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Paralytic Shellfish Poisoning: The Alaska Problem

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Imagine yourself, a few friends, and family at the beach. The weather is amazingly cooperative this time of year for Southeast Alaska, and you feel blessed to enjoy the sunshine. Even though the wind cools the temperature, the beauty of the Alaska landscape is cause enough for celebration. What a day this is! The ocean and the scenery are magnificent.

A seafood feast planned for mid-afternoon has members of your party busy harvesting shellfish from the rocky beach. In less time than expected, buckets of harvested shellfish arrive at the feet of the chef. A steamer pot of boiling salt water quickly cooks the bounty, and a few minutes later the harvest is devoured with gusto. What qualities could better represent a day in the Great Land?

Reluctant to disrupt the excitement of the outing, George tells you that he feels a strange tingling on his lips and face. Your spouse is also experiencing the same strange numbness on her face. You, too busy to eat much, don't understand as each guest complains of this strange ailment. Your spouse stumbles as she carries more food to the table. George becomes dizzy and nauseous. While helping him to a beach chair, you notice the volleyball team is leaving the playing area as each person becomes listless. The game is over, and unfortunately, so is the party.

What is happening to these people? Could seafood fresh from the ocean cause such a serious condition?

The problem is paralytic shellfish poisoning (PSP), and there is little you can do at this point except to get these victims to a medical facility and fast. A potentially lethal event, PSP is a crisis no one wants to experience. As many coastal residents know, eating personally harvested shellfish is risky. As Alaskans you need to know about PSP, what health dangers it presents, and how you can reduce your risk of contracting this dreaded ailment.

The Toxins

In Alaska microscopic single-celled dinoflagellate algae of the genus *Alexandrium* produce PSP toxins as a normal by-product. Bivalve shellfish (two shelled shellfish, like clams and mussels) feeding on these toxic algae may accumulate PSP toxins to concentrations unsafe for human consumption.

The singular term toxin is not an accurate term for PSP since there are at lease 21 molecular forms of PSP toxins. Collectively, these PSP toxins are termed saxitoxins, deriving the name from the butter clam, *Saxidomus giganteus,* where saxitoxins were originally extracted and identified. All the saxitoxins are neurotoxins that act to block movement of sodium through

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nerve cell membranes, stopping the flow of nerve impulses causing the symptoms of PSP which include numbness, paralysis, and disorientation (Mosher et al. 1964). The toxicity of PSP toxins is estimated to be 1,000 times greater than cyanide and symptoms appear soon after consuming toxic shellfish. There is no antidote for PSP, and all cases require immediate medical attention that may include application of life support equipment to save a victim's life. If the dosage is low and proper medical treatment is administered, symptoms should diminish in approximately nine hours (Kao 1993).

Saxitoxin molecules undergo chemical transformations that change one molecular form to another. Transformations are performed by the dinoflagellate cell and by many animals that acquire saxitoxins. One common transformation, termed epimerization, occurs when a portion of the original saxitoxin molecule rearranges. Scallop and mussel, for example, can perform epimerization of saxitoxin they receive from the toxic algae when the H and OSO_3^- switch locations on the number 11 position of the saxitoxin molecule (Figure 1) (Oshima et al. 1990). Such a transformation can decrease the toxicity of the original saxitoxin by 11 times. Some transformations increase toxicity. For example, a six-fold increase in toxicity occurs when a process termed acid hydrolysis separates the SO_3^- group from position 21 on the saxitoxin molecule (Figure 1) (Hall et al. 1990). Recall that your stomach is acidic and acid hydrolysis can occur after you eat the shellfish. Numerous other types of transformations occur as well as eventual detoxification that can render the shellfish safe for consumption.

The number of saxitoxin forms and their tendency for spontaneous transformation are major factors hindering development of a simple field test kit for measuring PSP toxins (Sullivan and Wekell 1988). Currently, only the mouse bioassay test is approved by Food and Drug Administration (FDA) because it simultaneously measures the total of all the saxitoxin toxicities from a sample of shellfish tissue. Simply stated, the mouse bioassay measures the saxitoxin level by timing the death of an 18-20 gram mouse following injection of fluid extracted from shellfish tissue. Because the mouse bioassay is so reliable, PSP is less of a human health problem than many other types of food born illnesses.

The Algae

PSP episodes in Alaska tend to be seasonal, occurring most often during late spring and summer. Off-season occurrences of PSP are most likely caused by retention of toxins from the summer. Shellfish become toxic when environmental conditions enable toxic dinoflagellate cells to rapidly reproduce causing a toxic bloom.

A bloom begins as a small population of toxic dinoflagellate cells in the lag phase or in the form of resting cysts residing in the bottom sediment (Hall 1982). Environmental conditions such as changes in salinity, warming water

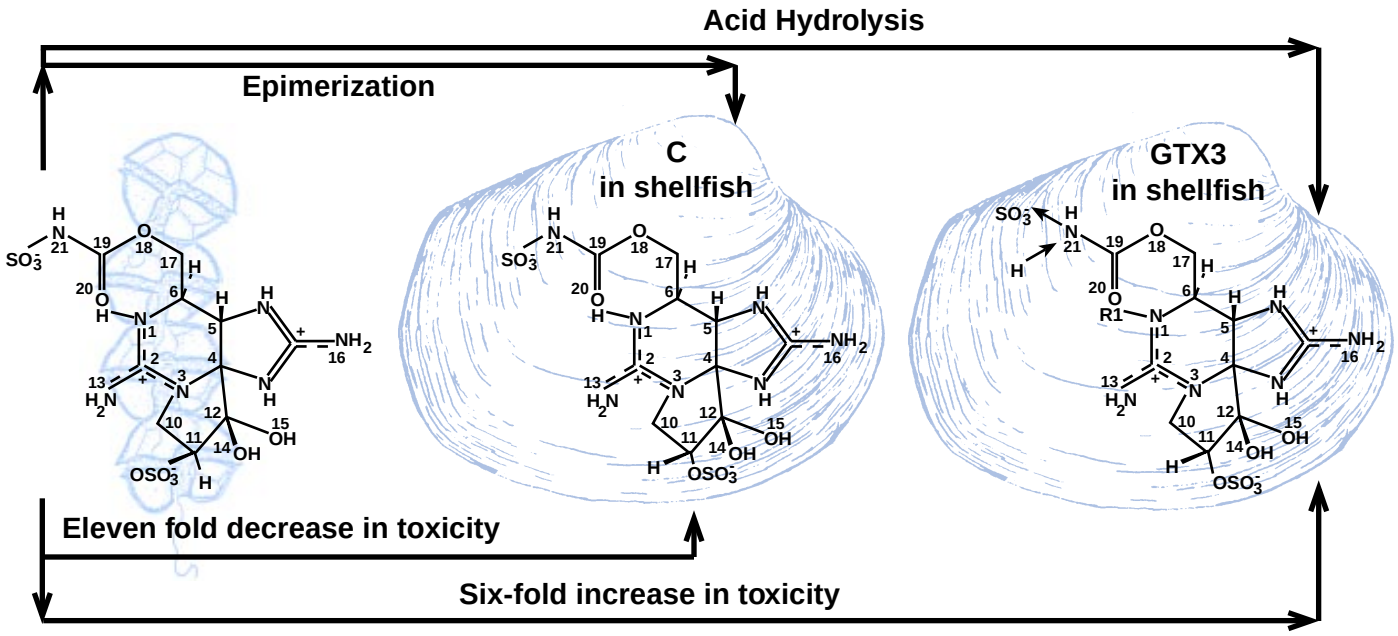

Figure 1: *Molecular transformations change the toxicity of the saxitoxin molecule. The diagram illustrates two common types of chemical transformations that occur when the saxitoxin is passed on from algae to shellfish.*

temperature, and increased nutrients and sunlight trigger cyst germination to a vegetative stage that enables rapid reproduction. Once the dinoflagellate bloom begins, an exponential growth phase causes a tremendous increase in their population. In time, depletion of nutrients and carbon dioxide in the water and degraded environmental conditions caused by the bloom decrease population growth. A stationary phase ensures leveling off the population. At this high level of the bloom, the water may assume a fluorescent reddish color referred to as a red tide. Continued environmental degradation increases cell death and ultimately leads to a population crash. At this phase of the bloom many dinoflagellate species form resting cysts that settle to the bottom, ready for the next bloom. Within this bloom cycle, the most toxic cells occur generally during the middle of the exponential growth phase, while older cells tend to undergo more toxin transformations (Anderson 1990).

PSP toxicity can exhibit a geographic pattern. For example, on the Northeast Coast of the United States dinoflagellates are more toxic in the more northern latitudes (Anderson 1990). In Alaska, varying toxin forms are found at different locations, but no clear pattern of toxicity has been determined (Hall 1982).

Toxic dinoflagellates produce more saxitoxin when nitrogen is abundant. Where phosphorus is deficient, individual algal cells become more toxic probably because the cells continue saxitoxin production but reduced cell reproduction prevents transfer of toxins to newly produced cells (Anderson et al. 1990). The net effect is that these non-reproducing cells continue to accumulate toxin.

Under laboratory culture, individual dinoflagellate cells tend to have a higher toxin concentrations when grown at lower temperatures (Anderson 1990). Again, like phosphorus limitation, the higher concentration may be caused by toxin production continuing during low temperature conditions while low temperatures reduce the rate of cell reproduction. The combined effect is higher toxin concentration in cells grown at a lower temperature.

What about a beach that has toxic shellfish while an adjacent beach has shellfish that are toxin free? This uneven toxicity is most likely caused by a patchy distribution of the toxic algae. In the ocean, cells of toxic algae are moved, concentrated, or dispersed by winds, tides, and water currents. For example, if winds and ocean currents flow in the same direction; their combined effect tends to concentrate drifting toxic algae. Opposing wind and currents often disperse the algae, decreasing the density of toxic cells. Shellfish feeding on the more concentrated patches of toxic algae will likely become more toxic (White et al. 1993).

The Shellfish

In Alaska's productive coastal waters, bivalve shellfish feed on a literal smorgasbord of microscopic algae. Bivalves are ideal conveyers of PSP toxin because they are relatively indiscriminate filter feeders, consume massive amounts of algae, are not generally killed by saxitoxins, and pass the accumulated saxitoxins on to any animal that eats them.

Six factors determine the concentration of saxitoxins in shellfish:

- The amount of toxic algae in the water as determined by the bloom size and patchiness.
- The toxin content of the individual dinoflagellate cell.
- The feeding rate of the shellfish.
- Avoidance of toxic algae by the shellfish.
- Transformation of the consumed saxitoxin by the shellfish into more or less toxic forms.
- Selective retention and excretion of the various forms of saxitoxins by the shellfish.

Shellfish nerve cells are not entirely immune from the effects of saxitoxins and degree of tolerance influences the shellfish's ability to feed and accumulate toxins. In Alaska, the blue mussel, *Mytilus edulis*, can accumulate in excess of 20,000 micrograms (mg) of saxitoxin per 100 grams of tissue, an extremely dangerous level considering that allowable limit enforced by the FDA is 80 micrograms per 100 grams of tissue. In the Kodiak area during the summer of 1993, one death and several illnesses were attributed to blue mussels containing 19,600 mg of saxitoxin. A concentration of saxitoxin that high will deliver a lethal dose of 480 mg saxitoxin by consumption of only 2.5 grams of mussel tissue or a single small mussel.

The extreme toxicity of blue mussels is due primarily to their relatively insensitivity to high toxin accumulations that enables them to continue feeding. Their high tolerance to saxitoxins and continued feeding on toxic algae can result in initially toxin-free blue mussels exceeding the FDA 80 microgram saxitoxin level in less than a 1 hour (Bricelj et al. 1990). Butter clams can be highly toxic partially because their nerve cells appear to have a special resistance to STX saxitoxin, one of the two most potent forms of the saxitoxins (Beitler and Liston 1990, Twarog et al. 1972).

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In addition, the butter clam has a distinctive ability to chemically bind the highly toxic STX saxitoxin in their siphon tissue (Beitler and Liston 1990), and they can retain PSP toxins for up to two years after initial ingestion (Hall 1982).

The Alaska steamer or littleneck clam, *Protothaca staminea*, becomes toxic but is generally less toxic than the butter clam. The lower toxicity of the littleneck clam is due partially to their ability to perform unique transformations that change highly toxic saxitoxins to the moderately toxic forms (Sullivan et al. 1983).

The combined effect of the littleneck clam's capability to transform saxitoxins to less toxic forms, and the ability of butter clams to concentrate and retain highly toxic forms can result in a wide difference in toxicity between these two species. This toxicity difference is particularly significant since butter and littleneck clams can coexist on the same beach, and, to the unskilled harvester, are similar in appearance. To exemplify the difference, one study testing for toxicity of a mixed butter/littleneck clam population found that littleneck clams were about 11-25% as toxic as butter clams (Kvitek and Beitler, 1991). The lesson here is that if you cannot distinguish the difference between a butter and littleneck clam, you should take the time to learn and return your harvested butter clams back to the clam bed.

The Pacific oyster, *Crassostrea gigas*, though not native to Alaska is an important species for aquatic farming. The Pacific oyster tends to consume toxic algae readily during initial contact but decreases and eventually stops feeding when tissue toxin levels become high (Bardouil et al. 1993).

Saxitoxin concentrations also differ among various shellfish tissues. For example, in the Pacific giant scallop, *Patinopectin caurinus*, the adductor muscle seldom accumulates saxitoxins above the FDA limit, but other tissues regularly have high levels (Table 1). It is these high saxitoxin concentrations in other tissues that have prevented development of a highly valued gonad/adductor muscle product.

Another endeavor to diversify the line of scallop products through aquaculture development in the Kodiak area was attempted on two bay scallop species; the pink scallop, *Chlamys rubida*; and spiny scallop, *Chlamys hastata*. This time the scallop were to be sold as a whole in-the-shell product. The effort ceased when persistent high saxitoxin concentrations, at times exceeding 11,000 mg, were encountered. While most of the PSP records for whole scallop has been confined to the Kodiak area, consumers should be cautious of eating whole scallop harvested anywhere in the state since toxin levels can be very high and scallop retain toxins for an extended time.

The purple hinge rock scallop, *Crassadoma gigantea*, is another popular scallop species found attached to subtidal rocky substrate, predominantly in Southeast Alaska. Peculiar to this scallop is its tendency to have a toxic adductor muscle (Beitler 1991). Although testing for saxitoxins in purple hinge rock scallop has not been done in Alaska, data from British Columbia and the West Coast of the U.S. provides us a warning (Table 2).

The razor clam recreational fishery in Cook Inlet brings thousands of harvesters to the beach during extreme low summer tides. A question often asked is "Are these clams safe to eat?" The answer to this question is, "Most likely, yes." Data collected by the ADEC from the Cook Inlet commercial fishery has consistently shown that PSP is not a problem in these razor clams. Other locations around the state, however, have recorded saxitoxin concentrations in razor clams that are above the FDA regulatory limit. Relying on a commercial fishery for PSP monitoring does have a major shortcoming because you, as a recreational harvester, do not have immediate access to the test results. Thus, you would have no idea if a sample submitted by a commercial harvester failed the PSP test.

Saxitoxins also migrate to different tissues and may undergo further transformation in the process. In the butter clam, for example, high saxitoxin concentrations begin to accumulate in the digestive system after initial consumption of toxic algae. Within one month, however, saxitoxins migrate to the siphon and undergo transformation from the relatively less toxic GTX saxitoxins to the highly toxic STX form (Beitler and Liston 1990).

Shellfish eventually clean themselves of saxitoxins through a process termed depuration. The time required for saxitoxin depuration is greatly

Table 1: PSP values for selected giant scallop tissues (in µg saxitoxin/100 grams of shellfish tissue).

Location	Date	Adductor	Viscera	Gills	Gonads	Mantel
Akhiok	June 1987	35	2,298	221	301	340
Izhut Bay	July 1987	58	4,945	504	1,361	243
Swikshak	Sept. 1987	<32	2,862	-	446	41

Data from Alaska Department of Environmental Conservation.
Note: All the locations in this table are in the Kodiak Island area.

influenced by environmental conditions and is extremely variable and unpredictable for wild grown shellfish. As an example, blue mussels can reduce saxitoxins from 700 mg to below the FDA 80 mg limit within 20 days, but the process may take over 50 days (Desbins et al. 1990). In the Skagway area, blue mussels required 40 days to reduce saxitoxins from 1,098 mg to below the 80 mg FDA requirement (ADEC data). Any attempt to estimate the depuration time for a shellfish population following a PSP event is dangerous; primarily because there is no way of knowing the size and duration of the toxic dinoflagellate bloom, and recurrent blooms can recontaminate shellfish.

The PSP problem in not isolated to just the bivalve shellfish. In recent years the Alaska crab fishery was drastically impacted when PSP was found in crab viscera. Although crab viscera is consumed in small portions, the discovery of PSP caused a flurry of regulations meant to assure consumer safety. A major concern that differs from bivalve shellfish is the fact that crab are opportunistic feeders, not filter feeders, and toxicity may vary significantly for each crab based upon the toxins contained in the food they choose to eat. Since initial concerns of PSP in crabs, regulations developed by the ADEC and cooperative agreements with the commercial crab fishery, now assure the safety of crab viscera. Since saxitoxins are water soluble, boiling live crab with the viscera in tack may spread the toxins from the viscera to other tissues. To prevent spreading of toxins, the ADEC recommends cleaning crabs of viscera before boiling.

The Food Web

How does PSP effect the marine environment? The answer to that question is difficult and extensive research reveals few conclusions.

Zooplankton, microscopic animals drifting in water, feed on toxic dinoflagellates and concentrate the saxitoxins, but these tiny animals are generally more sensitive to the effects of saxitoxins than adult bivalve shellfish (Hwang and Chueh, 1990). Although lethal to many zooplankton, saxitoxins can be passed along the food chain by zooplankton that limit toxin accumulation by reducing their feeding. High saxitoxin levels also impaired zooplankton; swimming ability causing them to become easy prey for fish, mammals, and birds (Buskey and Stockwell, 1993). Saxitoxin containing zooplankton have been implicated in fish kills (White 1981, Smayda 1992) and deaths of marine mammals after eating toxic fish (Geraci et al. 1989).

Some marine mammals and birds have adapted to living in an environment of marine toxins. For example, sea otters can detect harmful concentrations of saxitoxins and avoid eating toxic shellfish (Kvitek et al. 1991). The glaucous-winged gull has evolved an aversion to PSP and even young chicks regurgitate contaminated shellfish (Kvitek 1991). Marine biotoxins play a significant role in our marine environment and future efforts to measure the sublethal effects of toxic algae on marine organisms and the consequences for the marine ecosystems will be an elusive endeavor.

The Alaska Problem

Episodes of PSP in Alaska are centuries old, but on a global scale, toxic algae blooms are becoming an increasing menace. Attributed to man-caused nutrient enrichment of coastal waters (Anderson 1989, Smayda 1992), uncontrolled ballast water discharge from international shipping (Jones 1991), and possibly climatic changes, an international effort is now underway to explore solutions to the problem. Of practical significance is recognition that unpredictable changes in the ocean environment invalidates use of historical information as a sole source in forecasting toxic algal blooms and provides no guarantee that shellfish, historically free of PSP toxins, will remain in that condition.

Table 2: PSP toxin concentrations in the purple hinge rock scallop (µg saxitoxin/100 grams of tissue).

Location	Adductor	Viscera	Whole Body
British Columbia[1]	130	2,500	1,200
Washington[1]	229	2,036	295
California[2]	2,000	26,000	13,593

Data from: [1]Department of Fisheries and Oceans 1989
[2]Sharpe 1981

The economic consequences of the PSP problem has drastically impacted development of a clam fishery in Alaska where an estimated 50 million pounds are available for harvest (U.S. Department of Interior 1968). With harvest of 5 million pounds annually, a wholesale value of over $5 million could be realized.

In Alaska, widespread indifference of recreational and subsistence harvesters to PSP warnings causes considerable concern for the Alaska Division of Public Health and the Alaska Department of Environmental Conservation, agencies responsible for ensuring public health.

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A recent survey of Kodiak Island conducted by the Alaska Division of Public Health found that the level of risk of contracting PSP is not equally shared among all shellfish consumers. Survey results found that:

- Long-term residents (at least 23 years) are 11.8 times more likely to report symptoms of PSP than short terms residents.
- Alaska Natives are 11.6 time more likely to report symptoms of PSP than non-Natives.
- If you have eaten shellfish for longer than 20 years, you are 5.4 times more likely to report symptoms of PSP.
- Residents of the Alaska Native village of Old Harbor are 3 times more likely to report symptoms of PSP than residents of Kodiak.

One of the most disturbing findings of the study showed that people who knew nothing about the lethal potential of PSP had the same frequency of reporting symptoms of PSP as those who knew PSP could cause death (Gessner and Schloss 1996).

Non-English speaking residents may have greater risk of exposure to PSP because the communication barrier hampers alerting them of PSP warnings. One of the latest victims in Kodiak was a Laotian resident.

Many myths about PSP have lead to practices alleged to improve your chances of avoiding illness. The Kodiak study found two-thirds of the residents that consumed shellfish from untested beaches believed it was possible to collect, prepare, or test shellfish in such a way that PSP could be prevented. Rather than reducing the risk of PSP, these unproven practices may give the consumer a false sense of security that may actually increase their risk of a PSP incident.

PSP is a complex problem, but you can still reduce your risk of encountering PSP. Obviously, the most acceptable decision is not to consume untested shellfish but purchase shellfish from a seafood retailer or shellfish farm that is required to sell only tested product. However, many people will continue to consume shellfish despite the warnings, and willingly accept an unknown risk with each meal.

Some shellfish consumers take absurdly high risks. For example, eating whole blue mussels from the Kodiak area during the summer is an invitation for PSP. When considering harvesting shellfish the potential consumer must at a minimum consider:

- The recent history of PSP for the area.
- The species harvested and their ability to concentrate and retain toxin.
- The season of the year.
- The method of cleaning and preparing the shellfish (i.e., whole scallop vs. adductor muscle).

As a harvester of wild shellfish, you cannot have enough information to absolutely guarantee that untested shellfish are free of dangerous levels of PSP toxins.

Avoid myths surrounding PSP prevention. The mere fact that in all five outbreaks in Kodiak in 1993, none showed any evidence of a red tide should be ample evidence that water color is not a reliable indicator of PSP.

A major problem in Alaska is under-reporting of PSP by persons experiencing minor symptoms. In some instances, if victims had reported their PSP symptoms to a medical facility, more serious consequences could have been averted.

It is your obligation to report even minor symptoms of PSP to your local medical care unit. Your action may save someone's life.

An obvious problem in Alaska is the lack of data on toxic algae blooms, shellfish testing, and reporting of PSP outbreaks. The Alaska Division of Public Health and the ADEC are very interested in recruiting public assistance in PSP monitoring. The more information we collect about the frequency and distribution of red tides, toxic algae blooms, and PSP episodes the more likely we are to understand the environmental impacts of PSP and develop strategies to prevent illness.

References

Anderson, D.M. 1989. Toxic algal blooms and red tides: a global perspective. Pages 11-16. *in* R. T. Okaicki, D.M. Anderson and T. Nemto, editors. Red Tides: Biology Environmental Science and Toxicology. Elsevier Science Publications.

Anderson, D.M. 1990. Toxin variability in *Alexandrium* species. Pages 41-51. *in* E. Graneli, B. Sundstrom, L. Edler, and D.M. Anderson, editors. Toxic Marine Phytoplankton. Elsevier Science Publications

Anderson, D.M., D.M. Kulis, J.J. Sullivan, S. Hall, and C. Lee. 1990. Dynamics and physiology of saxitoxin production by the dinoflagellates *Alexandrium* spp. Marine Biology 104:511-524.

Bardouil, M., M. Bohec, M. Cormerais, S. Bougrier, and P. Lassus. 1993. Experimental study of the effects of toxic microalgal diet on feeding of the oyster *Crassostrea gigas* Thunberg. Journal of Shellfish Research. 12(2):417-422.

Beitler, M.K. and J. Liston. 1990. Uptake and tissue distribution of PSP toxin in butter clams. Pages 257-263 *in* E. Graneli, B. Sundstrom, L. Edler, and D.M. Anderson, editors. Toxic Marine Phytoplankton. Elsevier Science Publications.

Beitler, M. 1991. Toxicity of adductor muscles from the purple hinge rock scallop (*Crassodoma gigantea*) along the Pacific coast of North America. Toxicon 29(7):889-894.

Bricelj, V.M., J.H. Lee, A.D. Cembella, and D.M. Anderson. 1990. Uptake of *Alexandrium fundyense* by *Mytilus edulis* and *Mercenaria mercenaria* under controlled conditions. Pages 269-275 51 *in* E. Graneli, B. Sundstrom, L. Edler, and D.M. Anderson, editors. Toxic Marine Phytoplankton. Elsevier Science Publications.

Buskey, E.J. and D.A. Stockwell. 1993. Effects of a persistent "brown tide" on zooplankton populations in the Laguna Madre of South Texas. Pages 659-666 *in* T.J. Smayda and Y. Shimizu, editors. Toxic Phytoplankton Blooms in the Sea. Elsevier Science Publications. B.V. Amsterdam.

Department of Fisheries and Oceans. 1989. Summary of paralytic shellfish toxicity records in the Pacific region. Department of Fisheries and Oceans. Burnaby, British Columbia. Canada.

Desbins, M., F. Coulombe, J. Gaudreault, A.D. Cembella, and R. Larocque. 1990. PSP toxicity of wild and cultured blue mussels induced by *Alexandrium excavatus* in Gaspe Bay (Canada): Implication for aquaculture. Pages 459-463 *in* E. Graneli, B. Sundstrom, L. Edler, and D.M. Anderson, editors. Toxic Marine Phytoplankton. Elsevier Science Publications.

Geraci, J.R,. D.M. Anderson, R.J. Timperi, D.J. St. Aubin, G.A. Early, J.H. Prescott, and C.A. Mayo. 1989. Humpback whales (*Megaptera novaeangliae*) fatally poisoned by dinoflagellate toxin. Canadian Journal of Fisheries and Aquatic Sciences. 46:1895-1898.

Gessner, B.D. and M.S. Schloss. 1996. A population-based study of paralytic shellfish poisoning in Alaska. Alaska Medicine. 38(2):54-58.

Hall, S. 1982. Toxins and toxicity of *Protogonyaulax* from the northeast Pacific. Ph.D. Thesis. University of Alaska Fairbanks. 196 pp.

Hall, S., G. Strichartz, E. Moczydolowski, A. Ravindran. and P. Reichardt. 1990. The saxitoxins: sources, chemistry, and pharmacology. Pages 29-65 *in* S. Hall and G. Strichartz, editors. Marine Toxins: Origin, Structure, and Molecular Pharmacology. American Chemical Society Symposium Series. Washington, DC.

Hwang, D, and C. Chueh. 1990. Susceptibility of fish, crustacean and mollusk to tetrodotoxin and paralytic shellfish poison. Nippon Suisan Gakkaishi. 56(2):337-343.

Jones, M. 1991. Discharge ballast introduces unwanted organisms to Australian waters. Australian Fisheries. August edition. Bureau of Rural Resources. Canerra, Australia.

Kao, D.Y. 1993. Paralytic Shellfish Poisoning. Pages 75-86 *in* I.R. Falconer, editor. Algal Toxins in Seafood and Drinking Water. Academic Press, New York.

Kvitek, R.G. 1991. Sequestered paralytic shellfish poisoning toxins mediate glaucous-winged gull predation on bivalve prey. Auk 108(2):381-392.

Kvitek, R.G. and M.K. Beitler. 1991. Relative insensitivity of butter clam neurons to saxitoxin: a pre-adaptation for sequestering paralytic shellfish poisoning toxins as a chemical defense. Marine Ecology Progress Series. 69:47-54.

Kvitek, R.G., A.R. DeGange, and M.K. Beitler. 1991. Paralytic shellfish poisoning toxins mediated feeding behavior of sea otters. Limnology and Oceanography. 36(3):393-404.

Mosher, H.S., F.A. Fuhrman, H.S. Buchwald, and H.G. Fischer. 1964. Tarichatoxin-Tetrodotoxin: A potent neurotoxin. Science. 144:1100.

Oshima, T., K. Sugino, H. Itakura, M. Hirota, and T. Yasumoto. 1990. Comparative studies on paralytic shellfish toxin profile of dinoflagellates and bivalves. Pages 479-485 *in* E. Graneli, B. Sundstrom, L. Edler, and D.M. Anderson, editors. Toxic Marine Phytoplankton. Elsevier Science Publications.

Sharpe, C.A. 1981. Paralytic shellfish poison, California - Summer 1980. State of California Department of Health Services - Sanitary Engineering Section

Smayda, T.J. 1992. Global epidemic of noxious phytoplankton blooms in the sea: evidence for a global epidemic. Pages 275-307 *in* K. Sherman, L.M. Alexander, and B.D. Gold, editors. Food Chains: Models and Management of Large Marine Ecosystems. Westview Press. San Francisco.

Sullivan, J. and W.T. Iwaka, and J. Liston. 1983. Enzyme transformations of PSP toxins in the littleneck clams, *Prototheca staminea*. Biochemical and Biophysical Research Communications. 114(2):465-472.

Sullivan, J. and M. Wekell. 1988. Detection of paralytic shellfish toxins. Pages 87-106 *in* T. Tu. Anthony, editor. Handbook of Natural Toxins, Volume 3. Marcel Dekker. Inc., New York.

Twarog. B.M., T. Hidaka, and H. Yamaguchi. 1972. Resistance of tetrodotoxin and saxitoxin in nerves of bivalve molluscs. Toxicon. 10:273-278.

United States Department of the Interior. 1968. The potential of Alaska's fishery resources. Newsletter to Alaska Fishermen and Processors. Number 9, September 13, Juneau, Alaska.

White A.W. 1981. Marine zooplankton can accumulate and retain dinoflagellate toxins and cause fish kills. Limnology and Oceanography. 26:103-109.

White, A.W., S.E. Shumway, J. Nassif, and D. Whittaker. 1993. Variation in levels of paralytic shellfish toxins among individual shellfish. Pages 441-445 *in* T.J. Smayda and T. Shimizu, editors. Toxic Phytoplankton Blooms in the Sea. Elsevier Science Publications.

Alexandrium, the Dinoflagellate that Produces Shellfish Poisoning Toxins

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In Alaska, and elsewhere in the Pacific Northwest, paralytic shellfish poisoning (PSP) is caused by dinoflagellates in the genus *Alexandrium* (Figure 1). First described as a species in the genus *Gonyaulax* (Whedon and Kofoid 1936), the toxin-producing species were later transferred to *Protogonyaulax* (Taylor 1979), and recently to *Alexandrium* (Balech 1985, Steidinger 1990).

Much of the confusion has been resolved by a closer examination of cellular morphology of the toxin-producing species. In addition, the *Alexandrium* show much variation in morphology caused by natural variation, sexual reproduction that increases genetic variation, the discovery of the cyst stage that is structurally different from the vegetative cell stage, and environmental conditions. Currently, there are 22 species in this genus (Balech 1985).

The vegetative stage of *Alexandrium* is motile and have a theca (an outer covering or cell wall) made of cellulose plates. The arrangement of these plates, though there may be some variability, is a characteristic used for identification (Balech 1985). The plates are most easily seen if the cells are gently squashed to remove the cell contents. The cells are divided into upper and lower parts by a central groove (girdle) with the ends displaced about one girdle width. A longitudinal groove (sulcus) runs from the girdle to the posterior end of the cell (Figure 2). Two flagella, whip-like structures used for swimming, are present, one encircling the cell in the girdle, the other, lying along the sulcus and trailing behind the cell. Cells are round to oval in shape and range in size from about 20-50 mm in diameter. They may be single or occur in chains. Identification is difficult unless chains are present and single cells may easily be mistaken for other small, brown-pigmented dinoflagellates including *Scrippsiella trochoidea*.

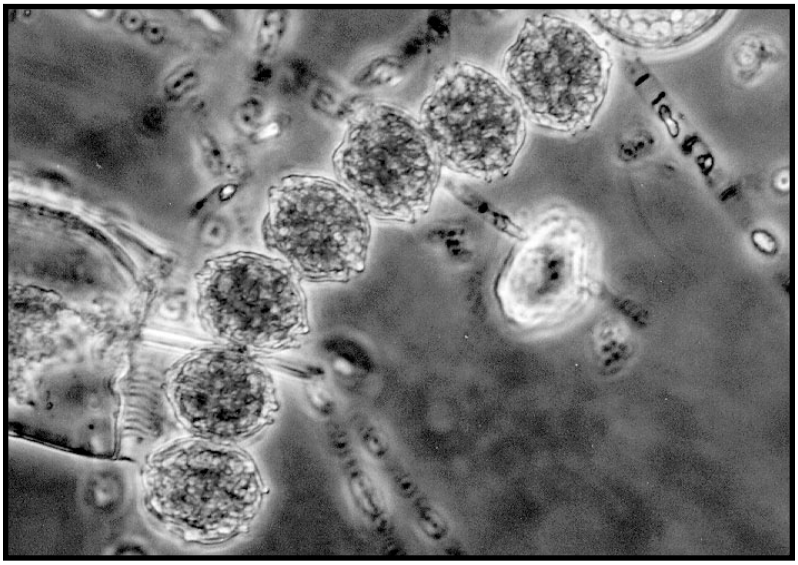


Figure 1: Alexandrium catenella 7-celled chain

General features of the genus include the characteristic shape and arrangement of surface plates, girdle displacement about one girdle width, no spines or horns, thin cell walls, a characteristic apical pore plate, the presence or absence of a ventral pore, and smooth-walled cysts. Species are distinguished by the size and shape of the cells, size and shape of some of the thecal plates, presence or absence of a ventral pore, size and shape of some of the girdle plates, and the relationship between the apical pore plate and the more-or-less diamond-shaped plate ventral to it. A key to the species is found in Balech (1985).

Based on analysis of small-subunit ribosomal RNA genes, three species of *Alexandrium* occur on the North American west coast (Scholin and Anderson 1994). *A. catenella* (Whedon and Kofoid) Balech occurs from southern California to southeast Alaska, forms chains, blooms when the water temperature is about 20°C, and occurs in both estuarine and open coast environments; it lacks a ventral pore. *A. tamarense* (Lebour) Balech, prefers cooler temperatures and less saline water than *A. catenella* and has a ventral pore. It has been found at Unimak Island in the Gulf of Alaska. *A. fundyense* Balech, originally described from the Bay of Fundy, is small, lacks a ventral pore, and has been found at Porpoise Island, Alaska. Other species identified using standard morphological characteristics include *A. acatenella* (Whedon and Kofoid) Balech, *A. ostenfeldi* (Paulsen) Balech, *A. hiranoi* Kita and Fukuyo (Taylor and Horner 1994). *A. catenella, A. acatenella*, and *A. tamarense* are part of the same species complex, but in British Columbia, at least, they tend to have different distributions (Taylor and Horner 1994). Elsewhere in the Pacific Northwest, their distribution is not well-known.

Two kinds of cysts may occur in the life cycle, both with smooth cell walls (Figure 2). Pellicle cysts are vegetative and are produced from motile, vegetative cells in response to environmental stress, including temperature changes and nutrient depletion. These cysts have limited durability and do not overwinter. They are smooth-walled, deeply pigmented, and have a required dormancy period. They are resistant to environmental extremes and may provide seed populations for future blooms if conditions for germination are right. These resting cysts may be transported in the same manner as sediment particles, including by normal water currents or catastrophic events such as hurricanes. As a result, they may germinate far from their place of origin and initiate blooms in new areas. Cyst formation may be a factor in the decline of blooms. Cysts are also toxic and are thus a source of toxicity to the food chain.

The distribution of *Alexandrium* in Alaskan waters is not well-known and historical records are sparse. Reasons for this include the long coastline and the lack of samples from many sites. Moreover, much of what is known or suspected about the distribution of toxic cells comes from records of toxicity in shellfish, not from knowledge of the biology of the dinoflagellates. *Alexandrium*-like cells have been found at a number of places in southeast and southcentral Alaska, but the problem has been to correlate the abundance of a causative organism, presumably *Alexandrium* spp., with the timing, levels, and geographic distribution of toxin in the shellfish (Hall 1982). Consequently, Hall (1982) sampled the water column and sediments from Dutch Harbor to Ketchikan for motile cells or cysts and isolated about 50 strains from 11 sites. In culture studies he found that toxin content per cell varied substantially within a strain, but toxin composition of a strain changed little with culture conditions or stage of growth. However, regional patterns of toxin composition were found where strains from one region had the same toxin composition, while strains from other regions had different compositions. Shellfish toxicity should vary in a similar manner according to Hall (1982).

Thus, it is apparent that there are no easy answers to the problem of shellfish toxicity and causative species in Alaska. Without comprehensive phytoplankton and/or shellfish monitoring programs, there is currently no way to ensure that shellfish are safe for human consumption.

References

Anderson, D.M. and D. Wall. 1978. Potential importance of benthic cysts of *Gonyaulax tamarensis* and G. *excavata* in initiating toxic dinoflagellate blooms. Journal of Phycology. 14:224-234.

Balech, E. 1985. The genus *Alexandrium* or *Gonyaulax* of the tamarensis group. Pages 33-38, *in* D.M. Anderson, A.W. White, and D.G. Baden, editors. Toxic dinoflagellates. Elsevier, New York.

Hall, S. 1982. Toxins and toxicity of *Protogonyaulax* from the northeast Pacific. Ph.D. Thesis, University of Alaska, Fairbanks. 196 pp.

Scholin, C. A. and D.M. Anderson. 1994. Identification of group and strain-specific genetic markers for globally distributed *Alexandrium* (Dinophyceae). I. RFLP analysis of SSU rRNA genes. Journal of Phycology 30:744-745.

Streidinger, K.A. 1990. Species of the *tamarensis/catanella* group of *Gonyaulax* and the fucoxanthin derivative-containing Gymnodinioids. Pages 11-16, *in* E. Graneli, B. Sundstrom, L. Edler, and D.M. Anderson, editors. Toxic dinoflagellate blooms. Elsevier, North Holland, New York.

Taylor, F.J.R. and R.A. Horner. 1994. Red tides and other problems with harmful algal bloom in Pacific Northwest coastal waters. Pages 175-186, *in* R.C.H. Wilson, R.J. Beamish, F. Aitkens, and J. Bell, editors. Review of the marine environment and biota of Strait of Georgia, Puget Sound and Juan de Fuca Strait: Proceedings of the BC/ Washington Symposium on the Marine Environment, Jan 13 & 14, 1994. Can. Tech. Rep. Fish. Aquat Sci. 1948.

Whedon, W.F. and C.A. Kofoid. 1936. Dinoflagellata of the San Francisco region. I. On the skeletal morphology of two new species, *Gonyaulax cantenella* and *G. acatenella*, University of California Publication. Zoology 41(4):25-34.

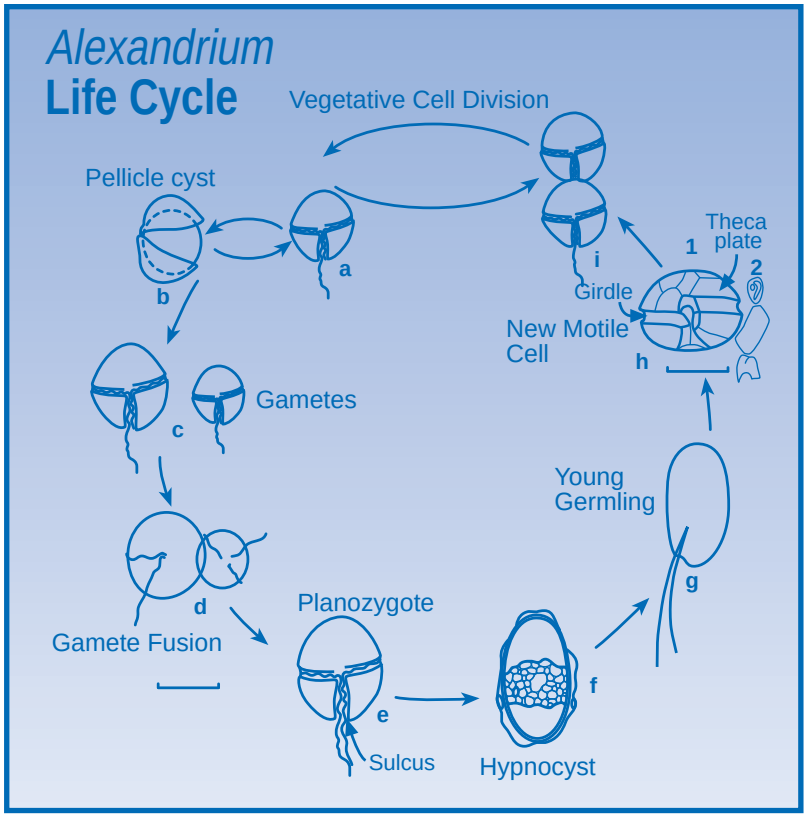
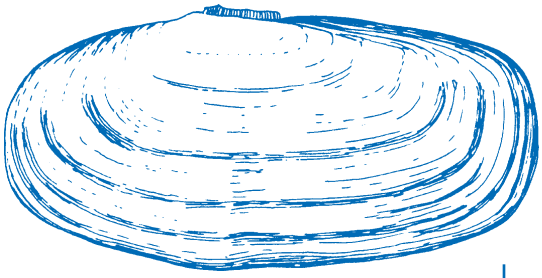


Figure 2: Generalized life cycle of Alexandrium. Figure h shows characteristic thecal plates for A. catenella; 1 is the ventral view, 2 is the apical pore plate, the thecal plate closest to the port plate, and a sulcal plate. Figures a-g, and i modified from D.M. Anderson; Figure h modified from Balech (1985).

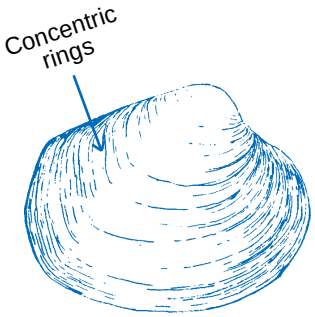
How Toxic Are Alaska's

Toxicity levels shown are the highest recorded in Alaska. The FDA



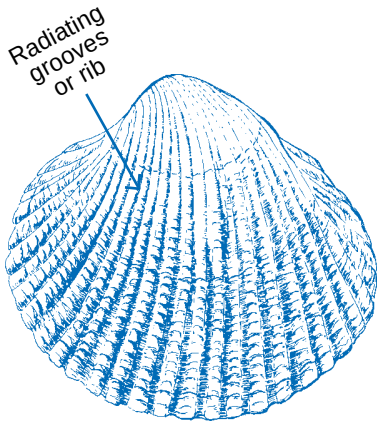
Pacific Razor Clam

Siliqua patula
Distribution: Alaska to mid California
Habitat: Intertidal zone, open coasts in sand
Size: up to 8"
Identification: Long narrow shell, thin and brittle, olive green to brown color
Toxicity: 3,294 µg toxin



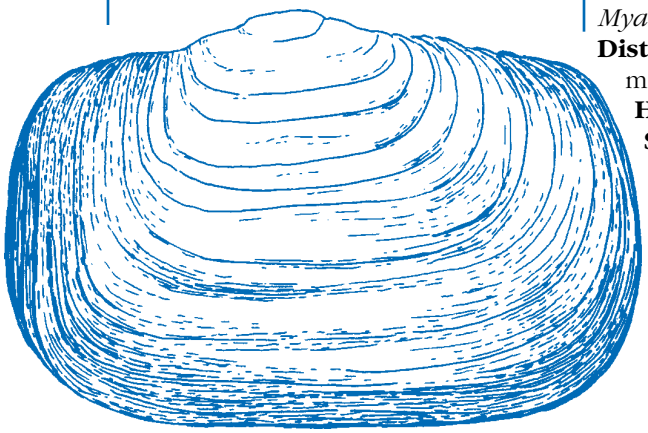
Butter Clam

Saxidomus giganteus
Distribution: Aleutian Islands to mid California
Habitat: Intertidal zone to 120 feet depth, on protected gravel, sandy beaches
Size: up to 5"
Identification: Dense shell, external surface with concentric rings, prominent growth rings
Toxicity: 7,750 µg toxin



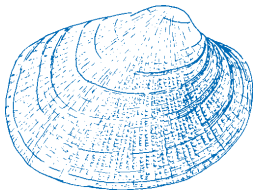
Cockle

Clinocardium nuttalli
Distribution: Bering Sea to Southern California
Habitat: Interidal zone to 90 feet, mud to sand beaches
Size: Up to 6"
Identification: Thick cupped shells, up to 35 strong ribs spreading from the hinge to shell margin
Toxicity: 2,252 µg toxin



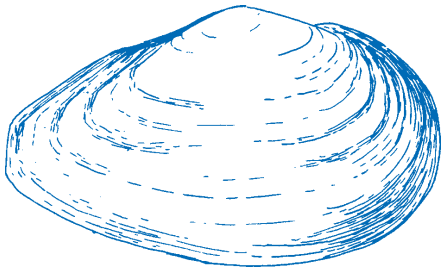
Geoduck

Panopea abrupta
Distribution: Sitka, Alaska to Gulf of California
Habitat: Intertidal to deep water, buried deeply in sand and mud bottom
Size: Shell up to 8"
Identification: Shells heavy, one end of shell rounded the other end flat, rough concentric grooves on shell surface.
Toxicity of viscera: 1,526 µg toxin



Pacific Littleneck Clam

Prototaca staminea
Distribution: Aleutian Islands to mid California
Habitat: Midtidal to subtidal zone, mud to coarse gravel beaches
Size: Up to 2 1/2"
Identification: External surface of shell with radiating and concentric grooves
Toxicity: 580 µg toxin



Softshell Clam

Mya arenaria
Distribution: World-wide north of mid California
Habitat: Upper tidal level mud flats
Size: Up to 6"
Identification: Shell soft, easily broken, one end of shell rounded, other end pointed, concentric rings only
Toxicity: 47 µg toxin

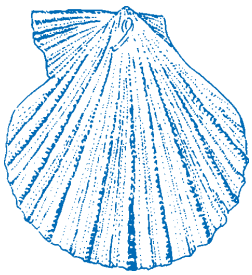
Blue Mussel

Mytilus edulis
Distribution: Northern Hemisphere
Habitat: Rocky intertidal areas of exposed and protected coastline
Size: Up to 4"
Identification: Blue/black to brownish shell, shell pointed at one end and round at the other, has a threadlike structure to attach to substrate
Toxicity: 20,000 µg toxin

Shellfish drawings from "Intertidal Bivalves: A Guide to Common Marine Bivalves of Alaska", Nora R. Foster. 1991. University of Alaska Press

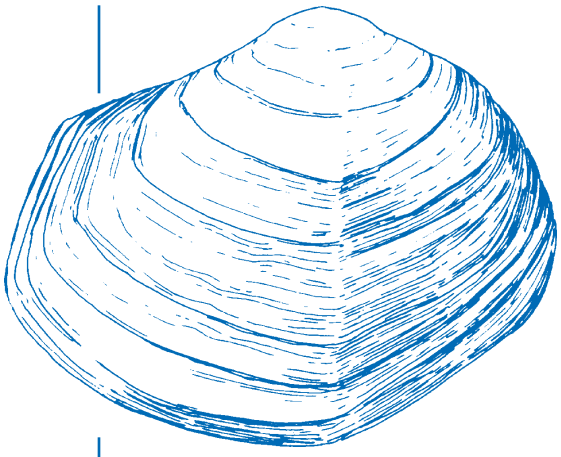
Most Common Shellfish ?

considers anything above 80 µg (micrograms) of toxin not safe to consume.



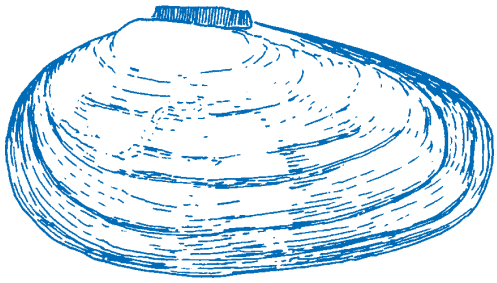
Spiny Scallop

Chlamys hastata
Distribution: Gulf of Alaska to California
Habitat: Low intertidal area to 400 feet depth
Size: Up to 3 1/2"
Identification: Shell thin and flattened, auricles uneven size, 20-30 ribs on each shell, ribs spiny textured
Toxicity: 11,945 µg toxin (whole)



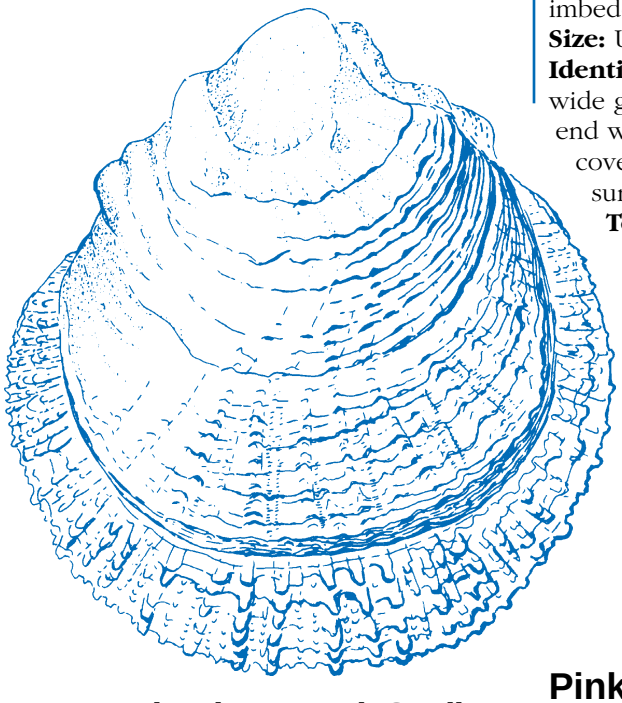
Horse (Gaper) Clam

Tresus capax
Distribution: Shumagin Islands, Alaska to California
Habitat: Intertidal zone imbedded deeply
Size: Up to 8"
Identification: Shell large and thick, wide gape between shells at posterior end when held together, dark covering (periostracum) on shell surface often partially worn off
Toxicity: 281 µg toxin



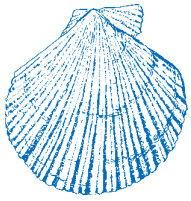
Alaska Razor Clam

Siliqua alta
Distribution: Bering Sea to Cook Inlet
Habitat: Intertidal zone to 30 feet on open sandy beaches
Size: Up to 6"
Identification: Long narrow shaped shell, shell thin and brittle, brown to olive green color
Toxicity: 3,294 µg toxin



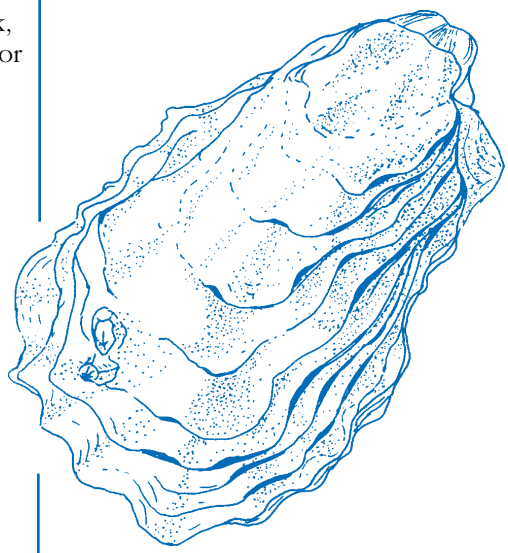
Purple Hinge Rock Scallop

Crassadoma gigantea
Distribution: Aleutian Islands to Southern California
Habitat: Low tidal area to 200 feet depth, attached to rocks and in crevices.
Size: Up to 10"
Identification: Very heavy rough shell, purple color hinge area when shell open
Toxicity: 2,000 µg toxin (whole)



Pink Scallop

Chlamys rubida
Distribution: Bering Sea to mid California
Habitat: Low tidal area to 900 feet depth, rocky shoreline
Size: Up to 2 1/2"
Identification: Shell thin and flattened, 20-30 ribs on each shell, auricles uneven size, red/pink on one shell, opposite shell, color pale
Toxicity: 11,945 µg toxin (whole)



Pacific Oyster

Crassostrea gigas
Distribution: Kachemak Bay to California
Habitat: Intertidal in mud to rocky beaches. In Alaska only on aquatic farms, but may be a few small populations in southern southeastern Alaska. Does not reproduce in Alaska waters
Size: Up to 8"
Identification: Shell irregular shape, rough surface, upper shell cupped while lower shell flat
Toxicity: 910 µg toxin

PSP: The Bacterial Connection

F.G. Plumley, Associate Professor, UAF Institute of Marine Science, and Z. Wei, Ph.D. graduate student

Paralytic shellfish poisoning (PSP) is a persistent problem in Alaska and along the West Coast of the United States. PSP is caused by a neurologically damaging saxitoxin that is assumed to be produced by a planktonic dinoflagellate, *Alexandrium cantenella* (Read: *Alexandrium*, the Dinoflagellate that Produces Shellfish Poisoning Toxins). This may very well be true, but recent data questions this assumption and surfaces suspicions that bacteria, not dinoflagellates, produce saxitoxins.

Questions about the role of dinoflagellates in Alaska producing saxitoxins began in the mid-1960s when a study by the University of Alaska in southeastern Alaska failed to find a relationship between the abundance of *A. cantenella* in the water and the occurrence of PSP (Chang 1971). Other studies found a correlation between the presences of *A. cantenella* and PSP, however, the very small number of *A. cantenella* collected in the water samples could not account for the high level of toxin (Sparks 1966, Neal 1967). In 1973, the first direct link between *A. cantenella* and PSP was recorded by Simmerman and McMahon (1976) when several families ate butter clams collected near the boat harbor in Tenakee. The case was proven when two unsuspecting victims developed PSP from eating clams harvested from a beach whereas others, having recently eaten clams from the same beach, had no toxic reaction. An analysis of the uneaten portions of clams, which included the gills and digestive gland, showed high levels of saxitoxin. Saxitoxin in these particular tissues indicated that the toxic conditions were recent since toxin in butter clams moves into the siphon after a period of time. Fortuitously, only five days before the toxin problem, the Alaska Department of Environmental Conservation RV Maybeso had been in the area and had observed unusual bioluminescence in Tenakee Harbor prompting the scientists to collect water samples. Later examination of the samples found high numbers of *A. cantenella*. Later, Hall (1982) confirmed the "dinoflagellate connection" when he induced resting cysts of *A. cantenella* to germinate and then subsequently produced saxitoxin.

During the same time period the PSP story was also unfolding in laboratories around the world. The general scenario emerging was similar to Alaska, in some locations and at certain times there were inconsistencies between toxin production and algae abundance, whereas in other locations there was consistent agreement. Adding to the confusion were findings that some geographical strains of dinoflagellates produce more toxin while others produced little or no toxin. Silva and Sousa (1981) made a remarkable discovery when they transformed a non-toxic dinoflagellate strain to a toxin producer by simply inoculating the non-toxic strain with a bacterium, *Pseudomonas sp.*, isolated from a toxin-producing dinoflagellate. This observation, though exciting, could not confirm which organism, the bacterium or the dinoflagellate, produced the toxin. Nonetheless, this observation, linked with the fact that dinoflagellates routinely harbor intra cellular bacteria (Bold and Wynn, 1979), prompted the question "are bacteria the real source of saxitoxins?" Since most scientists studying saxitoxins were phycologists (algae specialists), they emphatically responded: "no way!"

The implication that bacteria produce the saxitoxins has met with some resistance from phycologists. This resistance is due in part to the complex associations that occur between algae and bacteria, but there is clear implication that phycologists could say with a clear conscience: "bacteria are not producing saxitoxins, they are only inducing the alga to synthesize the toxins."

Most phycologists accepted the idea that bacteria may have a direct role in saxitoxin production by inducing the algae to produce toxin rather than directly producing saxitoxin. Many investigators started examining their algal cultures more closely and using the electron microscope to look for bacteria within the dinoflagellate cell. Kodama and colleagues, attempting to prove the hypothesis that bacteria can produce saxitoxin took a more risky approach (Kodama et al. 1988, 1990) by isolating bacteria from cultured dinoflagellates and even removing bacteria individually from inside the dinoflagellate cells. They found that under certain precise growing conditions bacterium could indeed synthesize saxitoxins. This finding was a shock to everyone, especially the phycologist, many of whom had spent a lot of time confirming that bacteria were NOT present in their toxic dinoflagellate cultures.

The race was on. Who makes saxitoxins? Could Kodama's work be repeated? For more than five years several labs attempted to culture saxitoxin producing bacteria, some labs even used Kodama's original strain, but without success. Was the toxin producing bacteria an artifact or a hoax? Could the toxin detected in the original experiment have been a residual amount accumulated and retained by the bacteria from toxin producing dinoflagellates?

Then, to everyone's surprise, a second lab demonstrated bacterial production of toxin (Doucette and Trick 1995, Doucette 1995). The

amount of toxin was very small, but the experimental methodology was performed with extreme care and the results were conclusive. This time the results were more acceptable to phycologists because more recent studies also showed that freshwater cyanobacteria (formerly known as blue-green algae) also produced saxitoxins (reviewed in Carmichael et al. 1990; Carmichael and Falconer 1993). The importance of this event is apparent in that the saxitoxin producing cyanobacteria are more closely aligned taxonomically to bacteria than algae. The fact that this process occurs in freshwater rather than marine systems was then, and still is, a matter of concern.

There remains, however, several unanswered questions. First, are dinoflagellates able to synthesize saxitoxins in the absence of bacteria? Second, if bacteria contained within the dinoflagellate cell are responsible for saxitoxin synthesis, how in nature, can they produce the large amount detected when in laboratory culture only minute quantities are produced? Third, if both the bacteria and the dinoflagellate have necessary roles in toxin production, how did such an evolutionarily separated pair of organisms develop such a capability?

The answers to the first two questions are now being investigated by a number of laboratories around the world. For the first problem, laboratories are again checking their toxin producing dinoflagellate cultures for bacteria.

A problem with this type of investigation is that theoretically you cannot prove that something does not exist, you can only demonstrate that you have been unable to find it. By the same logic, the absence of data cannot be taken as an absence of the event. Bacteria may indeed be in a dinoflagellate culture, but scientists have not been able to find them or detect their influence on toxin production. For the second question, several labs are growing bacteria under a variety of conditions to determine if saxitoxin production can be increased.

The answer to the third question is the area of research being conducted in the laboratory of these authors. We are attempting to clone one or more genes that encode the enzymes required to make saxitoxins. Once this task is completed, the cloned DNA fragments can then be used as "probes" to determine who else produces saxitoxins. To date our results have been less than encouraging, primarily because the bacteria that produce saxitoxin are difficult to analyze at the molecular level.

One hypothesis we are pursuing is that the toxin producing genes evolved only once, originating in the bacteria, then later were transferred to dinoflagellates by a recently discovered process called trans-kingdom sex (Amabile-Cuevas and Chicurel 1993). That bacteria may be able to transfer genetic information across kingdom boundaries from bacteria to algae cells. This has profound evolutionary implications for several controversial issues in biology, possibly including a better understanding of the PSP problem.

References

Amabile-Cuevas, C.F. and M.E. Chicurel. 1993. Horizontal gene transfer. American Scientist. 81:332-341.

Bold, J.C. and M.J. Wynn. 1978. Introduction to the Algae: Structure and Reproduction. Prentice Hall, Inc., N.J. 706 pp.

Carmichael, W.W., N.A. Mahmood, and E.G. Hyde, 1990. Natural toxins from cyanobacteria (blue-green algae). Pages 87-106, *in* S. Hall and G. Strichartz, editors. Marine Toxins: Origins, Structure and Molecular Pharmacology. American Chemical Society, Washington, D.C.

Carmichael, W.W. and I.R. Falconer. 1993. Diseases related to freshwater blue-green algal toxins and control measures. Pages 187-209, *in* I.R. Falconer, editor. Algal Toxins in Seafood and Drinking Water. Academic Press Limited.

Chang, J.C. 1971. An ecological study of butter-clam (*Saxidomus giganteus*) toxicity in Southeast Alaska. M.S. Thesis, University of Alaska Fairbanks, Alaska. 94 pp.

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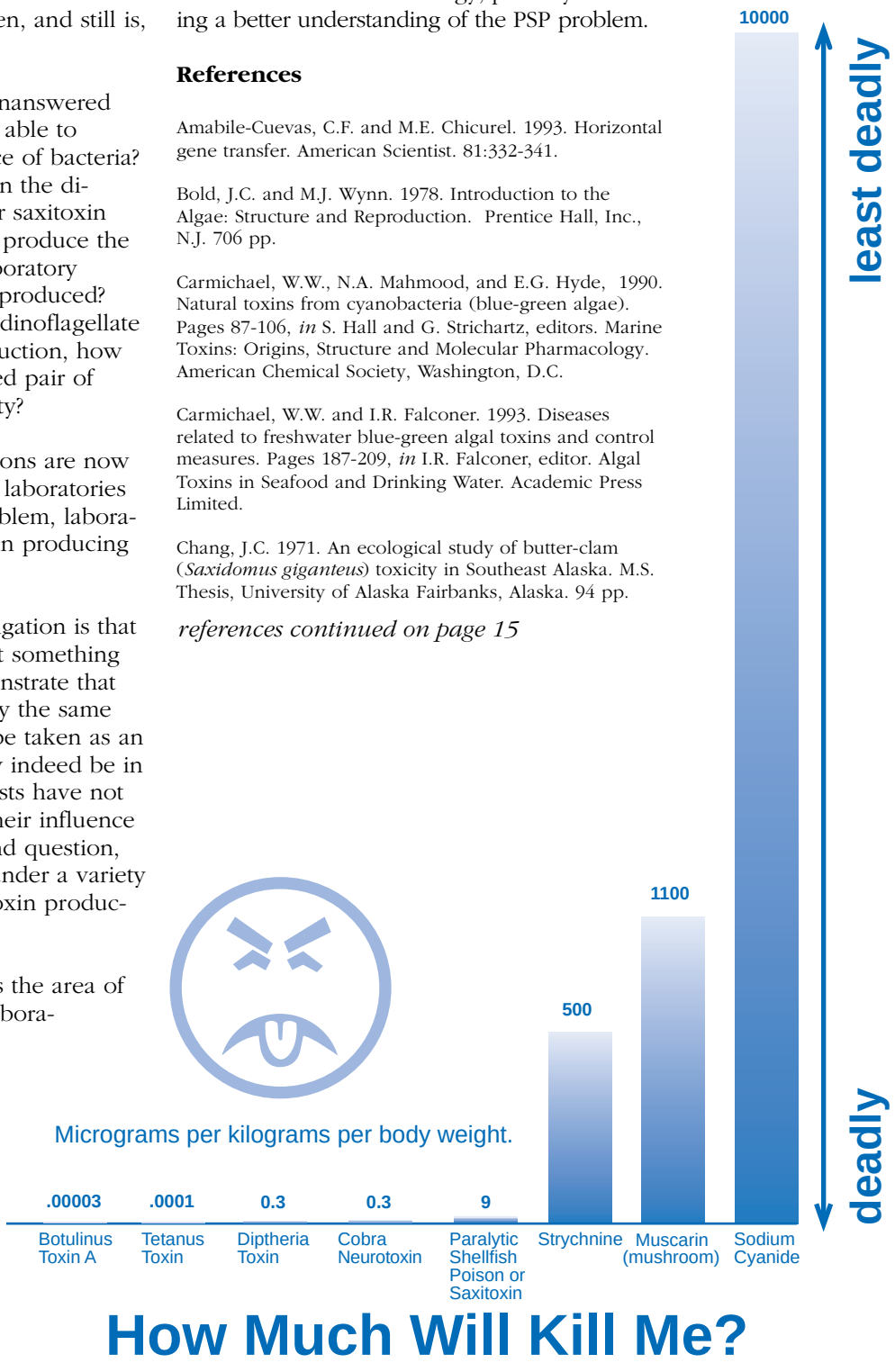


How Much Will Kill Me?

Truths and Myths about PSP

Are months with an "r" are safe for eating shellfish?

No. Months without an "r" occur during the summer when toxic dinoflagellate blooms that cause PSP most often occur. With the unlikely possibility that shellfish will become toxic outside the summer season, consumers assume shellfish are safe to eat. This answer is wrong in three ways.

1. In some locations in Alaska shellfish remain highly toxic in the spring and fall. PSP outbreaks have occurred in all seasons.

2. Toxic dinoflagellate algae can form cysts that reside in the sediment during the non-bloom seasons. These cysts are as toxic as the suspended vegetative form that are present during a toxic bloom. Shellfish, being bottom dwelling filter feeders, can continue to consume cysts during non-bloom periods and accumulate PSP toxin.

3. Some shellfish can retain the PSP toxin for a long period. Blue mussels in the Skagway area took 28 days before they were safe to eat. Such a long retention time could extend into the fall season. Other shellfish like the butter clams can chemically bind PSP toxin and retain it for as long as two years.

Is there an antidote for PSP?

No. PSP is a neurotoxin that blocks movement of sodium through membranes of nerve cells. Without sodium transmission, nerve cells cannot function. This leads ultimately to the symptoms of PSP: numbness, paralysis, respiratory failure, and coma. There is no specific antidote to stop the effect of PSP toxicity.

Is there a treatment for PSP?

Yes. Induce vomiting by sticking a finger down the throat, drinking warm saltwater, or taking Syrup of Ipecac to expel shellfish from the victim's stomach. Treat the victim for shock and transport to a medical facility. Application of life support services at the medical care facility may be necessary to sustain the life of the victim. Reduction of symptoms normally occurs within 9 hours and complete recovery usually within 24 hours. You must not underestimate the seriousness of PSP. Once the symptoms begin to appear, the victim must be transported immediately to a medical care facility.

Is a toxic algae bloom the same thing as a red tide?

Not always. A number of marine organisms in Alaska cause red tides, including non-toxic dinoflagellates of the genera *Noctacula* and *Mesodinium*. During bloom conditions, single celled organisms can cause the surface water to become red. Toxic dinoflagellate blooms turn red only when a certain density is reached. Individual toxic dinoflagellate cells may actually be most dangerous during the early part of bloom when the red color is less likely to appear. Red coloration often occurs in patches created by winds and water currents passing through the area. Shellfish left in the wake of these moving poisonous patches may remain toxic long after evidence of the algae bloom has passed. Thus, water color alone is not a consistent indicator of PSP toxicity. To emphasize this point, none of the five PSP outbreaks in Kodiak in 1993 were preceded by a red tide. However, if a red tide is in progress, do not eat the shellfish! You may not know what is causing the red coloration.

Is shellfish purchased at a seafood retailer safe to eat?

Yes. Shellfish sold for human consumption must meet the Food and Drug Administration standard of less than 80 ug of PSP toxin per 100 grams of shellfish tissue. Alaska regulations require regular monitoring of commercially harvested shellfish or batch certification that requires each commercially harvested or farm grown batch of shellfish to pass the PSP test prior to market.

Are there some clam beaches in Alaska certified to be free from PSP toxin?

No. Unlike other west coast states, Alaska does not certify recreational beaches for evidence of PSP toxin. The term "certified beach" is used in Alaska, but a certified beach is one that has passed a fecal coliform test. This test certifies a beach free from sewage caused pollution and indicates the shellfish are free of human pathogens like cholera or hepatitis.

Can I test for PSP in shellfish by chewing a small piece of shellfish tissue and see if I feel tingling in my lips? If no tingling or numbness occurs, is the shellfish OK to eat?

No. Only a mouse bioassay is approved by the U.S. Food and Drug Administration for detection of PSP toxins. The test procedure first extracts PSP toxins from 150 grams of shellfish tissues. The extract is injected into 3 Swiss Webster strain white mice 18-23 grams in weight. The amount of time required for the mice to die is recorded then converted to micrograms (ug) of toxin by substitution into a prescribed mathematical formula.

Chewing on a small piece of shellfish gives you no clue as to the PSP dosage in the tissue. In addition, PSP toxins in an acid pH environment undergo chemical transformations that may produce more potent toxins than originally found in the shellfish. Since your mouth has a nearly neutral pH, the toxins in your mouth may not have the potency as the toxins that are formed in acidic conditions of your stomach. With data collected during recent outbreaks, the Alaska Department of Epidemiology found evidence of toxin transformations in the digestive tract of humans. The amount of change in PSP toxicity caused by these transformations has not been confirmed and requires additional research.

Is my risk of getting PSP reduced if I dig clams in an area where there is an ongoing commercial fishery?

It depends. In the Cook Inlet region, PSP has not been a problem with razor clam harvesting. During the razor clam fishery for example, commercial harvesters submit a sample for PSP analysis at every other tidal change. The Alaska Department of Environmental Conservation then fills in the remainder of the sampling schedule. This massive testing program has not found PSP levels that exceed the FDA standard. The same is true for the littleneck clam fishery in Kachemak Bay. However, reliance on commercial fishery sampling has a major drawback since you do not have immediate knowledge of the commercial fishery PSP test results.

Shellfish from other locations around the state—Southeast Alaska, Prince William Sound, Kodiak, and the Aleutians; have PSP toxin problems. Commercial harvest of shellfish in these areas requires certification of the harvested batch before marketing. Again, as a personal use harvester, you do not have access to the PSP test results.

Does cleaning the intestinal contents of the shellfish make them safer to eat?

Sometimes. The digestive tract of the shellfish is the first tissue to accumulate PSP toxin from the food they consume, and cleaning the intestinal contents can reduce your risk if done during the early part of the toxic bloom. The problem, however, is that you have no indication of how long the shellfish have been consuming the toxic algae. After initial consumption by the shellfish, the toxin distributes to other tissues, and the level of toxicity these other tissues achieve depends on a number of factors. Butter clams store highly toxic forms of toxins in their siphon, the part most often eaten. Along with the intestinal contents the most toxic tissues tend to be gonad, siphon, foot, mantle, and gills. Several articles in this publication provide additional information of tissue accumulation.

Does cooking eliminate PSP from shellfish?

No. PSP toxins are heat stable. Even when pressure cooked at a temperature of 250°F for 15 minutes, PSP remains toxic.

PSP: The Bacterial Connection

references continued from p. 13

Doucette, G.J. and C.G. Trick. 1995. Characterization of bacteria associated with different isolates of *Alexandrium tamarense*. Pages 33-38, *in* P. Lassus, G. Arzul, E. Erard, P. Gentien, and C. Marcaillou, editors. Harmful Algal Blooms. Technique et Documentation - Lavoisier, Intercept Limited.

Doucette, G.J. 1995. Interactions between bacteria and harmful algae: a review. Natural Toxins 3:65-74.

Kodama, M., T. Ogata, and S. Sato. 1988. Bacterial production of saxitoxin. Agriculture and Biological Chemistry. 52:1075-1077.

Kodama, M., T. Ogata, S. Sakamota, S. Sato, T. Honda, and T. Miwatani. 1990. Production of paralytic shellfish toxins by a bacterium *Moraxella* sp. isolated from Protogonyaulax tamarensis. Toxicon 28:707:14.

Neal, R.A. 1967. Fluctuations in the levels of paralytic shellfish toxin in four species of lamellibranch molluscs near Ketchikan, Alaska, 1963-1965. Ph.D. Dissertation, University of Washington, Seattle, WA. pp. 1964

Schantz, E.J. and H.W. Magnussen. 1964. Observations of the origin of paralytic poison in Alaska butter clams. Journal of Protozoology 11:239-242.

Silva, E.S. and I. Sousa. 1981. Experimental work on the dinoflagellate toxin production. Arq. Inst. Nac. Sau´de 6:381-387.

Sparks A.D. 1966. Physiological ecology of the causative organisms including mechanisms of toxin accumulation in shellfish. pages 10-11, *in* W. Felsing (ed) Proceedings of Joint Sanitation Seminar on North Pacific Clams. Alaska Department of Health and Welfare and U.S. Dept. of Health, Education, Welfare and Public Health Service.

Zimmerman, S.T. and R.S. McMahon. 1976. Paralytic shellfish poisoning in Tenakee, Southeastern Alaska: a possible cause. Fisheries Bull. (U.S.) 74:679-680.

Epidemiology of Paralytic Shellfish Poisoning Outbreaks in Alaska

Dr. Brad Gessner, Section of Epidemiology, Alaska Department of Health and Social Services

The information presented below represents an update of information presented in a previously published article (Gessner and Middaugh, Am J Epidemiol 1995;141:766-70). Persons interested in further detail, including methodology, should consult this article.

Between 1973 and 1994, 66 outbreaks of paralytic shellfish poisoning occurred in Alaska, involving 143 ill persons. Of the 143 ill persons, the most common symptom was paresthesias including perioral or extremity numbness or tingling (n=137). Other common symptoms included nausea or vomiting in 57 persons, trouble with balance in 39, dizziness in 37, shortness of breath in 35, a floating sensation in 33, dry mouth in 23, difficulty seeing in 19, difficulty talking in 17, diarrhea in 10, and difficulty swallowing in 10. Eight persons had paralysis of a limb, eight required mechanical ventilator support, and two died. The time from ingestion of shellfish to illness onset ranged from 5 minutes to 11 hours (most commonly, 1 hour). The time from illness onset until resolution of symptoms ranged from 30 minutes to 8 hours (most commonly, 8 hours). The majority of persons had cooked their shellfish before eating it (76%).

Most outbreaks occurred during May and June with a smaller number during July (79%) (Figure 1). However, outbreaks occurred during every month except November and December. Among 61 outbreaks where the shellfish species was known, 57% involved butter clams (*Saxidomus giganteus*); 30% involved mussels (*Mytilis edulis* or *californianus*); 13% involved cockles (*Clinocardium nuttalli*); and 5% each involved razor clams (*Siliqua patula*) or littleneck clams (*Protothaca staminea*); some outbreaks involved more than one species.

For 1979-92, we determined the location of outbreaks (Figure 2). No outbreaks occurred north of the Aleutian Chain. Most outbreaks occurred on Kodiak Island, the southern edge of the eastern half of the Aleutian Islands, and in Southeastern Alaska. Interestingly, no outbreaks have resulted from eating shellfish collected from Cook Inlet, including Clam Gulch, and only one outbreak has resulted from eating clams collected from Prince William Sound, on Montague Island.

To evaluate the historical trends of paralytic shellfish poison levels in Alaska shellfish, we analyzed records from the Alaska Department of Environmental Conservation for all shellfish tested which had detectable paralytic shellfish poison (>39 ug/100 gm tissue) during July 1982-February 1992. These records roughly corresponded with data from outbreaks and showed that the mean paralytic shellfish poison level varied by month and shellfish type and that the highest toxin levels occurred among mussels and butter clams during May and June. All types of shellfish tested, except razor clams, had at least one sample with detectable levels during the winter (December-February).

Comment

Although suspected previously, a recent investigation provides evidence that most cases of paralytic shellfish poisoning go unreported (Alaska Division of Public Health, unpublished data). Cases of paralytic shellfish poisoning are sentinel events, signaling public health providers to warn local residents about the increased danger from eating shellfish. For this reason, persons who experience symptoms of paralytic shellfish poisoning, even if they only experience numbness or tingling, should immediately report their symptoms to a medical provider. Medical providers, in turn, should immediately report all suspected cases of paralytic shellfish poisoning to the Alaska Section of Epidemiology.

The data presented above indicates that the most dangerous shellfish consumption involves eating mussels or butter clams collected from south of the Aleutian chain during May, June, or July. Although less dangerous, outbreaks have also occurred with razor clams, cockles, and littleneck clams. Additionally, outbreaks have occurred during all months of the year except November and December. It is also important to recognize that saxitoxin and its analogues are heat stable toxins. Thus, unlike many other shellfish-borne illnesses, paralytic shellfish poisoning may occur even when eating cooked shellfish. While some persons believe siphon removal prevents illness, evidence indicates that sufficient toxin exists in the remainder of the shellfish to cause symptoms. Persons who harvest shellfish, including recreational and subsistence users, should familiarize themselves with the epidemiology of paralytic shellfish poisoning to minimize their risk of illness.

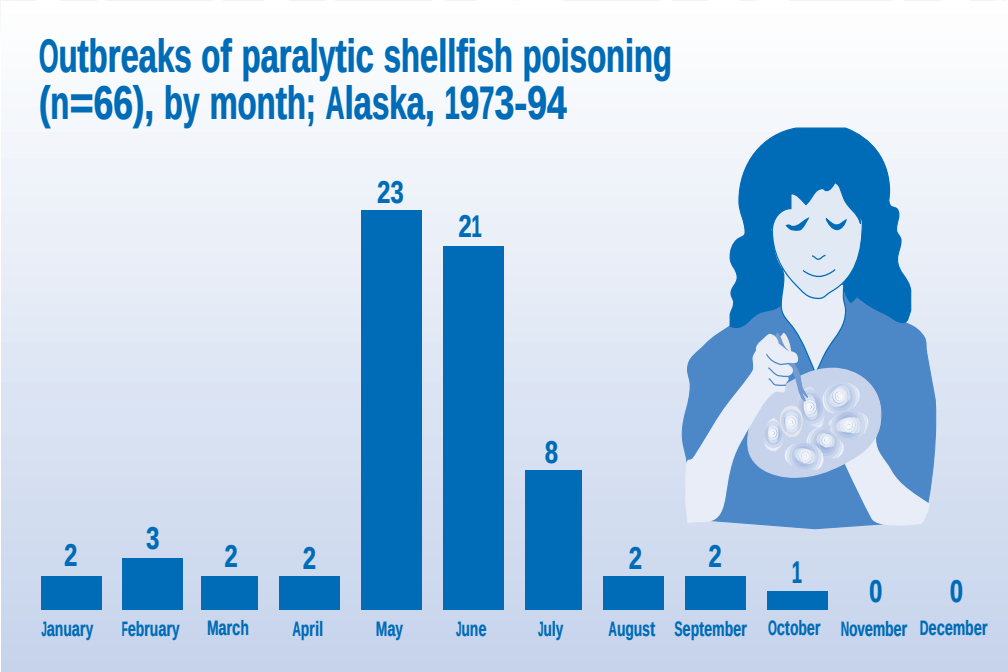
Symptoms of 143 people with paralytic shellfish poisoning, Alaska, 1973-94

Symptom	Number
Paresthesias (tingling on skin)	113
Perioral (lip) numbness	64
Perioral (lip) tingling	61
Nausea	45
Extremity numbness	43
Extremity tingling	39
Vomiting	34
Weakness	33
Ataxia (immobility)	32
Shortness of breath	29
Dizziness	28
Floating sensation	24
Dry mouth	23
Diplopia (double vision)	19
Dysarthria (difficulty speaking)	16
Diarrhea	10
Dysphagia (difficulty swallowing)	6
Limb paralysis	4

Case Histories: Paralytic Shellfish Poisoning

Case 1

Within one hour after eating 50 roasted mussels, a 28-year-old male Kodiak resident developed perioral paresthesias, nausea, and vomiting followed by headache, and difficulty talking, swallowing, and walking. Shortly after presenting to the Kodiak Island Hospital he had a respiratory arrest. The patient was rapidly incubated and placed on mechanical ventilation. A neurologic examination shortly after the respiratory arrest suggested the patient did not have cortical functioning and consideration was given to pronouncing him dead. The clinicians caring for the patient, however, recognized that the symptoms were consistent with paralytic shellfish poisoning and maintained supportive therapy. Several hours later the patient regained consciousness and within 24 hours had complete symptom resolution.



Case 2

Within 1 hour of eating at least 12 raw and cooked mussels, a 61-year-old female Old Harbor resident developed paresthesias, vomiting, weakness, and difficulty walking. Soon after presentation at the local health clinic she suffered a respiratory arrest. Because no trained personnel or equipment for endotracheal intubation were available, community health workers supported the patient with bag and mask ventilation. When emergency medical technicians arrived for air transport to Kodiak, the patient had no pulse or voluntary respirations. At the Kodiak Island Hospital, a cardiac examination suggested her heart had stopped working. Despite vigorous resuscitative efforts, she was pronounced dead approximately six hours after she had consumed mussels.

Comment

These two cases illustrate the potential severity of paralytic shellfish poisoning. Patient 1

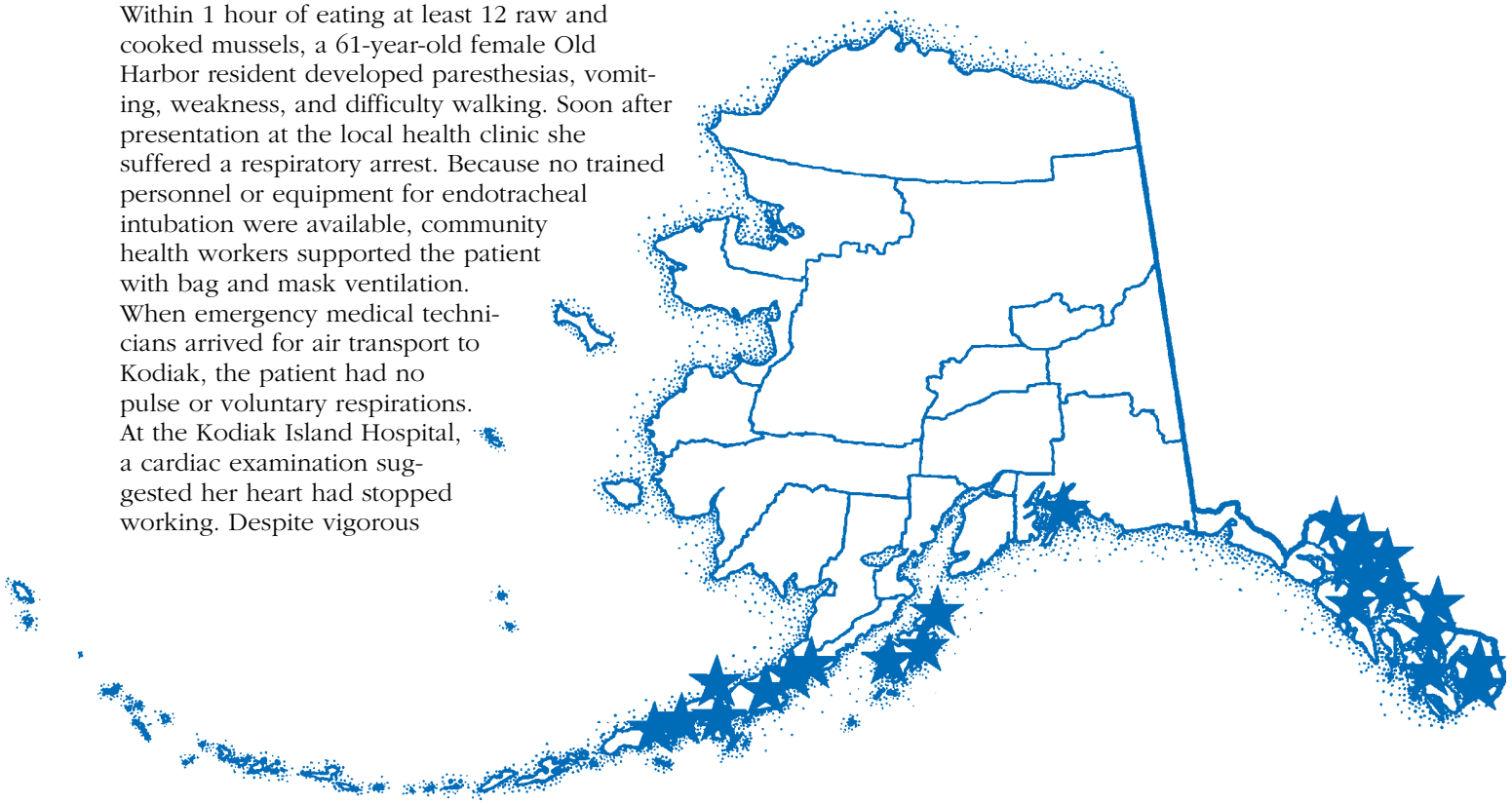


Figure 2: Location of paralytic shellfish poisoning outbreaks; Alaska, 1973-92
★ indicates ≥ 1 outbreak

Paralytic Shellfish Poisoning In The North Pacific: Two Historical Accounts and Implications for Today

Robert Fortuine, M.D.

Excerpt from "Chills and Fever", published by University of Alaska Press

The Natives of Alaska were exposed to several types of poisonous substances in their natural environment. Although general experience and cultural taboos protected them from frequent encounters with these hazards, illness and death could result from accidental (or sometimes intentional) exposure.

Paralytic shellfish poisoning, or PSP, is caused by the ingestion of a powerful toxin that is produced by severe species of plankton called dinoflagellates. These plankton sometimes "bloom" and are ingested by certain bivalve mollusks, such as mussels and razor clams. When the latter in turn are eaten by humans, a severe illness may result. The disease is characterized by numbness and tingling around the mouth, vomiting, diarrhea, and double vision, followed in severe cases by respiratory paralysis that may lead to death. This problem was first identified in the north Pacific nearly two hundred years ago and still claims periodic victims (Fortuine 1975b.)

What was undoubtedly an episode of severe PSP occurred in southeastern Alaska in July 1799, although early accounts differ on the date. Aleksandr Baranov himself, the chief manager of the newly formed Russian-American Company, has left a description of this tragic event, even though he was not personally a witness. A large party of Aleut hunters under his command had left the new fort on Sitka Island and were on their way back to Kodiak in their skin boats, when they stopped for the night at a place called Khutznov Strait, later called Peril Strait to commemorate the event. Although well supplied with provisions, the Natives could not resist eating some of the small, black mussels that were abundant in the area. Two minutes later about half the party experienced nausea and felt a dryness in the throat. By the end of two hours, says the account, about a hundred hunters had died. Some were saved, according to Baranov, by taking a mixture of gunpowder, tobacco, and spirits to induce vomiting. So far so good, but the chief manager goes on to describe how the illness then became infectious and others died without having eaten the mussels at all (Baranov *in* Tikhmenev 1979, 110-11; Khlebnikov 1973, 26-27).

The unique account of a Native witness—a Koniag named Arsenti—was preserved by Heinrich Johan Holmberg many years later (1985, 43):

> "When we found ourselves in Pogibshii proliv (Peril Strait), we turned to eating mussels (Mytilis) because of a shortage of fresh fish. They must have been poisonous at this time of year for a few hours later more than half of our men died. Even I was near death, but remembering my father's advice, to eat smelt (korushki) at such times, I vomited and recovered my health."

Arsenti's version is interesting because it shows that he knew mussels were poisonous in certain seasons of the year, and also knew of a traditional remedy, both of which point to previous experience with the disease.

The account of the same episode by Davydov (1977, 177) a few years later sheds some further light. According to him the Koniag were well acquainted with shellfish poisoning and knew that the mollusks could be harmless at some times and poisonous at others. In describing the events at Peril Strait (which he incorrectly dates in 1797), he recalled that the party camped at the mouth of a stream where there were many shellfish on both banks. Only those from the bank where there was no seaweed covering them caused illness. Within a half hour of eating the mussels a Chugach Eskimo had died, followed shortly by the death of five Koniag. According to Davydov, some eighty persons died that day. All who immediately ate sulfur, rotting fish, tobacco, or gunpowder survived, although some still had tingling sensations in the skin several years later. Davydov heard that pepper boiled in water was also an effective remedy, although no one seems to have tried it at the time. He also asserted that those who were affected felt some relief with the ebb tide.

The disaster at Peril Strait was an unforgettable one but certainly not unique. Veniaminov (1984, 364) mentioned that the Aleuts knew that clams and mussels were sometimes poisonous from May to September, while Holmberg (1985, 42-43) indicated that shellfish poisoning was well known in Kodiak in earlier times.

References

Davydov, G.I. 1977. Two voyages to Russian America, 1802-1807. Translated by C. Berne. Ed. R.A. Pierce. Kingston, Ontario: Limestone Press.

Fortuine, R. 1975b. Paralytic shellfish poisoning in the North Pacific: Two historical accounts and implications today. Alaska Medicine 17(5):71-76.

Holmberg, H. 1985. Holmberg's ethnographic sketches. Ed. Marvin W. Falk. Translated by F. Jaensch. The Rasmuson Library Historical Translation Series. Vol. 1. Fairbanks: University of Alaska Press.

Khlebnikov, K.T. 1973. Baranov: Chief Manager of the Russian colonies in American. Translated by C. Bearne. Ed. R.A. Pierce. Kingston, Ontario: Limestone Press.

Tikhmenev, P.A. 1979. A history of the Russian American Company. Volume 2 documents. Pertiod 1783-1807. Translated by D. Krenov. Ed. R.A. Pierce and A.S. Donnelly. Kingston, Ontario: Limestone Press.

Veniaminov, I. 1984. Notes on the islands of the Unalaska District. Translated by L.T. Black and R.H. Geoghegan. Ed. R.A. Pierce. Kingston, Ontario: Limestone Press.

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survived only because he was able to find medical assistance before he had a respiratory arrest and because of the clinical acuteness of the health care providers caring for him. This case emphasizes the need for health care providers to recognize the symptoms of paralytic shellfish poisoning and, when it is suspected, to maintain respiratory support regardless of adverse neurologic findings. Case 2 died despite receiving appropriate medical care before her respiratory arrest. It is possible that she died of a cardiac arrhythmia rather than respiratory arrest, a recognized complication when exceptionally high amounts of toxin have been ingested. Both of these patients ate mussels, the shellfish traditionally associated with the highest toxin levels; collecting during May, the month which usually has the highest toxin levels; from Kodiak Island, a location which has been the site of several previous outbreaks. This raises the possibility that culturally appropriate education could have prevented these outcomes.

Sea Watch

Have you seen:

- discolored ocean, bay or estuary waters?
- unusual behavior or illness displayed by a group of fish, birds, or mammals?
- an extensive bird, mammal, or fish kill?

If your answer is yes, call Sea Watch at 1-800-731-1312 and report your observations.

The Department of Environmental Conservation's (DEC) Division of Environmental Health is urging you to call this information so that it can be used with marine toxin data to help forecast toxic events. These forecasts could help with public health notices regarding the possibility of toxic shellfish or crab, and assist the department in monitoring commercial crab harvests for possible PSP.

Marine toxins, such as PSP and domoic acid, are produced under certain environmental conditions by marine phytoplankton—the source of "red tides" sometimes observed. The toxins may be concentrated in the bodies of filter-feeding shellfish and in the viscera or guts, of crab and can thus become a public health hazard.

When you call, the department needs to know the exact location of your sighting, in detail if possible, especially latitude/longitude, loran, or by landmark. They would also like you to collect a quart or more of the water in a clean container and refrigerate it. When you call they will give you instructions on what to do with it.

Contact any of the following offices for information on:

- **Fisheries Business Management**
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Alaska's Marine Resources

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