Those Old Dioxin Blues

Some small fry are exquisitely sensitive models of dioxin vulnerability

By JANET RALOFF

Since the 1940s, a wildlife poisoning episode has quietly been evolving in the Great Lakes. The problem has garnered almost no public notice, even though it may have contributed to the downfall of a 10,000-ton-per-year fishery—one that today would be valued at $40 million annually.

While local communities have decried the loss of their prized lake trout—one of the top aquatic predators in the Great Lakes—scientists had until recently all but brushed off the possibility that the fish might have been poisoned. Instead, the majority had attributed the extinction of this trout in most of its range to more conventional causes, principally overfishing, predation by lampreys, and oxygen-depleted water, or eutrophication.

John G. Shedd Aquarium

Juvenile lake trout in an aquarium. Though these fish take 4 or 5 years to mature, a female doesn't become a prime spawner until age 7.

Over the past decade, however, teams of aquatic toxicologists headed by Philip M. Cook of the Environmental Protection Agency office in Duluth, Minn., and Richard E. Peterson of the University of Wisconsin-Madison have been investigating a curious coincidence. The lake trout's demise began at about the same time that large amounts of dioxins, polychlorinated biphenyls (PCBs), and their chemical kin were pouring into these lakes. What's more, concentrations of these pollutants peaked around 1970—just when fisheries managers began noticing that, although restocked lake trout had reached sexual maturity, their lake-spawned young were not surviving.

Dioxinlike toxicants sabotaged these efforts to reintroduce a self-sustaining population of the fish, Cook and Peterson argue in several studies to be published later this year. They even make the controversial claim that exposure to these pollutants before 1960 "probably contributed to the decline and virtual extinction" of this trout in Lake Ontario and perhaps in the other lakes.

There's good news, however. After more than 25 years of restocking the trout, "we are just beginning to see success," says Clifford P. Schneider, a biologist at the Cape Vincent (N.Y.) Fisheries Station. In Lake Ontario, historically the most dioxin-contaminated of the Great Lakes, "each year now, for the last 4, we've had successful reproduction that's gone beyond the fry stage."

Still, the fish may not be home free. As pollutant concentrations fall, new laboratory tests suggest that less-than-lethal amounts of dioxinlike pollutants may cause harm. Moreover, several newly identified lake contaminants appear at least as harmful to fish as the 2,3,7,8-chlorinated dioxin (TCDD). Until recently, TCDD was considered the most potent dioxinlike toxicant.

The five Great Lakes constitute the world's largest freshwater system. Until midway through this century, the silvery lake trout, which can live to 30 years and grow to more than 30 pounds, reigned as undisputed king.

"The importance of lake trout in the food web cannot be overstated," says Marc Gaden of the Great Lakes Fishery Commission in Ann Arbor, Mich. This trout's collapse "allowed alewife and smelt populations to expand, which set off a disruptive chain reaction." Today, the Great Lakes' fish population is less diverse than it was before the trout's decline, Gaden notes, and species abundances oscillate far less predictably.

Fishers and resource managers have been anxious to understand why commercial catches of lake trout fell so precipitously in Lake Ontario during the 1930s, in Huron during the early 1940s, in Lake Michigan by the end of that decade, and in Superior by the 1950s.

Overfishing and predation by sea lamprey undoubtedly played a role, believes aquatic toxicologist Michael Mac of the U.S. Geological Survey (USGS) in Reston, Va. However, no one realized that toxicants might also have contributed to the decline until Cook began probing the ecological fallout of dioxins released from Superfund sites (SN: 5/31/80, p. 340) around Love Canal in Niagara Falls, N.Y.

Because contaminated sediment and particulates would collect downstream, the EPA team designed a Lake Ontario study in 1987. By 1992, they had strong evidence that lake trout embryos exposed to dioxin could develop a lethal syndrome called blue sac.

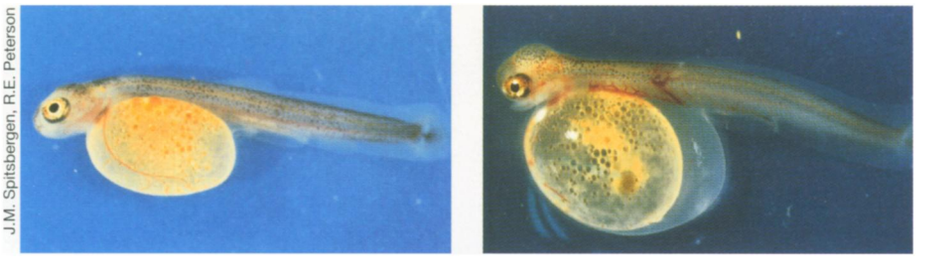
The yolk sac of healthy trout spawn gives them a rich golden hue. During the month or so after hatching, when the fry rely exclusively on that yolk for nutrients, they become vulnerable to blue sac syndrome. In this disorder, fluid leaks out of the blood vessels and into the yolk sac, which turns milky and slightly blue.

Though many conditions can cause blue sac, it has usually been linked to incubating fish eggs at too warm a temperature. In collaboration with Peterson's group, Cook's team has now confirmed that lake trout fry fall prey to blue sac syndrome if their eggs possess dioxinlike chemicals.

At concentrations of just 60 parts per trillion (ppt), TCDD kills 50 percent of lake trout fry—making this fish the most vulnerable known. The same mortality in rainbow trout requires 400 ppt.

Eleven types of PCBs exhibit dioxinlike toxicity, as do 6 other chlorine-containing dioxins and 10 of the 135 different chlorinated furans emitted by combustion (SN: 9/24/94, p. 206). Though none of these related compounds matches TCDD in potency, Peterson's group has demonstrated over the past year that their presence adds to any blue sac risk posed by TCDD.

Based on calculations of former concentrations of these dioxinlike chemicals



J.M. Spitsbergen, R.E. Peterson

Lake trout fry 4 weeks after hatching. The one on the left is normal; the one on the right exhibits blue sac syndrome from egg exposure to TCDD.

in Lake Ontario, Cook told SCIENCE NEWS, "you end up with a pretty significant period when exposures to lake trout embryos would have been 2.5 times greater than would cause 100 percent mortality."

What's more, certain brominated dioxins, furans, and biphenyls, though less persistent in the environment, are even more toxic than their chlorinated counterparts. For instance, the 2,3,7,8-brominated dioxin poses a higher risk of blue sac than TCDD does, and certain brominated biphenyls are 10 times more potent than PCBs, Peterson's group finds.

Since most of these brominated compounds come from the burning of flame-retardant materials—and the use of flame retardants is increasing—the pollutants may pose a growing risk to wild fish, Peterson and his colleagues concluded in the October 1996 TOXICOLOGY AND APPLIED PHARMACOLOGY.

In December, Peterson and Erik W. Zabel, also of Wisconsin, reported finding a component of paper mill waste (2,3,6,7-tetrachloroxanthene) that is about as potent as TCDD at inducing blue sac in trout. They worry that it may pose a risk to wild fish "even at low concentrations."

An even more arcane blue sac threat has just been identified by government scientists in Canada. This dioxin, present in a lamprey-killing compound used in the Great Lakes, contains not only a chlorine atom but also three fluorine atoms and several other chemical groups. Its discovery "shocked the pants off of us," says Mark R. Servos of Environment Canada in Burlington, Ontario.

While this pollutant does not appear to pose a major risk now, observes Cook, "it does suggest how, by doing something in the name of improving the lakes and their trout, we could actually risk aggravating the problem."

Since tough pollution control laws went into effect during the early 1970s, dioxinlike pollutants in the Great Lakes have been falling—generally to concentrations below which blue sac mortality might be expected. This has prompted many researchers to begin investigating possible sublethal effects.

Dioxinlike chemicals have long been known to retard growth, but studies in EPA's Duluth facility also indicate that sublethal exposures to TCDD can induce lethargy and a failure to avoid light—changes that might further compromise a fry's ability to evade predators, Cook notes.

Donald E. Tillitt of the USGS laboratory in Columbia, Mo., and his colleagues find that dioxins foster programmed cell death in vascular and neural tissue. One of Tillitt's students has just begun studying whether sublethal exposures might cause nerve damage that impairs behavior.

Meanwhile, John P. Giesy's team at Michigan State University in East Lansing has begun investigating immune function. George Noguchi there has preliminary evidence that certain PCBs damage infection-fighting T cells. PCB-induced suppression of immunity could have contributed to the large number of fish deaths from bacterial kidney disease in 1989, Giesy now believes.

"The real shocker from Peterson and Cook's work is that by 1940, levels of 2,3,7,8,-TCDD in Lake Ontario may have been so high that none of the lake trout were hatching," says Michael Gilbertson of the International Joint Commission in Windsor, Ontario.

Many others, like Mac and Giesy, argue that there are not and probably never will be sufficient data to indict pollutants as a major cause of the lake trout's extinction. Still, they share Cook and Peterson's concern that sublethal effects of chlorinated and brominated pollutants may continue to compromise the health of Great Lakes fish—and perhaps the wildlife and people that eat them.

Fish: Dioxins' most primitive victims?

Pollutants are defined as dioxinlike if they bind to the Ah receptor in cells, a protein that enables these pollutants to turn genes on or off. Only recognized in mammals 11 years ago, this receptor was first identified in a fish in 1988. Since then, researchers have been looking to see how far down the evolutionary ladder it—and vulnerability to dioxins—goes.

"We looked at fish and whales, mussels and lobsters," notes Mark E. Hahn of the Woods Hole (Mass.) Oceanographic Institution, "and cartilaginous fish—sharks—were the most primitive animals we've found it in." Hahn's group has acquired tentative evidence of something in lampreys that resembles the Ah receptor, but in this more primitive fish, he notes, the receptor doesn't appear to bind dioxinlike compounds.

The scientists' interest goes beyond the aquatic environment, however. These fish may offer a useful model of dioxinlike action in higher animals.

In a recent study conducted with colleagues at Cornell University, Richard E. Peterson of the University of Wisconsin-Madison found that dioxinlike chemicals target the cardiovascular system—as they do in mammals.

Using zebra fish, which have transparent embryos, they showed that the pollutants not only slow blood flow in the trunk veins feeding the head and gills, but also slow the heart's rate. Overall, Peterson observes, "there appears to be a pruning back of these blood vessels," which may account for the head malformations that often accompany blue sac syndrome. Dioxinlike compounds also appear to impair the fry's ability to maintain vessels once they form, which may explain why blood vessels become leaky in blue sac, he says.

Clues to this vascular vulnerability come from studies by Donald E. Tillitt of the U.S. Geological Survey and Mark Hannink of the University of Missouri, both in Columbia.

Cells lining the blood vessels of TCDD-exposed fry undergo inappropriate apoptosis, or programmed cell death, they found. Because oxidants can trigger apoptosis, the researchers gave antioxidants to fry that had received otherwise lethal doses of dioxin. The treatment "rescued nearly half of the fish from death," Tillitt says.

The presence of oxidants may trace to dioxin's ability to turn on genes that ratchet up the production of detoxifying enzymes—particularly in cells lining blood vessels—suspect biochemists John J. Stegeman and Jennifer J. Schlezinger of Woods Hole. When the enzymes bind to certain dioxinlike PCBs, they initiate a process that releases oxidative compounds and does not respond to normal controls. This overproduction of oxidants can damage the vessels.

Because there's no reason to suspect that this effect occurs only in fish, Stegeman says, "we're also looking for it in birds, reptiles, and mammals—including humans." —*J.R.*