

Researchers Track Killer Pathogen to Solve Aquaculture Mystery

Disease is a predominant concern in aquaculture: With fish confined in close quarters, an outbreak can be ruinous. So when disease does occur, determining the cause is a high-stakes mission.

"It was like one of those detective stories, isolating bacteria to see which one was causing the disease," comments Marta Gomez-Chiarri, URI fisheries, animal, and veterinary science assistant professor. With Sea Grant funding to uncover the killer pathogen, Gomez-Chiarri added her background in animal pathology to the specializations of Jennifer Specker, URI oceanography professor, and David Nelson, URI biochemistry, microbiology, and molecular genetics professor.

Immediate evidence of disease-distended abdomen and reddened anal area hinted at more profound damage that culminated in destruction of the intestine of diseased fish. Dubbed flounder infectious necrotizing enteritis (FINE), the disease progressed just as its name describes-inflaming and ultimately killing intestinal tissue. "The bacterium proliferates in the intestine and damages tissue until it cuts the intestine away from the anus," explains Gomez-Chiarri. In some of the fish the detached intestine had healed into a blind sac, indicating that these fish survived the initial infection.

Testing tissue from the kidneys and fluid from abdominal cavities of sick fish, Gomez-Chiarri and colleagues determined that the disease was bacterial, as suspected. To narrow the possibilities to the particular bacterium at fault, the scientists ran experiments with groups of healthy fish, injecting them with different bacterial clones isolated from solutions of fluid from the diseased fish.

Their results pointed to *Vibrio carchariae*, one of several *Vibrio* species known to afflict both flatfish and shellfish, especially in culture situations. This bacterium "had been identified before," according to Gomez-Chiarri, but why it appeared in this instance was uncertain.

"We don't know what triggers an outbreak, but it's probably a combination of transport stress and summer water temperatures," she says. The affected fish had been transported to the Rhode Island grow-out facility from a hatchery in New Hampshire, and the trip may have compromised their adaptability and disease resistance, as suggested by related research under way in Specker's laboratory. Also, the slightly elevated summer water temperatures in the grow-out environment could have contributed to bacterial growth. Proliferation of disease-causing organisms in culture facilities tends to be a warm-water, warm-weather phenomenon, according to aquaculture disease Web sites of the U.S. Fish and Wildlife Service, Michigan State University Cooperative Extension Service, and other organizations (<http://www.lsc.nbs.gov/fhl/fdl/fdl77.htm>, http://aq.ansc.purdue.edu/aquanic/publicat/usda_rac/efs/ncrac/efs101.htm).

With the cause of FINE identified and the pathology pinned down, the researchers turned their attention to the disease's aftermath. A month after the outbreak ended, Specker picked up the research, randomly selecting fish from the grow-out facility to examine for evidence of disease or residual symptoms. Fifteen percent of the fish she sampled exhibited the sealed pouch constituting a healed portion of the otherwise destroyed intestine. That these fish survived was a wonder in itself. That they could still maintain the vital balance between water absorption and salt elimination, with two-thirds of their intestines gone, was a real marvel.

"We assumed that osmoregulation would be disturbed in fish with intestinal stumps," Specker says. Because the intestine is a predominant organ for regulating the fluid-salt balance necessary for marine existence, intestinal damage of the extent exhibited by the FINE survivors should be incapacitating. To test whether or not this was the case, Specker and her colleagues examined survivors of the *Vibrio* infection three months after the outbreak ended.

What they found disproved their hypothesis and generated new suppositions about the summer flounder's mechanisms for homeostasis, or equilibrium. Possibly the entire gastrointestinal tract compensated for the loss of the intestine to keep salt and water uptake in proper balance, Specker suggests in an article in the journal *Fish Physiology and Biochemistry* (2001, in press).

If so, the struggle for homeostasis may have come at the expense of nutrition. Recovered fish did continue to feed: Specker and Gomez-Chiarri both report that survivors had food in their guts when examined. But these fish were quite small—less than half the size of healthy fish, Specker reports. She hypothesizes that the partial intestine remaining after the infection subsided worked harder to achieve the water-salt balance and so had less capacity to handle nutrient uptake.

The full consequence to aquaculture of *V. carchariae* and the impact of this new information are not yet clear. But spin-off studies will contribute to a knowledge base that may ultimately permit control of *Vibrio* infection. Specker is looking more closely at the role of stress in flatfish culture in order to identify industry practices that induce stress and to develop ways to reduce it. Gomez-Chiarri is expanding her investigation to other aquaculture sites, working with a major hatchery. So far, the *V. carchariae* bacterium is shown to infect juveniles in grow-out facilities. Gomez-Chiarri wants to know if it affects larvae and reproducing adults as well. And Nelson, who has been studying another deadly *Vibrio* bacterium, *V. anguillarum*, is continuing his work to develop vaccines against diseases caused by *Vibrio*.

—Tony Corey

Links

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- Marta Gomez-Chiarri <http://www.uri.edu/cels/favs/mg.html>
- Jennifer Specker <http://gso.uri.edu/faculty/specker.html>
- David Nelson <http://www.uri.edu/cels/cmb/faculty.html>