The Lurking Perils of *Pfiesteria*

This minute creature has been implicated in dramatic fish kills and has hurt people. But its most publicized actions may not be the most damaging. More subtle effects are raising new concerns

by JoAnn M. Burkholder

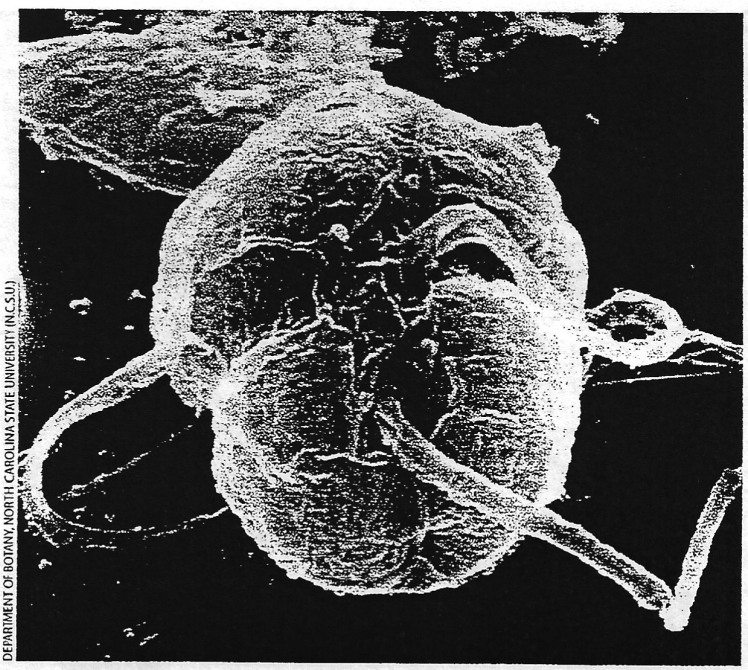
On a hot, humid October afternoon in 1995, I stood in a gently rocking boat, watching hundreds of thousands of bloody, dying fish break the mirrorlike surface of North Carolina's Neuse Estuary, where the Neuse River mixes with salty water from the Atlantic Ocean. Rising up out of the river, writhing, the fish gasped for air, then became still, floating on their sides. They were mostly Atlantic menhaden, small fish that serve as food for many larger species valued by commercial fishermen. An occasional flounder, croaker or eel also bobbed on the surface. Seagulls lined the shores of the nearly eight square miles of kill zone; a feast was in the making.

With my team from North Carolina State University (N.C.S.U.), I was collecting water samples from the area to try to determine the cause of the deaths. The bloody sores on the fish and their erratic behavior signaled a possible toxic outbreak of *Pfiesteria piscicida*, a single-celled microorganism that we had first seen in 1989 and had later linked to fish kills in several major estuaries. By the time this kill was over, 15 million silvery carcasses would carpet the water.

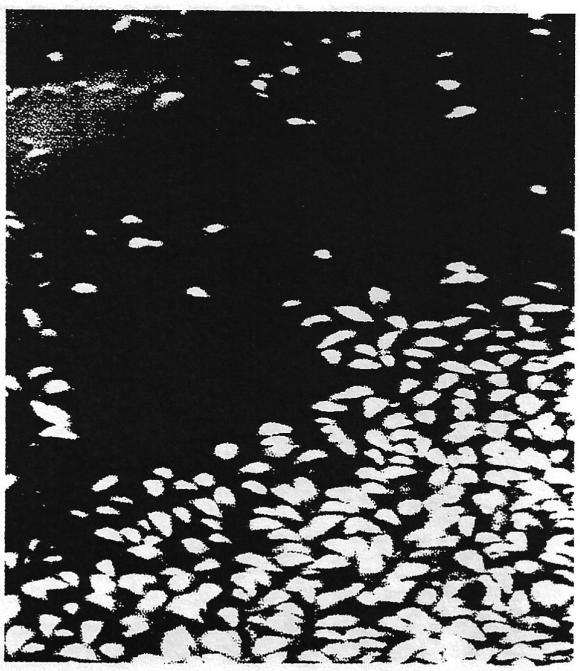
We quickly completed our sampling and pulled anchor, knowing it would be unwise to linger if *P. piscicida* was the culprit (as our test results later indicated was the case). People who have had contact with this creature in its toxic state have suffered from a range of symptoms, among them nausea, respiratory problems and memory loss so severe that it sometimes has been mistaken for Alzheimer's disease.

The scene on the river was all too familiar. In 1991 a billion fish died in the same way in this estuary. Since then, *P. piscicida*, occasionally with a closely related but unnamed toxic species, has been implicated almost yearly in massive fish kills in the estuaries of North Carolina (where it typically wipes out hundreds of thousands to millions of fish in a year) and in several smaller kills involving thousands of fish in Maryland waters of Chesapeake Bay.

These two species are the first members of the "toxic *Pfiesteria* complex," referred to hereafter as simply *Pfiesteria*. They (or still other toxic species that look the same but have not yet been identified definitively) have now been found as well in coastal waterways extending from Delaware to the Gulf Coast of Alabama, although they



DEPARTMENT OF BOTANY, NORTH CAROLINA STATE UNIVERSITY (N.C.S.U.)



have not been linked to fish deaths outside North Carolina and Maryland.

Over the past 10 years, my colleagues and I have learned a great deal about *Pfiesteria*'s life cycle and about the reasons for its proliferation and toxic outbreaks. We have also found it to be an astonishing creature, displaying properties never before seen in dinoflagellates—the larger group of microorganisms to which it belongs. Dinoflagellates, encompassing thousands of species, gain their name from the whiplike appendages (flagella) that they use for swimming in certain of their life stages.

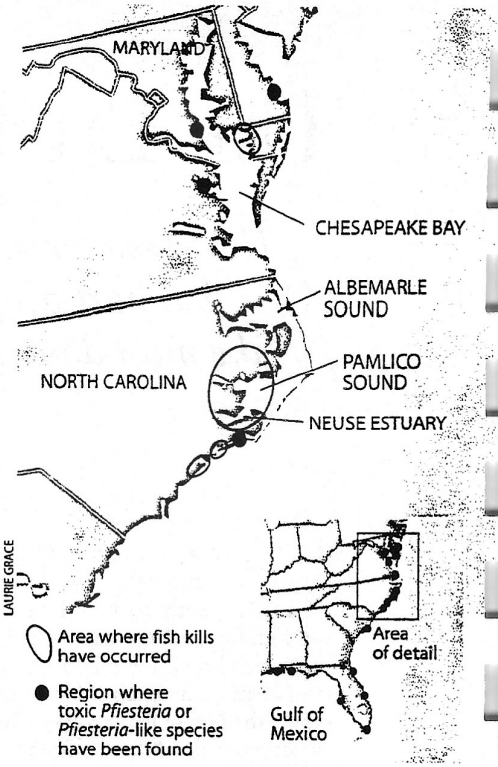
Other unexpected findings have prompted us to look beyond the floating dead fish to *Pfiesteria*'s additional untoward actions. Disturbingly, we have seen that aside from killing many fish at once, *Pfiesteria* can impair the health of finfish and shellfish in more subtle ways, such as by undermining their ability to reproduce and resist disease. These less obvious effects could potentially deplete fish populations more permanently than acute kills do.

Pfiesteria is not alone in its quiet treachery. Work by many investigators has also turned up insidious activities of other "harmful algae." As the term implies, this eclectic category encompasses certain true algae—primitive plants that make chlorophyll and carry out photosynthesis to make their own food. But it also includes various (usually unicellular) creatures, such as *Pfiesteria,* that look like algae but are not plants at all. The members of this ragbag group can hurt fish when they bloom, or proliferate—doing damage by producing dangerous levels of toxins or by other means, such as by growing so extensively that they rob the water of oxygen and cause fish to suffocate.

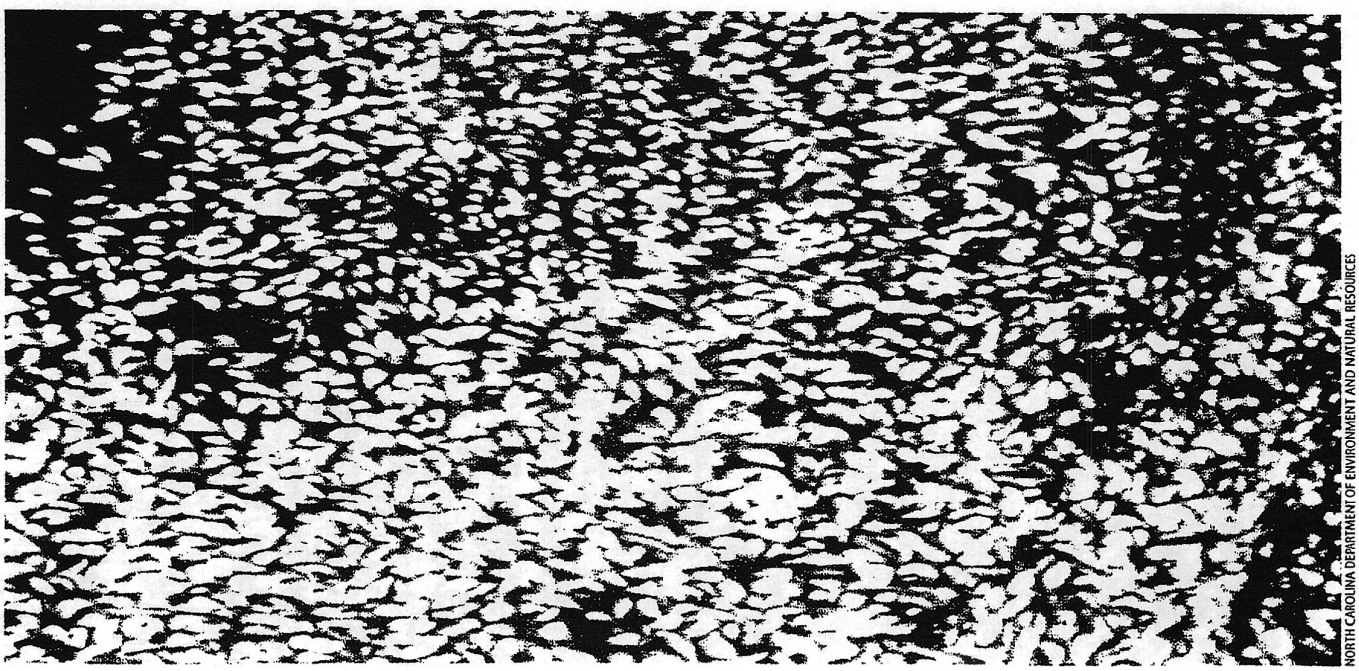
Various harmful algae are infamous for causing huge fish kills and for acutely poisoning animals or people who ingest toxin-laden seafood or water. Indeed, some of *Pfiesteria*'s dinoflagellate cousins account for the extraordinary red tides that have discolored and poisoned coastal waters worldwide for thousands of years. Yet the less obvious effects of harmful algae also need to be clarified and addressed if other serious illnesses and death in fish—and possibly in humans and other organisms—are to be avoided.

Pfiesteria was first linked to the death of fish in 1988, when tank after tank of fish in brackish water at N.C.S.U.'s College of Veterinary Medicine began dying mysteriously. The veterinarians noticed a swimming microorganism in the water and deduced through microscopy that it was a dinoflagellate. They subsequently noted that it became abundant in the aquarium cultures just before the fish died and seemed to disappear soon after the fish perished. But it reappeared if live fish were added to the tanks.

Because fish from around the world are studied at this laboratory, no one



TOXIC *PFIESTERIA PISCICIDA* (*micrograph on opposite page*), sometimes along with a close, unnamed relative, has been implicated in fish kills in estuaries of North Carolina and Maryland (*larger map*). But these species, forming the "toxic *Pfiesteria* complex," range much farther. Members of the complex, or very similar but not yet identified toxic microorganisms, have been found from Delaware to Alabama's Gulf Coast (*smaller map*). The carnage below occurred in North Carolina's Pamlico Estuary in 1991 and was the first kill linked to *Pfiesteria.*



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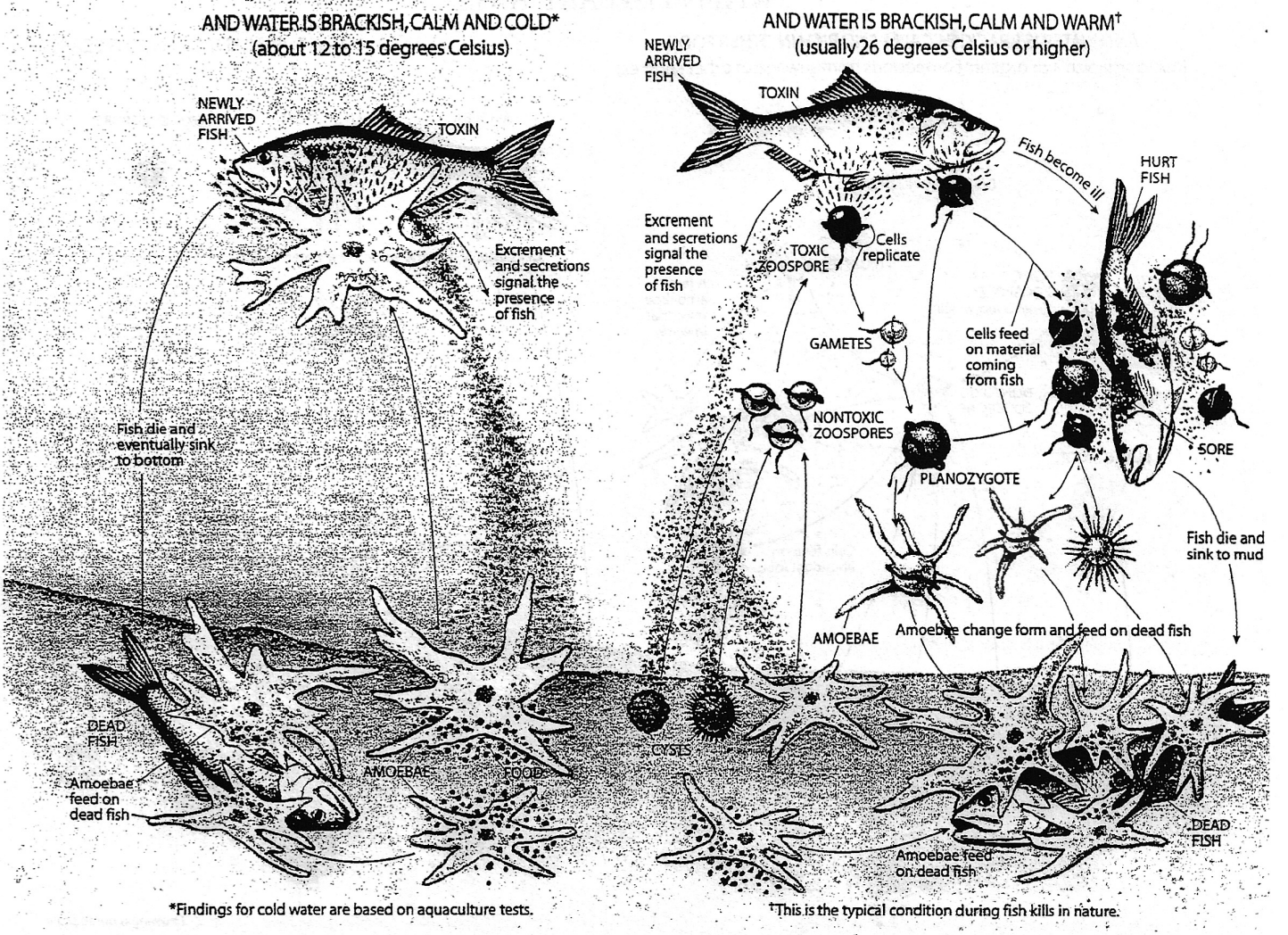
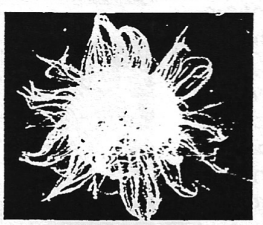
WHEN FISH ARE PRESENT...




LOBOSE AMOEBA

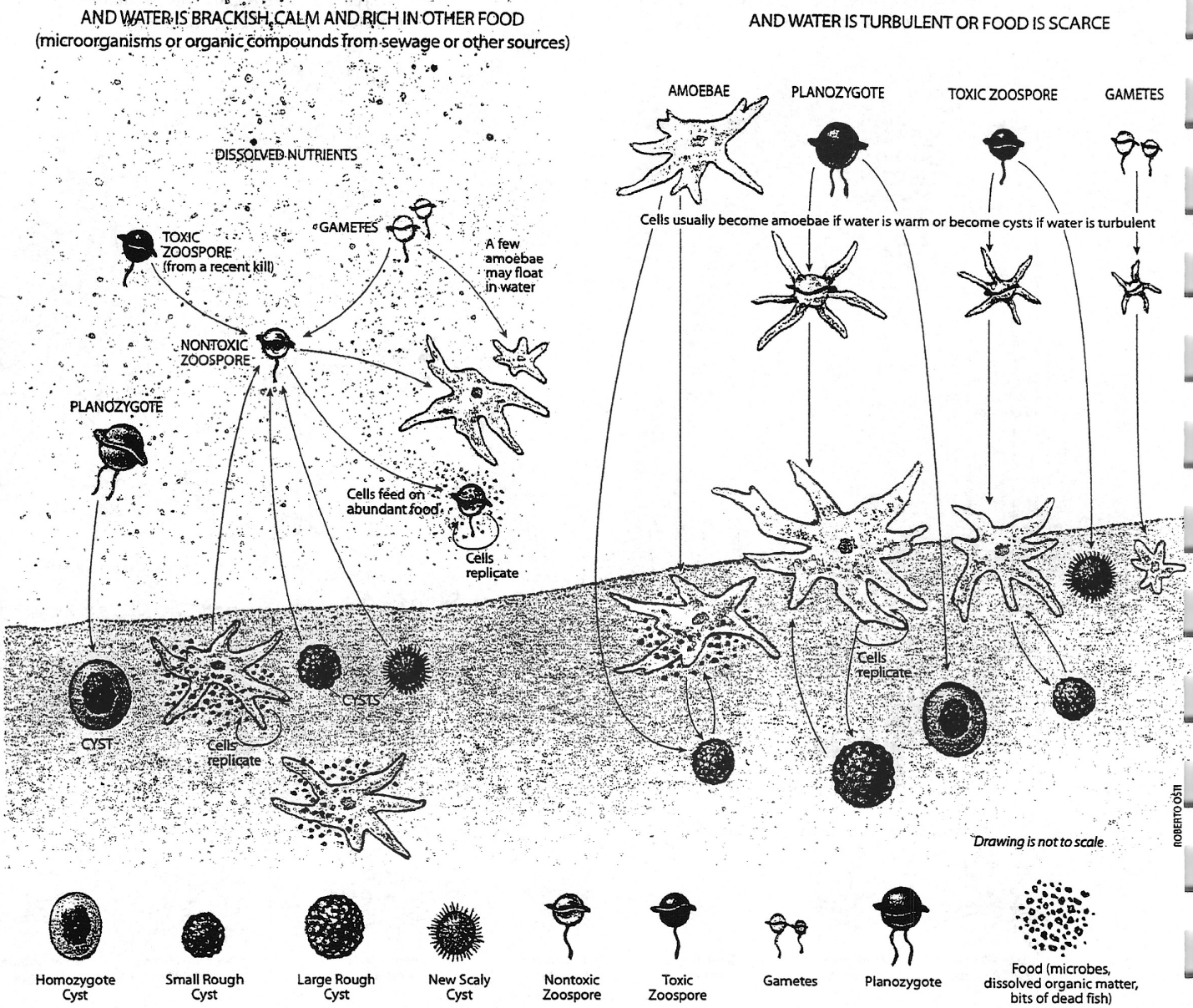

NEW SCALY CYST

Pfiesteria piscicida, a colorless single-celled organism, can change into at least 24 distinct forms—a rare feat. Only some are shown in the diagram (which is actually highly simplified) and micrographs here. The creature's shape and size depend on the type and amount of prey on the day's menu and on environmental conditions. That size can range from an invisible five microns (millionths of a meter) to a barely visible 750 microns.

The cells become toxic in nature when fish linger in their territory. Indeed, during the hotter seasons, the arrival of large schools of oily fish (*right panel above*) can trigger a "Jekyll and Hyde" personality transformation. Before fish enter the scene, the cells usually exist in any of three basic forms: various amorphous amoebae that quietly engulf algae and other prey in the bottom mud; encysted cells (also of many sizes) that hibernate, protected by a tough outer covering; or benign swimming cells known as nontoxic zoospores. When the fish arrive, the nontoxic zoospores become toxic (*unlabeled arrows indicate stage changes*). In addition, within minutes to hours, cysts and amoebae may give rise to nontoxic zoospores that soon become toxic as well. The altered zoospores send potent toxins into the water as they make a beeline for the fish.

The toxins drug the fish and destroy their skin, so that disease-causing bacteria and fungi can attack more easily as well. Meanwhile the toxic zoospores reproduce asexually and also produce gametes that fuse to form swimming, sexual products called planozygotes. As large sores develop on the fish, the toxic zoospores, planozygotes and gametes feed on substances that leak from the sores and on flecks of stripped skin, ingesting these materials by suction. When the fish

Cycle of *Pfiesteria*



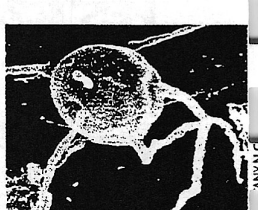
die, many of the cells may change into amoebae, attaching to the fish remains for a big meal.

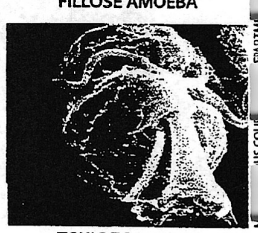
Laboratory tests and observations from aquaculture facilities suggest fish can face peril in cold water, too (*left panel on opposite page*). Large amoebae at the bottom of the tanks can quickly attack, kill and eat fish introduced into the system.

When dying fish disappear from the water but other nutrients, such as algal prey, are abundant, the toxic zoospores and gametes often revert to nontoxic zoospores (*left panel above*). Certain cells, meanwhile, may become amoebae or hypnozygotes (a kind of cyst). And amoebae and cysts in the bottom mud may produce more nontoxic zoospores. In the water the nontoxic zoospores feed well and multiply, but they will quickly become toxic attackers should another school of fish appear.

In more impoverished conditions (*right panel above*) the flagellated cells may opt to seek their fortune as scavenging amoebae in the mud. If the water is uncomfortably turbulent, though, swimming cells and amoebae may both turn into hibernating cysts, which are well suited for enduring adverse conditions. Twenty percent still survived even when we dried them for 35 days, immersed them in a concentrated acid or base for 30 minutes or held them in bleach for an hour.

The consummate opportunist, *P. piscicida* even resorts to thievery at times (*not shown*). It is unable to perform photosynthesis on its own. But in a process called kleptochloroplastidy, zoospores often steal chloroplasts, or photosynthetic organelles, from algae they have eaten and use them for days or weeks to help generate energy. —*J.M.B.*


FILLOSE AMOEBA


TOXIC ZOOSPORE

knew where the organism had come from or if it was a species already known to science. In 1989 the veterinarians asked my research group in the N.C.S.U. department of botany to help identify the microbe and determine whether it was responsible for the fish deaths.

The Nature of the Adversary

We soon realized that the creature was unique among both toxic and nontoxic dinoflagellates in adopting some forms, or stages, that do not resemble those of other dinoflagellates at all; in those stages it looks like a group of microorganisms called chrysophytes. It also stood alone among the small subset of dinoflagellates that are toxic. Those species (totaling about 60) produce some of the most potent poisons ever discovered in nature, although they make them for no obvious purpose. But the newfound organism not only appeared to poison fish—it ate them as well!

My research team learned that the extraordinary microbe we eventually named *Pfiesteria piscicida* is nontoxic when fish are absent. When it senses fish excrement and secretions in the water, however, it both emits toxins and swims directly toward the fish materials. The toxins strip away the skin of the fish, damage their nervous system and vital organs and make them too lethargic to flee. Then the fish commonly sustain attacks by other destructive microbes, and bleeding sores develop where the skin has been destroyed. With the fish unable to escape, the dinoflagellate cells feed on sloughed skin, blood and other substances leaking from the sores. Later the lethal cells change from flagellated, swimming forms to more amorphous amoebae that dine on the victims' remains, sometimes becoming so engorged that they can no longer move.

Toxic *P. piscicida* can be a very effective killer. In laboratory tests, toxin-contaminated water or cultures of the cells have killed many finfish and shellfish species. My research associate, Howard B. Glasgow, Jr., has found that young animals, as well as adults of more sensitive species, can die minutes after exposure, and most victims die within hours.

We also discovered another trait that had never been found in other toxic dinoflagellates. Remarkably, *P. piscicida* can transform into at least 24 distinct stages over the course of its life cycle. It alters its shape and size according to available food sources, which include prey ranging from bacteria all the way up the food chain to mammalian tissue. Some of these stage changes can involve a more than 125-fold increase in size and can take place in less than 10 minutes.

We studied *Pfiesteria* for two years in aquarium tanks without knowing where it might have come from. But the information we gathered indoors prepared us for that search. We began by looking in our own "backyard." Every year since at least the mid-1980s, massive fish kills had plagued North Carolina's Albemarle-Pamlico Estuarine System, which contains the Neuse River. With help from state biologists, we obtained water samples in 1991 during a kill of about one million Atlantic menhaden in the Pamlico Estuary.

The Adversary in Nature

When we examined the samples with a scanning electron microscope, we saw small dinoflagellates that looked identical to those we had found in the contaminated vet-school aquariums. Moreover, just as had happened in our tanks, the cells seemed to disappear after the kill ended—they were absent from water samples collected among the floating remains of fish one day after the fish died. This work not only tracked the vet-school contaminant to its probable origin but also implicated *Pfiesteria* as an important cause of fish death in nature.

What triggers toxic outbreaks of *Pfiesteria*? Laboratory and field experiments by many researchers indicate that, among other factors, an overabundance of nutrients such as nitrogen and phosphorus in the water help to set the stage for these events. The shallow, slow-moving waters of many North Carolina estuaries are easily polluted by materials from the surrounding land. These include nutrient-rich human sewage, fertilizers, certain industrial by-products (including some rich in phosphates) and animal wastes (from many swine and poultry operations in the watershed). When the waters become overnutrified, algae proliferate, much as houseplants grow much better when their soil contains added fertilizer. The abundant algae provide a rich food source for *Pfiesteria*, which then reproduces rapidly, creating legions ready to attack schools of fish should they swim into *Pfiesteria*-infested waters.

The estuaries of North Carolina turn out to be a very troubling place for *Pfiesteria* to wreak havoc. The Albemarle-Pamlico is the second largest U.S. estuarine system outside Alaska, and it provides half the area used by fish from Maine to Florida as nursery grounds. Many young fish come to these waters to grow and develop before heading north or south. If such fish die in large numbers in this crucial area, populations of affected fish species up and down the coast could eventually shrink.

Early in our research, as we established that *Pfiesteria* is highly lethal to fish, we also learned that fish are not its only victims; people can also be affected. Other toxic dinoflagellates generally hurt people by poisoning seafood. But studies by David P. Green of N.C.S.U. and his co-workers have found little evidence that *Pfiesteria* toxins accumulate in fish, a sign that seafood harvested from *Pfiesteria*-contaminated waters probably does not serve as a "middleman" in harming human beings. Instead the exposure route is more direct: people can become dangerously ill after getting toxin-laden water on their skin

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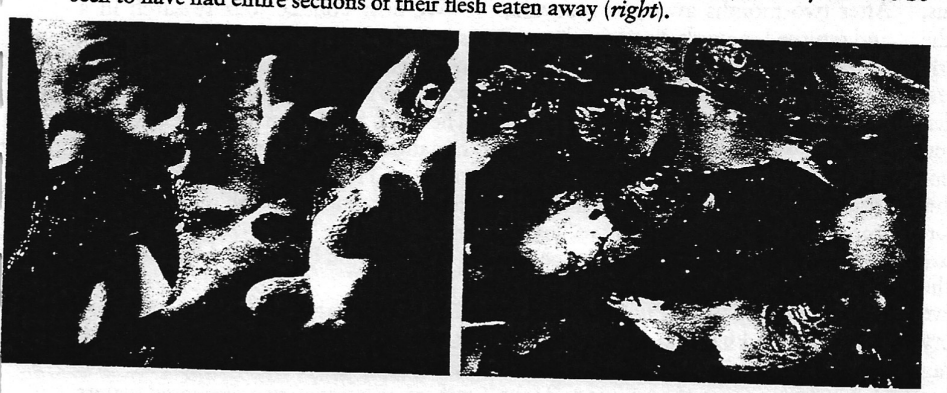


FISH KILLED DURING AN OUTBREAK OF *PFIESTERIA* (a term that encompasses any member of the toxic *Pfiesteria* complex) often display bloody sores (*left*); many can also be seen to have had entire sections of their flesh eaten away (*right*).

or after breathing the air over areas where fish are hurt or dying from their own encounters with toxic *Pfiesteria*.

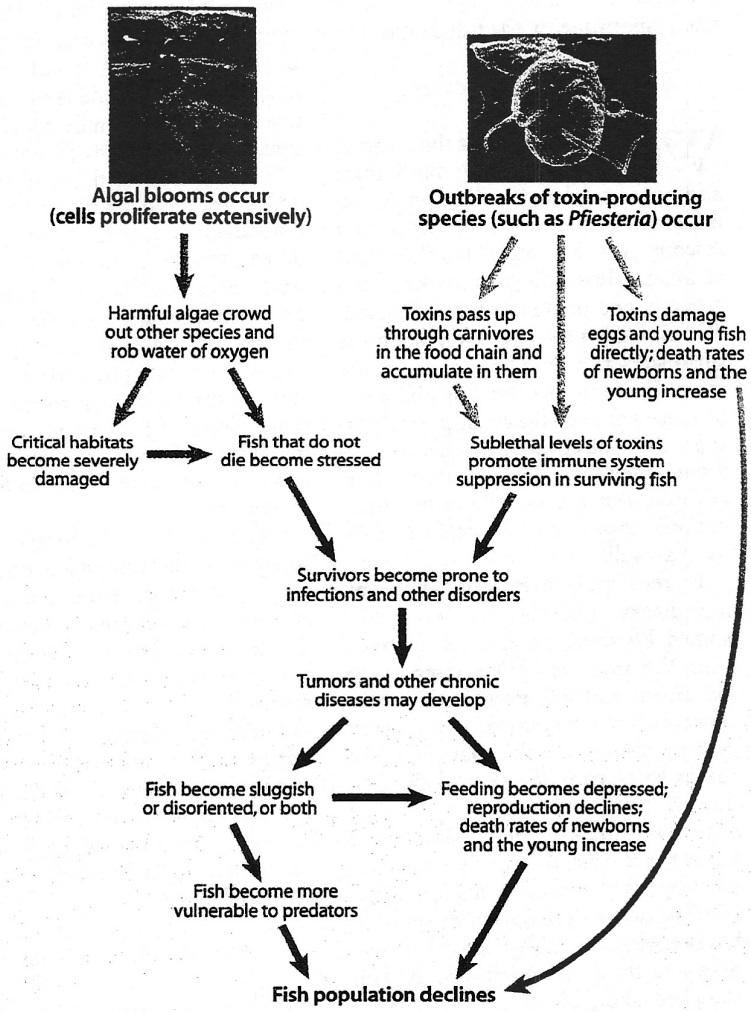
An Unwelcome Surprise

We learned about this last effect on people the hard way. When we first began our investigations, we followed established safety procedures for working with toxic dinoflagellates. We had been informed by specialists on other toxic dinoflagellates that in the laboratory contact with contaminated water was the only danger. We did not know that *Pfiesteria* produces an aerosolized neurological toxin that can seriously hurt people—the first dinoflagellate known to do so—and that we were inhaling it.

The symptoms were so subtle at first that we attributed them to other causes: shortness of breath that we ascribed to asthma; problems akin to allergy attacks, such as itchy or mildly burning eyes or a "catching" in the throat; and headaches and forgetfulness that we attributed to stress. Then one evening in 1992 Howard Glasgow went to a small laboratory where we originally had worked with *Pfiesteria*. Another department controlled the lab and had not cleaned it for some time. He found the walls caked with evaporated, toxin-laced *Pfiesteria* culture. He began trying to wipe up the mess, but after several minutes his eyes began to burn and he gasped for breath. He lost coordination, his legs went numb and he began to vomit. He managed to crawl out of the laboratory. We thought the extreme condition of the room was at fault and that he would not have fallen ill in a well-maintained lab.

We refused to use that laboratory again and had new facilities constructed. These were supposed to have been carefully ventilated, but unknown to us, the contractors mistakenly vented the air from the toxic-culture lab directly into Howard's office. Over the next few months, this normally cheerful, detailed scientist became extremely moody and sometimes seemed disoriented and unable to focus on even simple tasks. This highly intelligent man, with a razor-sharp memory, suddenly could not recall conversations from earlier in the day. Finally, after a period of intensive lab work, even his long-term memory suffered. He could not find his way home, remember his phone number or even read, and he struggled to speak.

How Harmful Algae May Cause Chronic Declines in Fish Populations



KEVIN SCHAFER *Peter Arnold, Inc. (left photograph);* DEPARTMENT OF BOTANY, N.C.S.U. *(right photograph)*

BEYOND KILLING MANY FISH AT ONCE, harmful algae can hurt fish in other ways. In the long run, these less obvious effects might lead to more persistent declines in fish populations than are caused by dramatic fish kills. The term "harmful algae" is a loose, eclectic category encompassing noxious algae as well as several species, such as *Pfiesteria*, that are more animallike than plantlike.

After two months away, he recovered and returned to work. But over the next two years, strenuous exercise caused relapses of aching joints, burning muscles and bouts of disorientation.

Before we realized that *Pfiesteria* can produce aerosolized toxin, 12 people from four different labs were sickened from toxic cultures. Three of us, myself included, have sustained some persistent problems we did not have before we began to study toxic *Pfiesteria*. In the past six years I have had chronic bronchial infections and 16 bouts of pneumonia; to cope with the infections, I take antibiotics for about a third of each year.

We now conduct our research in a specially designed biohazard III facility, using more precautions than are needed for most research with the AIDS virus. The lab is fitted with air locks, decontamination chambers and other safety features, and researchers wear full hooded respirators supplied with purified air.

Chronic Effects in the Field

People exposed to toxic *Pfiesteria* outbreaks in nature have reported similar symptoms. Divers, fishermen and others working in contaminated waters while fish were showing signs of *Pfieste-*

ria poisoning have described respiratory problems, headaches, extreme mood swings, aching joints and muscles, disorientation, and memory loss. Such anecdotal reports have recently been bolstered by formal clinical assessments.

In 1997, for example, three small outbreaks of *Pfiesteria* led Maryland's governor to close the affected waters in Chesapeake Bay for several weeks. Reports of strange symptoms in people who had been in the affected areas prompted the Maryland Department of Health and Mental Hygiene to organize a medical team to investigate. Among those who complained were heavily exposed fishermen—who described getting lost on a bay they had worked their entire lives or losing their sense of balance and concentration. Through neuropsychological testing, a medical team led by J. Glenn Morris, Jr., of the University of Maryland School of Medicine documented "profound" learning disabilities in the patients. The severity of their cognitive dysfunction was directly related to their degree of exposure, and the patients recovered their faculties over the next few months.

JEFFREY J. SPRINGER N.C.S.U.

RESPONSE OF BAY SCALLOPS to *Pfiesteria* in the laboratory is one of several indications that it can endanger the long-term health of fish it does not kill. When healthy scallops, such as the one on the left, were exposed to sublethal densities of toxic cells, they became unable to close their shells (*right*), a disability that would increase susceptibility to predation in the wild.

Doctors have difficulty diagnosing this "*Pfiesteria* syndrome" conclusively, however, because the specific toxins at fault have not yet been identified (as is the case with many toxic algae). Without that information, investigators cannot examine how the chemical acts in the human body, nor can tests be designed that definitively identify it in the blood or tissues. Fortunately, progress is being made. Peter D. R. Moeller and John S. Ramsdell of the National Ocean Service in Charleston, S.C., have semipurified components of *Pfiesteria* toxins that destroy fish skin and affect the nervous system in rats (which are studied as a model for humans).

Our own lingering health problems have led us to devote much attention to the possibility that *Pfiesteria* might cause chronic effects in fish that sustain nonlethal exposures. In lab experiments, we subjected fish to low concentrations of toxic *Pfiesteria* and monitored the animals for up to three weeks. The fish appeared to be drugged, and they developed skin lesions and infections. Tests revealed that white blood cell counts were 20 to 40 percent below normal levels, suggesting that *Pfiesteria* toxins may compromise the functioning of the immune system and make fish more susceptible to disease. Autopsies of fish that were affected have revealed damage to the brain, liver, pancreas and kidneys.

Weakened immunity, increased disease and periodic fish kills can all contribute to a decline in fish stocks. But other problems could seriously affect the ability of fish populations to recover. Research has shown that when toxic *Pfiesteria* is in the water, the eggs of striped bass and other commercially valuable fish fail to hatch. Experiments by Sandra E. Shumway of Southampton College and my graduate research assistant Jeffrey J. Springer have established that *Pfiesteria* also kills shellfish larvae, sometimes within seconds of contact, and causes young bay scallops to lose their ability to close their shells. In that condition, they would be highly vulnerable to predators.

The Bigger Picture

As we became increasingly concerned that *Pfiesteria* could threaten the viability of fish populations, we began to wonder whether this phenomenon was part of a broader trend. Dogma had long held that most finfish and shellfish exposed to sublethal doses of toxins from harmful algae suffer no ill effects. But could many harmful algae cause trouble that had been overlooked—perhaps by interfering with reproduction, with the survival of sensitive young fish or with resistance to disease? We also wondered whether there was evidence that these organisms could produce sustained or subtle health problems in people.

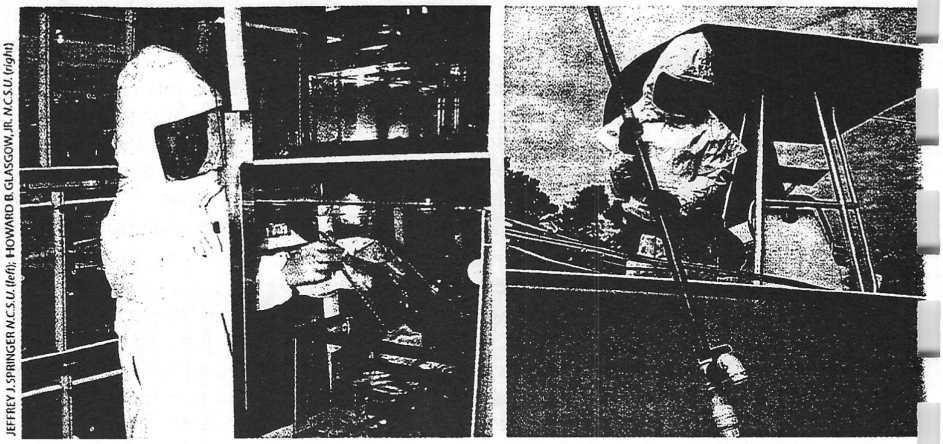
Few researchers have explored these questions or looked intently at the long-range effects of harmful algal blooms on the ecosystem as a whole. Nevertheless, a cluster of findings indicates cause for concern. These findings become especially disturbing when we note that as a group harmful algae are thriving. Some experts have pointed out that within the past 15 years, outbreaks of certain harmful algae seem to have increased in frequency, geographic range and virulence in many parts of the world.

Consider these examples. When bay scallops were exposed to small amounts of toxin from the dinoflagellate *Alexandrium tamarense,* their gut lining was eaten away, and their heart rate and breathing slowed. Other dinoflagellates produce ciguatera toxins that can accumulate in reef fish without killing them outright. The fish can grow large enough to be harvested as food for people, who then become sick. In fact, more human illness is caused by ciguatera-laden barracuda, red snapper, grouper and other tropical fish than by any other seafood poisoning. The symptoms can relapse for years, often triggered by alcohol consumption. Ciguatera toxins can also interfere with the normal function of white blood cells called T lymphocytes and thereby compromise the immune system. Recent work suggests that these toxins may take a similar toll on fish, resulting in impaired equilibrium, fungal infections and hemorrhaging.

Two types of cancer, disseminated neoplasia (similar to leukemia) and germinomas (which attack the reproductive organs), affect such shellfish as blue mussels and soft-shell clams. Studies have linked these cancers to certain dinoflagellates that produce saxitoxins, the same toxins that can cause sometimes fatal poisoning in people who eat contaminated shellfish. People who recover from acute saxitoxin poisoning may relapse with malarialike symptoms for years afterward. Ingestion of shellfish tainted with okadaic acid from toxic dinoflagellates along European coasts normally causes people to have diarrhea, but smaller, chronic doses have caused tumors in lab rats and human tissues. Okadaic acid can also destroy cells in the hippocampus of the

brain, an area important in memory, and can lead to suppression of the human immune system.

Chronic health problems from harmful algae are not restricted to marine environments. Blooms of blue-green algae (cyanobacteria) can take most of the oxygen from the water at night, so that fish become stressed and weakened and more vulnerable to disease. Moreover, toxins from these algae have caused liver, lung and abdominal tumors in mice, as well as mild to severe liver damage in humans.



JEFFREY J. SPRINGER N.C.S.U. (left); HOWARD B. GLASGOW, JR. N.C.S.U. (right)

HIGHLY PROTECTIVE EQUIPMENT is now de rigueur for researchers studying *Pfiesteria* and its close relatives. People can be harmed not only by having contaminated water touch their skin but also by inhaling *Pfiesteria* toxins from the air.

Fish as Canaries

To combat the unwanted effects of harmful algae, scientists must first "know the enemy" more thoroughly. Many harmful algae are so poorly understood that even fundamental facts about their life cycles remain unknown. Scientists must also chemically characterize more of their toxins, so that improved warning systems can be developed for determining when waters are unsafe.

Armed with that information, investigators will be able to assess how the toxins are processed in the human body and whether they are stored in our tissues. We will also be able to make progress in answering other important questions, such as: What is the range of acute and chronic effects of the toxins on the human nervous and immune systems, and how long do these effects last? What are the overall consequences to fish health? How do the toxins interact with other microorganisms and with pollutants to hurt fish, wildlife and humans?

For many species of harmful algae, the factors that stimulate increased activity are as incompletely understood as the organisms' life cycles. Clearly, nutrient pollution has stimulated the growth of *Pfiesteria* and certain other members of the group. Some ecologists believe that nutrient overenrichment and other types of pollution have contributed to a serious general imbalance in many aquatic ecosystems. Large algal blooms and toxic outbreaks, they assert, are symptomatic of this imbalance as well as participants in its perpetuation.

This ecological breakdown may have many causes. Continuing losses of the wetlands that act as the earth's kidneys hamper the ability of waterways to cleanse themselves. Some algal blooms have coincided with El Niño events, suggesting that warming trends in global climate may stimulate the growth of these species and extend their range. These climatic changes also create flooding that washes additional nutrients and other pollution into rivers and estuaries. Further, inadequate environmental regulations are providing too little protection for our waters at a time when nearly two thirds of Americans live within 50 miles of a coastline. There are more people on the earth than ever before. They are using relatively scarce freshwater supplies at an ever increasing rate, while they are also generating more and more wastes that degrade both fresh and marine waters.

As we pulled anchor during the October 1995 fish kill, many thoughts were in my mind. I was keenly aware that *Pfiesteria* is but one type of harmful microorganism that can disrupt both fish resources and human health. Ultimately, water quality, human health and fish health are strongly linked. All of us—scientists, politicians, resource managers, fishermen and other citizens—need to work together to learn much more about the chronic as well as the acute effects of harmful algae. We must also become more proactive in addressing the state of our waterways, instead of reacting to each fish kill as if it were a limited, isolated crisis. In protecting vulnerable fish, the health we spare may also be our own. SA

The Author

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Further Reading

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MARINE ECOSYSTEMS: EMERGING DISEASES AND INDICATORS OF CHANGE. Paul Epstein et al. Year of the Ocean Special Report. Center for Health and the Global Environment, Harvard Medical School, Boston, 1998.

The Aquatic Botany Laboratory at North Carolina State University site on the toxic *Pfiesteria* complex is available at www.pfiesteria.org on the World Wide Web.