Pollutant waits to smite salmon at sea

Canadian scientists have identified the likely culprit behind some historic, regional declines in Atlantic salmon. The researchers find that a near-ubiquitous water pollutant can render young, migrating fish unable to survive a life at sea.

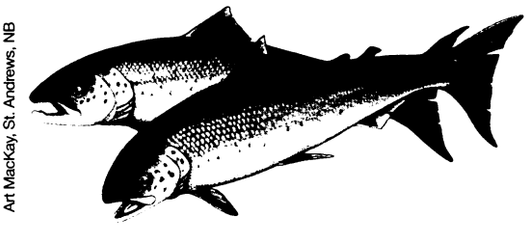
Heavy, late-spring spraying of forests with a pesticide laced with nonylphenol during the 1970s and '80s was the clue that led the biologists to unmask that chemical's role in the transitory decline of salmon in East Canada. Though these sprays have ended, concentrations of nonylphenols in forest runoff then were comparable to those in the effluent of some pulp mills, industrial facilities, and sewage-treatment plants today. Downstream of such areas, the scientists argue, salmon and other migratory fish may still be at risk.

Nonylphenols are surfactants used in products from pesticides to dishwashing detergents, cosmetics, plastics, and spermicides. Because waste-treatment plants don't remove nonylphenols well, these chemicals can build up in downstream waters (SN: 1/8/94, p. 24).

When British studies linked ambient nonylphenol pollution to reproductive problems in fish (SN: 2/26/94, p. 142), Wayne L. Fairchild of Canada's Department of Fisheries and Oceans in Moncton, New Brunswick, became concerned. He recalled that an insecticide used on local forests for more than a decade had contained large amounts of nonylphenols. They helped aminocarb, the oily active ingredient in Matacil 1.8D, dissolve in water for easier spraying.

Runoff of the pesticide during rains loaded the spawning and nursery waters of Atlantic salmon with nonylphenols. Moreover, this aerial spraying had tended to coincide with the final stages of smoltification—the fish's transformation for life at sea.

To probe for effects of forest spraying, Fairchild and his colleagues surveyed more than a decade of river-by-river data on fish. They overlaid these numbers with archival data on local aerial spraying with Matacil 1.8D or either of two nonylphenol-free pesticides. One contained the same active ingredient, aminocarb, as Matacil 1.8D does.



Art MacKay, St. Andrews, NB

Atlantic salmon.

Most of the lowest adult salmon counts between 1973 and 1990 occurred in rivers where smolts would earlier have encountered runoff of Matacil 1.8D, Fairchild's group found. In 9 of 19 cases of Matacil 1.8D spraying for which they had good data, salmon returns were lower than they were within the 5 years earlier and 5 years later, they report in the May Environmental Health Perspectives. No population declines were associated with the other two pesticides.

The researchers have now exposed smolts in the laboratory to various nonylphenol concentrations, including some typical of Canadian rivers during the 1970s. The fish remained healthy—until they entered salt water, at which point they exhibited a failure-to-thrive syndrome.

"They looked like they were starving," Fairchild told Science News. Within 2 months, he notes, 20 to 30 percent died. Untreated smolts adjusted normally to salt water and fattened up.

Steffen S. Madsen, a fish ecophysiologist at Odense University in Denmark, is not surprised, based on his own experiments.

To move from fresh water to the sea, a fish must undergo major hormonal changes that adapt it for pumping out excess salt. A female preparing to spawn in fresh water must undergo the opposite change. Since estrogen triggers her adaptation, Madsen and a colleague decided to test how smolts would respond to estrogen or nonylphenol, an estrogen mimic.

In the lab, they periodically injected salmon smolts with estrogen or nonylphenol over 30 days, and at various points placed them in seawater for 24 hours. Salt in the fish's blood skyrocketed during the day-long trials, unlike salt in untreated smolts. "Our preliminary evidence indicates that natural and environmental estrogens screw up the pituitary," Madsen says. The gland responds by making prolactin, a hormone that drives freshwater adaptation.

Judging by Fairchild's data, Madsen now suspects that any fish that migrates between fresh and salt water may be similarly vulnerable to high concentrations of pollutants that mimic estrogen. —*J. Raloff*

Dam the bacteria, drugs and vaccines ahead

Growing in a test tube, bacteria may appear harmless. When they infect a host, however, the microbes draw upon a concealed repertoire of molecular tools to dodge immune defenses and cause disease. Much like the famed Trojan horse, bacteria "hide their weapons until they're inside," notes Michael J. Mahan of the University of California, Santa Barbara.

In the May 7 Science, Mahan and his colleagues show that a protein called DNA adenine methylase, or Dam, regulates a bacterium's use of its armament. Without Dam, it's no longer virulent.

The findings suggest that Dam offers an appealing target for new antibiotics. Moreover, bacteria weakened by mutations in the gene for Dam might serve as vaccines against many diseases.

Like other methylases, Dam chemically coats DNA with clusters of atoms known as methyl groups. In doing so, it governs bacterial processes such as DNA replication and repair.

Mahan's group established the protein's role in virulence by disabling its gene in *Salmonella typhimurium*, which causes food poisoning in people and typhoid fever in mice. When researchers infected rodents with bacteria unable to make Dam, disease rarely resulted.

Mahan's team had previously identified around 250 genes activated in *S. typhimurium* when it infects a host. In the mutant bacteria, as many as 50 of these genes become active when the microbes are grown outside a host in test tubes. Dam seems to normally repress the genes until needed during an infection.

Unleashing the genes at the wrong time during an infection can prove troublesome to a bacterium. Furthermore, the mutant bacteria may overproduce some surface proteins, making them easier for the immune system to spot.

The investigators suggest that drugs that disable Dam will prove helpful in the fight against bacteria that have evolved resistance to traditional antibiotics.

"It's a nice target. It's a protein unique to bacteria, and its effect on virulence is spectacular," agrees Josep Casadesús of the University of Seville in Spain, who recently did experiments with results very similar to Mahan's group's.

While almost all bacteria employ Dam, investigators must still prove that the protein regulates virulence in more than this one species. The pervasiveness of Dam among bacteria raises a concern about any drug that targets the protein. "You could affect all the good bacteria in the gut," says Casadesús.

Disabling Dam weakens but doesn't actually kill bacteria. Salmonella bacteria without the methylase infect gut cells but don't advance deeper within a mouse, where they normally cause disease. Infections with the mutant bacteria protected mice from subsequent doses of unaltered salmonella.

"They stimulate the hell out of the immune system, but they don't proceed. It's the perfect vaccine," says Mahan, who has launched a company based on the new research. Its first goal is a vaccine that immunizes chickens from the salmonella that causes food poisoning. Dam-based vaccines may also thwart bacteria that cause cholera, the plague, and typhoid fever, Mahan says. —*J. Travis*