

Pfiesteria blamed unfairly for fish sores?

The so-called cell from hell has been unfairly blamed for opening bloody sores on fish, say government researchers.

All the fuss over fish-killing *Pfiesteria* has obscured a more probable culprit for the lesions, a fungal disease described in the 1980s, according to a statement released last week by fish pathologist Vicki Blazer of the U.S. Geological Survey's Leetown (W.Va.) Science Center.

Pfiesteria dinoflagellates have made headlines during the past decade as the main suspect in fish kills and a possible cause of neurological damage in people along coastal waterways in mid-Atlantic states, especially North Carolina and Maryland (SN: 9/27/97, p. 202). Yet one of the most striking images from these disasters, oozing lesions as big as a quarter on silvery menhaden, may not be the work of *Pfiesteria* at all.

"I do not dispute that you can produce lesions with *Pfiesteria*, but you cannot produce *these* lesions," Blazer says. *Pfiesteria* toxins act fast, certainly within several hours, she observes, yet the sores she analyzed microscopically showed inflammations that needed perhaps two weeks to develop. She checked sores on fish that died in Maryland's kills during 1997 as well as on fish collected in the same region but not during a kill. In more than 95 percent of all the fish, Blazer found fingerlike growths, strands of invading fungus surrounded by inflamed fish tissue with thick buildups of immune-system cells.

"The fish kills, I do believe, were caused by *Pfiesteria*," Blazer says. "We

need to separate fish kills from fish lesions."

Michael J. Mac, who is based in Reston, Va., directs the Geological Survey's fishery research program. He puts the implications simply: "If lesions can be caused by a fungus, then it's not a good indicator of *Pfiesteria*." That's bad news for fishery managers in Maryland, who use a high number of lesions as one of the warning signs in deciding whether to close off waterways to keep people away from a developing *Pfiesteria* outbreak and its toxins. Worse, Mac says "we don't have any other suggestions" for simple field tests for *Pfiesteria*.

That's why *Pfiesteria* researcher JoAnn M. Burkholder of North Carolina State University in Raleigh is troubled by recent publicity about the fungus. "I want to cling to the lesion method," she says. Fish kills and high numbers of the sores often show up together, she contends. "It's a very crude barometer," she acknowledges, but "why take a chance if you care about people's health?"

Burkholder has not swayed fish pathologist Wolfgang Vogelbein of the Virginia Institute of Marine Science in Gloucester Point. "There have always been doubts among fish pathologists," he says. "They have seen the condition in menhaden every year." His work parallels Blazer's analysis showing that the sores do not come from *Pfiesteria*. "It's impossible," he says. "They're old."

So is the fungus idea. Mycologist Michael J. Dykstra from North Carolina State and his colleagues started publish-



Open sores on menhaden from Maryland have sparked a whodunit debate: a slow fungus or a *Pfiesteria* toxin.

ing papers in the 1980s describing ulcers common on menhaden. The researchers saw no direct link to toxic algae blooms. "You can find fish with lesions when there's no kill," Dykstra points out. Instead, they focused on the fungi that showed up prominently in the sores, including *Aphanomyces*. Fungi of that genus drill gory sores in fish near Australia, Japan, India, and Thailand.

Aphanomyces species, with spores that have an uncommon ability to swim, also attack European crayfish and cause root rot in peas, notes David Rizzo, who teaches mycology at the University of California, Davis. Closely related fungi often have wide-ranging tastes, Rizzo says. He points out that a relative of the Dutch elm disease fungus infects human lymph glands.

However, the whole focus on finding the bleeding gun that causes fish sores distresses Dykstra. "Nutrient loading [phosphorus and nitrogen pollution] is probably more important than any specific pathogen because it's behind all the pathogens," he says. —S. Milius

Tabulating an enormous assortment of knots

A scout's handbook might show dozens of different knots—overhand, reef, granny, bowline, and so on. Mathematicians now have their own, considerably larger inventory of knots for their studies (SN: 5/3/97, p. 270).

Two new catalogs, produced independently, tabulate all knotted loops of a one-dimensional string having 16 or fewer crossings when the knot is laid flat. That's 1,701,936 different knots.

Jim Hoste of Pitzer College in Claremont, Calif., and Jeffrey R. Weeks of Canton, N.Y., worked on one listing, while Morwen Thistlethwaite of the University of Tennessee in Knoxville generated the other. They jointly describe their tabulation efforts in the fall issue of MATHEMATICAL INTELLIGENCER.

"With more than 1.7 million knots now in the tables, we hope that the census will serve as a rich source of examples and counterexamples and as a general testing ground for our collective intuition," the researchers say.

Mathematicians typically concern them-

selves with knots having their two ends connected to form a loop. One way to characterize such a knot is to lay it flat and determine its crossing number by counting the minimum number of times one part of the loop crosses over or under another part.

Efforts to tabulate knots began about 120 years ago, after British physicist Lord Kelvin hypothesized that atoms could be described as vortices in the ether, an intangible fluid then thought to fill all space. He proposed that different elements would correspond to vortices bent into different types of knotted tubes forming closed loops.

Inspired by this theory, fellow physicist Peter G. Tait began investigating knots and produced the first knot tables, organized according to crossing number.

Tait enumerated all possible knot diagrams up to a given crossing number, then grouped those diagrams representing the same knot type. He stopped at knots with seven crossings—a total of 15 knot types.

Using a similar strategy and with the



Example of a 16-crossing knot.

help of computers, Hoste, Weeks, and Thistlethwaite produced independent tabulations of knots with 16 or fewer crossings. Kept secret until they were finished, the two lists were in complete agreement.

Hoste and Thistlethwaite are now preparing the 17-crossing list. —I. Peterson