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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

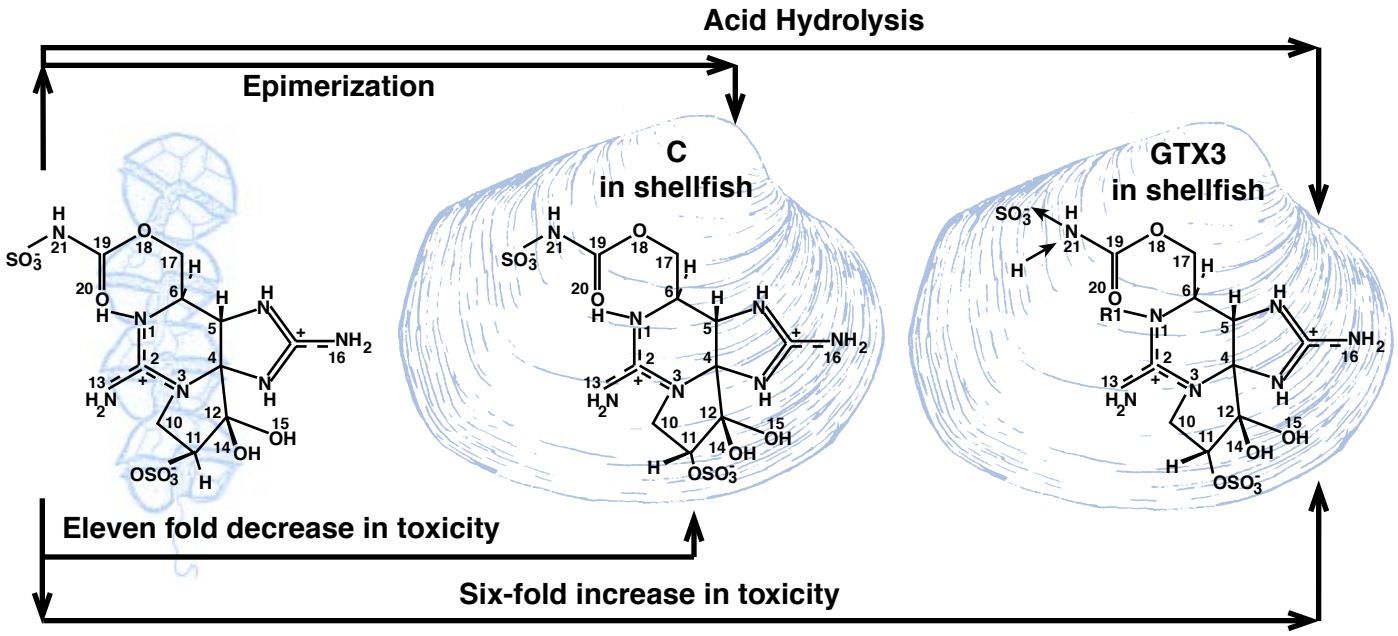

Figure 1: *Molecular transformations change the toxicity of the saxitoxin molecule. The diagram illustrates two common types of chemical transformations that occur when the saxitoxin is passed on from algae to shellfish.*

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.

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.

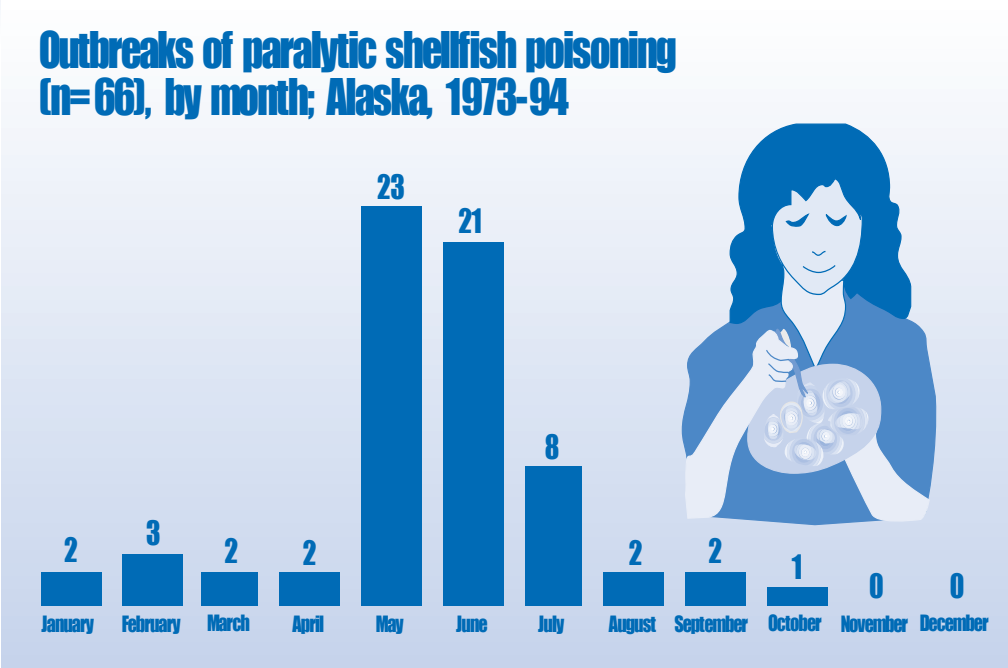
Symptoms of 143 people with paralytic shellfish poisoning, Alaska, 1973-94

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

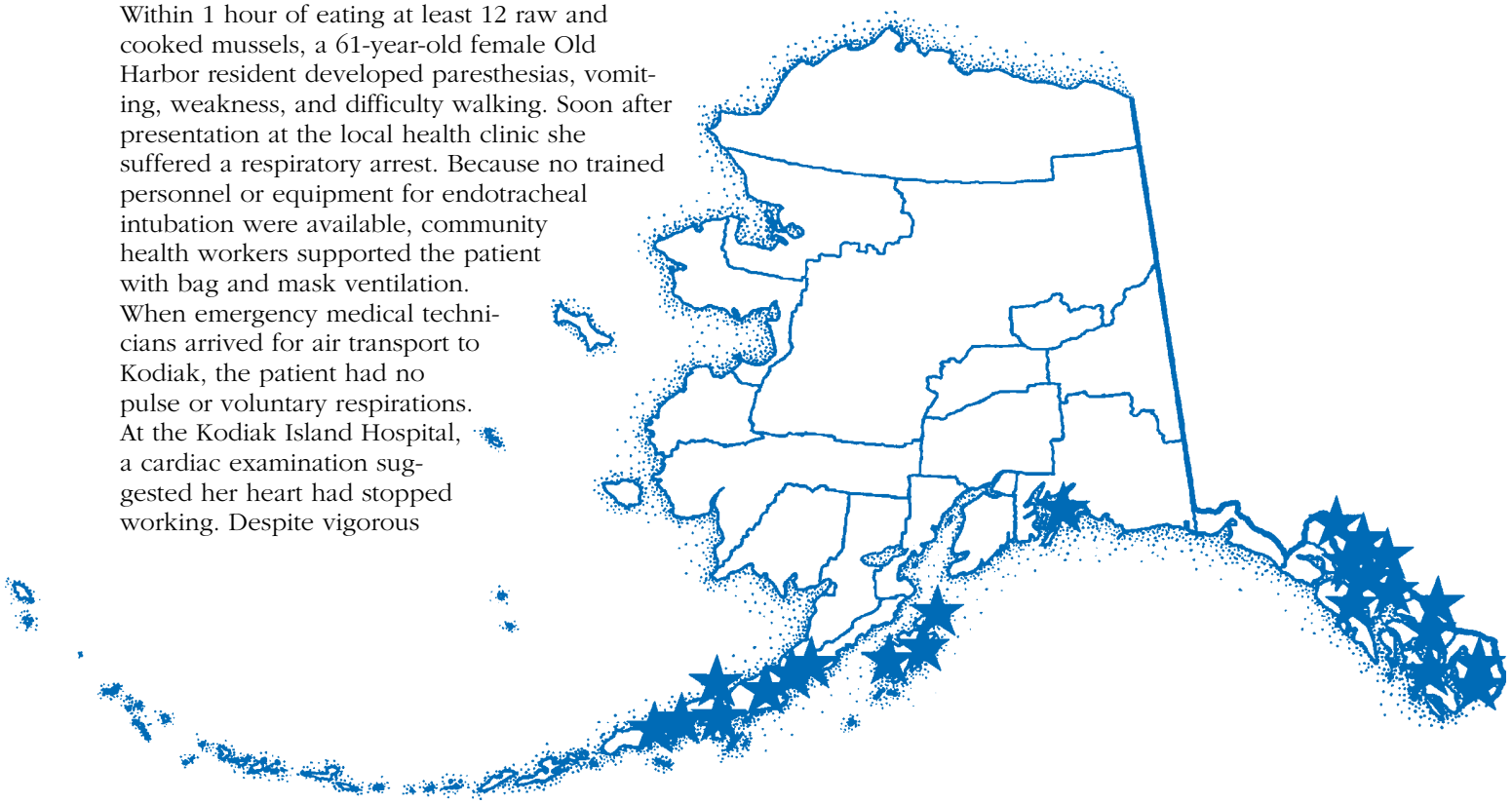


Figure 2: Location of paralytic shellfish poisoning outbreaks; Alaska, 1973-92
★ indicates ≥ 1 outbreak