California's quake deficit fades

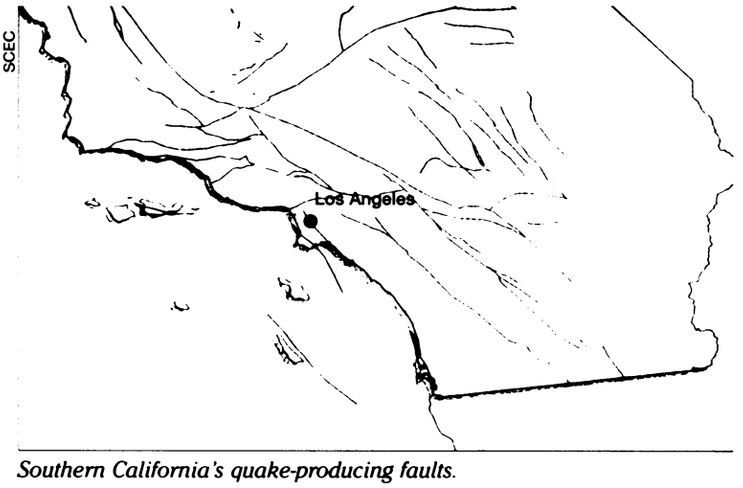
As Californians recover from El Niño-driven storms, they can take comfort from the news that the ground underfoot is less fickle than it once seemed.

In 1995, a panel of seismologists concluded that Southern California had experienced too few earthquakes since 1850 to relieve all the energy that had accumulated in the crust. The situation had created a quake deficit that would have to be paid back with larger or more frequent earthquakes in the future, they said (SN: 1/21/95, p. 37).

Now, two teams of researchers are disputing the evidence of a quake deficit in the region, and their studies are drawing positive reviews from other seismologists. "If you add all these things up, it's pretty clear now that this deficit can go away. It doesn't have to exist," comments Thomas L. Henyey, director of the Southern California Earthquake Center (SCEC), the Los Angeles–based consortium that issued the 1995 report.

Earthquake-producing stress in Southern California comes from the ongoing collision of the North American and Pacific tectonic plates—two large pieces of Earth's outer shell that grind past each other. The SCEC report estimated seismic hazard by looking at the rate of past earthquakes, the geology of faults, and ground movement. Written primarily by David D. Jackson of the University of California, Los Angeles, the report covered the southern 40 percent of the state.

The SCEC study concluded that magnitude 6 and larger earthquakes should have occurred once every 1.6 years—twice their actual rate. To relieve all the energy that had accumulated in the crust, earthquakes would eventually have to strike more frequently or smaller faults would have to join together to produce a giant quake, Jackson and his colleagues reported.



Southern California's quake-producing faults.

A reanalysis of the SCEC study has uncovered subtle flaws that combined to overestimate the amount of energy building in the crust, reported a team of scientists at a meeting of the Seismological Society of America in Boulder, Colo., earlier this week. Edward H. Field and James F. Dolan of the University of Southern California in Los Angeles and Jackson collaborated on the new study. When they constructed an improved model, no deficit emerged.

Thomas C. Hanks and Ross S. Stein of the U.S. Geological Survey in Menlo Park, Calif., questioned the SCEC study for other reasons. The report relied on a list of magnitude 6 and larger earthquakes since 1850, but Hanks and Stein argue that the list may overlook quakes from early in that period, when California's inland population was sparse. The rate of observed earthquakes since 1903 is 40 to 50 percent higher than that since 1850, which reduces the apparent deficit, they report.

While the new studies will please many Californians, they will not dispel the concerns of Los Angeles residents. Seismologists still believe that faults around metropolitan Los Angeles have a major quake deficit that will eventually lead to larger or more frequent tremors there. *—R. Monastersky*

Yeast cells point to human cancer gene

On the road to cancer, cells seem to lose the ability to count. When a normal cell divides, it painstakingly creates a pair of cells having the same number of chromosomes. Tumor cells often have extra or missing chromosomes, a condition known as aneuploidy.

Aided by clues from yeast cells, scientists have now found that specific mutations in human cancer cells may permit this chromosomal chaos to occur. The development of aneuploidy, researchers suspect, advances tumor formation by eliminating tumor suppressor genes or by increasing the number of growth-promoting genes.

Cancer investigators predict that more mutations abetting aneuploidy will emerge in the next few years and that their discovery may suggest new strategies for combating tumors.

"This is the first step through the door, which is going to be flung open wide in the near future," says Stephen J. Elledge of the Baylor College of Medicine in Houston.

That initial step was taken by scientists from the Howard Hughes Medical Institute at Johns Hopkins Medical Institutions in Baltimore and at Case Western Reserve University in Cleveland.

Some of those investigators had previously discovered that genes vital to the repair of damaged DNA in yeast have human counterparts that are mutated in some forms of cancer. Consequently, they decided to study whether several mutant strains of yeast might help explain the aneuploidy that occurs so frequently in human cancer cells.

Those yeast strains have similar trouble segregating chromosomes among dividing cells. This abnormality stems from the strains' inability to respond to problems in cell division, such as the misassembly of the spindle, a network of filaments that lets dividing cells partition their chromosomes properly.

In recent years, scientists have identified the mutated genes responsible for these yeast strains. The human copy of one such yeast gene, *BUB1*, is mutated in 2 out of 19 colorectal cancer cell lines examined, report Hopkins' Bert Vogelstein and his colleagues in the March 19 NATURE.

The investigators do not have direct proof that mutations in the human *BUB1* lead to aneuploidy, although they do have evidence that the mutant gene enables human cells to proceed with aspects of cell division after being treated with drugs that disrupt spindle assembly. Cells without the mutant gene halt the process completely, says Vogelstein.

The researchers have found a second human gene related to *BUB1*, and they are examining whether it—or human versions of the other yeast genes that cause chromosomal instability—is mutated in cancers.

Understanding how cells detect and respond to chromosomal problems while dividing and how mutations allow cancer cells to ignore those problems may eventually suggest new ways to kill tumor cells without harming healthy cells, says Vogelstein.

Other scientists note that this cancer study once again highlights how the study of simple organisms such as yeast can provide insight into human biology and illness.

"The mechanism by which aneuploidy might arise in tumor cells was never clear," says Terry L. Orr-Weaver of the Whitehead Institute for Biomedical Research in Cambridge, Mass. "Obviously, there's real value in sorting out the basic biology in model organisms first." *—J. Travis*