

Earthquakes: To live and shake in L.A.

More than 90 geologic faults thread the Los Angeles area. Many slash right through heavily populated parts of the city. A moderate shaking centered on one of these faults could cause as much or more damage than a catastrophic earthquake on the more distant but better known San Andreas fault, according to a new report from the U.S. Geological Survey (USGS).

The study is the first to present a detailed summary of recently developed methods for predicting the future effects of earthquakes and defining the hazards involved. Combined with pertinent geologic data, the techniques outlined in the report make it possible to predict in greater detail where surface cracks may appear, how strong the shaking would be from place to place and where soils would liquefy (SN: 10/12/85, p. 234).

"We've provided a framework summarizing existing knowledge," says Joseph I. Ziony, a USGS scientist in Menlo Park, Calif., and the report's editor. "It can be used almost as a textbook for evaluating earthquake hazards anywhere." Although the report focuses on the Los Angeles region, the methods apply just as well in urban areas like Salt Lake City, Seattle and Anchorage, Alaska, all of which are vulnerable to earthquake damage.

"We need to worry about all the potentially active faults in southern California," says Ziony, "not just the San Andreas." According to the report, slippage along these faults has produced more than 40 damaging earthquakes since 1800. "Potentially destructive earthquakes are inevitable in the Los Angeles region," the report states. Geologists currently estimate that the probability that a large earthquake will occur sometime during the next 30 years along the San Andreas fault is greater than 40 percent (SN: 12/24 & 31/83, p. 404).

"We have been waiting a long time for this report to come out," says Paul Flores, director of the Southern California Earthquake Preparedness Project, based in Los Angeles. Until now, "you could project potential building damage only on a regional basis," says Flores. "[The report enables us] to make clear distinctions for a particular earthquake which areas are most vulnerable and which ones will remain relatively undamaged."

Strategies for reducing future losses from earthquakes depend on the ability to predict the distribution and severity of an earthquake's effects, says Ziony. "There's been a tremendous advance both in geologic and technical knowledge of earthquakes and in using that information to reduce the hazards," he says.

The report should make it easier for geologists, structural engineers and

planners to decide where to build and the suitability of particular types of structures. Sometimes demolition of unsafe existing structures is the most effective safety measure, the study suggests.

Flores says something needs to be done to translate the report's information, barely known outside the research

community, into detailed maps and other more useful forms. "How do you get it from a well-presented piece of research into a practical format?" he asks.

The study itself notes that a comprehensive geologic data base is the most crucial prerequisite for improving evaluations of earthquake hazard potential. Even for Los Angeles, long known to be an area at risk, groundwater and soil data are still incomplete. — I. Peterson

Studies show DNA damage by long UV

Warnings about the sun's burning, aging and potentially carcinogenic rays are usually directed against overexposure to a portion of the ultraviolet (UV) spectrum termed UV-B—the medium-length UV wavelengths ranging from about 250 to 320 nanometers. For decades scientists believed longer-wave UV radiation had little biological effect. But research at Argonne (Ill.) National Laboratory is now demonstrating clearly identifiable damage to DNA—both in bacteria and in human cells—irradiated with the longer wavelengths known as UV-A. Moreover, this research is showing that the mechanism causing UV-A's damage is quite different from what occurs with exposure to shorter UV wavelengths.

The importance of UV-A-caused DNA damage has not been determined, says Argonne group leader Meyrick Peak. Though long UV produces DNA damage with only perhaps 1/1,000 the efficiency of mid-range UV-B, he points out that UV-A penetrates light-colored skin more deeply than UV-B does. "So my feeling is that the [longwave UV-A] might prove important in the formation of skin damage," he told SCIENCE NEWS.

Shorter UV wavelengths—in the B and C portions of the spectrum—will damage DNA directly. According to Peak, "The DNA of cells absorbs far UV [UV-B and -C] very strongly," transferring the photons' energy to the electrons in the DNA. This damage is powerful "and can clearly mutate cells and cause cancer very quickly—even at low doses," he explains.

By contrast, UV-A's effects on DNA are indirect. "DNA doesn't absorb near-UV [UV-A] as far as we can tell," Peak says. However, his group has identified cellular chemicals, which they call sensitizers, that do absorb the UV-A's photon energy. This absorption excites, or raises the energy level of, the sensitizers' electrons. But even they do not damage DNA, it appears. That's oxygen's role.

"We believe that these excited sensitizers can transfer energy directly to molecular oxygen," creating powerful, chemically reactive oxygen species, write Meyrick Peak and his wife, bio-

chemist Jennifer Peak, in the latest (autumn) issue of LOGOS, an Argonne publication. These reactive oxygen species quickly decay to molecular oxygen by releasing their excess energy. If they decay near DNA, they can damage it.

Their data show that when cells are irradiated in oxygen-free environments, virtually no UV-A damage occurs. However, when those same cells are irradiated in water (as they naturally would be in the body), eight different types of DNA damage will occur. And irradiation of cells in "heavy" water (where the hydrogen is substituted with deuterium) yields almost twice as much DNA damage. The reason, the Peaks believe, is that since reactive oxygen decays more slowly in heavy water, it has more time to diffuse into the vicinity of a DNA molecule.

Most recently, the Peaks have added porphyrin (a constituent of hemoglobin), bilirubin and riboflavin (vitamin B₂) to a growing list of sensitizer molecules, and identified superoxide anion (O₂⁻) as one of the reactive oxygen species. According to biologist Richard Setlow, acting director of life sciences at Brookhaven National Laboratory in Upton, N.Y., they also "have probably done the best work in trying to sort out what are the various [DNA changes] due to UV-A."

"For 60 or 70 years, it was felt that longwave UV radiation had very little if any biological effect," notes dermatologist Frederick Urbach, director of the Center for Photobiology at Temple University's Skin and Cancer Hospital in Philadelphia. In fact, he says, scientists drew the boundary between UV-A and UV-B at what they thought was the cutoff for DNA-active wavelengths. But research by the Peaks and others, using newly available lamps producing high-energy longwave UV, show that the initial UV-B cutoff of 315 to 320 nm was low, Urbach says. As a result, he expects that the boundary between UV-A and UV-B will eventually be changed to 340 nm—the point at which longer wavelengths cause almost entirely those indirect, oxygen-mediated DNA effects.

—J. Raloff