The waves are amplified as they pass beneath Los Angeles, explains Archuleta, because the city sits on a vast, sediment-filled basin. The soft sediments slow the waves as they enter, and the basin structure traps them—both factors that boost the size of the waves. The ground in some areas of Los Angeles moved at 1.4 meters per second, several times the rate expected.

If they struck an area with tall buildings, these exceptional waves could cause tremendous damage. But the simulation lacks sufficient resolution to show engineers whether the waves would hit the few sections of Los Angeles with high-rises. "It's going to take a fair amount of additional work to tell what this means for existing structures," says Heaton.

The study does not address short-period vibrations, the rapid jerking that tears small buildings apart. Some seismologists fear that the short-period waves from a San Andreas temblor would also exceed expectations.

Heaton disagrees, arguing that a giant quake outside Los Angeles is unlikely to shake the city with short-period waves much more destructive than those of last year's magnitude 6.7 Northridge shock. If big San Andreas jolts did create monster short-period waves, then the last superquake in 1857 should have devastated the city. Most of its 5,000 residents survived the tremor, however.

— R. Monastersky

U.N. to oversee methyl bromide phaseout

In Vienna last week, representatives of some 110 countries voted to strengthen the Montreal Protocol, a United Nations treaty to protect Earth's stratospheric ozone layer. On Dec. 8, industrial nations agreed to phase out their use of methyl bromide by 2010. The bromine released by this short-lived, gaseous pesticide is 50 times more destructive to ozone than chlorine is.

Previously, industrialized nations—which account for about 80 percent of methyl bromide use—had agreed only to freeze that use at 1991 levels.

"Phasing out methyl bromide offered the biggest percentage savings of future ozone loss" of any of three major treaty changes considered, explains Daniel L. Albritton of the National Oceanic and Atmospheric Administration in Boulder, Colo.

New analyses also indicate that further regulation of hydrochlorofluorocarbons (HCFCs) would offer the smallest ozone rewards, Albritton, a cochair of the U.N. scientific assessment on stratospheric ozone, told the delegates. After much debate, however, the delegates voted to accelerate by about 10 years—to 2020—the HCFC phaseout by most industrialized countries and to extend the phaseout to developing nations.

Last year, as scheduled, production of firefighting chemicals called halons came to an end. The Vienna meeting did not take action on Albritton's third proposal: prohibiting release of existing halons from unused fire extinguishers.

The United States had pushed for a 2001 phaseout of methyl bromide to coincide with its slated ban under the Clean Air Act. Environmental groups responded to the treaty change with a statement chastising the United States and other leaders for backing "an extremely weak compromise that will extend ozone depletion for several decades through continued use of methyl bromide."

The 2010 timetable "is ridiculous" and "far too long . . . for something that we are sure is contributing to ozone destruction," argues atmospheric physicist Rumen Bojkov, an adviser on global environmental issues to the secretary general of the World Meteorological Organization in Geneva. He noted, however, that most major users of methyl bromide categorically rejected earlier deadlines.

The 2010 deadline now threatens to derail the U.S. phaseout. A bill introduced by Rep. Dan Miller (R–Fla.) would delay the 2001 deadline until there is a worldwide phaseout or until cost-effective substitutes exist.

— J. Raloff

DNA manipulation goes large-scale

Geneticists have scored another victory on the playing field of the mouse genome. In what they call chromosome engineering, researchers have succeeded in deleting, inverting, and rearranging not single genes but large, selected blocks of mouse DNA.

In genetic engineering, investigators routinely pop extra genes into mice and knock out specific genes to create mice that develop without those genes' proteins. Now, with their new skills, researchers from Baylor College of Medicine and Texas A&M University, both in Houston, have robbed a mouse chromosome of 10 percent of its DNA.

"That's a mighty big chunk of DNA," marvels Kenneth Paigen, director of Jackson Laboratory in Bar Harbor, Maine, which collects mutant mice.

Chromosome engineering will speed the search for new genes, especially those that normally prevent uncontrolled proliferation of cells, says Allan Bradley, a Howard Hughes Medical Institute researcher at Baylor. The technique may also help create rodent examples of many human difficulties, since chromosomal rearrangements often cause failed pregnancies, familial diseases, and cancers.

"We're going to have quite a few different types of applications using this technology," says Mario Capecchi of the University of Utah in Salt Lake City.

Bradley and his colleagues, who describe their experiments in the Dec. 14 NATURE, use an unusual enzyme produced by a virus that infects bacteria. The enzyme, Cre recombinase, or simply Cre, recognizes short viral DNA sequences called loxP sites.

When Cre encounters two loxP sites that scientists have inserted into a mouse chromosome, the enzyme cuts out the intervening DNA (SN: 7/9/94, p.20). Depending on the orientation of the loxP sites, the enzyme then either inverts the DNA fragment and places it back in the chromosome or discards the snipped DNA, says Bradley.

Since every cell contains two copies of most chromosomes, the loxP insertions sometimes land on different copies of a particular chromosome. In those cases, Cre uses the loxP sites to define a region of one chromosome that it will cut off and attach to the other chromosome. "It puts one piece of chromosome onto another," says Capecchi.

To create mice with manipulated chromosomes, Bradley's group alters the DNA of stem cells and injects these immature cells into early-stage embryos. If the altered stem cells develop into the mouse's reproductive cells, all cells in the embryo's offspring will have the modified chromosomes.

Deleting large chunks of one chromosome does not generally kill the offspring, says Bradley, because the second copy usually contains a spare of each missing gene. The deletion does make it easier to search for tumor-suppressing genes, for example, since both copies of such genes must be deactivated before cancer results.

Bradley's group removed about one-tenth of one copy of chromosome 11 from a mouse strain. The deleted region resembles the part of human chromosome 17 that contains genes implicated in suppressing breast cancer. By mutating genes in the same region of the unaltered copy of chromosome 11 and observing whether cancer develops, investigators hope to identify the tumor-suppressor genes.

This search technique can also help identify genes whose functions are normally masked by dominant counterparts on the other copy of the chromosome. Chromosome engineering promises "to revolutionize mouse genetics," asserts Bradley.

— J. Travis