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." □

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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*