Ice crystals promote molecular eruption

Stacking randomly placed boxes after a hasty move clears a path from one room to another. Likewise, converting a disarrayed form of frozen water into ordered crystals creates channels that permit gas molecules trapped underneath to make their way into the open, researchers have found. Further study of such structural changes should help scientists understand how molecules dissolve in liquids and provide insight into the behavior of comets.

Frozen water can assume several forms. The investigators studied an unusual one, "much more like a liquid than a solid," according to R. Scott Smith of the Pacific Northwest National Laboratory in Richland, Wash. This material, called amorphous solid water (ASW), appeals to scientists interested in liquids because the molecules in it move extremely slowly, which makes them relatively easy to examine. Smith and his colleagues describe their work in the Aug. 4 PHYSICAL REVIEW LETTERS.

The investigators sprayed water vapor onto an oily substance at extremely low temperatures. Because the gas molecules stuck where they hit, they froze in random orientations. This process captured the water in its disorganized liquid form and trapped the oily layer underneath.

As the researchers raised the temperature, they initially observed no evaporation of the oily material, even after passing the point where vaporization would normally occur. As the heating continued, the gas suddenly burst through its icy restraint. The eruption occurred at exactly the temperature where ASW turns into the familiar crystalline ice.

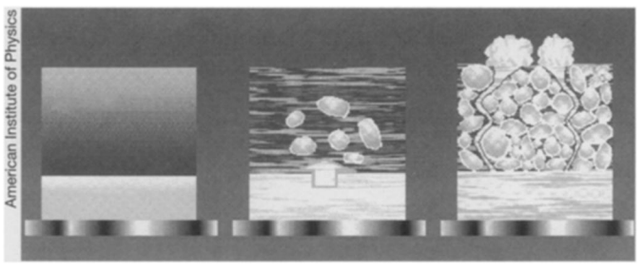
"The water molecules begin to move around and explore as they gain energy from heat," says team member Bruce D. Kay. "As they find the most stable arrangement, they settle into it." The individual crystals are separated by spaces, which create tiny ravines that act as exit routes for the gas trapped below. The researchers have dubbed this phenomenon "the molecular volcano."

"People knew that the transition between ASW and crystalline water happened, but they did not have a good way to detect the structural changes," says physicist H. Eugene Stanley of Boston University. "This gives a clear signal."

Researchers hadn't observed eruptions of this sort in the laboratory before, but such volcanoes are thought to occur frequently in space. Although ASW exists on Earth only in laboratories, it accounts for most of the ice in the universe.

"New comets occasionally release unexpected spurts of gas," says astrochemist Louis J. Allamandola of NASA's Ames Research Center in Mountain View, Calif. "They contain pockets of gases, which want to get out when they heat up. If the ice that seals these cavities goes through this structural change as the comet warms up, it'll release the gases."

—E. Strauss



Amorphous solid water (ASW) covers a layer of oily liquid (left). As the temperature increases, the ASW begins to form tiny ice crystals and the oil turns into a gas (center). Once continuous channels through the ASW have formed, gas molecules can escape (right).

Mutated gene can delay onset of AIDS

For years scientists have wondered why some HIV-positive people develop AIDS promptly, while others seem to ward off the disease indefinitely.

Researchers know that a mutation in a gene called *CCR5* renders roughly 1 percent of white people highly resistant to HIV infection. Such people receive the mutant gene from both parents. Individuals who inherit the *CCR5* mutation from just one parent can contract HIV, but the onset of AIDS is delayed 2 to 4 years.

Now, in a genetic déja vu, researchers have found a mutation in a second gene, called *CCR2*—this one carried by up to one-fourth of all people—that also seems to postpone the onset of AIDS in HIV-positive individuals by 2 to 4 years. The gene does not prevent infection, however.

The *CCR2* gene sits next to its cousin *CCR5* on chromosome 3. Both genes encode proteins called chemokine receptors, which act as docking stations on the outer surface of immune cells.

When cells are damaged, they produce chemokines, proteins that attract immune cells. To hijack these immune cells, HIV latches onto their chemokine receptors, preventing the chemokines from docking. The *CCR5* mutation results in a shortened receptor, thus thwarting HIV's ability to take over the immune cells (SN: 8/17/96, p. 103).

Discovery of the *CCR5* mutation last year spawned a flurry of research to identify other genes that encode chemokine receptors. The new study, led by researchers at the National Cancer Institute in Frederick, Md., indicates that HIV may also need normal CCR2 chemokine receptors to progress rapidly to AIDS. Defective CCR2 receptors, which appear in about 20 to 25 percent of people of all races, are probably difficult for the virus to commandeer. The prevalence of such faulty receptors may explain, in part, the wide variation in survival rates of HIV-positive people.

The researchers studied 3,003 blood samples from people at risk of getting HIV. Most were found to be HIV-positive. Roughly half of the HIV-positive individuals who survived more than 16 years have either the *CCR2* or *CCR5* mutation, the researchers report in the Aug. 15 SCIENCE.

The findings raise tantalizing possibilities. "If we could mimic the effects of these mutations, either by designing a drug or by gene therapy, then you would have the hope of delaying AIDS onset for a long period, if not indefinitely," says study coauthor Stephen J. O'Brien, who heads NCI's genomic diversity laboratory.

The NCI researchers don't know how the altered CCR2 receptor delays AIDS. The CCR5 receptor is used by the most prevalent HIV strains, while the CCR2 receptor is rarely used by viruses, says Robert W. Doms, a pathologist at the University of Pennsylvania in Philadelphia. "There might be some other mutation that we still haven't found yet," he says. Nevertheless, the study sheds needed light on the biology of chemokine receptors, he adds.

"They are the linchpins for HIV," says Dan R. Littman, an immunologist and Howard Hughes Medical Institute investigator at New York University. However, Littman doubts that the CCR2 receptor itself plays a major role. He suggests that it may regulate CCR5 receptor proteins in some way, possibly tipping the chemokine balance in the process and discouraging the virus from locking onto a cell.

Much is left to study, and the terrain is still new.

"It is striking to recall that a family of proteins as large and ubiquitously expressed as chemokines and their receptors was unknown less than 10 years ago," writes Barrett J. Rollins of the Dana-Farber Cancer Institute in Boston in the Aug. 1 BLOOD. *—N. Seppa*