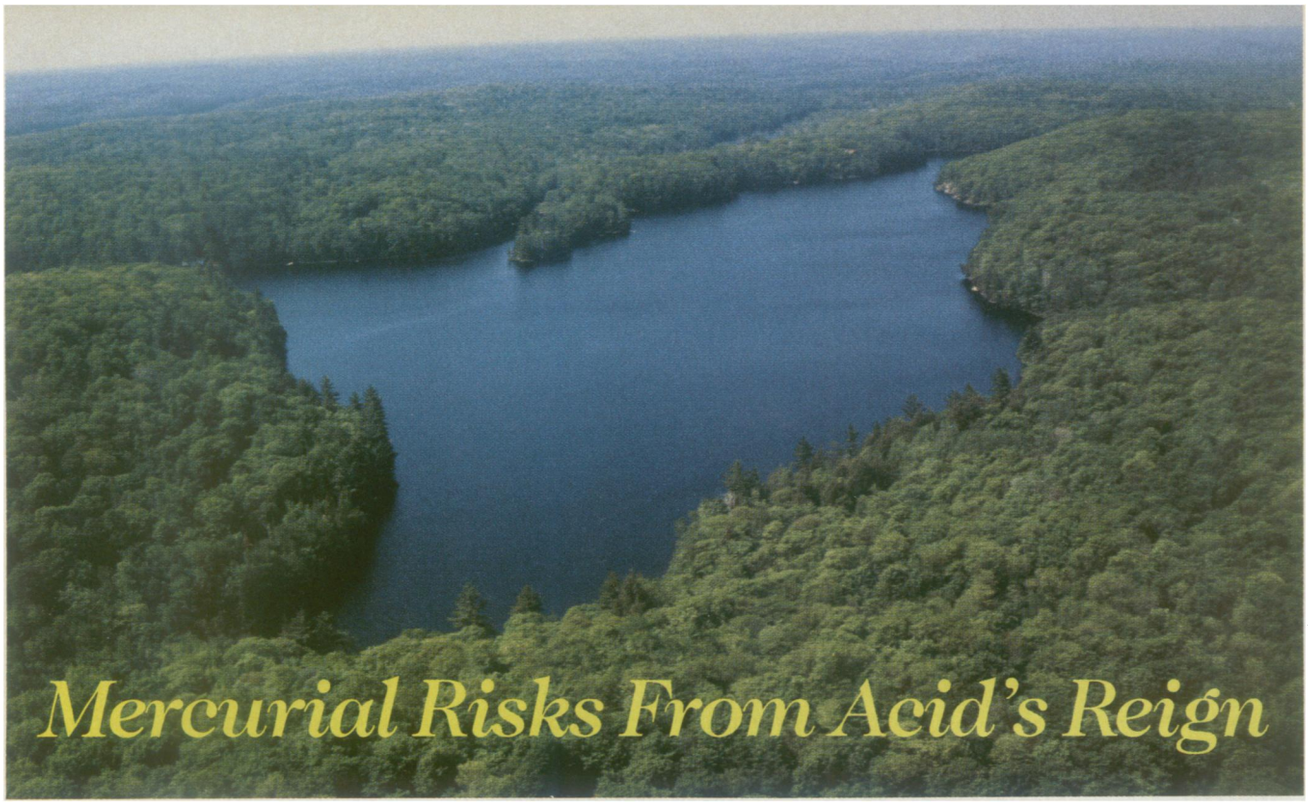


Mercurial Risks From Acid's Reign

Mierle/Dorset Research Center

Tainted fish may pose a serious human health hazard

By JANET RALOFF

Alerted by local health advisories, anglers in North America increasingly find that the catches they pull from their favorite lakes aren't fit to be fried, at least not in generous quantities. Most of those warnings focus on fish tainted with methylmercury, the form of mercury most toxic to humans.

Environmental chemists once viewed serious methylmercury contamination of fish as a threat only in waters downstream from large industrial polluters, such as pulp and paper mills, smelters or chloralkali plants. In the past decade, however, researchers have extended their lake-metals surveys to "pristine" waters in the northern woods of the United States, Canada and Scandinavia. And many of those surveys have unexpectedly turned up high levels of methylmercury in fish.

Today, one of the few features common to waters having a methylmercury problem is low pH or a steady rain of acidic deposition. Though researchers still labor to tease out precisely why fish in these waters are especially vulnerable to methylmercury contamination, the mercury-pH link remains fairly unassailable, according to James G. Wiener, a research biologist with the Fish and Wildlife Service's National Fisheries Contaminant Research Center in La Crosse, Wis.

Scientists have a better understanding of where the mercury originates: Some is natural, and some enters as fallout from distant air polluters – largely incinerators, smelters and coal-fired power plants.

Regardless of the source, the hazard remains the same. Adults eating large amounts of methylmercury-contaminated fish can suffer irreversible nerve damage, starting with a chronic numbness or tingling around the mouth or in the arms and legs. Children exposed *in utero* to even low doses of mercury can develop a range of more serious problems – from psychomotor retardation (including delays in speech or walking) to birth defects involving severe brain damage.

Because of the special vulnerability of children and developing fetuses, state health advisories on the consumption of local fish typically emphasize the risk to pregnant women and youngsters. But methylmercury also threatens adults, especially Native Americans living on subsistence diets in which protein comes mostly from local fish or from game animals that eat those fish.

"No known instances of mercury poisoning among humans have so far been reported from eating fish caught in lakes that do not receive a known discharge of the metal [in industrial wastes]," states a report issued last August by the Electric Power Research Institute (EPRI) in Palo Alto, Calif.

But the risk is there.

Chub Lake in south-central Ontario is one of many apparently pristine waters found to harbor fish with high methylmercury levels.

What is methylmercury?

A highly dilute vapor of inorganic mercury clouds much of the developed world's landscape. Though this gaseous metal is toxic to a wide range of animals, the quantities typically present in air – and washing out in rain – are usually too small to present a hazard. However, once this metal enters water, it becomes more susceptible to a range of transformations, including methylation – the attachment of one carbon and three hydrogen atoms.

Methylation, which chemically bonds a carbon atom to the mercury atom, creates an organic compound that can move more readily through biological systems, notes chemist Frank D'Itri of Michigan State University's Institute of Water Research in East Lansing. Because methylmercury is more fat soluble than inorganic forms of the metal, he says, it can easily cross the blood-brain barrier or placenta.

Fish are relatively unaffected by low-level methylmercury concentrations, and can thus accumulate substantial amounts in their flesh without visible effect. Other animals that eat these fish, however, risk methylmercury poisoning. And, notes Thomas W. Clarkson of the University of Rochester (N.Y.), because this pollutant is a neurological poison, species having the most advanced brain development – such as humans – tend to be most vulnerable to its effects.

In epidemiologic studies of residential communities poisoned by industrial mercury discharges, Thomas W. Clarkson of the University of Rochester (N.Y.) and others have detected a risk of fetal brain damage among pregnant women receiving daily exposures of 600 to 1,100 nanograms of mercury per kilogram of body weight. FDA diet surveys indicate that the average American adult consumes only 50 nanograms of mercury per kilogram of body weight daily. Clarkson points out, however, that studies conducted in communities that depend on fish for their dietary protein (including ones in Canada and Sweden) have identified individuals whose diets apparently provide 200 times the federally permissible mercury limit for fish sold in the United States – and 23 times the amount considered "tolerable" by the World Health Organization.

Acknowledging the potential for such extreme exposures, health departments in at least 21 U.S. states and two Canadian provinces have now issued advisories on methylmercury-contaminated freshwater fish. These warnings typically list affected waters by name and estimate how much fish from each can be safely consumed each week, and by whom. For instance, New York's latest advisory recommends that infants, children under 15 and women of childbearing age not eat *any* fish from the waters listed.

Researchers who study the methylmercury problem point to combustion pollutants in the air as the primary source of mercury in most highly contaminated lake fish, says Greg Mierle, a biophysical ecologist at the Dorset (Ontario) Research Center.

Yet as recently as a decade ago, scientists had all but discounted air pollution as an important source of the metal.

Why the turnabout? The few tests conducted in the early 1980s to measure mercury in rainwater turned up only scant amounts, Mierle recalls. Because mercury contamination plagued so many lakes – including ones in remote, nonindustrial regions – some researchers reasoned that the metal must have come from natural geologic deposits.

"The problem," Mierle says, "is that the analytical techniques used in the early '80s were not very good." That realization led Mierle and his co-workers to spend several years improving mercury assay methods. The Dorset team then set about measuring all of the mercury entering one Ontario lake, as the metal trickled in with rain and with drainage from surrounding lands.

"And somewhat to our surprise," Mierle says, "it turned out that the direct deposition from rain accounted for about half the mercury coming into the lake."

He adds that these data, published in the September 1990 ENVIRONMENTAL TOXICOLOGY AND CHEMISTRY (ET&C), also indicate that "there's more than enough mercury in rain falling on the watershed [lands draining into the lake] to account for what enters the lake from runoff."

"The scary thing," Mierle says, is that the "very low levels" of mercury typically found in rain can cause such dramatic contamination. Industrial mercury fallout, generally measured at only a few parts per trillion in rain, could add as little as 0.3 gram of mercury a year to a 25-acre lake, according to the August EPRI report. Yet this "is more than enough to account for all the mercury that we're seeing in fish and other biota," Mierle says.

And aquatic ecologists are indeed seeing a lot of it. In the mid-1970s, after dramatically reducing mercury levels in industrially contaminated waterways, the Ontario government initiated a survey of fish in seemingly pristine lakes. The study sought to establish a baseline by determining natural, background levels of the metal in nonindustrial areas. But project scientists have turned up far more than background levels in most of the 1,400 sites they have sampled thus far. Fish retrieved from 90 to 95 percent of these Ontario lakes have proved so heavily tainted with methylmercury that they have triggered health advisories, Mierle observes.

In recent years, several other jurisdictions have uncovered similarly disturbing trends. Methylmercury findings in Florida have led officials there to issue fish-consumption advisories for one-third of the state's rivers, lakes and streams. Minnesota has published consumption advisories for 285 bodies of water (more than half of those sampled), and Wisconsin has done the same for 154. Michigan has extended a blanket warning on methylmercury-tainted fish to cover all of its estimated 10,000 inland lakes.

And in Sweden, "more than 9,400 lakes ought to be blacklisted due to levels of methylmercury in fish higher than 1 milligram per kilogram," according to a report in the September 1990 ET&C by Ying-Hua Lee and Hans Hultberg of the Swedish Environmental Research Institute.

In its gaseous, elemental state – the form typically emitted by combustion sources – mercury poses little environmental hazard, says Wiener. Though elemental mercury *is* toxic, it has difficulty passing through a fish's gills and gut membranes, he explains. And when it does sneak in, fish tend to eliminate it rather quickly.

Methylmercury is another matter. Even more toxic to humans, this compound accumulates rapidly and efficiently in a fish's edible muscle. In fact, Wiener notes, data reported in the September 1990 ET&C by researchers at EPRI and four other institutions show that even though most lake-water mercury remains in inorganic forms, the organic, methylated species of this metal constitute 95 to 99 percent of what ends up in fish.

Since methylmercury itself rarely deposits directly into lakes, toxicity problems seldom develop unless microbes or chemical conditions within the lake transform elemental mercury into methylmercury (see box, p.152), notes bio-

"Late in the Day" by Taylor Oughton

Bald eagle delivers dinner to her nestlings. In some regions, such meals could carry toxic levels of methylmercury.

Health tips for hungry anglers

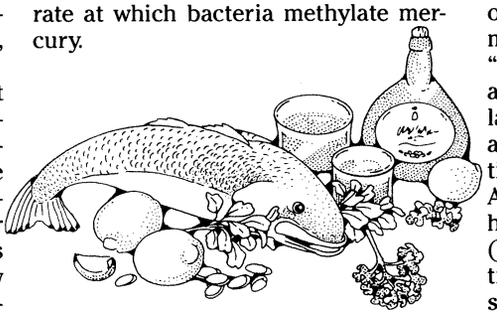
Methylmercury in lakes remains too diffuse to pose significant threats to humans and land-dwelling wildlife until it accumulates in the flesh of fish — especially the game fish that most anglers prefer. To date, researchers have detected potentially dangerous methylmercury levels in brook and brown trout, smallmouth and largemouth bass, yellow and European perch, pumpkinseed sunfish, walleye and northern pike, according to James G. Wiener of the National Fisheries Contaminant Research Center in La Crosse, Wis.

Two Canadian provinces and at least 21 U.S. states have issued fish-consumption advisories on methylmercury contamination. Yet many people ignore the warnings. University of Buffalo epidemiologist John E. Vena estimates, for instance, that 15 percent of the anglers fishing in Lake Ontario disregard New York State's recommendation to eat certain species of this lake's fish no more than once a month.

For those who choose to flout the advisories or who fish inland waters that have yet to undergo testing for methylmercury problems, several ongoing research projects offer hints that may limit potential health hazards. For example:

• Stick with small fries. Although large, spirited game fish are more fun to catch, their smaller brethren are safer to eat. Methylmercury accumulates over time, so the older fish will tend to have the highest levels. Many fish also change their diets — from zooplankton and bugs to fish — as they mature. Once a fish begins eating fish, its vulnerability to methylmercury pickup escalates dramatically, Wiener notes.

• Take your tackle box to larger, deeper lakes. John W.M. Rudd and his colleagues at the Freshwater Institute in Winnipeg are just completing a study of northern Ontario lakes that has turned up a strong correlation between lake size and methlymercury concentrations in fish. Rudd says he suspects the reason is that smaller lakes have warmer water, which can increase the rate at which bacteria methylate mercury.



• Consider going for bottom-feeding fish. Methylmercury concentrations can vary greatly among different species, even when those species share the same lake. For example, Rudd notes, suckers eating off lake-floor sediment can maintain low methylmercury levels while game fish feeding near the surface may sport dangerously high levels. The reason remains unclear, he says. Suckers might dine on less contaminated fare, or may just eliminate more of the methylmercury they consume.

• Avoid eating any fish from reservoirs less than two or three decades old. Rudd's investigation of artificially developed lakes associated with Canadian hydroelectric plants revealed that when flooded forest litter begins to decompose, mercury methylation rates "go through the roof" — even when the reservoir's pH and mercury deposition levels match those of lakes without a methylmercury problem. "The bacteria are just working harder to decompose all that forest debris," he says. And "because they're working faster, they run into more mercury atoms than normally — and they methylate them."

In Canada, Rudd says, creation of new reservoirs poses the most dangerous methylmercury threat to humans. In one northern Quebec reservoir, methylmercury levels in fish are running "about 10 parts per million — way above anything you'd ever see in an acidified lake. And those fish are healthy, will live a long while and are being eaten all the time," he says. The U.S. Food and Drug Administration forbids the sale of fish having more than 1 ppm methylmercury. (Some states have even more sensitive triggers for issuing advisories: Wisconsin's is 0.5 ppm and Minnesota's is just 0.16 ppm.)

At some point, when all the flooded forest litter has decomposed, mercury levels should return to normal, Rudd says. "But from what we've been able to determine, that will take at least several decades."

• During pregnancy, consider substituting saltwater species in recipes calling for freshwater fish. Although oceanic fish, including shark and tuna, can acquire high levels of methylmercury, they also pick up relatively high concentrations of selenium, Rudd notes. "And it's been shown that if you eat a little bit of selenium along with methylmercury, it tends to counteract methylmercury toxicity," he says. — *J. Raloff*

geochemist John W.M. Rudd of the Freshwater Institute in Winnipeg, Manitoba. Researchers are now trying to figure out how microbes and ambient water chemistry can methylate mercury so prodigiously in one lake and ignore the metal in another.

Ambient pH appears to be one of the most important factors.

For years, says Wiener, aquatic ecologists in the United States, Canada, Sweden, Finland and the Soviet Union observed a similar trend: Fish from acidic lakes tended to show substantially higher methylmercury levels than did fish from neutral or alkaline lakes. Wiener and his colleagues decided to investigate the role of pH by experimentally acidifying a portion of Little Rock Lake in northern Wisconsin.

By installing two "sea curtains," the researchers partitioned part of the lake into two nearly identical basins. At two-year intervals beginning in 1985, they have added sulfuric acid — the pH-lowering agent formed by the sulfates in acid rain — to one basin. This incrementally increased its acidity from an initial pH of 6.1 to a very inhospitable 4.7. The other basin has remained untreated.

Last year, the team published comparisons of methylmercury levels in fish caught from the basins during the first two years, when researchers maintained the treated basin at a pH of 5.6. And by the end of the second year, they report in the September 1990 ET&C, year-old perch from the acidified basin contained 16 percent more methylmercury than did perch of the same age from the untreated water. Because the adjacent basins derive nearly all their water from rain and should receive nearly identical doses of air pollution, Wiener now concludes that the experimental lake acidification somehow fostered the increased contamination in fish from the treated basin.

Rudd, who has conducted his own studies of freshwater systems, has found that any of three factors can increase mercury methylation and the subsequent contamination of fish. Those factors, he says, are waterway acidification, increased deposition of mercury into a waterway, and flooding of previously wooded land.

Since combustion pollutants can not only acidify surface waters but also enrich their mercury concentration, Rudd suspects that acid rain packs a double whammy. "In some of our experiments," he says, "if we both decreased [a water's] pH and increased its mercury concentrations, we got kind of a double effect" on methylation rates, compared with the effect of either factor alone.

Moreover, other findings suggest that independent of their pH-altering role, sulfates may foster the methylation of mercury in some waterways. Because the lakes studied in those investigations — led by Cynthia C. Gilmour at the Benedict

(Md.) Estuarine Research Laboratory — tended to have an acidic pH, Rudd says it's possible that sulfates may represent yet another additive factor contributing to the methylmercury problem in regions beset by acid rain.

It's possible that the remote waters identified as methylmercury problem sites over the last five or 10 years have actually held contaminated fish for decades. Ecologists essentially stumbled onto the discovery that these waters are far from pristine; and few of the lakes have been assayed regularly enough or long enough to reveal whether the methylmercury levels of their inhabitants are stable or increasing. But Rudd suspects that a growing acid-deposition problem is raising both the number of affected lakes and the methylmercury levels of the fish that swim in their waters.

Because mercury is so ubiquitous, and because so many factors can foster its methylation, "once you have a [methylmercury] problem it tends to stay with you" — especially in acidic waters, observes Ronald J. Sloan of New York's Department of Environmental Conservation in Albany.

But a number of researchers are investigating possible remedies for acid-prone lakes contaminated by airborne mercury.

Research undertaken by Rudd 10 years ago suggested, for instance, that adding selenium to freshwater systems would inhibit methylmercury uptake by susceptible fish. Swedish researchers are now conducting lake-scale experiments to explore the safety and effectiveness of such treatments. These new studies, says Rudd, indicate that "if you add a little selenium to a lake, it not only decreases the uptake of methylmercury by fish, but also reduces the toxicity of the mercury that has bioaccumulated in the fish."

However, concerns that the selenium

When fish eaters can't read the warnings

Humans aren't the only animals at risk of methylmercury poisoning. "You don't have to have real high levels in fish to get toxic levels in the animals that feed on them," observes Greg Mierle of the Dorset (Ontario) Research Center.

According to an August 1990 report by the Electric Power Research Institute, "some bass caught in the Florida Everglades contain up to 4.4 parts per million mercury" — enough to threaten predators such as eagles and osprey. Ronald J. Sloan of New York's Department of Environmental Conservation notes that dead loons found in Florida have had potentially lethal amounts of methylmercury in their brains.

Mierle's worries center on several mammals — such as otters and mink — that feed almost exclusively on fish at certain times of year, especially winter. He cites a study in which researchers determined that diets containing 1 part per million methylmercury were ultimately fatal to mink. "One part per million," he says, "is not an uncommon level in [Ontario] sport fish."

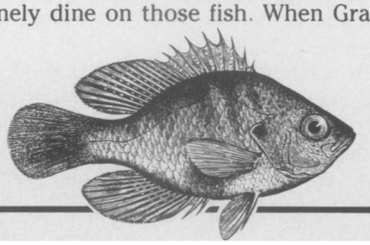
Granted, he says, such high levels tend to develop mainly in very large sport fish — larger than those usually eaten by mink and other piscivorous mammals. But Mierle suspects that if mercury levels in lakes continue to increase or environmental factors boost the methylation rate, methylmercury could become a very serious problem for these animals. Because health advisories do nothing to change the habits of wildlife, the threat of methylmercury poisoning in animals "is even more a concern to me than is the human health hazard," he says.

— J. Raloff

Hard-won appreciation for methylmercury's threat

So far, the provocative link between acidic waters and methylmercury buildup in freshwater fish has maintained a surprisingly low profile. For example, the National Acid Precipitation Assessment Program (NAPAP) didn't even mention mercury in its draft review of potential human health risks posed by acid rain, unveiled at a conference last February (SN: 2/24/90, p.119). Lester Grant of EPA, who summed up the findings of NAPAP's health-effects panel, cited five other toxic metals of concern: lead, aluminum, copper, selenium and zinc. In the end, he reported, lead appears "to be the only material of really high immediate interest."

One by one, scientists at the conference stood up to ask, "What about mercury?" At least four described studies linking a lake's pH to methylmercury accumulation in fish and in the hair of Native Americans who routinely dine on those fish. When Grant replied that he was unfamiliar with that research, William Sharpe, a water scientist from Pennsylvania State University in State College, remarked that NAPAP "hardly advances the state of science on mercury; it actually sets it back."



John W. Huckabee of the Electric Power Research Institute also challenged the panel's assessment of toxic metals. Huckabee, invited to formally critique this section of NAPAP's scientific review, concluded that human exposure to mercury "is more likely [than lead exposure] to be linked to acidic deposition." Noting that "mercury in fish increases as ambient water pH decreases, and atmospheric deposition is probably the main source of mercury for most watersheds," Huckabee advised NAPAP to investigate methylmercury's threat to people who regularly consume fish from acidic waters.

In its "integrated assessment" of the impacts of acid rain — scheduled for formal publication within the next few weeks — NAPAP now states that people eating fish from regions subject to chronic acidic precipitation (with a pH of 4.5 or less) may consume unacceptable levels of methylmercury — 70 to 200 micrograms a day. (Most people normally ingest just 1 or 2 micrograms per day.) The new report concludes that "although the number of individuals [in the United States] who might be affected by these extreme conditions is probably quite small," well below 10,000, the health impacts for these people "may be severe" and may well go undetected and untreated.

Biochemist John W.M. Rudd asserts that "the mercury people are well aware of the acidification literature and the effects of nitric and sulfuric acids on lakes." However, "the reverse is not true," he charges. "I was at an acid rain congress in Glasgow last fall, and there was no mention of mercury there."

Although methylmercury contamination is widespread and its link to acid lakes has been established by reports going back at least to the late 1970s, most acid-rain researchers "are not even aware of the problem," says Rudd, who studies water chemistry at the Freshwater Institute in Winnipeg, Manitoba.

Why not? "That," he says, "remains a mystery to me."

— J. Raloff

might adversely affect fish reproduction leave the treatment's merits still in question, says Ralph Turner, a geochemist at Oak Ridge (Tenn.) National Laboratory. Selenium can be highly toxic (SN: 7/4/87, p.8), and its therapeutic dose may be close to, if not overlapping, its toxic dose, Turner notes. "It certainly doesn't give you much margin of safety," he says.

Turner's own tactics for countering methylmercury buildup in freshwater fish focus on stimulating the activity of several detoxifying bacteria. In the water, elemental mercury can undergo a host of chemical transformations. Many of the resulting compounds render the metal ripe for methylation. Turner, who admits he's taking "a long-shot sort of approach," is participating in an EPRI-sponsored project to identify factors that might spur certain naturally occurring bacteria to reduce such oxidized compounds back to elemental mercury.

During the 1950s, classified defense activities at Oak Ridge resulted in extensive mercury contamination of a nearby creek. Yet despite the hundreds of thousands of pounds of mercury now present in this water system, "our levels of methylmercury in fish are similar to what you find in background, pristine lakes," Turner says. Part of the reason, his data suggest, "is that bacteria have intervened and reduced the production of methylmercury."

Bacteria don't methylate mercury on purpose. They apparently accomplish these transformations only inadvertently, "as a consequence of doing something else," Turner notes. For this reason, enticing them to methylate less hasn't proved easy, he says.

High levels of mercury-based compounds appear to stimulate the "reduction" activity Turner has seen in the waters around Oak Ridge. The challenge, he says, is to enhance the level of that activity through mechanisms that don't involve adding more mercury. This summer, Turner will work with researchers from EPA's microbial ecology lab in Gulf Breeze, Fla., to investigate how changing water pH or bacterial nutrient levels affects mercury's methylation rates.

In another EPRI-funded project, scientists at the University of California, Irvine, are exploring ways to spur the activity of *demethylating* bacteria. These microbes vary in their innate genetic ability to convert methylmercury back to the gaseous elemental metal. However, like athletes, demethylators can improve their skills with a training regimen that involves overcoming a succession of challenges.

Environmental scientist Betty Olson has established an Olympic-style training camp for such microbes in her lab, where the organisms exercise against mercury-contaminated river sediment from Oak Ridge in a microcosm of the natural environment.

When her organisms first arrived for training, they achieved "no measurable [mercury] demethylation," Olson recalls. Today, her recruits can transform 20 nanograms of methylmercury (per gram of sediment) back to elemental mercury daily. If they can sustain this "really exciting" rate once they return to their home waters, she calculates, it might be possible to naturally "clean up" Oak Ridge's contamination in just eight years. To test the idea, she plans to return her mighty microbes to their Oak Ridge home for micro-scale field exercises in just a few weeks.

Of course, unless the volatile, elemental mercury is subsequently trapped by activated charcoal or other filtering systems, the demethylated metal remains susceptible to remethylation, she notes. While such filtering at the lake surface is conceptually feasible, it's a long way from routine, Olson says.

For most of these scientists, the big hurdle has been winning national and international appreciation for the magnitude of the problem they're tackling. That hard-earned recognition — along with critical research funding — is coming, Rudd says. And that's fortunate, adds Turner, because there's still so much about the problem that "we just don't know." □

News of the week continued from p.151

showed a significant correlation between incidence and maternal age, and two of those — a heart defect called patent ductus arteriosus and a stomach defect called hypertrophic pyloric stenosis — actually *decreased* with increasing maternal age, the researchers report in the March 2 LANCET. The third, a congenital hip dislocation, showed increased incidence until age 30, but then decreased again in women over 30.

The results should be "very reassuring" to pregnant women over 35 who have undergone fetal testing that showed no detectable chromosomal disorders, Baird says. These women, especially if they have no other fetal risk factors such as diabetes or alcoholism, "are not at any greater risk [for birth defects] than if they were in their 20s," she told SCIENCE NEWS.

Robert J. Clayton, a birth defects specialist at the University of Texas Health Science Center at San Antonio, says the study is extremely valuable because the researchers were able to eliminate the "random drifts" in birth defect rates that confound smaller-scale population studies focusing on a single type of defect. The results, says geneticist Maureen E. Bocian of the University of California, Irvine, should extend to the general population, with the exception of isolated, inbred communities. — *T. Walker*

Drug duo uses synergy to fight AIDS virus

A pigment derived from red blood cells slows replication of the AIDS virus in cell cultures, especially when used in combination with the drug zidovudine, researchers report in the March 1 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

Zidovudine, currently the only compound approved by the FDA for clinical use against the AIDS virus (HIV), does not fight off the disease indefinitely, in part because the virus tends to develop resistance to it after six months or more of treatment, notes study coauthor Nader G. Abraham, a molecular hematologist at New York Medical College in Valhalla. The new findings may offer a way to overcome this drug resistance, he and his colleagues suggest.

The iron-rich pigment, called heme, helps hemoglobin carry oxygen through the body. A previous study by Abraham's group indicated that heme might help stave off the anemia brought on by zidovudine treatment, Abraham says.

In the new study, he and his co-workers used laboratory cultures of HIV-infected human T-lymphocytes. They found that heme alone slowed the replication of a zidovudine-sensitive strain of HIV and, when combined with zidovudine, slightly improved that drug's ability to slow viral growth. In a zidovudine-resistant strain, neither drug alone slowed viral replication. But when the researchers applied the two drugs together, the combo nearly halted replication of the otherwise resistant strain.

This "surprising" synergy, says Abraham, suggests that physicians could prescribe lower doses of zidovudine to limit its toxic side effects on bone marrow cells, while also using heme to help slow the disease's progression. Although the FDA has not yet approved heme for the treatment of AIDS, U.S. physicians do use it to treat several metabolic disorders, leaving open the possibility of prescribing the drug for some patients with AIDS. "AIDS patients now can start taking heme at a small dose," Abraham asserts.

Howard Z. Streicher of the National Cancer Institute in Bethesda, Md., notes that "myriad other drugs" have slowed HIV growth in cell cultures. Scientists still must prove that heme fights HIV in people and determine the best dosages for treatment, he says.

Nevertheless, the new study shows promise, Streicher adds. He's especially intrigued by the finding that the two drugs somehow conspire to stop the proliferation of a zidovudine-resistant HIV strain. — *W. Gibbons*