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PRELIMINARY OBSERVATIONS ON
PARALYTIC SHELLFISH POISONING IN CENTRAL PUGET SOUND*

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ABSTRACT

The first outbreak of paralytic shellfish poisoning (PSP) in central Puget Sound occurred in 1978, with toxicity levels as high as 30,000 ug toxin/100g mussel tissue. Research initiated at that time has shown that Gonyaulax catenella, the species believed to have been the causative organism, forms cysts and that a maturation period may be required before excystment can occur. A shellfish monitoring program throughout central Puget Sound delineated the geographical limits of toxicity in 1978 and demonstrated a southward spread in 1979. Differences noted in patterns of uptake and loss of toxin in butter clams (Saxidomus giganteus) and mussels (Mytilus edulis) may be partially explained by the presence of cysts. A study of environmental parameters has been initiated to determine whether any of these factors can be used in prediction of occurrences of PSP.

BACKGROUND AND PURPOSE

In September, 1978, paralytic shellfish poisoning occurred for the first time in Puget Sound, a fiord extending deep into the more populous portions of Western Washington (Fig. 1). Prior to that time, toxic shellfish had been found almost annually since the 1930's along the northern coast of the Olympic Peninsula, and since 1975 in the Bellingham area as well. In the 1978 outbreak red water and extremely high levels of toxicity (22,000 to 30,000 ug toxin/100g mussel meat) were reported in three different areas of the Whidbey Basin of Puget Sound. Lower levels of toxicity and discolored water, reported several days later in the northern part of the Main Basin of Puget Sound, suggested that a southward spread of the causative organism was occurring.

A research program was initiated by the College of Fisheries at the University of Washington to study several facets of the PSP problem: the extent of the geographical spread, the identity of the causative organism and questions regarding cyst formation and distribution, rates of uptake and loss of toxicity in shellfish in Puget Sound, and water quality conditions which influence growth of the toxic alga.

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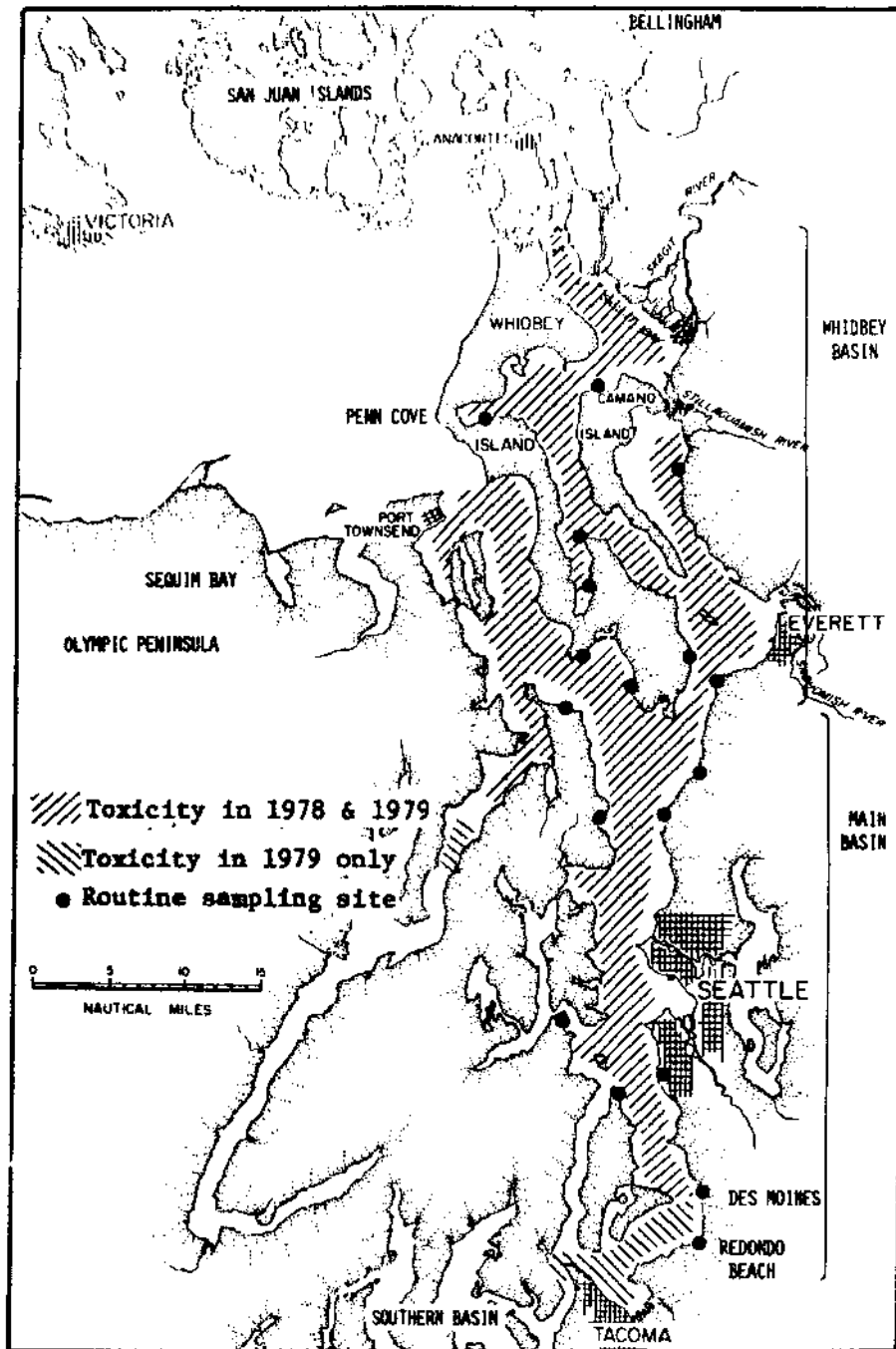


Figure 1. Distribution of PSP in Central Puget Sound, 1978-1979.

RESULTS AND DISCUSSION

A shellfish monitoring program with routine sampling at 16 sites and occasional sampling elsewhere on both sides of the Whidbey and Main Basins of Puget Sound showed that in 1978 shellfish were toxic throughout the Whidbey Basin and on both sides of the Main Basin as far south as Des Moines but not at Redondo Beach (Fig. 1). In 1979 shellfish toxicity occurred throughout the southern end of the Main Basin in areas which had not been toxic in 1978 with the highest levels of toxicity at Redondo Beach. This leads to the speculation that significant numbers of resting cysts may have been carried southward in the sediments by the bottom current, which has a net southward flow in this region (2), and deposited in the gyre off Redondo Beach. Such extension of geographical distribution by resting cysts has been proposed for Gonyaulax tamarensis and G. excavata (1). If, in the future, the southward extension of toxicity continues past Tacoma, it could jeopardize the Southern Basin oyster and clam industries with landing values of approximately \$5 million per year and pose very significant additional public health problems.

Plankton samples taken in the Whidbey Basin just after the bloom in 1978 contained a few motile cells of Gonyaulax catenella, a dinoflagellate known to cause shellfish toxicity on the Olympic Peninsula. It was considered probable this species had caused the toxicity observed. To determine whether this species forms resting cysts, as had been demonstrated for G. excavata (4), sediment samples were collected in Penn Cove, a bay on Whidbey Island in which toxin levels reached 22,000 ug toxin/100g mussel meat, and incubated in a suitable growth medium and under a temperature-light regime previously used for culturing G. catenella. In a series of such cultures set up at intervals beginning in October, motile cells first appeared in March. These results demonstrated the formation of cysts by G. catenella and suggested that a maturation period may be required before excystment can occur, as had been observed for G. excavata (5). Rough estimations of the density of cysts in Penn Cove were obtained by the technique of serial dilutions in microtiter plates suggested by Hall (6). Motile cells grew from 31 of the 34 sediment samples collected in three areas of Penn Cove (the head, middle, and mouth of the bay), with estimated densities ranging from 40 to 2,800 cysts/ml of the top centimeter of sediment. Thus, cysts were demonstrated to be patchily distributed throughout the bay and very dense in some samples.

The fact that benthic cysts are produced by this species contributes to our understanding of the patterns of uptake and loss of toxin, found to be similar in this study and in earlier work in Sequim Bay (9) and Alaska (7). Mussels became toxic when motile cells of G. catenella were present and, at most study sites, lost toxicity rapidly to undetectable levels within five to seven weeks after maximum toxicity was attained. An exception to that pattern was noted in those areas of the Whidbey Basin which had had very high toxicity in 1978. There, following the loss of 80% of the toxin in a few weeks, low levels of toxicity were maintained for three to twelve months. Butter clams became toxic several weeks after mussels and, in areas outside the Whidbey Basin, remained toxic longer than mussels. The different times of toxin increase in these two species observed by Neal in Alaska (7) led him to suggest that they may have different food

sources providing the toxin, namely motile cells of Gonyaulax for mussels and toxic detritus or possibly an encysted form of Gonyaulax for butter clams, as Bourne had suggested for sea scallops in eastern Canada (3). Our demonstration of resting cysts of G. catenella supports Neal's suggestion and appears to provide at least a partial explanation for the differences in timing of increase in toxicity in these two species. The longer maintenance of toxin in butter clams has been attributed to the binding of toxin to melanin and storage of the bound toxin in the siphon (8). It now seems probable that, in addition to such retention, continued feeding on benthic cysts may also contribute to the longer maintenance of toxin in butter clams. The exceptional pattern of prolonged toxicity in mussels, as well as fluctuations in toxin levels throughout the winter in areas which had had very high toxicity, raise the question of whether cysts may have been stirred up in the water column and ingested by mussels, thus providing a continued source of new toxin which compensated for metabolic loss.

During this study seven intertidal and three subtidal species of bivalves were found to be toxic. In addition, a large gastropod, Fusitriton oregonensis, from -55m dredges was found to be as toxic as the scallops (Chlamys hericium) in the same samples.

A study of water quality parameters, plankton composition, and toxin levels in shellfish was initiated in Penn Cove in 1979 to determine whether any measureable environmental conditions could be used in prediction of PSP. This study will be continued as funding permits. Because hazardous levels of PSP in shellfish in Washington occur much more frequently with low densities of G. catenella (20×10^3 cells/l) than with denser, visible blooms, continued studies of both population changes at very low densities and the role of cysts in intoxication of shellfish are essential to our understanding of the factors which may be of value in prediction of PSP.

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