

Dioxins Dominate Denver Gathering of Toxicologists

More than 2500 environmental scientists met in Denver from 30 October to 3 November for the 15th annual meeting of the Society of Environmental Toxicology and Chemistry. The theme—how to interject science into regulatory decisions involving environmental pollutants—figured prominently in many presentations, including three discussed below. One examines the possible role of dioxin and other toxicants in a mass die-off of seals in northern Europe, another looks at how those compounds may pose a continuing threat to fish in the Great Lakes, and a third looks at ways to reduce the amount of dioxin and other pollutants produced in the paper-bleaching process.

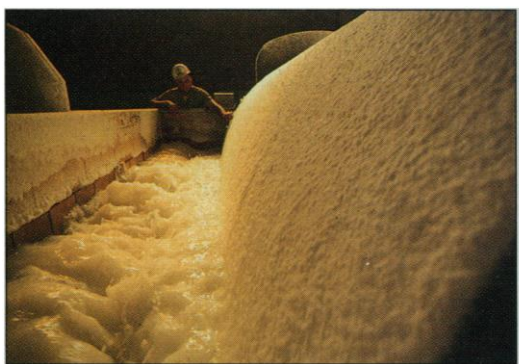
A Paper Solution

Paper isn't the only thing paper mills produce. Some also disgorge measurable quantities of dioxin into local streams as a byproduct. The dioxin stems from chlorine, used as a bleaching agent to turn the paper acceptably white. But new data presented at the meeting suggest that paper mills can sharply reduce their release of dioxin and other toxic chemicals by substituting chlorine dioxide for chlorine.

These findings reinforce the views of officials at the Environmental Protection Agency (EPA), which has waged a 3-year effort to characterize the health risks of dioxin. "We need to take prudent measures to protect the public from dioxin," says senior EPA official Lynn Goldman, adding that exposure to even minute levels of the compound could be dangerous. Fleeting exposure to mere nanograms of dioxin, for example, is enough to kill immature fish, says University of Wisconsin toxicologist Richard Peterson.

The estimated 110 grams of dioxin and related compounds that U.S. paper mills release each year into rivers and streams is only a fraction of the amount spewed into the air by incinerators, but scientists are still concerned about dioxin's effects on fish. "Paper mills need to reduce their dioxin releases to zero," says Peter DeFur, a toxicologist with the Environmental Defense Fund. And that's exactly what the paper industry is trying to do, says Barry Polsky, a spokesperson for the American Forest and Paper Association. Paper mills began switching voluntarily to chlorine dioxide in 1988, he says, and the industry has promised to reduce dioxin levels to below detection limits by 1996.

New data presented in Denver indicate that this switch offers an effective way to reduce dioxin emissions. A group of prominent environmental scientists, including toxicologist Keith Solomon of the University of Guelph in Ontario, Canada, and chemist



Clean stream. Paper mills are testing bleaching agents that do not produce dioxins.

Robert Huggett, now EPA's research chief, found that using chlorine dioxide as the sole bleaching agent brings dioxin emissions below current detection limits. The group also found that using chlorine dioxide dramatically cuts production of another class of compounds—phenols containing two or more chlorine atoms—that are harmful to fish.

Although reducing dioxins and phenols is a big step forward, in itself it's not enough to protect fish populations living downstream from a paper mill, Solomon says. Other steps in processing the raw material—such as dissolving lignin by cooking wood pulp in a soup of sulfides—generate potentially dangerous chemical byproducts, he notes. "That's a whole other area that needs to be investigated," he says.

Fatal Mix for Marine Mammals

Toxic pollutants dumped into the world's oceans may be taking a lethal toll on marine mammals. New evidence presented at the meeting suggests that polychlorinated biphenyls (PCBs), dioxins, and other organochlorine chemicals may have abetted a virus that killed 20,000 harbor seals in the North and Baltic seas in 1988.

Findings from Dutch researchers at four institutes* show the first evidence of immunotoxicity of chemicals in a wild animal

population. The results could play a role in a pending lawsuit in which the U.S. government will try to prove that several companies dumped pollutants into the Pacific Ocean off the California coast, possibly harming marine mammal populations.

After masses of seal carcasses began washing ashore in northwestern Europe in 1988, marine virologists suspected an unknown virus. That suspicion was borne out in 1989, when biologist Albert Osterhaus of Erasmus University in Rotterdam identified the pathogen as phocine distemper virus (PDV). PDV is a relative of canine distemper virus and a potent suppressor of the immune system. But several new lines of work suggest the virus wasn't working alone and that the pollutants exacerbate its effects.

In the follow-up study led by Osterhaus, the Dutch scientists found that the natural killer cells of 11 captive harbor seals fed a dioxin- and PCB-rich diet of Baltic herring were 25% to 50% less active than the killer cells of a control group fed relatively uncontaminated herring from the Atlantic Ocean. These cells kill tumor cells but are under different genetic control from immune cells. At the same time, activity of one type of immune cell—T cells—was also suppressed in the Baltic-fed seals. "There's no question there's a significant difference in the immune function in the two groups," says Robert DeLong, a field biologist at the National Marine Mammal Laboratory in Seattle.

Those studies were done in the test tube, but the Dutch researchers found even stronger evidence for a link between the pollutants and the seal deaths in a study of live animals, in which seals were immunized with egg albumin, a protein that their immune systems had never seen before. Ten days later, after another injection of egg albumin, the Baltic-fed seals mounted an immune reaction—swelling at the injection site—half as large as that of the control group. In addition, the amount of pollutants in the seal blubber varied inversely with the amount of swelling that occurred. "This hypothetically means that the seals would be more sensitive to a viral infection if it comes along," says biologist Peter Ross, a doctoral student working with Osterhaus.

A further confirmation that a link exists would be to challenge the Baltic-fed seals with the virus, but that's not in the cards. The Netherlands bars such tests on marine mammals, says Ross, and the researchers are opposed to conducting such work. Regardless, the definitive story behind the mass die-off may never be known: DeLong says re-

*Seal Rehabilitation and Research Centre, Pieterburen; National Institute of Public Health and Environmental Protection, Bilthoven; Erasmus University, Rotterdam; and DLO Institute for Forestry and Nature Research, Den Burg.

searchers lack good data on the levels of organochlorines in seals that died and on those that survived the PDV outbreak.

Something Fishy in Great Lakes

Environmental scientists applauded the news last May that several toxic pollutants in Great Lakes water had sunk to their lowest levels in two decades. But one finding left them puzzled: Many species of lake fish are just as tainted by pollutants—including PCBs and DDT—as they were several years ago. How can levels of these damaging compounds remain high in the fish when they're falling in the lakes?

New evidence presented in Denver suggests that invaders are to blame. Not humans, but two organisms introduced into the Great Lakes within the past 15 years. These two, the zebra mussel and a zooplankton known as BC (*Bythotrephes cederstroenii*),

seem to have been sucking organochlorine pollutants out of the water and concentrating them in the food chain. The bad news for humans is that the two species are favorite snacks for little fish that are consumed by the Coho salmon, another non-native species that is fished commercially and for sport.

The levels of DDT and PCBs have been declining since the U.S. government banned the compounds in the 1970s. PCB levels in Lake Superior, for example, fell from about 2 nanograms per liter in 1978 to less than 0.5 nanogram per liter in 1992. But David DeVault, an aquatic biologist with the U.S. Environmental Protection Agency (EPA) in Chicago, David Anderson of EPA, and Robert Hesselberg of the National Biological Survey found that levels of PCBs and DDT in lake trout, walleye, and Coho salmon have held steady in some Great Lakes regions—and even gone up slightly over the past few years in others.

One probable culprit is BC, a species of zooplankton brought to the Great Lakes in the ballast of Eurasian ships in the late 1970s or early 1980s. BCs, which contain elevated levels of organochlorine pollutants drawn from the water, are eaten by another exotic species, the alewife fish. Great Lakes Coho salmon feed almost exclusively on alewife, says DeVault.

Ohio State University scientists who presented findings at the meeting also put the blame for the high toxicity in fish on zebra mussels, but through a different mechanism. The researchers believe feces from the pollutant-laden mussels (which are so numerous in Lake Erie that the population can filter the lake's entire volume in one week) are consumed by invertebrates living in lake sediment. The invertebrates are then eaten by fish, which leads to the buildup of the pollutants in predators higher up the food chain.

—Richard Stone

PALEONTOLOGY

Crowding Innovation Out of Evolution

Life in the sea, with the exception of a brief creative frenzy half a billion years ago, has been in a rut. And scientists wonder why. About 530 million years ago, during the Cambrian Period, after a long period in which animals were essentially jellyfish or worms, marine animal life exploded into a variety of fundamentally new body types. Arthropods turned up inside external skeletons, mollusks put on their calcareous shells, and seven other new and different body plans appeared; an additional one showed up shortly thereafter. But since then, nothing—at least in terms of basic body types, which form the basis of the top-level classification of the animal kingdom called phyla.

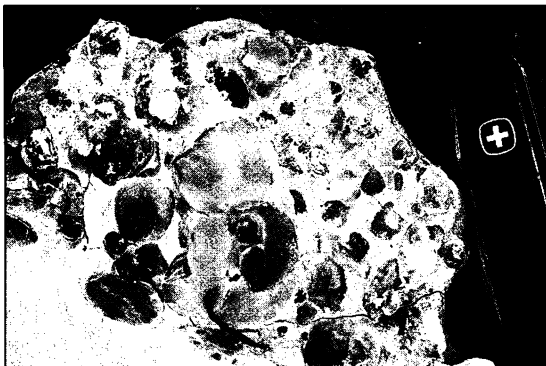
A new look at the worst of times in the history of life suggests there may not have been any more room for invention. The analysis, presented last month at the Geological Society of America (GSA) annual meeting in Seattle, lends support to the idea that once evolution fills the world with sufficient variety, further innovation may be for naught.

There are only so many ways marine animals can feed themselves—preying on others or scavenging debris, for example. And there are only so many places to do it—on the sea floor, beneath it, or some distance above it. When all the nooks and crannies of this “ecospace” are filled, latecomers never get a foot in the door.

Paleontologists David Bottjer of the University of Southern California, Jennifer Schubert of the University of Miami, and Mary Droser of the University of California,

Riverside, tested this theory by zeroing in on a period when ecospace should have been emptier than at any time since the Cambrian: the immediate aftermath of the mass extinction between the Permian and the Triassic periods 251 million years ago, when perhaps 95% of all marine species perished. Yet no new body plans appeared.

That might seem to contradict the ecological hypothesis, but, in fact, Bottjer and



Simple life. This monotonous community of bivalves typifies life forms after the Permo-Triassic extinction.

his colleagues reported at GSA that extinction's bite out of ecospace may not have been large enough to allow new phyla to appear. In part they sized up that bite by analyzing the abundance of stromatolites—pillars or reefs of blue-green algae and cemented sediment. Stromatolites thrived during the 2 billion years before the Cambrian, then took a nose dive as the first animals appeared in the fossil record. The algae pillars retreated to harsh environments like briny lagoons where the new creatures that

grazed on them couldn't follow. Through field work and literature searches, Schubert and Bottjer found that while stromatolites rebounded during the first few million years after the Permo-Triassic blight, they didn't quite rebound to their pre-Cambrian abundance. This limited return indicates there were still predators around to keep the stromatolites in check—in other words, postextinction ecospace wasn't as empty as it was when the Cambrian explosion struck.

More direct evidence that ecospace was occupied comes from animals that live on the sea floor, such as mollusks. Bottjer, Schubert, and Droser found that, after the extinction, the vertical range occupied by bottom-dwellers narrowed: They extended upward 5 centimeters and burrowed down 12 centimeters. But their range prior to and even during the Cambrian explosion was even smaller. The researchers therefore conclude that there was less latitude for evolutionary diversity after the Permo-Triassic extinction—because the mollusks and their relatives were occupying too much ecospace.

Although these findings are certainly consistent with ecological control of phylum innovation, they do not exclude the possibility that control lies elsewhere—in the genes, for example. However, the strengthened ecological hypothesis looks like the best explanation, according to the paleontologist who helped introduce both the ecological and genomic hypotheses, James Valentine of UC Berkeley. If, as recent studies suggest, control “isn't in the genome,” he says, “it must be in the ecosystem.” And if that is so, life's resilience in the face of mass extinction also ensured 500 million years of monotony.

—Richard A. Kerr