# Tiny earthquakes tamed in the laboratory

In the heart of an earthquake, action moves at a furious pace. Fractures spread through rock at more than 7,000 kilometers per hour, literally faster than a speeding bullet. That lightning motion has long hampered scientists who model earthquakes in the laboratory. But a team of U.S. and Soviet researchers has slowed the clock on the cracking process, providing new insight into the way rocks fracture.

David A. Lockner of the U.S. Geological Survey in Menlo Park, Calif., and his colleagues developed a system that retards crack growth within a rock sample, prolonging for minutes or hours an event that normally takes less than a millisecond. In one experiment, says Lockner, "We went almost a day. I went home and went to bed and let it keep going."

The researchers studied cylinders of granite a little larger than beer bottles. Pressure exerted on the end of the cylinder by a metal ram causes the granite to fracture.

Lockner's group altered the standard experimental procedure by mounting six sensors on the cylinder to listen for the ultrasonic "sound" of microcracks that develop as the granite breaks. By feeding the acoustic information into the system that pushes on the granite, the researchers can control the rate of fracturing: If the rock starts cracking too fast, the apparatus quickly compensates by easing off the pressure to slow down the breaking process.

The sensors also serve as a miniature version of the seismometer networks used to locate earthquakes and faults. A computer determines the position of the microcracks by analyzing the time it takes the ultrasonic waves to reach each sensor. This allows the researchers to monitor action inside the cylinder and provides an unprecedented image of the way fractures grow, Lockner says.

The first microcracks develop in an unorganized fashion throughout the granite sample as it absorbs stress from the advancing ram, he and his co-workers report in the March 7 NATURE. But at some point, the action concentrates in one spot as the microcracks coalesce to form a virtual fracture. That crack expands until it cleaves the cylinder.

Among the surprises to come out of the experiment was the finding that as the fracture advances through rock, it leaves a quiet zone in its wake. Scientists see the same activity in real earthquakes but had not observed it in lab experiments. Because rock samples in the lab are so small, researchers had presumed it impossible to catch this type of behavior in experiments, says Lockner.

While scientists in Japan have previously succeeded in slowing crack growth, Lockner's group is the first to both retard fracturing and use acoustic emissions to track the developing crack, says Teng-fong Wong of the State University of New York at Stony Brook. This technique will improve mathematical descriptions of the way rocks fracture, aiding efforts to study and predict earthquakes, Wong says. — *R. Monastersky*

# Intimate chemistry of a symbiotic odd couple

Symbiosis, like a happy marriage between seemingly mismatched lovers, succeeds through a subtle chemistry. Each partner benefits, even though they may differ so radically that one wonders how they ever communicate.

By eavesdropping on a chemical conversation between one symbiotic couple — the soybean plant and its bacterial sidekick — two biochemists have discovered a surprisingly intimate collaboration.

In the March 8 SCIENCE, Indu Sangwan and Mark R. O'Brian from the State University of New York at Buffalo report that the soybean and the bacteria seem to share in the making of a vital molecule called heme. "It's the only example I know of where a higher organism supplies an intermediate product in a pathway of another organism," says biochemist Winston J. Brill, who has studied these bacteria and who now runs a scientific consulting firm in Madison, Wis.

Soybean plants, like many other legumes, thrive in poor soils because they link up with bacteria that convert air's nitrogen into a form the plant can use. In return, the plant encases the nitrogen-fixing microbes within pea-sized nodules along its roots and keeps them supplied with nutrients.

To fix nitrogen, the bacteria need lots of energy and oxygen — and, consequently, lots of heme. They use the molecule to make energy-generating proteins called cytochromes. Nodules hold a rich supply of heme, which helps transport oxygen and tints the nodule interior red.

Scientists have long wondered where all this heme originates. Does it come from the plant, the bacteria, or both? The nodules seem to contain heme only when bacteria are also present, suggesting the plant relies on supplies from its microbial partners. To find out more about the bacteria's role, O'Brian and Sangwan mixed a genetically altered strain of the nitrogen-fixing *Bradyrhizobium japonicum* with newly germinated soybean seedlings. The mutant microbes could not make a chemical called ALA (delta-aminolevulinic acid), the initial precursor for heme.

The researchers found that the bacteria could not make heme on their own, but heme was still produced in the nodules. "What it hints at is that both the plant and the bacteria play a role in making the heme," says Brill.

"We'd love to know if this is what happens normally," adds Mary Lou Guerinot, a molecular geneticist at Dartmouth College in Hanover, N.H., who engineered the mutant strain in 1986 and proposed that soybean might "rescue" heme-deficient bacteria.

O'Brian thinks that even unaltered *B. japonicum* may rely on its plant partner for some ALA and may somehow prod the plant into producing more of it. For example, he and Sangwan found that the bacteria produce about the same quantity of ALA whether or not they have linked up with a plant, but the amount of *heme* increases when the bacteria live symbiotically. In soybeans with bacterial collaborators, "the ability to make ALA is almost 10 times as good as in a plant that has not seen any bacteria," O'Brian explains. "The bacteroid [symbiotic bacterium] seems to be telling the plant to turn up the [ALA-making] activity."

— *E. Pennisi*

# Most birth defects don't rise with age

Many women who postpone childbearing until their late 30s wonder whether their age increases the odds of having a child with a birth defect. For certain chromosomal disorders such as Down's syndrome, the unfortunate answer is yes. But a study of birth defects that result from unknown causes — representing more than three-quarters of all congenital defects — offers good news for thirty-something women.

Researchers at the University of British Columbia in Vancouver have completed what they call the first rigorous analysis of whether nonchromosomal birth defects increase with maternal age. The team, led by Patricia A. Baird, obtained records for the more than 500,000 live births occurring in British Columbia between 1966 and 1981. These records, they say, provide reliable data on maternal age and defects observed at birth. In order to include congenital defects not diagnosed at birth, the researchers tracked each child for up to seven years. In all, they identified roughly 27,000 children with birth defects of unknown cause.

In analyzing data on women who gave birth between their early teens and late 40s, Baird and her colleagues found no association between birth defect rates and advancing maternal age. Of the 43 types of birth defects studied, only three

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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*