Quakes Pose Greater Threat to Bay Area

The U.S. Geological Survey is sending a clear message to the San Francisco Bay area: Prepare now.

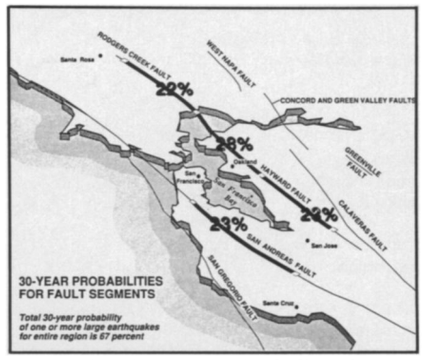
The federal agency last week released a report estimating a 67 percent chance of a major quake striking the region within the next 30 years, a significant increase over its previous estimate of 50 percent. That warning will hit the streets later this summer when USGS, in an unprecedented action, distributes 2.5 million copies of an informational magazine designed to help people plan for the likely event.

"We hope the main consequence of releasing this updated information is that it will act as another inducement for people in the Bay area, and perhaps other earthquake prone regions of the country, to take the actions now that can make a big difference in how well they fare when an earthquake does happen," says William L. Ellsworth, a seismologist with USGS in Menlo Park, Calif., and a member of the 12-person panel that drafted the new report.

According to calculations by the panel, the Bay area faces 2-to-1 odds — a 67 percent chance — that one of the major faults in the region will unleash a magnitude 7 or larger quake in the next 30 years. A 1988 USGS report gave even odds — a 50 percent chance (SN: 7/16/88, p.37).

An earthquake of this size, centered under the populated Bay area, would wreak significantly more damage than last October's Loma Prieta temblor, a magnitude 7.1 shock that originated in the rural Santa Cruz mountains. Comparing the two epicentral regions, Ellsworth says, "instead of 125,000 living in the immediate area where the ground shaking was hardest, there will be 1.2 million people."

The panel produced its estimates by reviewing the available information on major Bay area faults: the San Andreas, Hayward and Rogers Creek faults. The scientists based their calculations on the theory that earthquakes occur when stress on a fault segment builds to a critical level. The probability of a major shock in the near future therefore depends on the date of the last quake on that segment, the rate of stress build-up on the fault, and the average interval between quakes.



USGS

Probability that a magnitude 7 quake will occur by 2020 on a specific fault: San Andreas Peninsula segment (23%), Rogers Creek (22%), southern Hayward (23%) and northern Hayward (28%).

The increase in Bay Area risk reflects several new findings that have emerged in the last few years. Most importantly, geologists working north of San Pablo Bay have determined that the Rogers Creek fault caused major tremors in the past and again may be nearing its breaking point (SN: 12/16/89, p.388). In 1988, scientists knew too little about this fault to assess its earthquake risk.

Recent investigations have also determined that stress builds along the Hayward and San Andreas faults faster than previous work had suggested. Moreover, movement along the San Andreas during the Loma Prieta quake slightly increased the stress along the same fault near San Francisco.

Specialists often describe the job of forecasting earthquakes as an inexact science at best. "Everyone realizes that the actual data we have are quite meager," says seismologist Lynn Sykes of the Lamont-Doherty Geological Observatory in Palisades, N.Y.

In grading the reliability of their Bay area estimate, the panel gave it a B on an A to E scale. The group also calculated the chances of an earthquake on each of the individual fault segments in the region, but it had less confidence in these estimates, giving them marks of Cs and Ds.

USGS is preparing to inform millions of Bay area residents about the quake hazard through a magazine supplement to local newspapers. Several nonprofit groups will fund printing costs for the magazine, which will appear in English, Spanish and Chinese. — *R. Monastersky*

Cystic fibrosis gene: Too many mutants

To their growing surprise and disappointment, researchers analyzing the CF gene in cystic fibrosis patients around the world have found dozens of different genetic mutations capable of causing the debilitating respiratory disease. Although each mutation reveals something new about the poorly understood genetic basis of cystic fibrosis (CF), the added complexity has geneticists scaling back their hopes for rapid development of a useful CF-screening test.

Last year, when researchers discovered the gene mutation responsible for most CF cases, they expressed hope that just a few other mutations in the CF gene might account for the remaining cases of this relatively common inherited disease (SN: 9/2/89, p.149). Instead, they've found more than 40 CF-causing mutations, and the number may grow.

That was unexpected, says Lap-Chee Tsui, the University of Toronto geneticist who co-discovered the CF gene. Tsui described some recent progress — and several problems — along the road to understanding and curing CF this week during the Short Course in Medical and Experimental Genetics held at the Jackson Laboratory in Bar Harbor, Maine.

Tsui said analysis of 13,000 chromosomes from people with mutated CF genes reveals striking racial and geographic differences. In Denmark, for example, 90 percent of CF mutations involve the loss of an amino acid, phenylalanine, at position 508 in the 1,480-amino-acid protein. This protein is made under the direction of the CF gene. In Israel, among Ashkenazic Jews and Arabs, only 30 percent of CF mutations involve this loss. There and elsewhere, other CF-causing mutations arise with characteristic frequency, and some evidence suggests different mutations may determine the disease's severity.

This genetic variability has slowed development of a prenatal screening test for CF, which, to be useful, must detect the vast majority of CF-causing mutations. However, the newly found mutations spotlight parts of the CF gene critical to its function. That information might open the door to new drugs or genetic therapies.

Along these lines, an analysis of four new CF mutations, described by Tsui and others in the July 26 NATURE, lends support to the theory that the CF protein plays a role in shuttling chloride ions across cell membranes.

The research, led by Garry R. Cutting at the Johns Hopkins University School of Medicine in Baltimore, suggests to some that the CF protein regulates the activity of protein-lined "channels" that grant chloride ions access through cell membranes.

Tsui says researchers wishing to investigate the CF protein remain hampered by their inability to coax genetically engineered bacteria or mammalian cells to produce useful quantities of the protein. — *R. Weiss*