Pollution conundrum has fishy solution

To understand why industrial chemicals taint even uninhabited Arctic regions thousands of miles from where the pollutants were released, scientists have focused on air pollution. Many organic pollutants leapfrog the globe, periodically vaporizing from sites on the surface to ride the winds in a slow, polar-bound trek (SN: 3/16/96, p. 174).

Few scientists had considered biological means of transport, yet ecotoxicologists now report that much of the DDT, polychlorinated biphenyls (PCBs), and other persistent organic chemicals in one seemingly pristine lake arrived via spawning salmon.

A research team headed by Göran Ewald of Lund University in Sweden compared organic pollutants in grayling—a top predator and game fish—from two neighboring Alaskan lakes: Round Tangle, which is self-contained, and Lower Fish, which drains into the final leg of a spawning run for sockeye salmon. Grayling in Lower Fish Lake have up to four times the concentrations of organic pollutants in their fat as grayling in the salmonfree lake, the team reports in the just-published March ARCTIC.

Ewald explains that the salmon go to sea after spending a year or two in the freshwater lake. A few years later, they return to the lake to spawn. His team collected the fish throughout their 410-kilometer migration from the Gulf of Alaska.

Analyses showed that as the fish burned fat to power their trip home, they didn't metabolize the pollutants in it. Instead, the chemicals became concentrated in the remaining fat. After spawning, the fish died, and their roe and carcasses introduced these pollutants into the food chain of Lower Fish Lake. Concentrations in the water, expected to be low, were not measured.

Sockeye salmon (above) can share pollutants with lake-bound grayling (below).

Ewald's team reports that the proportions of DDT breakdown products and PCBs in Lower Fish Lake grayling match the pattern in salmon rather than the concentrations present in the air. Since the data show that both lakes received equivalent inputs of airborne pollutants, the grayling in Lower Fish Lake must be picking up most of their pollutants "from eating the fish and roe," Ewald says.

"This finding is exciting and really important," says Derek C.G. Muir of Environment Canada's National Water Research Institute in Burlington, Ontario. "It might explain much of the variation [in pollution] in regions where there are a lot of migratory fish," he says. It could also help regulators identify areas that might need more stringent advisories to limit consumption of tainted fish.

In reality, people know little about freshwater-spawning marine fish, observes Phyllis Weber Scannell of the Alaska Department of Fish and Game in Fairbanks. For instance, biologists had thought that the repeat-spawning Dolly Varden char stay in local ocean waters between successive Alaskan migrations. In fact, some fish tagged in Alaska by her group later visited Russia. "What an opportunity for picking up pollutants," she speculates. —*J. Raloff*

Mouse tests hint at protein's role in lupus

Complement proteins are aptly named. These molecules help antibodies round up foreign invaders in the body and assist immune cells in removing dead cells. Like so many things, however, complement isn't fully appreciated unless it's missing.

About 90 percent of people who lack the complement protein C1q, for example, have systemic lupus erythematosus, an autoimmune disease. In such diseases, the patient's immune system attacks normal tissue.

The causes of lupus remain unclear—some patients don't lack C1q, for instance—but researchers are now shedding some light on the connection between lupus and C1q deficiency.

They have found in experiments on mice that immune systems lacking the C1q protein failed to clear away cells undergoing apoptosis, or programmed cell death, says Mark J. Walport of the Imperial College School of Medicine in London. He and his colleagues report their findings in the May NATURE GENETICS.

Apoptosis weeds out badly functioning or mutated cells.

Normally, complement helps immune cells called macrophages remove such dying cells. Macrophages "move in and sweep up" before the rest of the immune system can recognize the dying cells as foreigners and mount an immune response, says John D. Mountz of the University of Alabama at Birmingham.

What happens next is mysterious, says immunologist Michael C. Carroll of Harvard Medical School in Boston. "Apoptotic cells just sort of disappear." Neighboring cells probably absorb the components of the dying cells, he suggests.

However, if apoptosis is slowed and the dying cells linger—as seems to be the case when C1q is missing—the ordeal stimulates the immune system to produce more antibodies. In lupus patients, these autoimmune reactions can cause kidney damage, skin problems, central nervous system disorders, and other symptoms.

"We wanted to see if the C1q-deficient mice developed the signs of lupus," Walport says. In fact, the researchers found, many of the mice suffered kidney damage. He suggests that a graveyard forms in the kidneys as dead cells pile up faster than the macrophages can process them.

In two experiments comparing C1q-deficient mice to normal mice, the researchers found that deficient mice died earlier and had more autoimmune antibodies, or autoantibodies, than the normal mice.

Of the 82 normal mice, none died, none had kidney damage, and 13 showed high concentrations of autoantibodies. Of the 40 C1q-deficient mice, 11 suffered kidney inflammation, including an accumulation of apoptotic cells, and 6 died of it. Among the animals that survived, 19 had high concentrations of autoantibodies. Even C1q-deficient mice that did not suffer kidney damage had more apoptotic cells in their kidneys than the normal mice did. Roughly similar results emerged from a subsequent test of 226 C1q-deficient mice and 108 controls.

"Lupus is such a complicated disorder that it's really been hard to [assess] what goes wrong," Carroll says. "It's really through these animal models that we're beginning to unravel what's happening."

He speculates that C1q plays another essential role in healthy people by removing some aggressive immune cells that might otherwise attack normal cells.

The findings support other scientists' concerns. Ultraviolet rays from sunlight, for example, can cause serious autoimmune reactions in lupus patients, says Mountz. In addition, viral infections can trigger a reaction. It could be that cells in these cases are being broken down but not cleared away promptly, Mountz says.

Lupus affects more than 1 million people in the United States, attacking women roughly nine times as frequently as men. —*N. Seppa*