Earth Science

Northwest squeeze: Quake in the making

Plate tectonic forces are warping the Pacific Northwest in a manner that will eventually lead to a large, potentially giant earthquake there, according to scientists who have studied geodetic measurements in the region. No one knows when the future shock might hit, but its size could rival the largest the world has seen this century.

The Pacific Northwest lies in the middle of a collision zone where the small Juan de Fuca plate crashes into and dives under the larger North American plate. In other parts of the world this "subduction" process has generated huge earthquakes, and geoscientists have long wondered whether the Northwest is also prone to such massive temblors.

Some researchers discount the possibility, suggesting that the Juan de Fuca plate—moving about 4 centimeters per year—is sliding smoothly beneath the North American continent. But many geoscientists think the small plate has become stuck as it scrapes underneath North America. Such locking action would build energy until the Juan de Fuca plate suddenly bolts forward, generating a tremendous jolt possibly larger than magnitude 9.

To find out what gives deep beneath the Northwest, James C. Savage and Michael Lisowski of the U.S. Geological Survey in Menlo Park, Calif., examined measurements made by bouncing laser light off reflectors in the Olympic Mountains of Washington state to detect subtle warping in the region. They also looked for signs of movement in tide gauge records along the Washington-Oregon coast. Both sets of data indicate the region is storing strain from a locked subduction zone beneath, the researchers report in the April 5 SCIENCE.

The work supports geologic findings along the coastline suggesting the region has experienced extremely large jolts in the past (SN: 2/17/90, p.104). From that evidence, investigators have deduced that the most recent quake probably occurred about 300 years ago. While the interval between quakes runs on average about 600 years, some intervals appear much shorter.

Methane lasts longer in atmosphere

Laboratory experiments indicate that the greenhouse gas methane remains in the atmosphere about 25 percent longer than previously suspected—a finding that may clear up some nagging questions about this important player in the global warming drama.

The concentration of methane gas rises almost 1 percent per year, but scientists lack a good explanation for the increase. A problem arises when researchers tally the known sources for methane and compare those with the "sinks" that remove the gas from the atmosphere. The calculations suggest methane levels should remain constant or even decrease, despite what current measurements show.

The new research, conducted by scientists at the National Oceanic and Atmospheric Administration in Boulder, Colo., examined the speed of a chemical reaction between methane and the hydroxyl radical, the principal methane sink in the atmosphere. The experiments revealed hydroxyl reacts about 25 percent slower at atmospheric temperatures than suggested by previous, less accurate experiments. That means the average methane molecule remains in the atmosphere about 12.5 years rather than 10 years, the scientists report in the April 4 NATURE. Methane gas comes from natural wetlands as well as from such human activities as rice cultivation, raising domestic animals, biomass burning and landfilling wastes.

Because the findings suggest a weaker removal process for methane, they help resolve the discrepancy between the calculations and the observed methane increase. They also suggest that methane, with its longer lifetime, contributes more to the greenhouse effect than previously thought.

Genetics

Of mice and men: Sharing locator genes

Beginning life as a microscopic, amorphous blob, an embryo undergoes numerous divisions to form clusters of specialized cells. Some eventually become nerves; others develop into skin, muscles or blood. But even before these cells take on their destined shapes and functions, certain genes in the developing embryo lay out a master blueprint, specifying from head to tail where major sections of the body will lie. Now, researchers have discovered compelling new evidence that creatures as diverse as fruit flies and mice share a common set of genes for organizing the body's architecture.

While the new study focuses on manipulating embryonic development in the mouse, investigators say the striking similarity between this animal's genetic makeup and that of humans indicates the work may also provide a better understanding of human fetal development. Osamu Chisaka and Mario R. Capecchi of the University of Utah School of Medicine in Salt Lake City report their findings in the April 11 NATURE.

"It's the most beautiful and amazing and fantastic thing—that there is a common, unifying link between flies and humans," declares developmental biologist Brigid Hogan of Vanderbilt University Medical School in Nashville. "You can use the genetics of flies and the relevance of the mouse model to humans to start to ask very, very detailed questions about human embryonic development," she adds.

The gene that the Utah researchers studied belongs to a group called homeobox genes, which scientists discovered a decade ago in fruit flies. Each gene, depending on its location, helps determine which groups of cells will develop as a particular body segment, and where their structure may reside. Looking at fruit flies, investigators had found that genes lying closer to the head determined development of features there; those nearer the tail orchestrated posterior development. In the early 1980s, geneticists reported that disrupting one of the front-end fruit fly genes caused a body alteration worthy of a sci-fi movie: Instead of antennae, legs grew from the fly's head. Other researchers later discovered homeobox genes with identical DNA sequences in mice, humans and other vertebrates. But whether these shared genes also orchestrate development remained largely unknown.

In their new experiment, Capecchi and Chisaka used a method called gene targeting to insert into mice embryos two copies of a mutant homeobox gene, called *Hox-1.5.* Mice born with the resulting mutation had no thymus, an organ critical for regulating the immune system. The mice, which all died within 12 hours, also had abnormal parathyroid function and defective hearts and arteries—characteristics of a life-threatening human birth defect called DiGeorge syndrome.

Although the defective gene in mice appears on a different chromosome than the one implicated in about 20 percent of people with DiGeorge syndrome, *Hox-1.5* might still play a role in the human disorder, Capecchi told SCIENCE NEWS. He speculates that several genes working in concert, including *Hox-1.5,* may cause the syndrome, or that the gene may trigger the disorder by activating a gene above or below it.

"Any lingering doubts that homeobox genes are important regulators of vertebrate [development] should be dispelled" by Capecchi's study, write Hogan and her Utah colleague Christopher Wright in a commentary accompanying the NATURE paper.

Capecchi plans to disrupt several other of the 40 or so homeobox genes in mice in hopes of elucidating their function. "We want to get a feeling for what the whole complex of 40 are doing... which genes are talking to each other," he says. He also seeks to determine whether embryonic cells destined to form part of the nervous system, which interact with homeobox genes, directly cause defects—or if these cells must migrate elsewhere in the embryo to do damage.