
Humpback Whales (*Megaptera novaeangliae*) Fatally Poisoned by Dinoflagellate Toxin

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During a 5-wk period beginning in late November, 1987, 14 humpback whales, *Megaptera novaeangliae*, died in Cape Cod Bay after eating Atlantic mackerel, *Scomber scombrus*, containing saxitoxin (STX), a dinoflagellate neurotoxin responsible for paralytic shellfish poisoning in humans. We propose a line of evidence to explain how whales, by virtue of their diving adaptations, may be particularly vulnerable to this systemic neurotoxin. Absence of STX in New England waters and shellfish during the episode suggests that the mackerel, representing the northern stock which spawns in the Gulf of St. Lawrence, accumulated the toxin there and delivered it to the Gulf of Maine and Cape Cod Bay in the fall of 1987. These findings challenge common perceptions of the manner in which planktonic toxins move through the food chain, and offer new insights into natural mortality and strandings of marine mammals. It seems appropriate to search for STX and other phytotoxins when investigating marine mammal mortalities.

À partir de la fin novembre 1987 et sur une période de 5 sem, 14 rorquals à bosse, *Megaptera novaeangliae*, sont morts dans la baie de Cape Cod après avoir mangé du maquereau, *Scomber scombrus*, contenant de la saxitoxine (STX), une neurotoxine produite par les dinoflagellés et responsable de l'intoxication paralysante des humains par les mollusques. Cet article tente d'expliquer en quoi les baleines pourraient s'avérer particulièrement vulnérables à cette neurotoxine systémique à cause de leurs adaptations liées à leur comportement de plongée. Le fait que les eaux et les mollusques de la Nouvelle-Angleterre ne contenaient pas de STX durant cette période laisse penser que les maquereaux ingérés par les rorquals, qui constituent le stock nordique et fraient dans le golfe du Saint-Laurent, auraient transporté cette toxine dans le golfe du Maine et dans la baie de Cape Cod à l'automne 1987. Ces résultats bouleversent les idées reçues sur la manière dont les toxines planctoniques cheminent dans la chaîne alimentaire et ouvrent de nouvelles perspectives sur la mortalité naturelle et l'échouage des mammifères marins. Il semble indiqué de tenter de détecter la STX et d'autres phytotoxines au cours des recherches portant sur la mortalité des mammifères marins.

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Between November 28, 1987 and January 3, 1988, 14 humpback whales (7 females; 7 males, including the only calf), *Megaptera novaeangliae*, were stranded dead along the beaches of Cape Cod Bay and northern Nantucket Sound,

MA. Although not unusual for toothed whales, group mortality of this kind is unprecedented for baleen whales, which come ashore rarely and singly. Based on the nature and timing of the episode, behavioral observations of a terminally-ill animal,

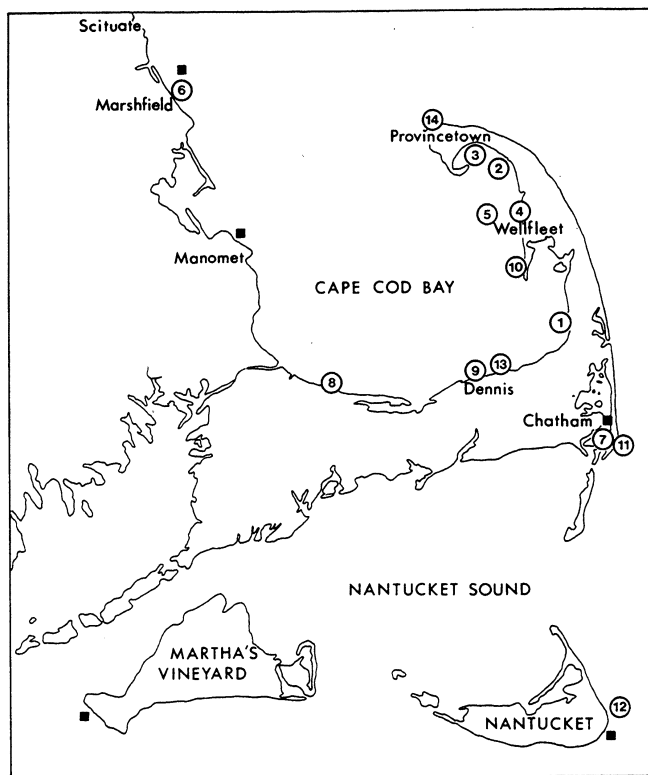


FIG. 1. Location and stranding sequence (numbered circles) of humpback whale carcasses washed ashore in Cape Cod Bay. The event peaked Dec. 15–22, with the landing of whales 4–12. Black squares designate the location of two fin, *Balaenoptera physalus*, and three minke, *B. acutorostrata*, whales cast ashore during the same period.

condition of the carcasses, and laboratory analyses of food-fish and whale tissues, we conclude that the whales died by consuming Atlantic mackerel (*Scomber scombrus*) containing saxitoxin (STX). This neurotoxin, which is the cause of paralytic shellfish poisoning (PSP) in humans, is produced by marine dinoflagellates and certain bacteria (Shimizu 1987). This is the first report of STX accumulation and transfer through a living, commercially important pelagic fish, and of its role in killing marine mammals.

The whales died at sea. Except for one which was towed ashore dead, the carcasses sank to the bottom, were buoyed to the surface by decomposition gasses, and deposited on land by wind, tides, and currents. Hence there was no temporal or geographic pattern to the distribution of beached whales within the Bay (Fig. 1), and none was fresh when examined; six were moderately and eight extremely decomposed.

Death was quick; one whale observed close to shore seemed to behave normally and 90 min later was dead (C. Carlson, International Wildlife Coalition, Falmouth, MA, pers. comm.). Gross examination of 12 whales, and limited histopathologic analyses on 3, revealed no significant lesions. The carcasses were robust, with abundant hypodermal fat (blubber). Six of 9 stomachs examined contained incompletely digested fish including mackerel, indicating that the animals had been feeding just before they died.

The pattern of deaths prompted the search for an acutely toxic substance. We focused on STX because of its periodic presence in New England shellfish (Shumway et al. 1988). Mackerel caught in Provincetown harbor on Dec. 15, 1987, as well as

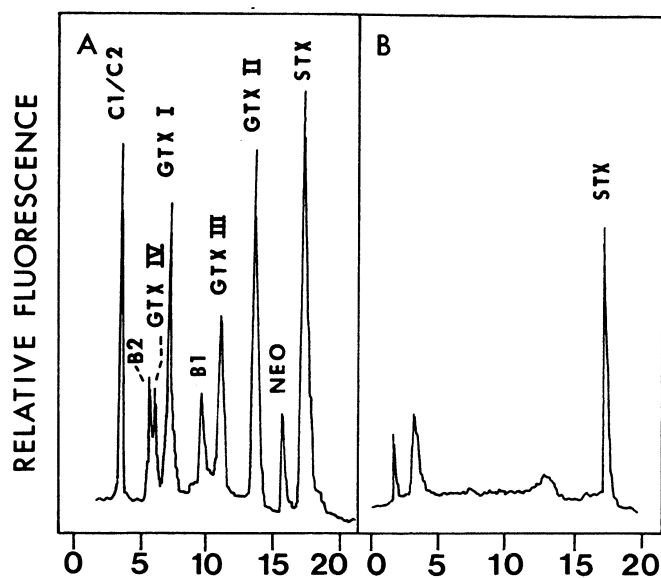


FIG. 2. HPLC chromatograms of paralytic shellfish poisoning toxins. A. Elution profile of a standard toxin mixture containing saxitoxin (STX), neosaxitoxin (NEO), and nine derivatives: B1, B2, C1, C2, GTX (gonyautoxin) I, II, III, IV. B. Elution profile of an extract of mackerel viscera. The toxin in the fish existed only in the parent form.

stomach contents and tissues from the whales were analyzed for STX and its derivatives using standard AOAC mouse bioassay (Association of Official Analytical Chemists 1984). Extracts found to be positive were verified using high performance liquid chromatography (HPLC) (Sullivan and Wekell 1988) (Fig. 2), and by correlating bioassay dose-response curves for serially diluted preparations. Saxitoxin acts by blocking the entry of sodium ions to nerve and muscle cells; selected samples were tested for this effect using neuroblastoma tissue culture (Kogure et al. 1988). During the outbreak, plankton samples were collected in the vicinity of feeding whales near Provincetown, using vertical tows with a 30 μ m mesh plankton net. Samples were concentrated by centrifugation, freeze-thawed three times to lyse the cells, and extracted with 0.1 M HCl.

Saxitoxin was universally present in viscera, especially liver, of mackerel caught at the time and place the whales were feeding, and in others collected between the Isles of Shoales, NH, and Pt. Judith, RI, (approximately 80 km north and south of the study area) on Dec. 16 and 18, 1987. Only STX was detected, and not any of its 11 derivatives (Fig. 2). Five analyses of composite tissues from 17 fish showed STX in liver at a mean concentration of 153 μ g/100 g. Average concentration in 4 fish was 52.3 μ g STX/100 g of viscera (range 40.2–71.2 μ g/100 g), equivalent to a total body burden of 80 μ g STX/kg fish. No toxin was detected in fish muscle, a reassuring finding in a commercially valuable fish. Pacific mackerel, *Scomber japonicus*, tested as controls, contained no STX.

Extracts from 3 of 8 whale kidneys, 4 of 7 livers, and the contents from 7 of 9 stomachs (macerated flesh, bones, and fluid) caused mortality characteristic of STX in mice; control liver samples ($n = 21$) from two humpback whales, one minke whale, *Balaenoptera acutorostrata*, one fin whale, *B. physalus*, four common dolphins, *Delphinus delphis*, and 13 bottlenose dolphins, *Tursiops truncatus*, that died unrelated to this incident showed no such toxicity. One each of the positive kidney and liver samples from the whales was also tested and found

positive in the tissue culture assay for sodium channel blockage (Kogure et al. 1988). However, HPLC analysis showed no STX peak. Given the certainty with which STX was present in fish, we assume that once digested or in tissues, STX was transformed into compounds that still elicited characteristic signs in mice, but that were not identifiable by the standard HPLC protocol, as has been observed in shellfish (Sullivan et al. 1983).

How much toxin did the whales consume? We calculate that a whale eating 4% of its weight in mackerel daily (Sergeant 1969) would have consumed STX at a dosage of 3.2 $\mu\text{g}/\text{kg}$ of body weight, enough to cause illness perhaps, but not death in humans for whom the minimum lethal oral dose is estimated to be 7–16 $\mu\text{g}/\text{kg}$ (Schantz et al. 1975). There are no data that can be used to determine the effects of this amount of toxin on whales. However, conventional pharmacological dogma suggests that larger animals require proportionally less for the same effect (Casarett 1975). Furthermore, lethal dose estimates are based on a single administration of toxin, and not on cumulative effects of sublethal concentrations, as would occur in actively feeding whales.

Beyond this are physiological factors that are expected to increase the effect of a given dosage of STX in the whales. Thirty percent of the mass of a humpback whale is poorly perfused, metabolically inactive blubber (Ash 1957). Water-soluble STX would by-pass this depot and concentrate in more physiologically sensitive tissues. During a dive, marine mammal blood is channeled to the heart and brain (Ridgway 1972), thus funneling the toxin to those organs while limiting access to liver and kidney where metabolism and elimination occur. The same processes that lead to reversible peripheral nerve impairment, mild hypothermia and muscle fatigue in poisoned humans (McFarren et al. 1960) may cause a whale to lose control over vital peripheral heat-conserving mechanisms (Ridgway et al. 1974) or affect its ability to return to the surface to breathe. The unusual sensitivity of a cetacean's respiratory system to anesthetics (Ridgway and McCormick 1971) and the importance of ionic metabolism in their cortical neurons (Glezer et al. 1987) further suggest that the respiratory center would be a vulnerable target for a centrally active neurotoxin such as STX (Borison et al. 1980).

Apart from its implications for whales, humans, and other consumers of fish, the discovery of STX in mackerel challenges our understanding of accumulation and retention of the toxin in fish. No STX was detected in New England shellfish, or in plankton sampled in the vicinity at the time of the outbreak, nor did the plankton assemblage contain algal species associated with PSP. The toxin in mackerel could have been a metabolite of endogenous bacteria (Kodama et al. 1988). More likely the fish, representing the northern stock which spawns in the Gulf of St. Lawrence (Anderson and Paciorkowski 1980), accumulated STX from planktonic sources there (White 1982; J. Martin, Canada Department of Fisheries and Oceans, Biological Station, St. Andrews, N.B., pers. comm.) converted the STX derivatives to the parent form (Shimizu and Yoshioka 1981), retained it as some shellfish do (Schantz and Magnusson 1964), and were already contaminated when they entered Cape Cod Bay during October–November. Burdened by STX at levels toxic to other fish (White 1981), the mackerel were perhaps easier prey.

Given the history of PSP in the region (Prakash et al. 1971), it is likely that related poisonings are broader in scope than we present. In fact, during the same period, 5 decomposed carcasses of 2 other whale species washed ashore on Cape Cod (Fig. 1). And in November 1988, a year after the original dis-

covery, we found STX in mackerel from the stomach of a dead humpback there. Toxic dinoflagellates are distributed worldwide, and associated poisonings may have occurred (White 1984) unnoticed against the background of occasional strandings. It now seems appropriate to search for STX and other phytotoxins when investigating these events.

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