Microorganisms create a line in the ocean


NASA


William M. Balch

A long line of diatoms forms at the boundary of two ocean currents (left). Closeup of the organisms (right).

If you flew over the Pacific Ocean about 100 miles north of the equator, roughly halfway between Hawaii and Tahiti, you might notice something resembling a deep green snake hundreds of kilometers long and less than 10 kilometers wide floating in the sapphire sea. Researchers recently identified this mysterious line as a surprisingly rich collection of single-celled diatoms, a species of the phytoplankton *Rhizosolenia*.

Sailors and astronauts have reported seeing the green streak, but few oceanographers even knew it existed, asserts James A. Yoder of the University of Rhode Island in Narragansett. He and his colleagues analyzed water samples as well as photographs taken from the space shuttle *Atlantis* in 1992, satellites, aircraft, and a ship, they report in the Oct. 20 NATURE. They did the work as part of the long-term Joint Global Ocean Flux Study.

Coastal waters commonly harbor unusually dense patches of plant or animal life, Yoder notes. Also, sargassum (gulfweed), jellyfish, and anything else that floats will line up along a current boundary, such as the edge of the Gulf Stream. However, rarely do such living lines form in the open sea.

The diatoms creating the streak in the Pacific congregate and thrive at the convergence of the North Equatorial Countercurrent and the cooler, denser water of the South Equatorial Current sinking below it, the scientists assert. The microorganisms feed in the cooler waters, then float up to the sunlight.

Studies by Tracy A. Villareal of the University of Massachusetts at Boston show that phytoplankton "adjust their buoyancy and migrate vertically between surface waters and nutrient-rich waters deeper in the water column," Yoder and his colleagues note.

No other research has demonstrated on such a large scale how the diatoms' buoyancy influences their concentration, Villareal says.

So-called instability waves, which run east to west, propagate along the boundary of the two currents and probably help the diatoms to accumulate, Yoder suspects. The line appears to form only during summer and fall. At other times, the organisms disperse.

The team speculates that the same mechanisms that help these diatoms gather may have caused a similar accumulation that geologists found recently in deep-sea sediments laid down 4.4 million to 15 million years ago in the same area. — *T. Adler*

Immune cell triggers attack on plaque

The sudden, often fatal rupture of plaque clogging an artery involves a host of possible villains, including the scavenging immune cells called macrophages. Now, a Finnish team presents evidence that another immune cell, the mast cell, may also take part in this deadly assault.

Atherosclerosis results from the buildup of cholesterol, fatty debris, and other substances on the interior wall of an artery. Normally, a tough protein cap covers this goo and keeps pieces of the plaque from breaking off into the bloodstream. But a fissure in that protective cap may lead to the formation of a clot (thrombus), which can block blood flow to the heart and cause a heart attack.

Petri T. Kovanen of the Wihuri Research Institute in Helsinki and his colleagues studied sections of human coronary arteries removed during 32 autopsies of men and women age 13 to 67. All 32 had some sections of the interior artery wall that appeared healthy and free of fatty buildup. However, 25 of the 32 showed regions with fatty streaks, the beginnings of atherosclerosis, and 19 of the 32 had areas with hardened plaque.

The researchers discovered mast cells in 50 percent of the normal vessel sections, in 84 percent of the fatty streaks, and in 95 percent of the so-called shoulder, a rupture-prone area of the plaque (see illustration).

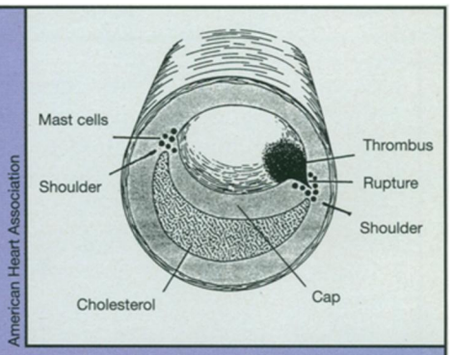
The Finnish team proposes that mast cells help crack a coronary plaque by releasing enzymes that help dissolve collagen and other components of the plaque's protein cap.

The group also showed that mast cells in the shoulder regions of plaque are much more likely to be activated than mast cells elsewhere in the artery. Once revved up, these cells spit out a cocktail of cap-melting enzymes and the chemical histamine as well.

Kovanen believes that histamine makes matters worse by causing the coronary artery to contract. If a clot forms when the plaque splits, the histamine-narrowed vessel may become completely blocked, thus leading to a massive heart attack.

The Finnish team reports its findings in the October CIRCULATION.

Mast cells, which are present in the airway and nasal passages, play a key role in hay fever and asthma attacks. However, the researchers say there's no evidence that people with asthma or allergies have an increased risk of heart attack.



American Heart Association

The shoulder region of the plaque joins the healthy artery wall to the protein cap on the plaque.

What causes mast cells to release their risky products? Smoking may. Kovanen points out that smoking increases IgE, an antibody that helps stimulate mast cells. However, elucidating the basic biology of these cells will require much more research, he says. For example, no one knows how mast cells function in healthy artery walls.

If future research confirms the role of the revved-up mast cell, researchers might attempt a preventive strike with drugs that quiet these cells, comments L. Maximilian Buja, a plaque researcher at the University of Texas Medical School at Houston. — *K.A. Fackelmann*