PARALYTIC SHELLFISH POISONING

Paralytic shellfish poisoning, also termed "Red Tide", struck the Massachusetts, New Hampshire and Maine coasts in mid-September. The shellfish poisoning is caused by the planktonic dinoflagellate *Gonyaulax tamarenensis*. The dinoflagellates are unicell organisms with both plant characteristics (i.e. chloroplasts for photosynthesis) and animal traits (i.e. locomotion). They are enclosed in a smooth theca (case) of cellulose plates arranged in a definite pattern. The two flagella, one in an equatorial groove, provide limited propulsion.

*Gonyaulax tamarenensis* is thought to be present in Massachusetts waters throughout the year, but this plankton is such a small component of the ecosystem that its effect is negligible. However, when certain conditions are right, *G. tamarenensis* can explode into a bloom that may or may not discolor the water to a rusty brown and may leave the water slimy to the touch. High water temperature, light intensity and nutrient content, along with low salinity and stability of the water
column all seem to enhance the chances of a bloom. In Massachusetts the outbreak of PSP occurred Labor Day weekend after a major rainfall that lowered the salinity in the estuaries. This salinity drop was concurrent with the fall phytoplankton die-off and the subsequent release of nutrients into an ocean that was at its warmest temperature of the year. With these coinciding factors, *G. tamarensis* multiplied so fast that it became the dominant planktonic life. The *G. tamarensis* bloom first appeared off Gloucester and spread south to Manchester. Other outbreaks occurred in Nauset Inlet area and Pleasant Bay, Orleans, with traces in Scituate Harbor and Green Harbor. By far the highest levels of PSP were recorded from Manchester northward.

Although this planktonic bloom has been termed a "Red Tide" by newsmen and the public, it is an ambiguous name at best. There are many species of dinoflagellates that cause colored blooms: *Bodo* sp.; *Gynodinium splendens*; *Gynodinium breve*; *Mesodinium* sp.; *Noctiluca scintillans*; *Gonyaulax tamarensis*; *Gonyaulax catenella*, etc. However, only three of these have any detrimental effect on the environment. *Gynodinium breve* secretes a poison that will kill finfish, but leaves shellfish unharmed, while *Gonyaulax catenella* on the west coast and *Gonyaulax tamarensis* on the east coast affect only molluscs. In Canada, a *G. tamarensis* bloom is not called a red tide because that would confuse it with similarly appearing non-toxic blooms, or one that is poisonous to finfish. They have named it Paralytic Shellfish Poisoning, which is what it should
be called in Massachusetts.

Paralytic shellfish poisoning occurs every summer in the Bay of Fundy and the Gulf of St. Lawrence in Canada. Since 1880, there have been 187 reported cases of illness and 24 deaths attributed to PSP in eastern Canada. Most poisonings were from ingestion of clams and mussels, but some were from consuming whelks. PSP is an accepted way of life in Canada with effective governmental regulation of the shellfisheries. The unknowing tourists seem to be the ones most often poisoned.

Bivalve molluscs are the only seafood that can ingest, maintain and build up the poison of *G. tamaeensis* (whelks can be poisonous because they may prey on poisoned bivalves, and thus absorb their poison, but whelks are not commonly eaten in Massachusetts). Clams (*Mya arenaria*), mussels (*Mytilus edulis*), quahogs (*Mercenaria mercenaria*), scallops (*Placopecten magellanicus*), and oysters (*Crassostrea virginica*) are all bivalve (two shell) molluscs that feed on plankton they filter out of the sea water. The plankton is siphoned into the shellfish, selected according to preference and stored in the gut. Seawater and other unwanted materials are removed through the excurrent siphon. Clams and mussels accumulate the highest poison counts of the shellfish. Mussels seem to prefer dinoflagellates to other plankton, as shown by some mussels being three or four times more poisonous than soft shell clams taken from the same area. Quahogs were comparatively unaffected by PSP: there was not a single contaminated quahog (*Mercenaria mercenaria*) found in the sampling areas which produced contaminated soft shell
clams and mussels. The quahogs may selectively reject dinoflagellates as food, much like the mussels seem to select for dinoflagellates, but not enough is known to be sure of either hypothesis. Scallops are never banned in Canada, even in the worst PSP areas, for one important reason. Although the scallop viscera absorbs relatively high amounts of poison, it absorbs very small amounts into the adductor muscle, which is the only part that is commonly eaten. Many scallops sampled in Massachusetts had viscera that proved positive while the adductor muscle was negative. So few oysters inhabit the areas where *G. tamarensis* occurred that their susceptibility to PSP is unproven. Some evidence points to lobsters being poisoned by eating PSP infected shellfish, but not being a filter-feeder, the lobster would never accumulate enough toxin to poison anyone.

*Gonyaulax tamarensis* produces a non-protein poison that dangerously affects the nervous system of warm-blooded animals. *Gonyaulax catenella* and *Gonyaulax acatenella*, west coast species of the same genus, produce a similar but weaker form of poison. When *Gonyaulax* contaminated shellfish are consumed by warm-blooded animals, the toxin immediately attacks the nervous system. One researcher experienced a numbing and prickly sensation in his lips five minutes after chewing a toxic mussel. The symptoms of paralytic shellfish poisoning usually appear within 30 minutes of ingesting the shellfish. The first signs are, as mentioned, a tingling and numbness in the lips, spreading to the face and neck, then to the fingertips and toes.
Headache, dizziness, and nausea follow. In more severe cases, the numbness invades the arms and legs, bringing incoordination, general weakness and lightheadedness. The first signs of breathing difficulty appear along with a rapid pulse. The poisoning becomes extreme when muscular paralysis sets in, accompanied by severe respiratory difficulty and a choking sensation. Most fatalities occur within 12 hours of ingesting toxic shellfish and are a result of respiratory paralysis and cardiovascular collapse.

The poison that produces these dangerous effects is a neuromuscular toxin that impedes peripheral nerves and reflex transmission, and leads to malfunction of the respiratory and cardiovascular systems. The interference of neuromuscular transmission is a result of the toxin reducing the permeability of the muscle membrane to sodium ions, thus preventing the build-up of muscle action potential, which, in turn, causes muscle paralysis.

Paralytic shellfish poisoning has no antidote, so the treatment can only be given to the symptoms. The best primary treatment is drinking plenty of warm water. This will act as an emetic to empty the stomach of the toxic shellfish, and will bring about urination to remove the poison from the blood. Apomorphine is a recommended emetic and Lloyd's reagent can be used to absorb any toxin remaining in the stomach. In case of respiratory difficulty, artificial respiration should be used until drugs such as neostigmine, DL amphetamine, epinephrine, and similar respiratory-stimulating drugs are available.

Canadian studies show that susceptibility to PSP seems to
depend on a variety of unrelated factors. Non-coastal residents are more affected by the toxin than are coastal residents, probably because the coastal residents have experienced low levels of *Gonyaulax* toxin over a long period of time. Age seems to lessen the effect of the toxin, as shown by a two-year-old, after having eaten 2 steamed clams (dosage 96µg) and a 65-year-old man, after consuming 2 dozen raw clams (dosage 8300µg). Both exhibited the same severe poisoning symptoms. Women are likely to suffer more than men after ingesting toxic shellfish. Even when body weight is accounted for, the young and the female sex are more prone to PSP.

The outbreak of PSP in New England caused 26 poisonings in Massachusetts and 7 cases in New Hampshire. Fortunately, there were no fatalities. Shellfish poisoning has occurred in Scotland, Germany, Norway, France, Ireland, Wales, Australia, New Zealand, South Africa, and Japan. The total world-wide PSP cases on record number approximately 1023 poisonings with 227 deaths.

Most of the recent poisonings in Atlantic Canada can be attributed to tourists' ignorance of the danger or the lack of respect for the posted warnings. New Englanders were caught completely by surprise with the *Gonyaulax* bloom, and as a result, 33 people were poisoned and the fishing industry suffered severe financial damage. To prevent such a catastrophe from occurring again, steps must be taken to assure the public safety and safeguard the fishing industry. Shellfish monitoring programs have been laid out to assure the public of advanced warning of any potential new outbreak. The public must be made aware that

* micrograms
PSP does not make lobsters, shrimp or finfish harmful. And a study of the biology and ecology of *Gonyaulax* should be implemented. A rational, scientific approach to the paralytic shellfish poisoning will prevent another panic that badly injured the seafood industry.


PARALYTIC SHELLFISH POISONING BIOASSAY

The bioassay was first published as an official method of the Association of Official Agricultural Chemists in 1960. The mouse unit is defined as the minimum amount of poison required to kill a 20 gram mouse in 15 minutes when 1.0 ml of shellfish extract is injected intraperitoneally. By use of purified shellfish poison as a reference standard, the response of mice can be expressed in terms of a definite weight of poison generally expressed as micrograms per 100 grams of shellfish meat (μg/100g).

Outline of method of analysis:¹

Materials needed:

1. Mice from a healthy stock weighing 19-21 grams.
2. Paralytic shellfish poison standard solution.

Method:

A CF value (correction factor) must first be determined for the mice to be used for testing. This is done by inoculating mice with serial dilutions of pure toxin to determine their resistance to the toxin compared with the standard.

The shellfish sample must be thoroughly cleansed on the outside and without damage to the body, carefully shucked into a glazed dish. They are then placed in a #10 sieve without layering and drained for 5 minutes,
then transferred to a blender and homogenized. One hundred grams of homogenate is added to a tared beaker, stirred with 100 ml of 0.1 N HCl. The pH should be about 3.0. The mixture is then boiled for 5 minutes and let to cool to room temperature. The pH of the cooled mixture is adjusted to 2.0 - 4.0 and must never exceed 4.5. The mixture is diluted to 100 ml and allowed to settle until a translucent supernatant can be decanted.

Each mouse is inoculated intraperitoneally with 1 ml of extract using a 26 gauge hypodermic needle. The time for death is recorded.

Time for death is converted to mouse units by use of a standard table (APHA, 1970). If necessary, a correction factor for weight is applied from the table in APHA, 1970. Mouse units are converted to ug poison per ml by multiplying by the CF value. Then ug poison/100g meat=(µg poison/ml X dilution factor) X 200.

Any value greater than 80 µg/100g meat is hazardous and unsafe for human consumption.

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2 An acid environment is necessary for the stability of the toxin.