Unraveling a fish killer's toxic ways

On August 6, large numbers of dead fish began floating to the surface of Maryland's Pocomoke River, which flows into the Chesapeake Bay. The scene continued for the next 4 days, during which an estimated 10,000 fish died, many pockmarked with red lesions. Three weeks later another 2,000 or so fish went belly-up in Virginia waters nearby.

In the estuaries of North Carolina, such scenes have played out regularly since at least the 1980s. In 1991, researchers there linked the fish kills to a microscopic, toxin-producing organism that was new to science. They named this dinoflagellate *Pfiesteria piscicida*, or fish killer.

Exactly how the single-celled organism kills—and how harmful it is to other wildlife and humans—is still unknown. New details about its modus operandi came to light at a meeting last week in Research Triangle Park, N.C., as researchers reported progress in isolating and characterizing two of the several toxic compounds produced by *P. piscicida*.

Participants at the National Institute of Environmental Health Sciences meeting welcomed the news, even though the chemical structure and identity of the toxin complex have yet to be worked out.

"They're closing in on it," says Jeffrey L.C. Wright of the National Research Council of Canada in Halifax, Nova Scotia. "It means progress is being made." Once the toxins are fully identified, researchers can work on detection methods and treatments or preventives.

JoAnn M. Burkholder of North Carolina State University in Raleigh reported that she and her collaborators at the National Marine Fisheries Service in Charleston, S.C., have isolated a water-soluble compound from laboratory-grown *P. piscicida* that quickly kills fish. When fish are exposed to a high concentration of the chemical, they become moribund within a few seconds and are dead in 3 to 5 minutes.

"It's a very small, rapidly acting toxin," says Burkholder. The researchers have also found that it doesn't fall apart when heated and is toxic to mammalian neural and pituitary cells grown in the lab.

Burkholder says the compound is unlike any of the known toxins produced by other dinoflagellates. In the early stages of studying *P. piscicida*, she and her colleagues suffered a range of health problems, including memory loss, apparently from intense exposure to the unknown toxins. They now work with the toxins under contained conditions, in a trailer apart from the main campus.

Also at the meeting, Daniel G. Baden of the University of Miami presented a photo of a crystallized compound that his coworker Kathleen S. Rein recently separated from laboratory-grown *P. piscicida*. This second compound is also relatively low in molecular weight but is soluble in fat rather than water and acts on the fish's skin. In tests of the isolated compound, "we watch the epidermis pill up and fall off the fish," Baden told SCIENCE NEWS.

The descriptions of the two compounds fit into the emerging picture of the strange life of *P. piscicida,* which has at least 24 stages. The lethal water-soluble toxin may be excreted when the sediment-dwelling microorganism detects and swims towards a school of fish, earning *P. piscicida* the reputation of an ambush predator. The skin-damaging compound would allow the microorganism to feed on the fish's tissues.—*C. Mlot*

Ambush predator: Microscopic Pfiesteria piscicida *(inset, magnified several thousand times), in one of the 24 or more cellular forms it can take, produces powerful toxins that are responsible for mass killings of fish, such as these Atlantic menhaden.*

Caries: Legacy of mom's lead exposure?

Several studies have linked a high incidence of dental cavities to lead exposure, offering parents yet another reason to shield their youngsters from old house paint and other potential sources of this toxic heavy metal. However, a new animal study now suggests that for some of these children, one of the more potent sources of lead—and caries vulnerability—may be all but unavoidable: their mother.

Throughout the United States in recent years, "there's been a big drop in the prevalence of childhood caries," notes William H. Bowen, a dentist and microbiologist at the University of Rochester (N.Y.) School of Medicine and Dentistry. Of those cases that do show up, however, "some 80 percent are occurring in just 20 percent of kids"—mostly those living in inner cities, where lead exposures can still be relatively high, he observes.

Because lead mimics calcium, the body normally stores most lead in bone. During pregnancy and lactation, when the body breaks down bone to liberate calcium for the developing young, lead can be released back into the blood.

"We know that lead crosses the placenta," Bowen says, "so there was good reason to suspect it could affect [tooth] development." His group decided to probe maternal transmission of lead in pups born to female rats that had been raised on drinking water spiked with relatively high concentrations of lead (34 parts per million). Resulting blood concentrations, about 40 micrograms per deciliter, are at the high end of what can be found in humans.

In the September NATURE MEDICINE, the researchers report that pups from lead-exposed moms developed 40 percent more dental cavities and produced 30 percent less saliva than did those born to mothers raised on leadfree water.

The two observations may be related, the Rochester scientists note. Saliva not only helps wash away food particles that might contribute to the development of caries but also possesses natural antibiotics to retard the growth of bacteria. Saliva can even supply teeth with the mineral building blocks needed to repair incipient cavities (SN: 4/19/86, p. 252).

Moreover, the Rochester study turned up evidence that moms may remain an important conduit for delivering lead after birth. "We found surprisingly high levels of lead in [breast] milk," Bowen says—concentrations roughly 10 times as high as those in the mothers' blood. "This indicates that there is clearly some concentrating mechanism" in mammary tissue, he says.

Previously, there had been a suspicion that any lead-induced vulnerability to decay traced to a substitution of the metal for calcium or some other constituent in tooth enamel, creating weaknesses in its crystalline structure, say Martin E. J. Curzon and K. Jack Toumba of the University of Leeds School of Dentistry in England in an accompanying commentary. The primary exposures of concern had been lead-based paint and drinking-water pipes.

Now, the Rochester findings "reveal two hitherto unrealized aspects of lead toxicity," they observe—the role of lead stored in the mother's body and the salivary glands' vulnerability.

These new data may even have some historical implications, the Leeds pair muses. While some toxicologists have suggested that IQ declines associated with drinking water from lead pipes (SN: 1/27/90, p. 63) may have contributed to the fall of the Roman Empire, Curzon and Toumba offer an alternative speculation. Perhaps, they say, chronic toothaches "afflicted the Roman legions, rendering them incapable of defending the Empire." —*J. Raloff*