NOTES

PARALYTIC SHELLFISH POISONING IN TENAKEE, SOUTHEASTERN ALASKA: A POSSIBLE CAUSE

PSP (paralytic shellfish poisoning) has been reported from much of the west coast of North America. Recent reviews (Halstead 1965: 157-240; Quayle 1969) summarizing many aspects of the problem have emphasized its causative organism, *Gonyaulax catenella* (and possibly *G. acatenella*).

Chemical studies (Schantz and Magnusson 1964) indicate that the poison is chemically similar throughout the range of *G. catenella*–California through Alaska. Because of this similarity, and the reported occurrence of *G. catenella* in Alaska (Meyers and Hilliard 1955; Sparks 1966; Neal 1967), it has often been assumed that this species is the cause of PSP in Alaska. This assumption has not been well verified, however. A 2-yr study in southeastern Alaska by the University of Alaska failed to find a significant correlation between the occurrence of PSP and *G. catenella* (Chang 1971). Sparks (1966) and Neal (1967) reported a correlation in their occurrence near Ketchikan, but the number of *G. catenella* was so low that very long toxification periods would have been required to cause lethal clams.

The difficulty in verifying the relationship results, in part, from the very low densities of *G. catenella* in Alaska plankton (Schantz 1966; Chang 1971). Sparks (1966) stated that it has even been difficult to demonstrate that *G. catenella* occurs in Alaska waters. Since toxic shellfish occur quite frequently in southeastern Alaska, some observers (Schantz and Magnusson 1964; Neal 1967; Chang 1971) have concluded that organisms other than *G. catenella* might also cause PSP.

We believe the events reported in this paper provide the first demonstration of a localized *G. catenella* bloom followed by a PSP outbreak in Alaska waters.

Methods and Results

On 20 September 1973, 5 days before an outbreak of shellfish poisoning in humans occurred, very high bioluminescence was seen in Tenakee Harbor (lat. 57°48'N; long. 135°14'W). During darkness, glowing outlines of large individual fish and schools of fish were clearly seen moving in the water. Long-time residents remarked that it was the greatest amount of "phosphorous" (bioluminescence) they had ever seen there.

The RV *Maybeso*, Alaska Department of Environmental Conservation, was in the area at the time, and curiosity about the bioluminescence prompted the crew to collect a small (100-cm^3) water sample, which was preserved with Formalin.[1] Water temperature at the time of collection was 11.5°C, and salinity was 22.18‰. The water could not be microscopically examined until 1 October, when the *Maybeso* returned to Juneau. At that time the sample was given to the senior author, who was coordinating a PSP research program for the Department of Environmental Conservation. Large numbers (235,000/liter) of *G. catenella* were found in the sample. Other dinoflagellate species were present but only in trace amounts. No organism other than *G. catenella* was found in high enough numbers to cause intense bioluminescence.

We learned that on 25 September 1973, several families had dug the butter clam, *Saxidomus giganteus*, near the boat harbor in Tenakee. After eating the clams, two people reported severe symptoms of PSP to the Alaska Department of Health and Social Services. When interviewed, the victims, as well as other Tenakee residents, stated that they had eaten clams from the same area earlier in the year without any toxic reactions.

Using conventional methods (Quayle 1969; Prakash et al. 1971), the Alaska Division of Public Health Southeast Regional Laboratory determined that the level of toxin in the uneaten portion of some of the cooked clams from Tenakee was 4,550 μg/100 g. The toxin was distributed throughout the body and was not concentrated in the siphons. Indeed, one of the illnesses was caused by ingesting clams from which the siphons had been removed before cooking.

We flew to Tenakee on 5 October, about 2 wk after the outbreak, but found no *G. catenella* in the water. We did not test any clams for toxin levels at that time, but the mussel, *Mytilus edulis*, growing on harbor pilings had high levels of toxin (2,300 μg/100 g).

[1]Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA.

Discussion

The fact that toxin was distributed throughout the bodies of the clams, rather than being concentrated in the siphons, indicates that the contact between the clams and the toxin-producing organisms had been recent. The lack of a concentration of toxin in the siphon may even indicate that toxification was in progress (Quayle 1969). The high toxin levels in mussels also reinforces the probability that toxification had occurred recently; mussels lose their toxin rapidly (Prakash et al. 1971) and the high levels indicate that the toxicity was acquired shortly before our sampling.

There is presently no information on the pumping rate, particle retention, or assimilation efficiency of *Saxidomus giganteus* (K. Chew pers. commun.). Pumping rates of the American oyster, *Crassostrea virginica*, can be as high as 20 liters/h and probably average about 10 liters/h (Loosanoff and Engle 1947; Galtsoff 1964). By using the rate of 10 liters/h, which is conservative for the larger *S. giganteus*, and assuming a particle retention of 25%, which is also conservative when particles the size of *G. catenella*, 25-55 μm, are ingested (Loosanoff and Engle 1947), a toxification period may be calculated.

Approximately 3,000 *G. catenella* will produce one mouse unit (approximately equal to 0.2 μg) of toxin (see discussion in Neal 1967). Filtering 10 liters/h of water containing 235,000 *G. catenella* /liter and retaining 25% of the *G. catenella* will result in an increase of 40 μg of toxin/h in each clam. The *Saxidomus* sampled at Tenakee contained 4,500 μg/100 g or approximately 2,250 μg/clam (an average clam probably weighs less than 50 g). Thus, using these conservative figures, it would have taken slightly more than 2 days (57 h) of filtering to reach the levels found in Tenakee clams.

From the known background of this event, it is apparent that the shellfish must have become toxic shortly before the illnesses were reported. The occurrence of the *G. catenella* bloom approximately 1 wk before the PSP outbreak indicates that even though this species is normally found in very low densities in Alaska, it can occur in high enough numbers to rapidly toxify clams.

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Literature Cited

CHANG, J. C. C.
1971. An ecological study of butter clam (*Saxidomus giganteus*) toxicity in Southeast Alaska. M.S. Thesis, Univ. Alaska, College, 94 p.

GALTSOFF, P. S.
1964. The American oyster *Crassostrea virginica* Gmelin. U.S. Fish Wildl. Serv., Fish. Bull. 64:1-480.

HALSTEAD, B. W. (editor).
1965. Poisonous and venomous marine animals of the world. Vol. 1. U. S. Gov. Print. Off., Wash., D.C., 994 p.

LOOSANOFF, V. L., AND J. B. ENGLE.
1947. Effect of different concentrations of micro-organisms on the feeding of oysters (*O. virginica*). U. S. Fish Wildl. Serv., Fish. Bull. 51:30-57.

MEYERS, H. F., AND D. R. HILLIARD.
1955. Shellfish poisoning episode in False Pass, Alaska. Public Health Rep. 70:419-420.

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. Thesis, Univ. Washington, Seattle, 164 p.

PRAKASH, A., J. C. MEDCOF, AND A. D. TENNANT.
1971. Paralytic shellfish poisoning in eastern Canada. Fish. Res. Board Can., Bull. 177, 87 p.

QUAYLE, D. B.
1969. Paralytic shellfish poisoning in British Columbia. Fish. Res. Board Can., Bull. 168, 68 p.

SCHANTZ, E. J.
1966. Chemical studies on shellfish poisons. *In* W. Felsing (editor), Proceedings of joint sanitation seminar on North Pacific clams, p. 18-21. Alaska Dep. Health Welfare and U. S. Dep. Health, Educ., Welfare, Public Health Serv.

SCHANTZ, E. J., AND H. W. MAGNUSSON.
1964. Observations on the origin of the paralytic shellfish poison in Alaska butter clams. J. Protozool. 11:239-242.

SPARKS, A. D.
1966. Physiological ecology of the causative organisms including mechanisms of toxin accumulation in shellfish. *In* W. Felsing (editor), Proceedings of joint sanitation seminar on North Pacific clams, p. 10-11. Alaska Dep. Health Welfare and U.S. Dep. Health, Educ., Welfare, Public Health Serv.

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