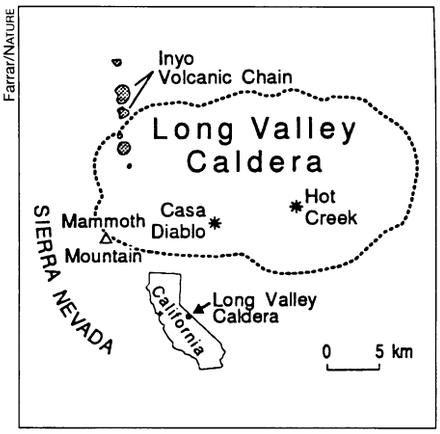
Smothered pine trees reveal unseen killer

Nobody could understand why trees were dying in California's Inyo National Forest. When stands of lodgepole pine, hemlock and Western white pine started turning a pallid gray in 1990, rangers first focused on the obvious suspects: disease, pests, and the ongoing drought. But none of these factors could explain the dead zones growing in cancerous patches around Mammoth Mountain, a restless volcano in the eastern Sierra Nevada.

Another curious fact: Since 1990, several people had experienced bouts of dizziness, nausea, and even convulsions after entering small cabins and other confined spaces on the mountain.

Geologist Christopher D. Farrar solved the double mystery by measuring gases in the soil around Mammoth Mountain over the last year. The soil beneath the dead trees contains toxic amounts of carbon dioxide gas, which is seeping out of faults on the side of the volcano, Farrar and his colleagues report in the Aug. 24 NATURE.

Carbon dioxide made up an average of 30 to 90 percent of all the gases in soil beneath the 30 hectare region of dead trees, whereas under healthy trees that proportion did not exceed 1.5 percent, says Farrar, a researcher with the U.S. Geological Survey in Carnelian Bay, Calif. By analyzing the ratio of different carbon isotopes, the researchers established that the gas had bubbled out of magma within the volcano.



Mammoth Mountain and the Long Valley Caldera.

The elevated carbon dioxide abundance can also explain why people suffered strange symptoms when entering confined cabins, says Farrar. As the gas seeps out of the soil, it gets trapped in any unventilated structure. Farrar measured carbon dioxide concentrations of 25 percent in a small cabin and 89 percent in an underground utility vault—both amounts that could quickly kill a person.

He also measured concentrations above 1 percent in campsite lavatories and small tents—levels far from lethal but still in excess of U.S. health standards for occupational safety. The Forest Service closed a popular campsite near Horseshoe Lake this summer because of the elevated gas levels and also because of the threat posed by standing dead trees.

The campsite will reopen in a few weeks, says Thomas Heller of the Mammoth Ranger Station in Mammoth Lakes, Calif. Mammoth Mountain, a popular ski and summer recreation spot, attracts 3.5 million visitors a year.

The carbon dioxide provides a reminder that hot magma sits beneath Mammoth Mountain, comments volcanologist Stanley N. Williams of Arizona State University in Tempe. The mountain could experience repeats of the small steam explosions that last blasted out craters roughly 500 years ago.

"Seeing a large amount of carbon dioxide coming out in a geologically sudden event gives us a reason to at least start paying more attention to the so-called dormant volcano and make sure there aren't other indications that are suggesting it's ready to go into a new phase of activity," says Farrar.

Volcanologists are also concerned about the potential for a truly gigantic eruption in the area. Mammoth Mountain sits on the edge of a large volcanic crater known as the Long Valley Caldera, which scientists are closely monitoring for signs of unrest. *— R. Monastersky*

Energy-starved mice hint at preemie woes

Like lightbulbs, genes turn on and off throughout an organism's life. Acting as genetic light switches are proteins known as transcription factors, many of which physically latch onto DNA to exert their influence.

Researchers from Baylor College of Medicine in Houston have now created mice bereft of one such transcription factor, a DNA-binding protein called C/EBP-alpha. These mice normally die within hours of birth, apparently starved for energy. Furthermore, the troubles confronting these "C/EBP-alpha knockout" mice, so named because of the inactivation of the transcription factor's gene, may provide a clue to the challenges facing premature infants.

Investigators have studied C/EBP-alpha for almost a decade. It belongs to a family of DNA-binding proteins, each of which activates a medley of genes. Although present in tissues as varied as lung and brain, C/EBP-alpha abounds most in the liver and adipose tissue.

Researchers quickly picked up on the latter two's functional similarity: both store energy. While fat cells accumulate in the adipose tissue, the liver stores glycogen, an energy-rich molecule formed from blood sugar. This suggested that C/EBP-alpha plays a role in energy metabolism, says M. Daniel Lane of Johns Hopkins University School of Medicine in Baltimore. Studying cultured cells, he and others added to that suspicion, demonstrating that C/EBP-alpha facilitates fat cell formation and activates a number of energy-related genes.

Since test tube experiments don't always reflect the complexity of a living organism, researchers hoping to confirm the protein's role needed to study it in an animal, explains Gretchen J. Darlington, who led the Baylor team that created the C/EBP-alpha knockout mice.

At birth, she says, these genetically engineered animals look identical to normal mice. But the knockouts soon become lethargic and die within 8 hours, Darlington and her colleagues report in the Aug. 25 SCIENCE.

Examination showed that blood-sugar concentrations in the knockout mice begin falling shortly after birth and that the livers of these animals store almost no glycogen. As a result, "Our animals get so weak they don't even feed," observes Darlington.

Through glucose injections into the mice, the Baylor team was able to provide the infant mice with enough energy to feed, but the animals remained unable to utilize nutrients or energy-rich fat from their mother's milk. As a result, even glucose-injected mice do not survive much beyond one day.

These problems arise because C/EPB-alpha is not available to turn on crucial genes, including ones relevant to energy metabolism, says Darlington. For example, the Baylor group found that the knockout mice synthesized reduced amounts of a variety of proteins, some of them needed for the production of glucose or glycogen.

C/EPB-alpha's gene lies dormant until the end of gestation, when fetuses begin storing energy for the birthing process and afterwards, Darlington notes. And that's why the C/EPB-alpha deficit doesn't kill mouse fetuses; they obtain nutrients and glucose directly from the mother.

Many characteristics of the knockout mice—such as immature lungs, low body fat and low blood sugar—resemble problems of premature infants. That may be because these babies are born before their own C/EPB-alpha genes become active, suggests Darlington. As they more fully characterize the role of C/EPB-alpha, the Baylor researchers hope the information may one day allow physicians to compensate for the transcription factor's presumed absence in premature infants. *—J. Travis*

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