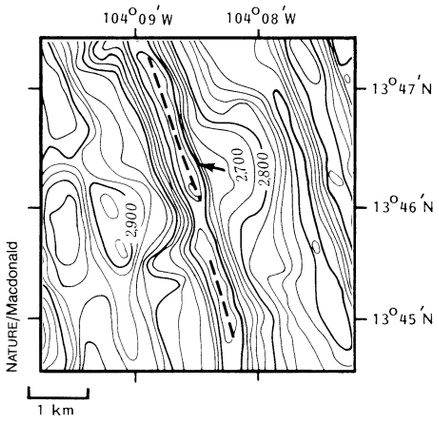
What's SNOO at ocean ridges?

Just as the scars and wrinkles on a person's skin can tell the story of a life, the faults and topography of the seafloor may reveal a history of the ocean crust as it is churned out at mid-oceanic ridges. In particular, scientists have been studying the geometry of ocean ridges for clues to how the process of seafloor spreading works.

"We've known for a long time in a general way what happens [at ridge crests]," says Rodey Batiza, a researcher at the National Science Foundation in Washington, D.C. "Now we have the kinds of tools in marine geology and geophysics to take a closer look at ridge geometry so that we can ask more detailed questions about exactly how it works."

With this goal in mind, Batiza and Steven H. Margolis of Washington University in St. Louis have studied what they say are among the smallest and least understood ridge structures — called small nonoverlapping offsets, or SNOOs — that have been mapped along the East Pacific Rise in the last few years. In the April 3 NATURE, Batiza and Margolis outline a simple model explaining how these SNOOs — regions in which one segment of the ridge crest is displaced, or offset, from an adjacent segment by several hundred meters — are formed.



In this depth-contour picture of part of the East Pacific Rise, the ridge axis (dotted line) is interrupted by a SNOO. Batiza and Margolis interpret the region to the southwest of the axis, in which the contour lines veer sharply away from the ridge, as a fault that grew perpendicular to the ridge during the spreading process.

Their model assumes that the eruption of magma from each section of the ridge crest is episodic and independent of any other segment. SNOOs arise when the spreading or growth of the seafloor along one segment is asymmetric, so that the segment moves away from the line of adjacent segments. As the seafloor coming from one segment moves relative to that arising from another, crustal faults are created perpendicular to the ridge axis.

This model "makes a major contribution in pointing out that structures even on that small a scale represent something significant about the underlying processes," comments Ken Macdonald of the University of California at Santa Barbara, who has mapped and modeled larger-scale structures along the East Pacific Rise.

But Peter Lonsdale at Scripps Institution of Oceanography in La Jolla, Calif., disagrees. He says there are ocean floor data, which are much more detailed than those used by Batiza and Margolis, that fail to support aspects of the researchers' description and model of SNOOs.

Batiza concedes that the small size of the SNOOs is approaching the resolution limit of the data set, which was collected using the Seabeam sonar mapper (SN: 3/15/86, p. 170). But he thinks there is enough information to characterize SNOOs and to speculate on the processes that make them.

"If the model that we present is correct," says Batiza, "then it suggests that there is a very direct link between the processes of magma emplacement and the tectonics, such as faulting, that go on at the ridge." This link between volcanic and tectonic processes is supported by the results of dredging studies conducted last year along the East Pacific Rise by Batiza, Charles Langmuir at Lamont-Doherty Geological Observatory in Palisades, N.Y., and John Bender at the University of North Carolina at Charlotte. They found a strong correlation between the occurrence of SNOOs and changes in the chemistry of the rocks that make up the rise.

Batiza says he would like to test some of the model's predictions by examining sections of the East Pacific Rise in detail with a submersible. If SNOOs do indeed reflect magmatic processes, he says, then the next step is to see if SNOOs are somehow preserved in the seafloor as it moves away from the ridge. If so, researchers might be able to reconstruct how the magmatic systems feeding the ridge have evolved over time.

— S. Weisburd

Cell aging: A process of oxidation?

While the outward signs of human aging are all-too-apparent, what's going on at the cellular level is more obscure. Researchers now propose that oxidation processes are one cause of cellular aging. Put less prosaically, we may all be rusting.

More than a decade ago, Marguerite M.B. Kay of Texas A&M University in Temple discovered a protein in the membranes of red blood cells that changes shape as the cells age. Eventually the protein displays what Kay has termed senescent cell antigen, an area of the protein that signals the immune system to destroy the cell. Her group and others have since found the antigen on many other cell types, including neurons.

In the current work, she and her colleagues at Texas A&M and at Hoffman-La Roche, Inc., in Nutley, N.J., monitored the protein and other age-related parameters in an oxidation-inducing situation. They fed rats a diet deficient in vitamin E, which acts as an antioxidant by halting the formation of free radicals — highly reactive molecules generated in cells by natural oxidation reactions.

The vitamin E deficiency accelerated the aging process in the red blood cells, presumably because of damage by free radicals, the researchers report in the current PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES (Vol. 83, No. 8). Among other changes, the cells were more likely to display the senescent cell antigen and to be destroyed by the immune system.

"I don't think [oxidation] is the only mechanism of aging," says Kay. "But I would suspect that it affects other cells besides red cells." Taken into account with her previous work, the findings suggest that blocking the oxidation process might lengthen the life of irreplaceable cells such as neurons, she says. "If you could prevent breakdown of this protein, conceivably you could lengthen the life span of cells."

Vitamin E megadoses aren't the way to do it, she says. "I would make sure people have a sufficient quantity of vitamin E," says Kay, "but it has been shown experimentally that an excess won't slow down aging."

Edward L. Schneider of the Bethesda, Md.-based National Institute on Aging is not convinced the work establishes an oxidant/free-radical connection to aging. Red blood cells differ from other cells in that they lack nuclei, and a vitamin deficiency isn't a good test system for aging, he says. "I don't doubt that it's possible," he says. "I think we need more evidence."

But Kay's theory has its adherents as well. "Of all the mechanisms [proposed for cellular aging], Kay's is probably the only one on very firm ground," says red blood cell researcher Lawrence Dalton of the University of Southern California in Los Angeles. "It's a very important result and gives the scientific community a direction to follow." *— J. Silberner*