Electron Mix Binds Water Molecules

Life owes its existence to a relatively weak connection called the hydrogen bond, which joins molecules or regions within a molecule. Without it, liquid water would be scarce on Earth and biological machinery involving DNA and proteins would halt. Despite intense scrutiny, the bond has remained mysterious in many ways (SN: 7/20/96, p. 37).

A new study of ice now shows experimentally that the frail hydrogen bond between water molecules taps into a molecule's internal covalent bonds, formed when atoms share electrons. The late Nobel laureate Linus Pauling first suggested this might be the case in 1935.

Although scientists have long assumed that hydrogen bonds are partly covalent, the experimental proof ranks as a major milestone, some hydrogen-bond experts say. Demonstrated in water, the findings apply to all hydrogen bonds, they add. The results may help investigators better understand properties of the bonds, such as why they are strongest in a certain direction, and improve models of their behavior.

A report on the experiment in the Jan. 18 PHYSICAL REVIEW LETTERS is "certainly a very, very important new paper," comments Jose Teixeira of the Saclay research center of France's Atomic Energy Commission.

In water, hydrogen bonds forge links between hydrogen and oxygen atoms in adjacent molecules. Such a bond's character derives mostly from attraction between unlike electric charges that the two types of atoms acquire.

However, the new findings show that an electron contributing to that charge separation spends roughly 10 percent of its time mingling with an electron covalently binding the hydrogen and oxygen atoms within the adjacent molecule. "That's what Pauling said, and it's consistent with our data," says Eric D. Isaacs, the leader of the new study and one of three scientists at Lucent Technologies' Bell Labs in Murray Hill, N.J., who took part in the work. The specific 10 percent estimate has not yet been published, he says.

In the new experiment, Isaacs' team, which also included scientists at Northeastern University in Boston, the European Synchrotron Radiation Facility in Grenoble, France, and the Canadian National Research Council in Ottawa, Ontario, shone X rays at millimeter-thick crystals of ultrapure ice. X rays lose some energy and change direction as they strike electrons in the crystal. Their transformations reveal the spatial distribution of the ice's electrons—considered waves, according to quantum mechanics. By studying X rays bounced off various planes in the crystal with different numbers of hydrogen bonds, the team highlighted features of the bonds.

In the portrait of the electron waves that emerged, the team found fluctuations like those observed when overlapping light waves interfere with each other—their crests and troughs adding and canceling. The researchers deduce that the electron wave in each hydrogen bond is interfering with the wave in an adjacent covalent bond. Consequently, the electrons in both bonds must overlap to some degree, indicating that the electron in the hydrogen bond is circulating around two linked atoms—the hallmark of covalency. —*P. Weiss*

Lucent Technologies' Bell Labs

An X ray (squiggles) glances off an electron in a hydrogen bond (arrow) between two water molecules. X-ray beams at newer synchrotrons are now bright enough to reveal sharing of electrons between hydrogen bonds and bonds within molecules.

Multiplied immune cells combat HIV

Cytotoxic T lymphocytes, or CTLs, richly deserve their nickname—killer cells. CTLs spot cells that have been invaded by viruses and kill them off. They even target immune cells infected by HIV, the virus that causes AIDS. Some HIV-positive people who have avoided AIDS for years have strong CTL counts.

However, some scientists doubt that CTLs play a great role against HIV. HIV-specific CTLs lurk mostly in the bloodstream, they note, whereas HIV tends to invade lymph nodes. There, the virus infects an immune cell called the helper T cell, which directs a range of immune responses. Moreover, some researchers fear that CTLs revved up to kill HIV-infected helper T cells may run amok and destroy healthy helper T cells or allow mutating HIV to escape and reproduce. Both would pose risks to a compromised immune system.

Researchers now have evidence that CTLs indeed attack HIV in lymph tissue and seem to distinguish between healthy and infected helper T cells. To gauge CTLs' effect on HIV, researchers took CTLs from the blood of three HIV-positive people who were receiving antiviral treatment. Over an 8-week period, the scientists mass-produced the CTLs, marking some with a slight genetic difference for tracking purposes. The researchers then injected CTLs derived from each person back into that individual, a total of about 13 billion cells in five infusions at 2-week intervals.

The CTLs seemed to know where to go to fight HIV, says study coauthor Stanley R. Riddell, an immunologist at the Fred Hutchinson Cancer Research Center in Seattle. The scientists found that many migrated to lymph nodes—in particular, to portions of lymph tissue that harbored the most HIV-infected helper T cells—Riddell and his colleagues report in the January NATURE MEDICINE. The number of infected helper T cells in the bloodstream declined precipitously, to undetectable levels. However, the overall count of free HIV in the patients' bloodstreams changed little.

The virus showed little sign of mutating to escape the infused CTLs, Riddell says. Meanwhile, concentrations of healthy helper T cells in the patients remained steady.

Seven days after each infusion, the patients' blood concentrations of CTLs dropped to previous levels, and the number of infected helper T cells in blood samples climbed back to pretreatment levels. "This is a very transient effect," says Riddell. "We need to make ways to maintain the response."

The study bolsters the argument that CTLs play a part in the fight against HIV, particularly by migrating to lymph tissues, but this method of reconstituting and reinjecting CTLs is still far from having practical use in therapy, says Xia Jin, an immunologist at the Aaron Diamond AIDS Research Center in New York City.

Riddell agrees. "Our data say the CTL numbers are not sufficient," even when bolstered this way, he says. "If we could do things that maintain this [CTL] response at really high levels, we could get antiviral activity." —*N. Seppa*