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# What Controls Toxic Phytoplankton Blooms in Long Island Sound?

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# What Controls Toxic Phytoplankton Blooms in Long Island Sound?

by Hans G. Dam, Michael Finiguerra,  
Christina Senft-Batoh and Hayley Flores



NASA image

This satellite image shows a phytoplankton bloom in 2004 in the Barents Sea, off the Coast of Norway: The turquoise swirls in the image are the bloom. The color of the swirls suggests that the bloom is probably comprised of cells coated with calcium carbonate (chalk), known as coccolithophores.

Mention the word bloom and most people think of green foliage on trees and brightly-colored flowers. Indeed, come springtime in temperate regions of the northern hemisphere, we witness the beauty and majesty of blooms. But these blooms are more than a pretty sight; they are essential for the balance of nature as

they start a seasonal cycle of growth for most animals as well. Yet the most spectacular blooms on the planet are not obvious to most of us, for they occur in the ocean.

The blooms are so large that they are easily seen from space. Furthermore, unlike the case of terrestrial blooms, which happen slowly as

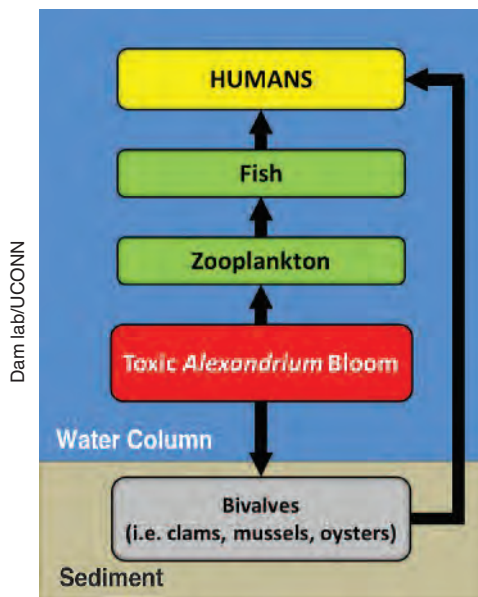
spring unfolds, blooms in the ocean can happen in a matter of days because the tiny single-cell plants that dominate the ocean (called phytoplankton) can divide in a matter of hours to days. When light and nutrient conditions are right, phytoplankton populations can take off in geometric fashion; that is, one cell begets two, which beget four, which then beget eight, and so forth. If we start with one cell that divides once a day, a week later we end up with more than a 250-fold increase in cell numbers! Long Island Sound is no exception. It turns from a cold, barren body of water in late winter, to a world of teeming biological activity during the spring bloom (which technically can happen from early February on). The shellfish and finfish we consume from the Sound depend on these phytoplankton blooms.

Now, no organism wants to become a meal for another. Phytoplankton have evolved features that reduce their chances of being eaten; for example, many have long and sharp spines; others form long chains that effectively make them too big to be easily eaten; yet others have heavy plates that resemble the shields used by warriors to defend themselves in ancient times. For those of us who love to eat fish and shellfish, the details of phytoplankton defense would seem at first sight to be of little interest. That is, as long as the phytoplankton blooms occur and sustain the fish and shellfish that we can eat, life is good. But life is not always so rosy. Some blooms are bad news for humans because certain phytoplankton may use toxins as a form of defense

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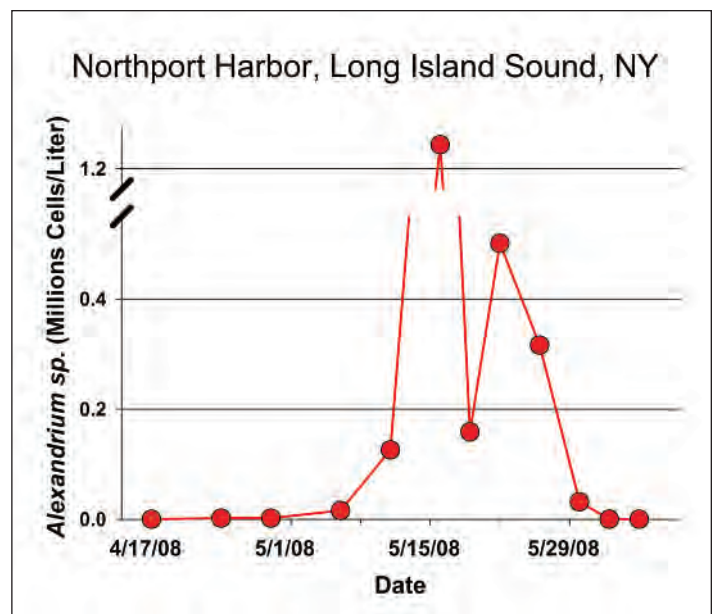
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against their consumers. For example, some species of the genus *Alexandrium* (a type of phytoplankton known as a dinoflagellate), which is found from Maine to New York, produce a suite of toxins that interfere with nerve transmission. Ingestion of the toxin by animals may lead to a condition known as paralytic shellfish poisoning (PSP), which in its most extreme form leads to respiratory failure and death. Filter-feeding bivalves, such as clams and mussels, ingest toxic *Alexandrium* and accumulate PSP toxins. These toxins are in turn passed to humans when they eat these bivalves. In recent years local municipalities and states have become rigorous in monitoring for toxin-contaminated shellfish. PSP toxin contamination results in closure of shellfish beds, negatively impacting local fishermen, seafood distributors and tourism. PSP toxins may also accumulate in the tiny floating animals (zooplankton) that eat phytoplankton. The toxins are then passed up the food chain, resulting in mass mortality of fish, sea birds, and even whales.



Toxin Transfer through the food web. Arrows depict the flow of paralytic shellfish toxins (PSTs) from a bloom of *Alexandrium* through organisms living on the seafloor and the water column.

Toxic *Alexandrium* was first detected in Long Island Sound in the early 1980s. A Sound-wide survey showed that it was mostly found along the north shore of Long Island, in Mattituck Inlet and Northport Bay. The highest cell concentrations at that time were about 3,000 cells per liter. To put that number in perspective, a typical (non-toxic) phytoplankton bloom would have cell concentrations 1000-fold higher. The Spring of 2005 marked the first full-scale toxic event caused by *Alexandrium* in the Sound. Unfortunately, the situation has considerably worsened in recent years. During 2008 the most intense



*Alexandrium* bloom in Northport Harbor during Spring 2008. Note that the vertical axis is in millions of cells per liter, reaching a peak density greater than 1.2 millions cells per liter. Local shellfish beds had to be closed as the result of the bloom. Modified from Hattenrath *et al.* 2010, *Harmful Algae* 9 : 402–412.

*Alexandrium* bloom ever recorded in Long Island Sound occurred in the vicinity of Northport Harbor, on the north shore of Western Long Island. The bloom, which led to the closure of more than 7,000 acres of shellfish beds, persisted from April through June and exceeded one million cells per liter!

Researchers at University of Connecticut and Stony Brook University are now trying to understand how PSP blooms wax and wane. The problem is akin to understanding how money fluctuates in a bank account. We all know that the amount of money in the account is the net balance of deposits minus withdrawals. This is why it is dangerous to balance your check book by only recording deposits. The only way money accumulates in the account is if withdrawals are smaller than deposits. As long as this condition is met, money will always grow in the account, no matter how small the deposits. Also, the larger the difference between the deposits and the withdrawals, and the larger the amount of money to begin with, the faster money accumulates in the account. In this analogy, money represents the phytoplankton concentration, deposits represent the “birth” (new growth) rate and withdrawals represent the death rate of the phytoplankton. The analogy also leads to a subtle, but important insight. Blooms wax not because the birth rate of phytoplankton increases, but rather because the mortality rate does not keep pace with the birth rate. When the mortality rate exceeds the birth rate of phytoplankton, blooms must wane.



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Earlier, we mentioned that when conditions of light and nutrients are right, blooms can take off. Light and nutrients are factors that enhance the birth rate of phytoplankton. But what accounts for the mortality? One possibility is that phytoplankton sink out of the sunlit waters, where they can no longer photosynthesize, and die from starvation. But the time it takes to sink out of the sunlit portion of the water is usually much longer than the time it takes cells to divide. So, sinking is usually not the major source of mortality to phytoplankton. This leaves us with the possibility that most phytoplankton are eaten (grazed in the oceanographic parlance).

Most of the phytoplankton grazing in the ocean is done by tiny, single cell consumers called microzooplankton. Because both the abundance and the birth rate of microzooplankton are similar to those of phytoplankton, microzooplankton grazing can keep the phytoplankton in check. In the bank account analogy, the withdrawals are the same as the deposits. Put another way, most phytoplankton cells in the ocean do not die of old age. They die as they become meals for grazers. In fact, many oceanographic studies are consistent with this view that a very

large fraction of the phytoplankton mortality is due to microzooplankton grazing.

Coming back to the idea of a bloom, relaxation of microzooplankton grazing allows development of phytoplankton blooms. In other words, microzooplankton control blooms. However, is that also the case for toxic phytoplankton? To date, very little research has addressed this important question. To answer it, we do experiments in which we measure microzooplankton grazing on all of the phytoplankton (measured as chlorophyll, a pigment that is present in all plants and that gives them their green color) and also specifically on toxic *Alexandrium*.

We can identify *Alexandrium* by labeling the cells with a fluorescent dye that binds only to the DNA of *Alexandrium* cells. Through these experiments we can calculate the birth rate in the absence of any grazers for all phytoplankton as well as, specifically, for toxic *Alexandrium*. This way, we can compare “birth” rate of phytoplankton to the mortality rate exerted by microzooplankton. We run these experiments during the progression of the bloom from its very beginning to its demise.

What we have found so far is that early in the *Alexandrium* bloom microzooplankton exert heavy mortality on the phytoplankton community as a whole, but almost none on the toxic *Alexandrium*. This is reassuring in that it is consistent with the notion that toxic *Alexandrium* can bloom because it is not being grazed by microzooplankton. This result is also important because it challenges the notion that microzooplankton can always keep toxic blooms at bay. In contrast, during the bloom’s demise, microzooplankton continue to graze heavily on the whole community, but also exert grazing mortality on *Alexandrium* that exceeds the birth rate.



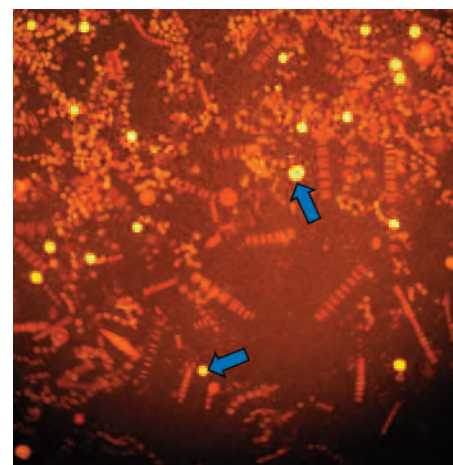
Microzooplankton species from Long Island Sound feeding on *Alexandrium* cells. (A) *Favella* sp. (a tintinnid) (B) *Polykrikos* sp. (a dinoflagellate). Arrows point to *Alexandrium* cells. Scale bars = 50 micrometers.

Dam lab/UCONN



The UConn Plankton Group getting ready for a field day.

Dam lab/UCONN



Fluorescent *Alexandrium* sp. cells (arrows) shown along with other phytoplankton species. The molecular probe only attaches to *Alexandrium* DNA and shines under a specific wavelength of light. This allows rapid and accurate enumeration of *Alexandrium* cells. Coiled colonial diatoms and smaller flagellated phytoplankton can be seen not fluorescing.

Photo by Theresa Hattenrath, Stony Brook University.

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This is reassuring because it explains why the *Alexandrium* bloom wanes. This result also settles an old dispute among oceanographers, namely, whether nutrient depletion controls the bloom. This hypothesis argues that as cells accumulate they deplete the nutrients from water and once cells run out of nutrients they stop growing.

Our experiments indicate that this is not the case. That is, the phytoplankton birth rate is still positive during the bloom's demise, but it cannot keep pace with the grazing mortality rate. The bloom does not end because of nutrient depletion; it ends because of grazing control.

Our experiments also raise questions about the nature of grazing control of toxic phytoplankton blooms. Why is it that microzooplankton did not readily graze the toxic *Alexandrium* early in the bloom, but did so during the bloom's demise? We can readily discard the hypothesis that *Alexandrium* poisons the microzooplankton early in the bloom. This is because in our experiments, the measurements of grazing on total phytoplankton and *Alexandrium* cells are simultaneously done on the same microzooplankton. If the microzooplankton had been poisoned from eating toxic *Alexandrium* they would not have been able to exert heavy mortality on the total phytoplankton (as we observed). A more likely explanation is that the microzooplankton avoid eating the toxic *Alexandrium* cells. Another possibility is that the kinds of microzooplankton that are found early in the bloom are different from those found during the bloom's demise. Indeed, this is something that we have repeatedly observed in our experiments. From week to week, there is a great deal of variability in the composition of the microzooplankton. It appears that it is the microzooplankton community structure, not their sheer numbers, that affect grazing on toxic *Alexandrium*. Further work will be required to tease out these possibilities.

The experiments also raise questions about toxin transfer up the food web. Specifically, what happens to these toxins once they are consumed? Our experiments indicate that microzooplankton do not seem to accumulate appreciable amounts of *Alexandrium* toxin. Therefore, microzooplankton may represent a sink of toxins out of the water column. Conversely, if there is no microzooplankton grazing on toxic *Alexandrium* (think early bloom), then there are more cells available for both filter-feeding bivalves that live on the seafloor and for the larger zooplankton grazers (such as crustaceans) that live in the water column. In a day's time, clams, mussels and oysters are capable of consuming millions of toxic cells and accumulating as much as 40% of the ingested toxin within their bodies. Thus the weaker the grazing control, the sooner shellfish beds get contaminated, and the sooner they are closed.

Our own observations on crustacean zooplankton from Northport Harbor indicate that they accumulate 5 to 20% of ingested *Alexandrium* toxin within their bodies. These toxin-laden zooplankton represent a potent vector to zooplankton-eating fish, which can ingest hundreds to millions of the intoxicated zooplankters every day, and the seabirds that in turn consume the fish. However, because there is no toxin monitoring program for these animals, it is impossible to know when toxic blooms affect these organisms at the top of the food web. As you can see, the ecological consequences that result from microzooplankton grazing on toxic cells can be very complex.

Understanding whether or not grazing by tiny animals controls the cycles of toxic phytoplankton blooms and toxin transfer to larger animals in the ocean is essential to plans for sustaining healthy shellfish and finfish fisheries in Long Island Sound. So the next time you see the word "bloom", think about plankton!

#### **About the Authors:**

Hans G. Dam is a professor in the Department of Marine Sciences at the University of Connecticut (UConn). His interests are the ecology and evolution of plankton. He loves to play bongoes, has never taken a lesson and it shows. To contact Professor Dam via e-mail: [hans.dam@uconn.edu](mailto:hans.dam@uconn.edu)

Michael Finiguerra and Christina (Tina) Senft-Batoh are Ph.D. candidates at UConn, working with Professor Dam. Michael is interested in the advantages and costs of mutations that confer toxin tolerance to copepods. Michael makes a killer limoncello. Tina is interested in how zooplankton induce toxin production in phytoplankton. She is also a competitive marathon runner and makes awesome cookies.

Hayley Flores is Director of Algal Culturing at Algenol Biofuels. She was formerly a postdoctoral investigator at UConn working with Professor Dam. Her interests are the biology and ecology of aquatic protists. Rumor is that Hayley is a plankton culture whisperer.

#### **Editor's Note:**

Hans Dam is the lead investigator in a project to study *Alexandrium* blooms and their control by predators, in Long Island Sound, on the north shore. The 2-year research project is funded by Connecticut Sea Grant.