# Why is the Pacific so big? Look down deep

When geoscientists stare at a globe, they see a puzzle. Basic physics and simple computer models suggest that Earth should have tectonic plates no more than 3,000 kilometers wide. Yet the planet somehow bypassed that limitation. The vast plate underneath the Pacific Ocean stretches 12,000 km across, and many other plates span two to three times the theoretical width.

A solution to this long-standing problem has now emerged from a massive computer model that simulates the movement of the surface plates and the underlying mantle. Hans-Peter Bunge and Mark A. Richards of the University of California, Berkeley report that characteristics of the deep mantle apparently govern the size of the plates.

"It really appears that the lower mantle is the unifying theme that makes plate tectonics work just exactly the way we observe it," says Bunge. He and Richards will publish their results in the Oct. 15 GEOPHYSICAL RESEARCH LETTERS.

Their model, which ran on a supercomputer at Los Alamos (N.M.) National Laboratory, depicts the rising and sinking currents of rock in the mantle by breaking the region into 20 million blocks. Although the mantle is solid, pressures and temperatures reach such a feverish state that the rock flows, but at the sedate pace of a few centimeters per year.

Most previous models have suggested that the 3,000-km depth of the mantle naturally sets the scale for the rock currents. Convection circulates rock up from the deep mantle and carries it a few thousand kilometers horizontally just below the surface plates before taking it back down again. The surface plates would then, in theory, match the size of these convection cells.

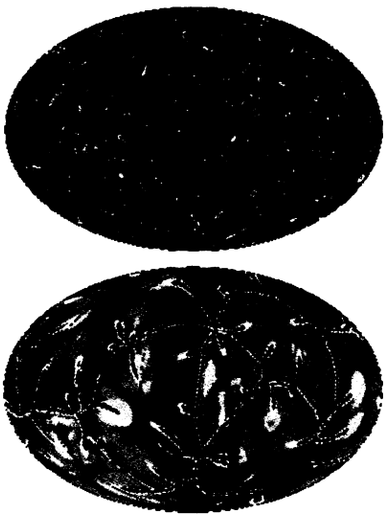
To explain the reach of the Pacific and other large plates, some scientists suggested that the plates exert a controlling influence on mantle circulation, stretching the convection cells far beyond their natural breadth. In other words, the plates determine their own size and the mantle follows.

In Bunge and Richards' model, however, the mantle retains the dominant role. Their modeling differs from previous experiments by including a more complex, realistic representation of the mantle's viscosity, or stickiness. When the scientists made the lower mantle more viscous than the upper mantle, the convection cells approached the size of Earth's cells. When they kept the viscosity uniform, the convection cells remained puny, the scientists reported in the Feb. 1 NATURE.

In their more recent experiments, Bunge and Richards gauge the importance of Earth's plates by adding simplified versions of them. Without the increase in viscosity, the model with the plates failed to produce realistic convection patterns. It showed broad convection cells only when the lower mantle was 20 times more viscous than the upper mantle.

These results suggest that the lower mantle, rather than the plates, organizes the whole system. "Plates and their size basically reflect the scale of the mantle," says Bunge. "Since the mantle convects on such a large scale, the plates are allowed to reach large sizes."

According to Richard J. O'Connell of Harvard University, Bunge and Richards are the first to simulate plate motion in a three-dimensional model of a spherical Earth. Because the simplified plates they included are unbreakable, a next step would be to include more realistic plates, which can fracture. This would provide further insight into why plates assumed the shapes and sizes they have today. — *R. Monastersky*



Bunge and Richards/GEOPHYSICAL RESEARCH LETTERS

*In a model of Earth's mantle, reds show rising rock and greens denote sinking material. When the viscosity of the mantle is uniform from top to bottom (top), the model produces a blotchy convection pattern. Increasing the lower mantle's viscosity leads to larger, more realistic convection cells (bottom).*

# Faulty gene adds the fever to a vaccine

Fever can be a patient's best friend. At elevated body temperatures, the immune system goes into overdrive and some pathogens stop reproducing. Now, researchers studying vaccines have discovered how one virus slips into cells without firing up a fever.

"Probably, we've found the first example of a virus that blocks the induction of fever and the mechanism by which it operates," says Geoffrey L. Smith, a virologist at the University of Oxford in England.

Smith and his colleague Antonio Alcamí report in the Oct. 1 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES their comparison of three vaccines that had been used against smallpox. In tests on mice, all three vaccines—each containing a weakened strain of the cowpox virus—stimulated antibody production.

Two proved relatively benign, producing only symptoms of a mild cold, but the third, known as the Copenhagen vaccine, also produced a fever.

The difference arose from a gene in the vaccines. When infected, cells release messenger proteins, or cytokines, to alert the immune system. A gene in the milder vaccines coded for a protein that latches onto a cytokine. This action prevented the cytokine from triggering an immune response that includes fever. The Copenhagen vaccine had a damaged version of the gene, which let the cytokine go undeterred. When the messenger reached the pituitary gland, the body turned up the heat.

"It's a fascinating study," says Raymond M. Welsh of the University of Massachusetts Medical Center in Worcester. "This clearly shows in this case that one protein causes the fever and one gene can suppress it."

To verify their findings, the virologists repaired the damaged gene and then inoculated mice with the mended Copenhagen vaccine. The mice responded with no rise in temperature.

"Take the gene out, get a fever," says Smith. "Put it back in, you don't." The researchers believe the vaccines would act in people just as they do in mice because both use the same cytokine.

"The study paves the way for asking fascinating questions about the evolution of viruses and the immune response," says biologist Rustom Antia of Emory University in Atlanta.

Antia suggests that future studies compare two colonies of mice, one inoculated with a genetically intact vaccine virus and the other with a defective, fever-causing version. If suppressing fever gives a virus an evolutionary advantage, he would expect the genetically undamaged strain to spread through a population more easily than the damaged one.

The study's authors warn that their results may point to a potential pitfall in plans to defang viruses through genetic engineering.

"You can't be certain that taking a gene out of a virus is going to make it less virulent," says Smith. "It might do the opposite." — *D. Vergano*