

Loper's group suggests that a layer of hot mantle material close to the core periodically becomes unstable, releasing mobile plumes of hot mantle material that rise to the surface and feed volcanoes. The researchers argue that the release of the plumes also leads to field reversals. The loss of material, they say, thins the layer, allowing more energy to escape from the core. This enables the heat engine in the core to drive the geodynamo harder, and as the flux of energy to the geodynamo increases, the reversal rate increases as well. In a rough, preliminary calculation, Loper estimates that the layer becomes unstable about every 22 Myr.

Loper's group is conducting laboratory experiments to learn more about the behavior of the lower mantle layer, which

seismologists have dubbed the D'' layer. The researchers place a layer of dyed water, representing the mobile D'' layer, under viscous corn syrup representing the colder overlying mantle. "We get what looks like episodic behavior" in the rising plumes of water, notes Loper.

Neither Loper nor Pal and Creer are the first to suggest and model links between these different periodic processes. But what is relatively new, say several observers, is that the findings of periodicity in a number of records are enjoying a resurgence of respectability. When all these hunts for periodicity resumed several years ago, says one scientist, "I thought it would be just a flash in the pan. But the idea has grown and periodicity is spreading waves throughout geologic thought." — S. Weisburd

Pacific's CO₂ levels: Cause for concern?

One of the greatest concerns associated with the world's burning of some 5 billion tons of fossil fuels annually is the large amount of carbon dioxide (CO₂) it generates. Right now, only about half of that combustion-generated CO₂ stays in the atmosphere. Much of the rest, it is generally believed, is taken up by the oceans. But new research indicates that the ocean might not remain as robust a sink for CO₂ as it has been. That would leave even more of the gas to accumulate in the atmosphere, potentially triggering a more rapid and devastating global warming from the so-called "greenhouse effect."

Researchers from the University of South Florida in St. Petersburg have been studying the calcium carbonate shells of pteropods (planktonic mollusks) in the north Pacific (SN: 12/15/84, p. 376). Not only have their shells incorporated some of the carbon that entered the water as CO₂, but the creatures are also a mechanism by which a portion of that carbon is eventually removed from upper ocean waters; as the creatures die, their shells fall toward the ocean bottom, carrying the carbon along. If a shell falls a long way before dissolving, it carries the carbon far from the surface, potentially making it easier for more CO₂ to enter, helping to reduce atmospheric CO₂ levels. This is usually the case, since high acid levels, which help dissolve the pteropod shells, are normally present only at great depths.

But Robert Byrne and his colleagues have identified regions in the north Pacific where the pteropod shells begin to dissolve at depths of only 170 meters — well within the top 10 percent of the ocean depth — and in far shallower water than generally expected. Since CO₂ is one source of water acidity, Byrne notes, the regions of shallow acidity they've identified may be an indication of higher CO₂ levels beginning to accumulate in the

surface waters.

If true, this suggests a couple of causes for concern, he says. First, the more acidic water is, the less CO₂ it will absorb. So a trend toward more acidic surface waters could spell a long-term decline in the amount of CO₂ the ocean will accept from the atmosphere. Moreover, if shell dissolving begins too high in the water column, there is a risk that the shells will be less effective at removing carbon from surface waters. That could exacerbate the acidity problem and the potential inability of the ocean surface to accept as much atmospheric CO₂. — J. Raloff

EPA suspends permit

Because a tree does not a greenhouse make, Advanced Genetic Sciences, Inc. (AGS) has at least temporarily lost its permit to conduct field tests of its genetically engineered bacteria. The Environmental Protection Agency (EPA) this week charged AGS with violating regulations when it injected "Frostban" bacteria into about 50 trees on its Oakland, Calif., rooftop (SN: 3/8/86, p. 148). The agency also charged that the company "knowingly falsified" data by describing its tests as greenhouse experiments and failing to inform EPA of cankers that developed on the bark of several trees after the injections. The EPA adds, however, that it has concluded that the bacteria are not pathogenic. The agency is levying a \$20,000 fine.

AGS has said that it believed the bacteria were always contained during the rooftop tests, first within syringes and then within the trees. According to EPA, the company plans to repeat the tests in its greenhouse in the next two months; after review, the EPA will decide whether to reinstate the permit. □

Antibody cocktail to fight bacteria

A mixture of seven human antibodies, produced by a new laboratory procedure, is proposed as an effective protectant against a deadly bacterium. The bacterium, called *Pseudomonas aeruginosa*, is the most lethal of the microorganisms that patients commonly acquire while hospitalized. Mark E. Lostrom and his colleagues at Genetic Systems Corp. in Seattle focused on this bacterium in devising a new strategy to develop prophylactic "cocktails" made of human antibodies.

The bacterium *P. aeruginosa* comes in at least 17 varieties, each having a characteristic surface molecule. The Genetic Systems scientists decided to work only with the seven varieties that are responsible for 90 percent of hospital infections, Lostrom said last week in Washington, D.C., at the American Society for Microbiology Conference on Biotechnology.

Invading bacteria expose many surface components to a host's immune system. Some of these components are shared by all bacteria of a species, whereas others provide the means of distinguishing the varieties, called serotypes. Using standard methods for producing large amounts of specific mouse antibodies (monoclonal antibodies), Lostrom and his colleagues employed a laboratory model of an immune system attack directed at each of the different surface components of *P. aeruginosa*. The serotype-specific antibodies, only one of which attacks a given bacterium, were the most efficient at triggering destruction of the bacteria. The antibodies against components found on all the serotypes were not effective.

The serotype-specific antibodies were also effective at protecting live mice against a bacterial attack. The mice given antibody prophylactically, before a large dose of the bacterium of the same serotype, all survived and showed few symptoms. Mice not given the antibody were killed in 1 to 3 days by the same dose of bacteria.

Antibodies against one shared bacterial product did confer some protection. *P. aeruginosa* makes a potent toxin; a monoclonal antibody that binds this toxin protected mice against a toxin dose that kills unprotected mice within a day. Lostrom says the toxin's role in human disease is unclear.

Lostrom proposes that a preparation of seven or eight monoclonal antibodies — one for each of the seven important serotypes and perhaps one for the toxin — should protect patients against most hospital infections.

Because administration of mouse antibodies to patients may create an undesirable immune response, the next