Although the laboratory was able to keep up with these crises, this 'shot-gun' method of monitoring toxicity levels can not be considered as a responsible public health protection program. Further, the lack of precise knowledge as to the areas which were toxic led to unnecessarily large areas being closed in order to give adequate public health protection.

Late in 1974, funding was obtained from the New England Regional Commission (NERCOM) to develop a monitoring program and to investigate the possibilities for the depuration of toxic shellfish. Work carried out under this contract was directed towards finding ways to reduce the impact of toxic dinoflagellate blooms on the shellfish industry of Maine. A greatly expanded monitoring program was implemented so as to provide more precise information about the temporal and spatial distribution of toxicity during bloom periods. To this end, 119 sampling stations were established along the coast of Maine.

The monitoring program at that time consisted of a series of 18 primary, 35 secondary, and 63 tertiary sampling stations. These were established on the basis of previous data. Primary stations were sampled on a weekly basis throughout the peak period of toxicity, i.e. April-October. Primary sampling stations were those which, in the past, were shown to be good indicators of the presence of toxins when present at low levels. Once toxicity was established at a primary sampling station, samples were taken at secondary and tertiary sampling locations. Secondary sampling stations were chosen on the basis of past results, as good indicators of what might be expected to

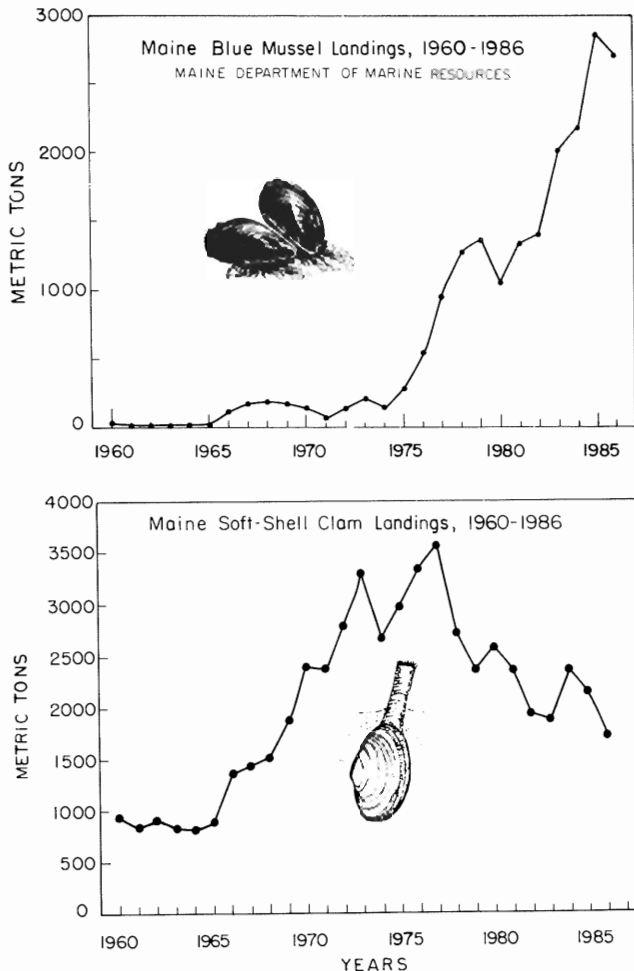


Figure 1. Landings data for the blue mussel, *Mytilus edulis*, and the soft-shelled clam, *Mya arenaria*, in the State of Maine from 1960-1986.

occur in a given clam growing area. Tertiary stations were chosen to fill in the gaps between the secondary sampling locations and to further localize the distribution of toxic areas. It was then, as it is now, the goal of the program to provide a safe method of monitoring the coast to ensure public health safety while at the same time causing the least disruption to harvesting activities.

Current Monitoring Procedures and Modifications

Years of practical experience have afforded us the opportunity to continually modify the sampling program. Presently, we instead consider areas. The coast of Maine is divided into 18 different areas from southwest to northeast with Area 10 the most southerly and area 27 in Cobscook Bay, the most northerly (Figure 2, Table 2).

At the beginning of the PSP testing year, we collect shellfish samples (mussels, *Mytilus edulis*, and clams, *Mya arenaria*) from each of these areas to determine the background level of toxicity (hopefully below quarantine levels). Four or five stations are sampled per area, usually

TABLE 1.
Maine landings of selected bivalve mollusks.

Year	(Thousands of Bushels)					Total
	Scallop	Clam	Mussel	Quahog	Oyster	
1980	539	378	155	?	1.0	1073.0
1981	639	346	197	1	1.1	1181.1
1982	266	452	208	7	3.1	936.1
1983	329	382	299	27	2.3	1039.3
1984	269	347	289	30	2.5	937.5
1985	135	317	406	38	5.1	901.1
1986	121	252	440	98	45	956.0

Year	(Thousands of Dollars)					Total
	Scallop	Clam	Mussel	Quahog	Oyster	
1980	10,752	8,554	546	?	62	19,914
1981	15,246	8,409	852	15	69	24,591
1982	6,295	10,236	903	98	200	17,732
1983	10,881	10,040	1,408	450	152	22,931
1984	9,437	11,606	1,668	510	168	23,389
1985	4,523	12,132	2,079	684	383	19,801
1986	4,160	12,300	2,319	1,960	3,600	24,339

including the original 'primary' station. These 'base' stations are sampled each week from April-October regardless of toxicity patterns. They are set up based on our historical information (and general trends) so that a closure can be made and the area described without having to return and resample the area before making the decision. A large part of the success of our program is based on the speed at which a closure can be made. When the shellfish show toxicity, we expand our sampling, from the points of land inshore, until we find stations of little or no toxicity. With more expanded sampling, we can adequately describe toxic areas. This relatively heavy sampling concept has allowed us to manage around PSP closures, e.g., Casco Bay in 1979.

In 1979, using the information derived from our sampling program, we were able for the first time as a part of our management plan, to keep open a portion of the area, with the exception of mussels, which would have normally been closed for all shellfishing. This entailed a heavy-handed sampling program. Although the growing area which we sampled intensively was relatively small, during the 55 day closure approximately 155 shellfish diggers harvested 17,050 bushels of soft-shelled clams with a landed value of \$426,250 and an estimated consumer value of \$2,770,625 (Maine DMR).

Another area which we have been able to manage successfully is Cobscook Bay. By examining our extensive data base, we selected potentially safe areas. These areas are sampled twice a week throughout the extent of the potential problem. As the demand increases, we can develop more areas that can be similarly managed.

Mussels, *M. edulis*, are our best prospect as an "alert" species and at the present time are they are being used as

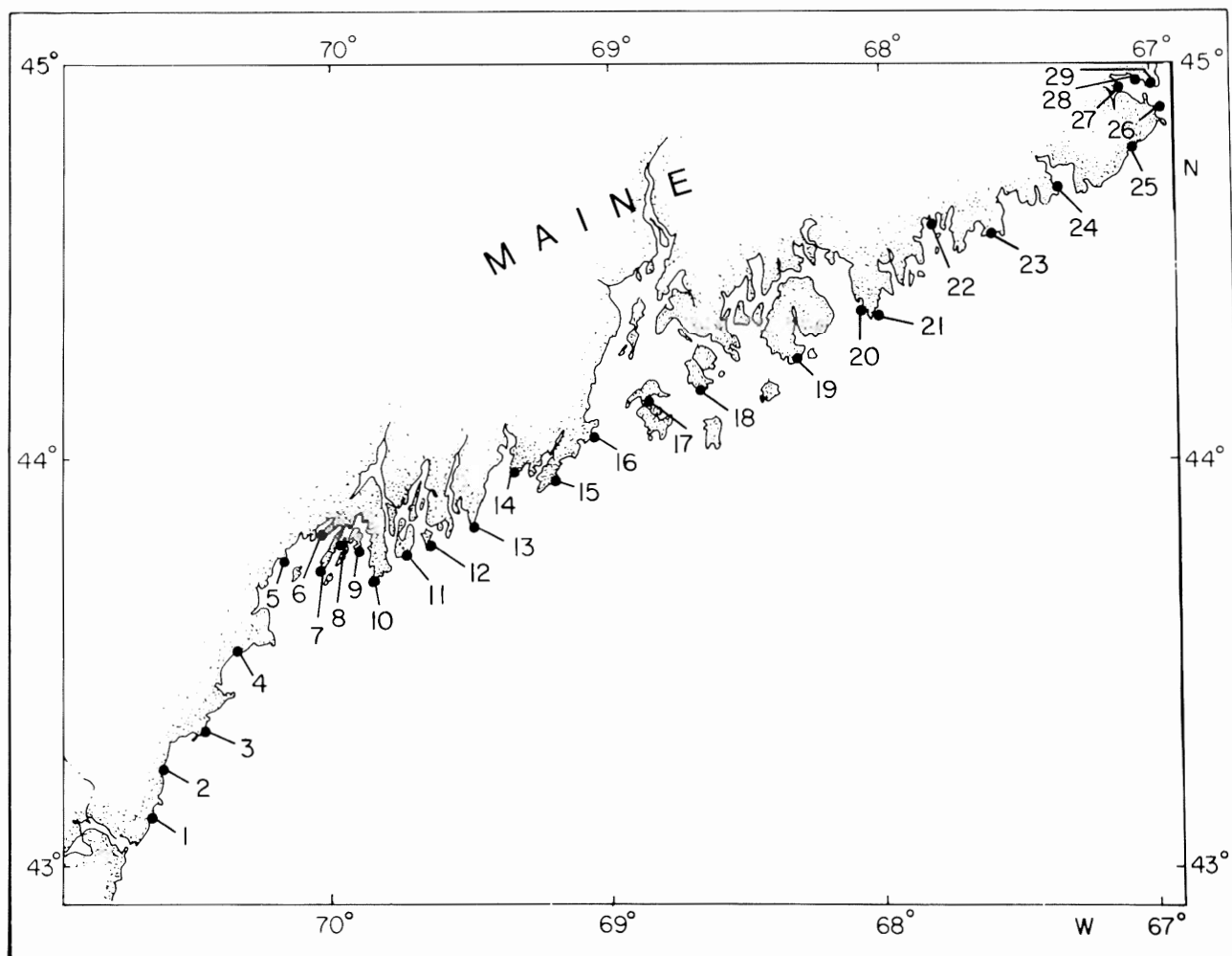


Figure 2. PSP sampling stations along the coast of Maine.

such. They become toxic approximately one week before *Mya arenaria* (Hurst and Gilfillan 1975, Shumway and Cucci 1987). With the recent risk in the harvesting of *M. edulis* and its gaining popularity (see Figure 1) as an inexpensive substitute for the soft-shelled clam, we take a greater risk of toxic shellfish on the market. Further expansion of our mussel sampling, possibly to the offshore islands, could give us a better early warning. Rises of toxin level in *M. edulis* and *Modiolus modiolus* have occurred without apparent alert. The best example of this is demonstrated by a rise in toxicity at Pemaquid Point in August of 1980 where routine sampling saw toxin levels at detection level rise to $8000 + \mu\text{g} \cdot 100\text{g}^{-1}$ in two days (Figure 3). This rise resulted in the prohibiting of harvesting of mussels coastwide. Regular sampling offshore may help us to avoid this in the future or at least give warning.

In addition, winter sediment samples have been collected since 1980 along the coast of Maine for determination of the distribution of the resting cyst stage of *P. tamarensis*. The patterns of cyst distribution generally follow those of toxicity shown in Figures 4 and 5, but are not reli-

able as a predictive index for increases in shellfish toxicity (Thayer et al. 1983). Greater concentrations of cysts are also found in deep water sediments off the coast of Maine (Dale et al. 1978) than in the inshore waters. The direct role of the resting cyst stage in shellfish toxification is still being evaluated.

One thing that is evident from our records is the "PSP sandwich", the area of the mid-coast that is relatively free from the toxic dinoflagellate, beginning at the west side of Penobscot Bay extending northeast to Mt. Desert Island and beyond (Figures 4 and 5).

Maine imposes shellfish closures whenever toxin levels reach $80 \mu\text{g} \cdot 100\text{g}^{-1}$. While it is theoretically possible to suffer poisoning from eating shellfish with low toxicity scores, there is no evidence that shellfish up to $80 \mu\text{g} \cdot 100\text{g}^{-1}$ toxicity are unsafe. The available evidence indicates that the current quarantine level gives a significant safety factor as yet undefined.

Unlike Canada where the 'key' early warning monitoring stations are in areas closed for shellfishing year round, Maine's 'key' stations are adjacent to the principal

TABLE 2.
Locations of Sampling Stations (see Figure 2).

Station Number	Location
1.	York River (York)
2.	Ogunquit River (Ogunquit)
3.	Cape Porpoise (Kennebunkport)
4.	Spurwink River (Scarborough)
5.	Littlejohns Bridge (Cousins Is., Yarmouth)
6.	Mere Point (Brunswick)
7.	Potts Point (So. Harpswell)
8.	Lumbos Hole (Orrs Is. Harpswell)
9.	Cundys Harbor (E. Harpswell)
10.	Head Beach (Phippsburg)
11.	Little River (Georgetown)
12.	Newagen (Boothbay Area)
13.	Pemaquid Point (Bristol Area)
14.	Garrison Island Bar (Friendship)
15.	Port Clyde
16.	Spruce Head
17.	Vinalhaven Island
18.	Stonington (Deer Isle)
19.	Bass Harbor (Mount Desert Island)
20.	Mosquito Harbor (Winter Harbor)
21.	East Pond Cove (Schoodic Point)
22.	Ray Point (Milbridge)
23.	Henry Point (Jonesport)
24.	Starboard Island Bar (Machiasport)
25.	Moose River (Trescott)
26.	Lubec Channel
27.	Halowell Island (Whiting)
28.	Leighton Point (Pembroke)
29.	Perry-Eastport Causeway

shellfish harvest areas. Canada's experience indicates that a rise in toxin levels in clams at a key station will give, in most years, approximately a ten day warning of an expected increase in toxin levels in their principal shellfish areas. This toxin increase can be regarded as a significant 'alert' warning which allows safe management and planning of monitoring.

In Maine, the spring season mussels become toxic approximately one week prior to a rise of toxin in clams. Thus, mussel toxin levels present a fairly reliable prediction of a rise of toxin levels in clams. During fall blooms, rises of toxin levels in mussels have been observed over a period of 1–2 tides. This sharp rise in toxin has led to the speculation that an alert toxin level should be established below $80 \mu\text{g} \cdot 100\text{g}^{-1}$. As an alternative for an alert toxin level, Maine has opted for critical observation of the mice for low level toxin reactions. This subjective observation takes considerable experience and is not a good substitute for a defined alert level which can be noted by anyone. The mouse bioassay is not sensitive enough to give reliable low toxin levels thus a substitute testing method such as high performance liquid chromatography (HPLC) must be used. With Maine's intense monitoring in place, whether an alert level below $80 \mu\text{g} \cdot 100\text{g}^{-1}$ would add significantly to a public safety monitoring plans remains to be seen.

Toxicity in scallops, *Placopecten magellanicus*

There has been a continued interest in developing an economic use for the discarded portion of scallops, the so-

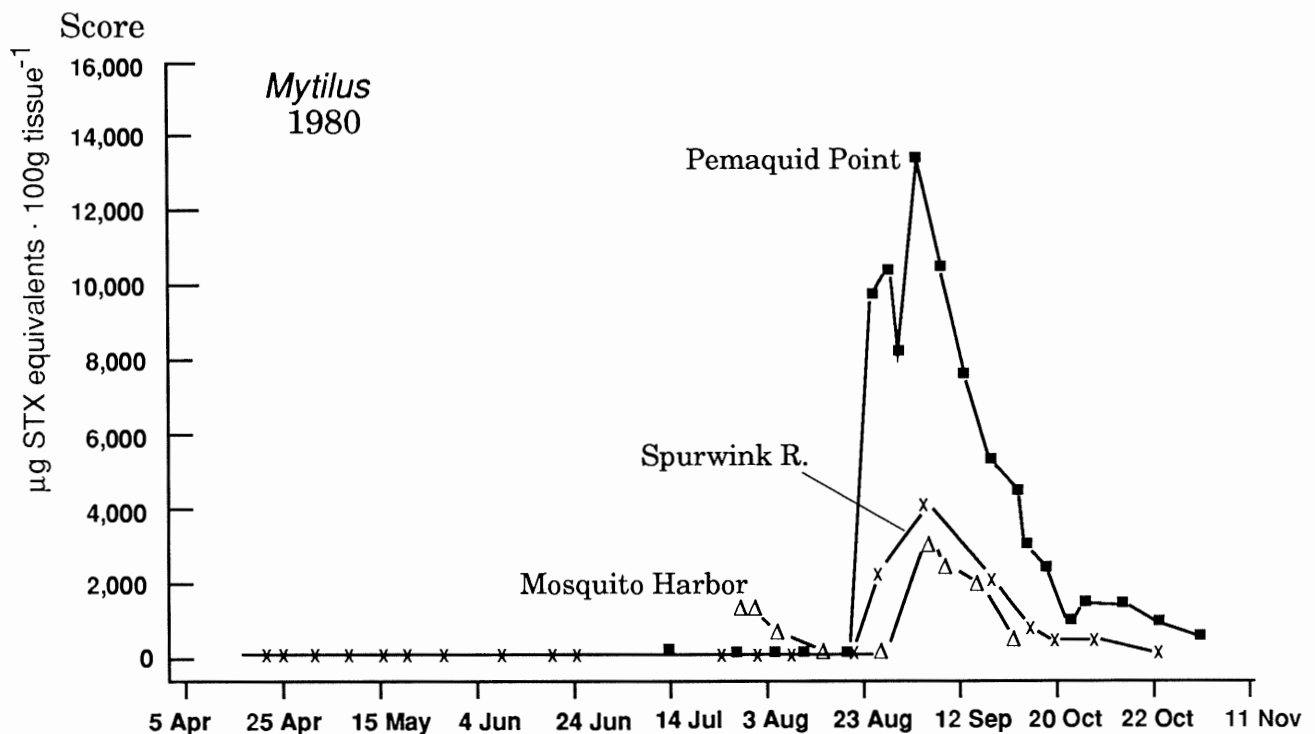


Figure 3. Toxicity levels (score) in μg toxin 100g tissue^{-1} for the blue mussel, *Mytilus edulis*, from various localities on the Maine coast.

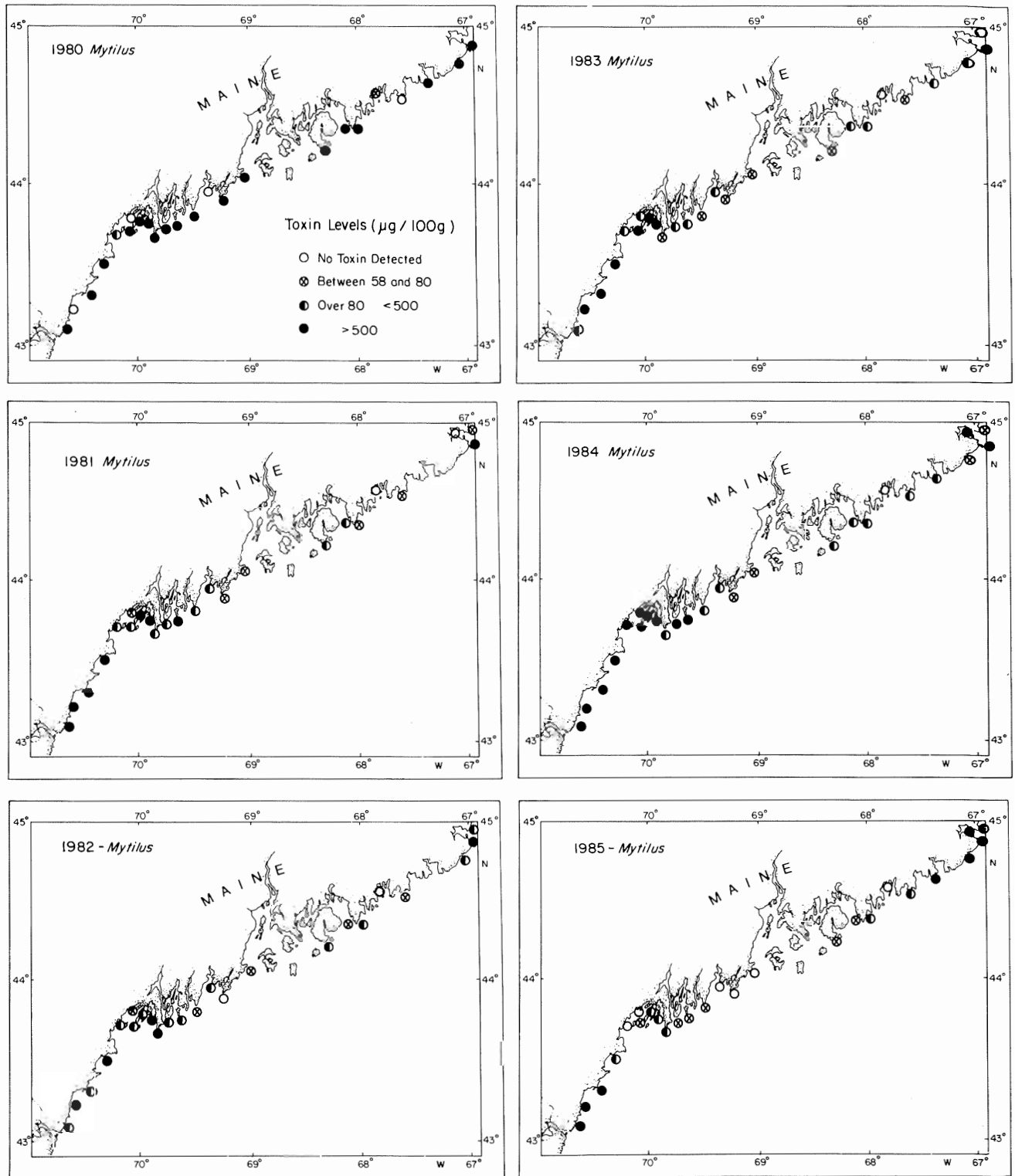


Figure 4. Toxin levels for the blue mussel, *Mytilus edulis*, along the coast of Maine from 1980–1985.

called 'rings and roes' (Bourne and Read 1965, Dewar et al. 1971). These tissues are comprised of the mantle, gonad, gill and digestive glands and can constitute weights equal to that of the excised adductor muscles. While there are several factors which have precluded the use of these

parts (e.g., keeping quality, market acceptability), the most prevalent problem in New England and Canadian waters is the accumulation of neurotoxins derived from dinoflagellates, i.e., *Protogonyaulax tamarensis*.

A number of authors have reported on the levels of tox-

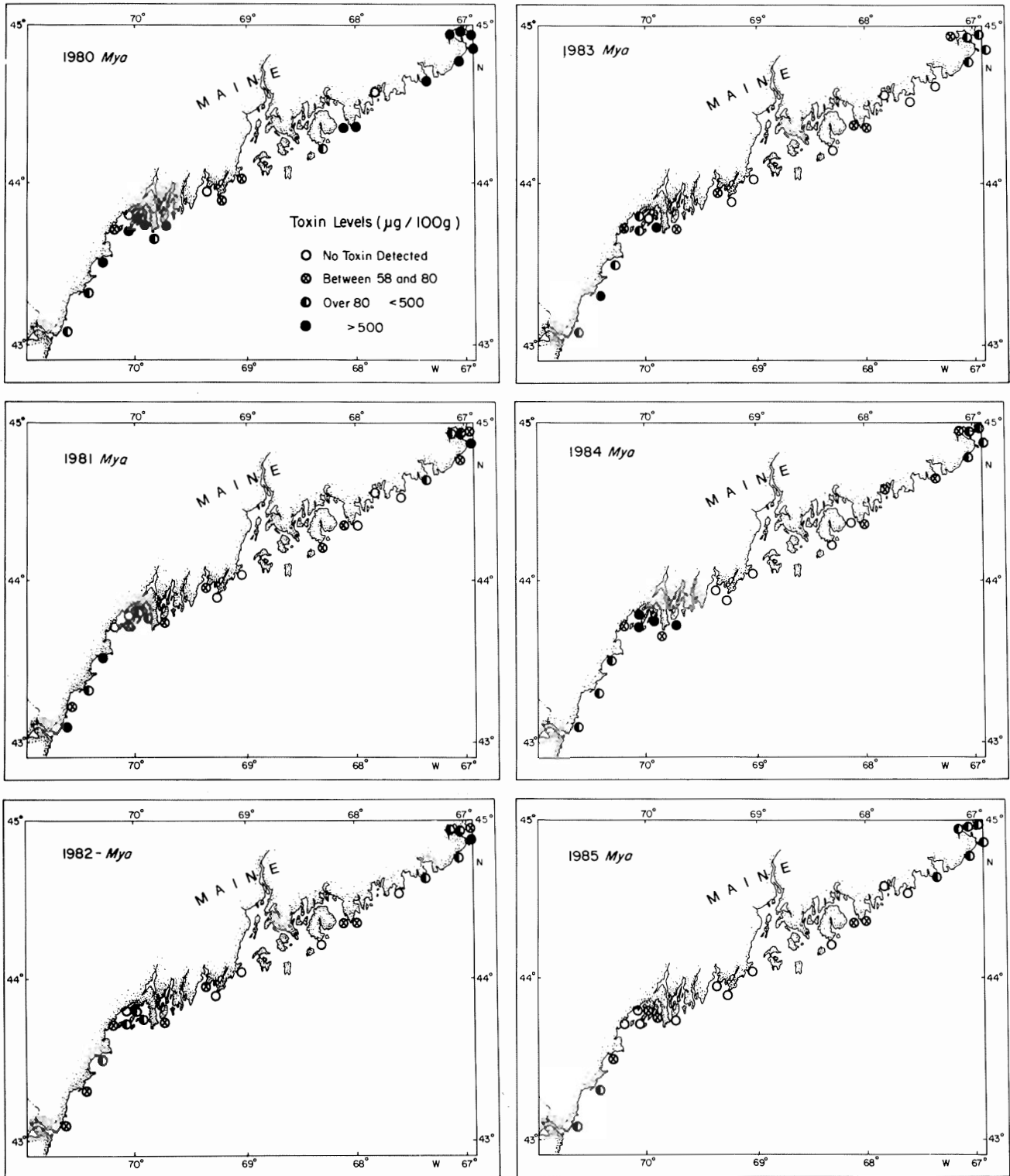


Figure 5. Toxin levels for the soft-shelled clam, *Mya arenaria*, along the coast of Maine from 1980–1985.

icity of various scallop tissues (Bourne 1965, Hsu et al. 1979, Shimizu and Yoshioka 1980, Noguchi et al. 1981, Ogata et al. 1982, Ueda et al. 1982, Jamieson and Chandler 1983) and a number of generalities have emerged:

1. The adductor muscle does not accumulate toxins and has, in fact, been shown to inactivate the toxins

when present (Shimizu and Yoshioka, 1980). One exception has been reported in the purple-hinged scallop, *Hinnites giganteus*, where toxin levels reached $2000 \mu\text{g} \cdot 100 \text{g tissue}^{-1}$ (Anonymous 1980),

2. Digestive gland, mantle, gonad and gill tissues all

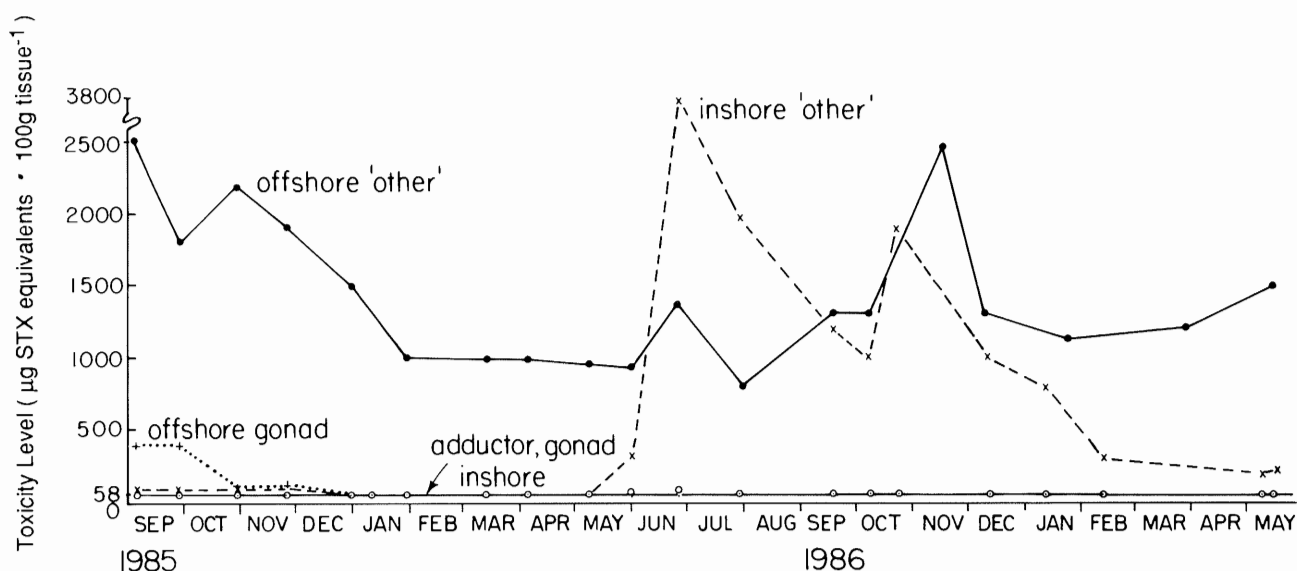


Figure 6. Toxicity levels of scallops, *Placopecten magellanicus*, from inshore (~30 m depth) and offshore (~150 m) collection sites. Data are given for gonad, adductor muscle and 'other' (including mantle gills and digestive glands).

retain the toxins although the levels vary between tissues and between species (Ueda et al. 1982, Jamieson and Chandler 1983, Bourne 1964, Medcalf et al. 1947),

3. There are seasonal variations in toxicity level of the various tissues (Bourne 1965, Jamieson and Chandler 1983).

A recent renewed interest in trying to market not only scallop meats with roes attached, but whole scallops, has prompted further investigation in our laboratory regarding the seasonal distribution of toxin levels in the various tissues of the giant scallop, *Placopecten magellanicus*. Our results are given in Figure 6. Gonad tissues from inshore scallops were only toxic during May and June of 1988. The remaining tissue (digestive gland, mantle and gill) was toxic from September–November of 1985, May–December of 1986 and January–May of 1987. Scallops from offshore areas showed consistently high levels of toxicity in the 'other tissue' and high levels of toxicity in the gonads in September and November of 1985. These findings are similar to those of Bourne (1965) who demonstrated seasonal fluctuations in toxicity levels of the livers (digestive glands) and mantles of *P. magellanicus* from the Canadian east coast. Jamieson and Chandler (1983) also demonstrated seasonal variations in toxicity levels with peak toxicities occurring during fall and winter months.

Two interesting points emerge from our data. First, the scallops from the inshore water demonstrated high levels of toxicity even during periods when blooms were not evident. Second, the scallops collected from offshore areas (depths greater than 180 m) remained toxic throughout the year and showed some of the highest levels recorded in our study. The source of toxin for these deep water scallops is not clear. Bourne (1965) first suggested the possibility that scallop toxicity could be caused by the resting cyst stage of

P. tamarensis. These cysts are present in sediment samples and have been shown to be considerably more toxic than their motile cell counterparts. This possibility was later suggested again by Yentsch and Mague (1979) and Jamieson and Chandler (1983). Data available on the distribution and abundance of cysts (Lewis et al. 1979, White and Lewis 1982, Thayer and Lewis 1983) suggest that there is a broad distribution of cysts, sometimes in high densities, in the sediments off the Coast of Maine and in the Bay of Fundy. However, the abundance of cysts in locations of commercial scallop beds has been shown to be low (Sherman-Caswell unpublished). More recently, Anderson (1984) argued that a scallop would require consumption of as many as 100 million cysts to achieve the toxin levels recorded in deep water scallops! He recommended caution in assigning toxic events to cyst ingestion and our results support his skepticism. In our most toxic individuals ($>1500 \mu\text{g} \cdot 100\text{g}^{-1}$), careful examinations were made to identify and count the cysts present in the guts. In no instance could we locate any cysts. Further, in a series of experiments to determine the time necessary for these scallops to detoxify, we found that after a period of 4 months, toxicity levels had only decreased by approximately one third (Figure 7).

We suggest that the toxicity observed in the deep water scallops may be due to the ingestion of resting cells of *P. tamarensis* during the bloom period (early spring and fall) and the consequent slow rate of detoxification. It is also possible that the scallops transform the toxins and store them from one season to the next.

Our results, along with those of previous authors, suggests that the gonad tissue of *P. magellanicus* could be safely marketed from most geographic locations tested. Caution should still be observed and frequent sampling for toxin levels should be maintained. We strongly caution

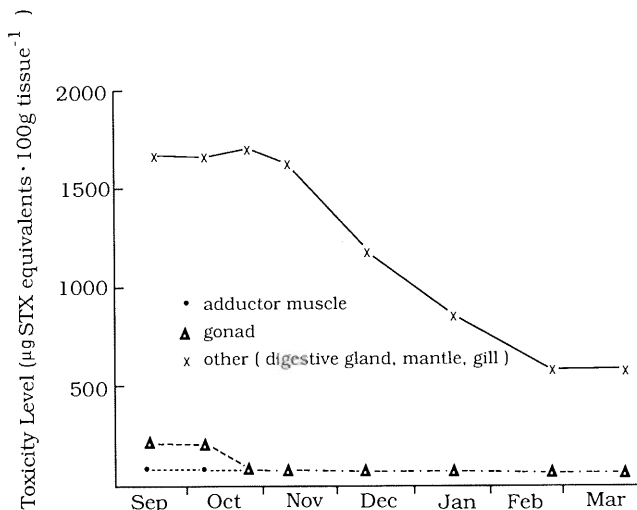


Figure 7. Toxicity levels of scallop tissues (*Placopecten magellanicus*) during detoxification. Animals were placed in filtered, running sea water at the beginning of the experiment (September) and maintained under these conditions through March.

against the marketing of whole scallops from any area of the coast of Maine or Canada due to the high levels of toxins recorded from digestive glands and other tissues and to the unpredictable seasonal variation in those toxin levels.

Toxicity in ocean quahogs, *Arctica islandica*

In 1985, we began more intensive screening of the ocean quahog, *Arctica islandica*, for paralytic shellfish toxins. The quahog fishery is relatively new with the majority of harvesting done around the Jonesport and Machias Bay areas. Around late August, there was a significant rise in the toxicity level of these shellfish. Prior to this period, only low levels of toxin had been detected. Samples taken from commercial dealers showed levels well over quarantine, with a maximum of $1895 \mu\text{g} \cdot 100\text{g}^{-1}$ in August. Quahogs remained toxic into October. Sampling stations off of Jonesport (east and south) and Machias Bay, remained toxic through the spring of 1986 with levels ranging from $80\text{--}532 \mu\text{g} \cdot 100\text{g}^{-1}$. The area from the Jonesport-Beals Bridge to the International Border (seaward to the U.S. jurisdiction) was closed to the harvesting of ocean quahogs on August 6, 1985. After samples of quahogs were tested as safe, a portion of these waters, Machias Bay and inshore of a line from Jonesport to Cutler, were reopened. A part of this closure still remains in effect.

The source of the toxin for these quahogs has yet to be determined although the original rise in toxicity in late August was also seen in blue mussels, *M. edulis*, at Starboard Island Bar, Point of Maine, Jonesport, which lies adjacent to the quahog beds as seen in Figure 8. Soft-shelled clams, *Mya arenaria* from the same area also showed a rise in toxin level during that period although not as severe. Other stations in the Machias area did not reflect the rise at that time. Further east at Lubec, West Quoddy Bar and Cob-

scook Bay, evidence was seen of a 'fall' bloom of *P. tamarisensis*.

A clearer understanding of the mechanisms of intoxication of near and offshore subtidal species (e.g., *Arctica*, *Placopecten*) is needed. They do not seem to follow the patterns of intoxication that are seen in the intertidal species. Once understood, this knowledge could aid in developing a more effective management plan.

Due to a lack of historical data, it is only speculation that *M. edulis* at Starboard Island Bar and Lubec would serve as a warning of a rise of toxin in the quahogs. A more intense sampling program is needed to understand this problem. As the fishery expands to other parts of the Maine coast, more extensive sampling will also be needed to manage the resource.

Toxicity in hen clams, *Spisula solidissima*

Regular testing of the surf or hen clam, *Spisula solidissima*, began in 1975. There is a small, bottom dredge fishery in southern Maine for these clams, which can also be harvested from the shores/beaches on the low drain spring tides. Eight sampling stations were established from Scarborough south to York with two stations in the Phippsburg/Georgetown area as well. These are sampled regularly throughout the year depending on the toxicity of the shellfish. *S. solidissima* has been shown to retain the toxins for over a year (Medcof et al. 1947, Blogoslawski and Stewart 1978, Shumway unpublished) and therefore these stations are sampled in the spring and summer each year and through the fall and winter months during years of toxicity. In 1980 and 1981, surf clams became quite toxic in some areas as seen in Table 3; Figure 9. The Maine coast from Georgetown to the New Hampshire border was closed to surf clams on October 1, 1980 and remained closed until May 2, 1981 when a portion between Pine Point, Scarborough and Fletchers Neck, Biddeford was opened, only to be closed again on May 29. This remained in effect until September 10, 1981, then the exception was included again and this closure remained until December 27, 1982. A large part of this closure can be accounted for by the sequestering of the toxin by these shellfish. The Higgins Beach station (Figure 9) stayed well over quarantine level for 2+ years from the initial bloom in 1980. There may have been some retoxification in the spring of 1981 as seen at Higgins Beach but Head Beach and Scarborough Beach reflect the slow depuration pattern seen in *S. solidissima*.

SUMMARY

Like other successful monitoring programs (see Chiang this volume), the objectives of the Maine PSP program are public safety and the optimum utilization of the resources. The frequent and intense sampling of growing/harvesting areas works well, prohibiting the taking of potentially toxic shellfish. Rapid implementation of closures avoids the costly problems associated with seizing commercial catches although closed areas must be patrolled frequently.

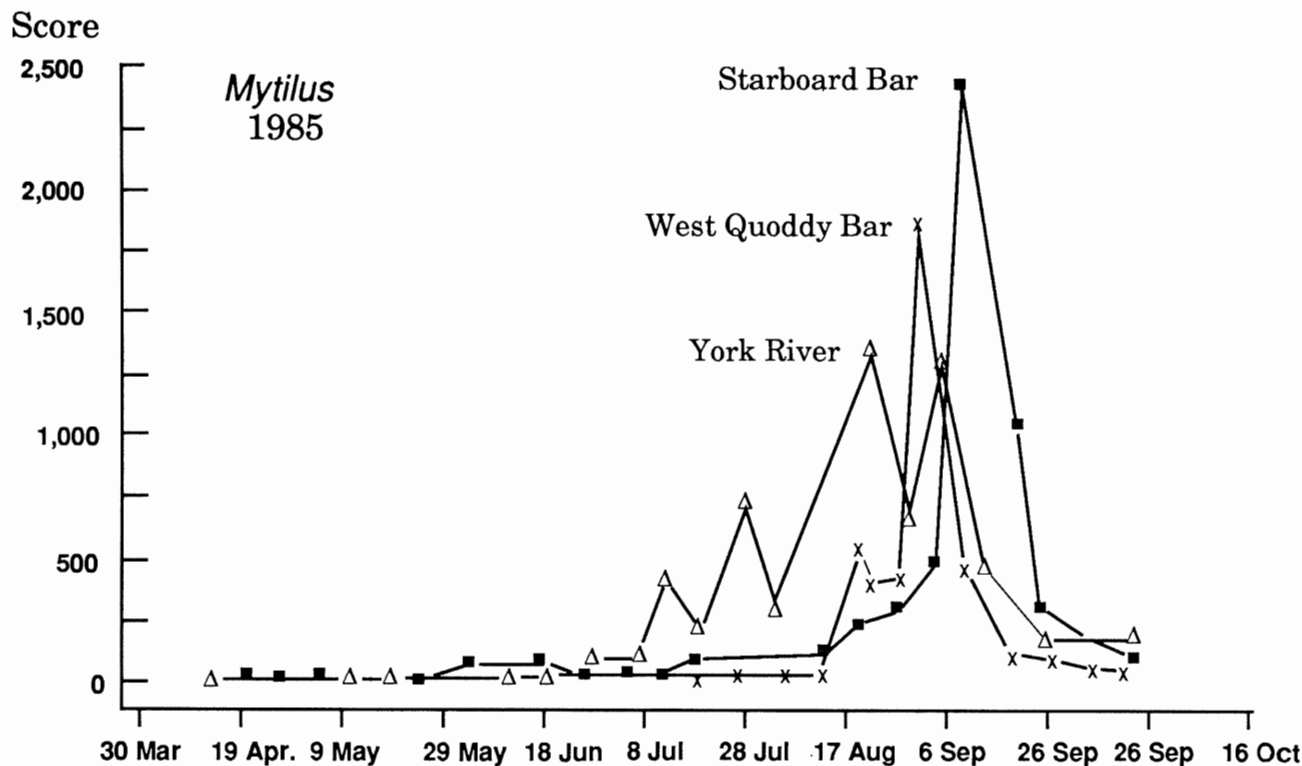


Figure 8. Toxicity levels (score) for the blue mussel, *Mytilus edulis*, from various localities on the Maine coast.

Further, the program should be in touch with the industry and aim for optimum use of the resources. It must be flexible enough to include new species entering the fishery, e.g., *Arctica islandica*, as well as account for new ways to utilize species already monitored, e.g., *Placopecten magellanicus*. Up to date knowledge of the shellfishing industry and its demands is required as the closures of large areas of

the coast that are not species specific can be very costly. The program must be intensive and distinguish larger harvestable areas.

To be effective, a monitoring program not only should be intensive in its screening of shellfishing areas, but have a good early warning agent. Plankton samples can be collected fairly easily, but must be analyzed immediately to be of any help. This requires a large staff. In addition, total numbers of cells may not reflect the predicted toxicity of the shellfish (Cembella et al. 1987). Better sampling of offshore populations (e.g., *Mytilus*) could aid in early warning; it has been observed that the offshore islands can become toxic before the mainland (Maine DMR). This, too, requires a larger staff.

Finally an effective monitoring program must be flexible

TABLE 3.
Maximum Scores ($\mu\text{g} \cdot 100 \text{ g tissue}^{-1}$) for the surf clams, *Spisula solidissima*.

	1980	1981	1982	1983	1984	1985
Higgins Beach	4518	5316	612	193	244	<58
Scarborough		(viscera only)				
Scarborough Beach	1752	928	161	101	470	59
Pine Point						
Scarborough Old Orchard	91	64	<58	<58	<58	—
Beach	<58	<58	<58	<58	59	—
Moody Beach						
Wells	672	365	71	174	715	162
Ogunquit Beach	78	331	152	98	172	303
Long Sands						
York	—	90	—	—	—	179
Head Beach	4993	7934	557	202	690	64
Phippsburg		(viscera only)				
Sagadahoc Bay						
Georgetown	5104	202	87	69	68	<58

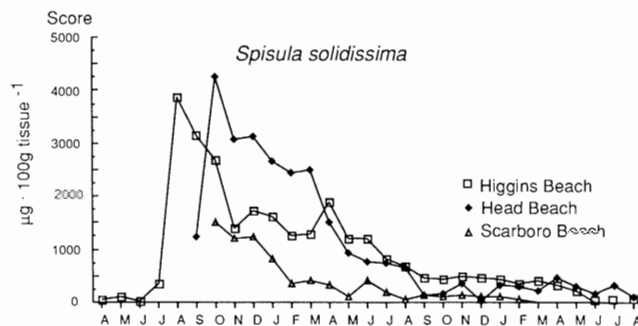


Figure 9. Toxicity levels (score) for the surf clam, *Spisula solidissima*, from various localities in Maine (1980-1982).

and able to handle day to day "emergencies" e.g., unexpected outbreaks of PSP or incidents such as the recent outbreaks of unidentified poison from Canadian waters.

The PSP monitoring program in Maine has grown continuously and is constantly being modified in an attempt to serve both the consuming public and the working industry. Economic losses due to rises in toxicity levels have been lessened. More importantly, no deaths or illnesses have been attributed to PSP from shellfish harvested in the State of Maine. All documented illnesses have resulted from recreational harvesting. This can only be corrected by increased public awareness. To this end, notices are posted that can be seen by land access to all closed areas. Shellfish dealers and towns involved are notified of all closures.

Closure notices are published in newspapers and are announced on the weather broadcasting radio station. Finally, several articles (newspapers and magazines) have been written on the subject of toxic shellfish. The goal is to keep the public aware of their responsibility to use the resource wisely without seriously affecting the market for Maine shellfish and ensuring the safety of the product. The program has become more effective with time but there is always room for improvement.

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