Crystalline hydrogen gets its first X ray

For decades, theoretical physicists have explored solid hydrogen—the simplest of elements—with their minds. Experimental physicists, however, have had a difficult time of it, largely because X rays tend to fly past hydrogen atoms with little interaction. Now, a team of U.S. and French physicists announces it has overcome this challenge and has peered at a single crystal of hydrogen.

"To take a direct look at solid hydrogen is a real breakthrough," says Paul Loubeyre of the University of Paris. "It was considered impossible." In the Oct. 24 NATURE, he and his coauthors describe how they carefully squeezed hydrogen gas until it became solid, then captured its image at the European Synchrotron Radiation Facility, the world's brightest X-ray instrument.

The researchers injected hydrogen and helium gas into a tiny sealed vise, or diamond anvil, made of two diamonds that can be squeezed together. For several days, the physicists slowly closed the vise, increasing the pressure on the gases to force them into a fluid state. Because of electrical interactions, hydrogen and helium repel one another.

At a pressure of more than a million times atmospheric conditions, the hydrogen solidified into a single crystal, pushed into the center of the vise by the liquid helium surrounding it. The researchers also created a crystal of deuterium, a heavier isotope of hydrogen.

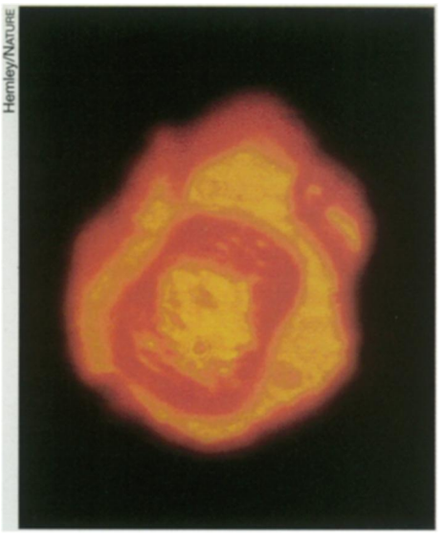
"It's a really clever idea, to grow the crystal in helium," says Isaac F. Silvera of Harvard University. The more compressible fluid cushioned the crystal, preventing it from shattering.

"The single crystal is the reason we could take an X ray," says Russell J. Hemley of the Carnegie Institution of Washington (D.C.), a coauthor of the report. To capture an image, the researchers fired an X-ray beam into the diamond anvil. The radiation passed through one diamond, diffracted off the atoms in the hydrogen crystal, exited through the second diamond, and finally reached a detector. "Diamond is transparent not only to the eyes but to X rays," says Hemley.

Although the imprisoned crystal was little more than 3 micrometers thick, the physicists were able to align the X-ray beam with the immobilized hydrogen. Instead of slipping past randomly moving hydrogen atoms, the beam interacted with the electrons confined within the fixed crystalline pattern.

As pressures approached 1.1 million atmospheres, the scattering of the X-ray beam off the hydrogen atoms revealed that the crystalline structure was becoming increasingly ordered, compressing 25 percent more than expected. Building on earlier experiments (SN: 4/20/96, p. 250), the researchers hope someday to squeeze the atoms tightly enough to convert from crystalline to metallic form, in which electrons can move freely among the atoms. Metallic hydrogen may be a superconductor at room temperature.

"It's the simplest system in nature," says Silvera. "But it's also one of its great challenges." —*D. Vergano*



Square deuterium crystal surrounded by helium fluid.

Viral protein disables a cellular alarm

Cells have a sophisticated alarm system to alert the human body to viral infections. When viruses start replicating inside a cell, the cell chops up viral proteins into fragments called antigens. It displays these viral antigens on its surface, a distress signal to the immune system, the body's police force.

Some of the craftiest viruses have developed tools for disabling this cellular alarm system. In the Oct. 24 NATURE, scientists describe one such tool, the protein pp65, employed by a virus called cytomegalovirus (CMV).

This virus, which can cause blindness, pneumonia, and other illnesses in AIDS patients and other people with weakened immune systems, infects an estimated 60 to 90 percent of the U.S. population, usually during childhood.

Though CMV rarely causes obvious illness in people with strong immune systems, some research has linked it to heart disease, and few people ever rid themselves completely of the virus. "We harbor the virus lifelong once exposed," notes Mark J. Gilbert of the Fred Hutchinson Cancer Research Center and the University of Washington, both in Seattle.

To establish such a long-lasting residence, a virus must carefully conceal itself from patrolling cells of the immune system. A short time after entering a cell, CMV begins to make proteins that prevent the cell from displaying antigens on its surface. "It's almost as if the virus cloaks the cell from the immune system," says Gilbert.

Even before that cloaking takes place, CMV needs to deceive the cell, says Gilbert. Right after infection, the virus synthesizes a large molecule called the immediate-early protein, or IE. This protein, known as a transcription factor, turns on other viral genes. "It's the main transcription factor that initiates replication of the virus," says Gilbert.

Why don't cells dice IE proteins into bits and display those antigens?

That's where pp65 enters the picture. Gilbert and his colleagues used another virus, to which they had added the CMV gene for IE, to infect human cells. Immune cells that recognize IE antigens destroyed the infected cells. But if the engineered virus also carried the CMV gene for pp65, the immune cells ignored any infected cells.

Gilbert's group concluded that pp65, a small protein that CMV releases as soon as it infects a cell, interferes with a cell's ability either to chop up IE into antigens or to display the fragments on its surface. In effect, the virus carries inside it a ready-made tool that conceals the initial signs of its break-in. It may be that pp65's actions buy the virus enough time to make the other proteins that further deactivate a cell's alarm system.

To confirm their hypothesis, Gilbert and his group created a version of CMV that has no pp65. As expected, cells infected with this mutant virus were killed by immune cells that target IE antigens.

Gilbert and his colleagues have evidence suggesting that pp65 modifies IE by attaching to it a group of atoms that includes phosphorus. Without affecting the protein's function, the addition seems to prevent a cell from processing IE into antigens.

"That's totally new, a unique mechanism nobody would have predicted," says Linda R. Gooding of the Emory University School of Medicine in Atlanta.

While an understanding of how pp65 silences a cell's alarm may inspire new strategies for treating viruses, Gooding says that a more immediate impact will be a clearer picture of the complicated machinery cells use to present viral antigens. One of the best ways to study how this cellular alarm system works is to explain how viruses cripple it, she says. —*J. Travis*