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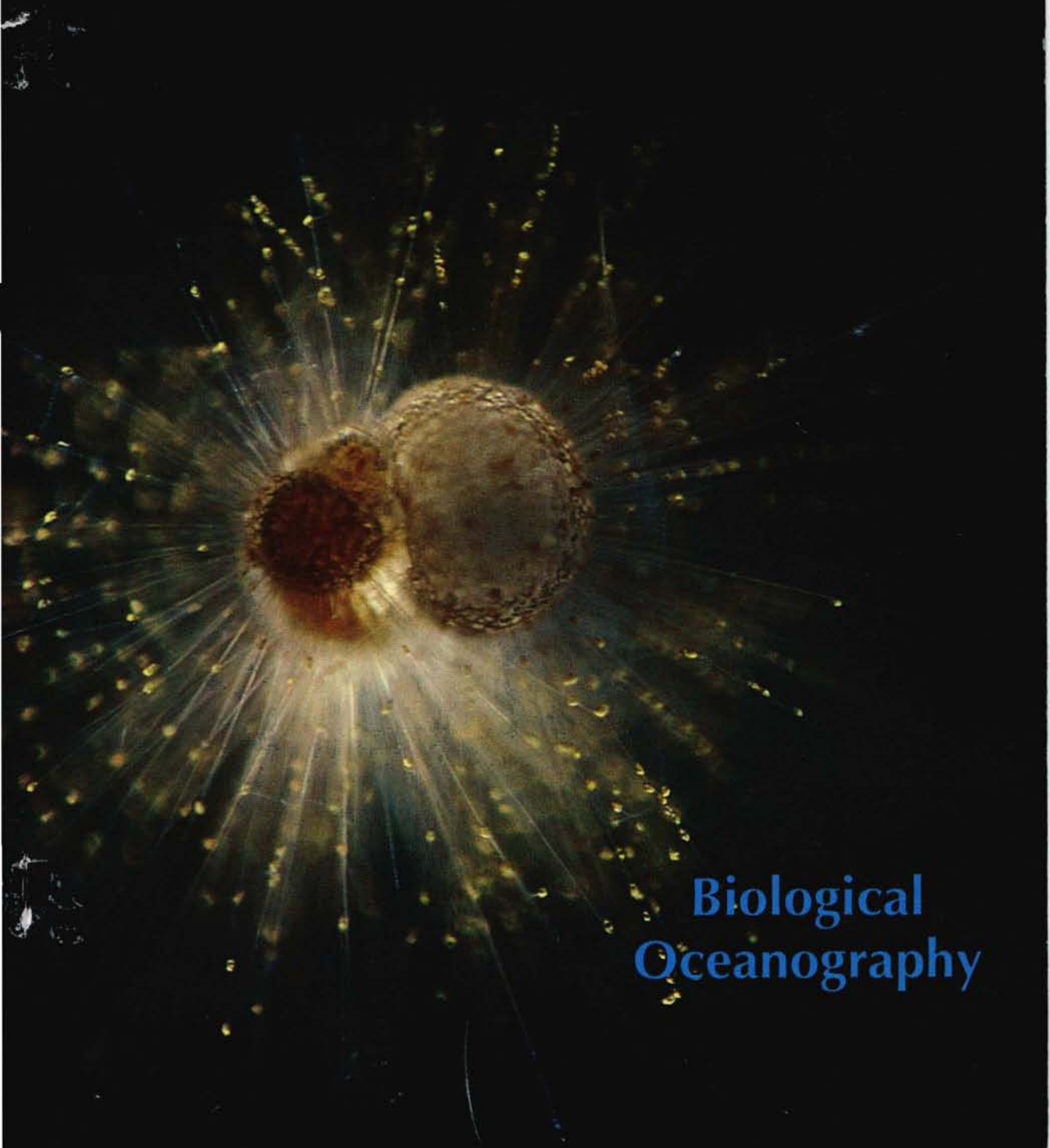

Biological Oceanography

Marine Biotoxins at the Top of the Food Chain

Donald M. Anderson and Alan W. White

In November 1987, whale-watch cruise participants off Cape Cod, Massachusetts, delighted in the antics of a large humpback whale known to many as "Torch" by his tail-fluke markings. Those observing Torch's energetic feeding behavior and breaching were horrified to see him floating dead at the water surface just 90 minutes later. This was not a typical stranding, when whales (or other marine animals) swim into shallow water and die. One pilot whale and 13 other massive humpbacks (*Megaptera novaeangliae*) died at sea in a very rapid and unusual manner that mystified marine pathologists. The whales floated ashore in five weeks; other victims undoubtedly washed out to sea and were never noticed. During the previous 10 years, only three humpbacks were found by a stranding recovery network in this same area. The 1987 mortality rate thus appears to be equivalent to nearly 50 years of "typical" mortality.

Florida Marine Research Institute/Florida Department of Natural Resources

A toxic "red tide" bloom shows as the darker color in Florida waters.

What caused this extraordinary event? No one will ever know for sure, but strong evidence indicates that the whales died from natural biotoxins originating in single-celled algae or phytoplankton. Thousands of microscopic algae species constitute the base of the marine food chain, and among these are a few dozen species that contain potent toxins. Some of these toxins accumulate in shellfish, fish, and other marine animals, and move through the food chain, affecting humans, marine mammals and other top-of-the-chain consumers. Blooms of these algae are commonly called "red tides," since in some cases these tiny plants can increase in abundance until they dominate the planktonic community, changing the water color to red, brown, or even green. Although human illness and death occur from the consumption of

Greg Early/New England Aquarium

Yasuwo Fukuyo/WESTPAC, IOC

A dead humpback whale on the shore of Massachusetts Bay. Evidence suggests that the massive animal died from an algal toxin, probably produced by the dinoflagellate Alexandrium *(small photo) accumulated by mackerel that it had eaten.*

seafood containing algal toxins, especially in developing countries, humans are usually well protected by federal and state monitoring programs that detect the toxins at an early stage and restrict harvesting or sale of the affected resources. Marine animals at the top of the food chain are not so fortunate. It is now clear that these toxins affect marine animals and seabirds in important ways that have long gone unnoticed.

Whales

The humpback whales that washed ashore in 1987 in Cape Cod Bay died suddenly at sea. Many still had fish in their stomachs, evidence of recent feeding. They had considerable blubber and seemed in good health. The pattern of the deaths prompted a search for an acutely toxic substance, but tissue analyses did not reveal unusual levels of metals, organic chemicals, or other toxic agents.

Because the whales died in waters where the red-tide dinoflagellate *Alexandrium* causes annual outbreaks of paralytic shellfish poisoning (PSP), natural biotoxins were investigated. This seemed an unlikely prospect, however, since it was November, a month when *Alexandrium* is not typically found in the region. Furthermore, throughout all of 1987, there was no PSP toxicity anywhere along the New England coast. It was thus not surprising when visual and chemical examination of plankton net-tow material from the whales' feeding area revealed no signs of *Alexandrium* cells or their toxins. However, an analysis of the mackerel that the whales had been eating revealed the presence of saxitoxin (STX), one of the PSP toxins that *Alexandrium* produces. This was an unexpected finding, in part because of the lack of PSP in the region in 1987, but also because, until then, PSP toxins were known to kill fish but not to accumulate in the tissues of living vertebrates.

Further analyses revealed that the STX was not present in the mackerel flesh, but was concentrated in the liver, kidney, and other organ tissues. Average concentrations were 52 micrograms per 100 grams of tissue, equivalent to a total body burden of 80 micrograms of STX per kilogram of fish. Most of the mackerel samples from the northeastern United States tested positive for STX, whereas Pacific Ocean

mackerel tested negative. Extracts of whale kidneys, livers, and stomachs also tested positive for STX or STX-like compounds, whereas similar samples from other whales and dolphins that had died unrelated to this incident showed no such toxicity.

The implication was that the whales died by consuming STX-laden mackerel. But how much STX did the whales consume, and how much is lethal to these animals? The humpbacks had begun their migration to warmer, southern waters, and were feeding heavily, at about 4 percent of their body weight per day. Feeding on mackerel, this corresponds to a daily STX dosage of 3.2 micrograms per kilogram of whale body weight. Unfortunately, no data exist that directly address the effect this amount of toxin has on a whale. The minimum lethal STX dose for humans is estimated at 7 to 16 micrograms per kilogram, which is two to five times higher than what scientists believe the whales received.

Such a low dose might still be lethal to a whale, however. First, 30 percent of a humpback's body mass is metabolically inactive blubber, so water-soluble STX would bypass this tissue and concentrate in more physiologically sensitive areas. Second, the whales would have received continual toxin doses as they fed, whereas human mortality statistics are based on a large dose obtained at a single feeding. Furthermore, during a dive, marine mammal blood is channeled predominantly to the heart and brain (known as the mammalian diving reflex), exposing those sensitive organs to the toxin while limiting contact with the liver and kidney where metabolism and excretion would occur. Most importantly, perhaps, is that the STX need not have killed the whales directly: Even a slightly incapacitated animal might have difficulty orienting to the water surface and breathing correctly. The affected animals may actually have drowned following a sublethal exposure to STX.

These are all speculations, but the evidence is strong that these enormous animals died from a natural marine biotoxin that originates in microscopic algae, but is transmitted through the food chain to the top consumers. Many questions remain to be addressed, including how the mackerel became toxic, how they survived carrying a potentially lethal toxin dose, and why this kind of whale mortality was never observed before 1987 or thereafter. This event and the ensuing research have taught us how important it is to search for STX and other red-tide toxins when investigating marine mammal strandings and mortalities.

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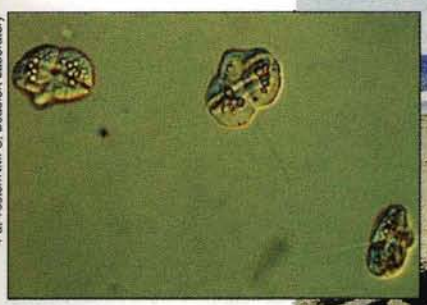
Dolphins

Another significant event that overlapped with the humpback whale deaths was a massive mortality of bottlenose dolphins (*Tursiops truncatus*). From June 1987 to February 1988, over 740 dolphins were found dead along the Atlantic coast between New Jersey and Florida. Again, undoubtedly many other dead animals drifted out to sea or were scavenged. Unlike the healthy humpbacks in Cape Cod Bay, which died quickly, most of the dead dolphins exhibited pathologies typically associated with chronic physiological stress. These observations and the temporal and spatial patterns of the dolphin deaths were not indicative of a primary infectious disease, and no known chemical pathogen was consistently isolated from dolphin tissues. Organic contaminant levels (PCBs and DDT) in some of the dead dolphins were high, but in many

other dead animals the levels were within the same range as those from control (aquarium dolphin) tissues. The evidence suggested that the dolphins were dying from opportunistic infections and other causes that were only fatal because the animals were already physiologically weakened by an unknown agent.

Success in tracing the humpback whale deaths to a red-tide biotoxin suggested that a similar investigation was warranted with the dolphins. However, the geographic range of the STX-producing alga is far north of



Pat Tester/NMFS, Beaufort Laboratory

US Dept. of Agriculture

This bottlenose dolphin, washed up on Virginia Beach, Virginia, is one of approximately 750 that fell victim between 1987 and 1988 to an enormous die-off along the US coast between New Jersey and Florida. Toxin from the alga Gymnodinium breve *(small photo) may have been responsible for weakening the animal's immune system so that it eventually died from infections or other secondary causes.*

where the dolphins were dying, so attention shifted to a different red-tide alga that causes fish mortalities and shellfish toxicity predominantly along Florida's west coast. Called *Gymnodinium breve*, this alga produces a suite of toxins called brevetoxins. In the ensuing investigation, 8 of 17 dolphin livers tested positive in a rigorous set of analyses for brevetoxin-like compounds, compared to negative results for 17 control samples obtained from dolphins killed in other incidents. The only sample of dolphin stomach contents tested proved to be positive, as did viscera samples from menhaden caught where some of the mortalities occurred.

There were insufficient data to support a rigorous conclusion, and the results indicated only that brevetoxin-like compounds were present in some of the animals and their prey; however, the suggestion that brevetoxins were involved in at least some of the dolphin deaths was strong. How could the dolphins have been exposed to the toxin, especially since these mammals tend to migrate along the US Atlantic coast, whereas *G. breve* red tides occur primarily within the Gulf of Mexico? One explanation has been offered that is quite controversial among scientists familiar with the mortality event. In October 1987, a major *G. breve* red tide struck the North and South Carolina coasts, having been transported from Florida in the Gulf Stream. This places the toxic alga in waters where the dolphins were dying, but does not explain the deaths that occurred between June and October. However, from January through April of that year, a *G. breve* red tide occurred in the eastern Gulf of Mexico, and, though evidence is sketchy, traveled south, possibly

around the Florida peninsula to the Atlantic coast. Menhaden, which filter plankton such as *G. breve* from the water as food, and Spanish mackerel, which are menhaden predators, were abundant in the coastal region where this bloom was presumably transported. Dolphins feeding on menhaden and Spanish mackerel could have received continuous low doses of brevetoxin as they migrated along the Atlantic coast. If this scenario is valid, the dolphins could have received brevetoxin doses that were not directly lethal, but nonetheless stressed them physiologically; the weakened animals were then eventually infected with bacterial and viral pathogens that ultimately caused the mortalities.

Evidence for this scenario is circumstantial. Other explanations are possible, but no clear alternatives have been proposed. Though unresolved, this investigation led to two important conclusions:

- brevetoxin can accumulate in planktivorous fishes and perhaps in their predators, and
- bottlenose dolphins can encounter red-tide biotoxins through the food chain.

As we write, another unusual dolphin mortality is occurring, this time in the Gulf of Mexico. Perhaps the lessons from five years ago will help unravel the mysteries surrounding yet another marine-mammal die-off.

Fish

For years algal toxins have been associated with mass mortalities of marine fish; for example, in the Gulf of Mexico, nearly annual red tides of the toxic dinoflagellate *Gymnodinium breve* cause spectacular fish kills. In this case, the fish are directly exposed to the algal toxins. When fish swim through *Gymnodinium* blooms, the fragile algae break open as they pass through the gills, releasing toxins that the gills absorb. The toxins burst the fish's red blood cells, and the fish die of asphyxiation. It is not uncommon for Florida's west-coast beaches to become covered with tons of dead fish, causing million-dollar losses to tourism and other recreation-based businesses.

Algal toxins can also cause fish kills as a result of toxin transfer through the food web. On a pleasant day in July 1976, the St. Andrews Biological Station, on the Bay of Fundy, received reports that hundreds of tons of adult herring were dead off Grand Manan Island. The news was particularly alarming because the herring fishery is an economic mainstay of that region. A flurry of research activity followed and determined that the herring had succumbed to toxins of the PSP-producing, red-tide dinoflagellate *Alexandrium*, which reaches the peak of its annual bloom during this period. Examination of the herring stomachs revealed pteropods (small planktonic snails that are herring's favorite food) that had fed on *Alexandrium* and were full of its toxins.

The possibility that Bay of Fundy herring might be contaminated with these potent toxins sent a shudder through the fishing industry. Fortunately, in terms of human health, subse-

Florida Marine Research Institute/Florida Department of Natural Resources

Dead fish on a beach in Florida following the red tide event shown on page 55. Beaches are often littered with dead and rotting fish for miles during these outbreaks. The cost of removal and disposal is significant, as is the loss due to tourist avoidance of these areas.

quent laboratory studies found that herring and other fish are very sensitive to these toxins. Unlike shellfish, herring die before they accumulate the toxins in their flesh to levels that would be dangerous to humans. So the human risk is thus confined to consumption of animals that eat whole fish, including the viscera, such as other fish, marine mammals, and birds. (It remains an enigma how and why Atlantic mackerel, mentioned earlier in connection with the 1987 humpback whale kill, live with substantial levels of the toxins in their guts).

Through similar food web events, *Alexandrium* toxins have been implicated in fish kills along the Atlantic coast, including herring, menhaden, sand lance, bluefish, dogfish, skates, and monkfish.

Algal blooms present a tremendous problem for aquaculturists around the world. Blooms can wipe out entire fish farms within hours. Many different algae have been implicated in such blooms, sometimes with indications that toxins are involved. Often, however, biotoxins are not the problem. Some bloom algae form a thick slime on fish gills that prevents the fish from breathing. Others possess sharp spines that stick into the gills and destroy them. Or some blooms simply strip all the oxygen from the water, either during the night as the concentrated algae respire, or when the bloom crashes and all the algae die at once, leaving no escape for fish in aquaculture cages.

Thierry Work, California Department of Fish and Game

A brown pelican on a beach near Santa Cruz, California, in September 1991 paralyzed by domoic acid from eating anchovies that had eaten toxic algae (diatoms).

Birds

A connection between marine algal toxins and seabird kills has been recognized for years, especially along the North Sea coast of the United Kingdom. In September 1991, an entirely unexpected and novel event occurred along the coast of northern California. A sudden mass mortality of brown pelicans and Brandt's cormorants near Santa Cruz puzzled wildlife experts. They conducted assays for pesticides, heavy metals, and other pollutants, but were unable to determine the cause of death. Thinking biotoxins might possibly be involved, they then conducted mouse bioassays (the standard method for PSP detection) on the birds' stomach contents, as well as on anchovies that the birds had been eating. The injected mice started behaving in strange ways, arching their backs and scratching behind their ears.

The clue was the itchy ears. The veterinarian in charge of the study recalled an article mentioning mice with these scratching symptoms during toxin bioassays. His continued sleuthing led him to suspect a new kind of algal toxin, first reported in 1987 when more than 100 people in eastern Canada were poisoned from eating contaminated mussels; three of the victims died. Investigating that episode, Canadian researchers found that a new toxin, domoic acid, was involved, and that it was produced by a marine diatom. As with other algal toxins, domoic acid was concentrated in tissues of mussels that had fed on the diatoms. This new type of poisoning was named Amnesic Shellfish Poisoning because a number of the victims experienced memory loss; about 10 of them still do.

Back to California and the pelicans. Samples of pelican and anchovy stomach contents were sent to Canadian investigators for analysis. Bingo! They found domoic acid at high levels, both in the birds' and the anchovies' stomach contents. The culprit was the diatom *Pseudonitzschia australis*, a close relative of the diatom that caused the Canadian problem. The anchovies ate the diatoms and passed the domoic acid along to the birds. Unlike the birds, it appears that the anchovies were not affected by the toxins. Tests conducted later not only confirmed the presence of domoic acid in anchovy viscera, but also suggested the presence of the toxin in the flesh, leading to the closure of the anchovy fishery because of public health concerns. Domoic acid has since been detected in shellfish at several locations in the US and has caused closures of razor clam and Dungeness crab fisheries in the Northwest, but so far the pelican and cormorant kill in California is the only reported incident where marine animals have been harmed by this toxin.

Looking Ahead

Toxic "red tides" have long been recognized as problems for human health and the fishing industry, but in recent years, these problems have been expanding throughout the world: There are more toxic species, more resources are affected, larger areas are affected, and blooms are more frequent. Although red tides are natural phenomena, they can be stimulated by human activities such as pollution or habitat alteration. As scientists struggle to understand the many factors underlying this disturbing trend, it is increasingly apparent that humans are not the only ones affected: Many innocent and unsuspecting marine animals are dying or being debilitated by algal toxins. We may be able to explain more mortality events with this knowledge, but our challenge is to ensure that human activities are not making a natural phenomenon worse, for our sake and theirs.

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Donald M. Anderson is a Senior Scientist in the Biology Department of the Woods Hole Oceanographic Institution, where he is actively involved in research as well as national and international activities related to harmful algae and their effects. Having received his Ph.D. from the Civil Engineering Department of the Massachusetts Institute of Technology in 1977, he is living testimony to the fact that a career in biological oceanography is possible without any prior appropriate training. His research has focused on the toxic red-tide phenomenon for nearly two decades. He admits that solutions to the problem are no nearer now than they were when he started, though he is confused at a higher level and about more important things.

Alan W. White received a Ph.D. in biology from Harvard University in 1972, conducting his thesis research at the Woods Hole Oceanographic Institution. Since then he has lived and worked in Canada, Japan, and Israel, investigating toxic red-tide algae, marine biotoxins, and their effects on fisheries and public health. In 1990 he joined the National Marine Fisheries Service in Woods Hole to guide responses to the new problem of contamination of offshore shellfish in the Gulf of Maine with red-tide toxins.