# SCIENCE NEWS of the week

## New Clues Link Leukemia Virus to AIDS

Scientists working to unravel the mystery of acquired immunodeficiency syndrome (AIDS) may now have a prime suspect: a virus that causes a rare type of leukemia in humans. In a series of investigations, laboratory and clinical researchers from the National Cancer Institute, Harvard University School of Public Health and the Pasteur Institute in France have established a promising link between human T-cell leukemia virus (HTLV) and patients with AIDS or lymphadenopathy, a condition that often leads to AIDS. The evidence, reported in four articles in the May 20 SCIENCE, includes isolation of the actual virus from a few AIDS patients, detection of the genetic sequence of an HTLV in the white blood cells of two cases, and the determination that a high percentage of AIDS patients harbor antibodies against HTLV.

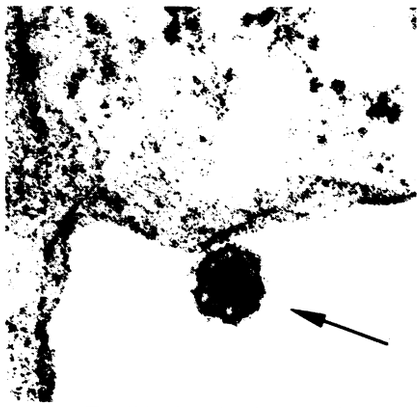
Robert C. Gallo of the National Cancer Institute in Bethesda, Md., and colleagues isolated the virus from one AIDS patient in the United States and found antigens to HTLV in white blood cells from two cases in France. A similar virus was isolated from a patient with lymphadenopathy by F. Barré-Sinoussi and colleagues at the Pasteur Institute. Another group at NCI, led by Edward P. Gelmann, detected the genetic material of an HTLV in the white blood cell DNA of two of 33 AIDS patients. At least 25 percent of the AIDS patients in a group studied by Myron Essex and colleagues at Harvard University School of Public Health had evidence of exposure to HTLV. Fewer than 1 percent of the control subjects in this study had antibodies to HTLV. A related article in the same issue of SCIENCE reports findings by Ze'ev Trainin and colleagues, also at Harvard, linking an AIDS-like disease in cats with the feline leukemia virus.

One of the strongest links between HTLV and AIDS is that the virus attacks and transforms T lymphocytes, the same cells that are disturbed in AIDS patients. Victims of AIDS have decreased ratios of helper to suppressor T cells. Helper T cells activate the cell-mediated immune response, which kills infected cells, while suppressor T cells exist to moderate this response and turn it off after the infected cells are eliminated. In AIDS patients, the predominance of suppressor cells leads to profound suppression of the cell-mediated response. Normally innocuous microorganisms, usually kept in check by the immune system, are free to cause serious and often fatal infections. The feline leukemia virus, which resembles HTLV, causes a similar immunosuppression in cats.

As of May 9, the Centers for Disease Control in Atlanta report 1,410 cases of AIDS in the United States alone, and the death toll stands at 541 — a 38 percent fatality rate. Although male homosexuals remain the group at highest risk, intravenous drug abusers and Haitians, and their children, along with hemophiliacs, also risk contracting the disease (SN: 4/16/83, p. 245).

The HTLV-Haitian connection is important because, while the other high-risk groups are connected either through intimate contact or transfer of blood products, most of the Haitians deny the homosexual practices, drug abuse or exposure to blood products that would provide this link. In the Caribbean (and southern Japan) from 4 percent to 37 percent of healthy adults have developed antibodies to HTLV.

Gallo contends that "it's still too early to say" whether HTLV is the agent that causes AIDS, or just another opportunistic infection. "It's going to be extremely difficult to prove" HTLV causes the disease, he says, because the targets, helper T cells, slowly disappear as the disease progresses, removing the sign of infection.



Gallo/Science

*Human T-cell leukemia virus particles were isolated from a patient with AIDS.*

"It's almost like a hit-and-run," he adds. In fact, when the two cases that initially demonstrated HTLV genetic sequences were later tested, the evidence of HTLV infection had indeed vanished. One possible mechanism, suggested by Gelmann, is that HTLV induces proliferation of a suppressor T-cell clone by integrating its genetic material into a lymphocyte. By studying the actual sites of integration of the HTLV genetic material into the T lymphocyte, the researchers hope to find whether this is the case. *—P. Taulbee*

## Fast pulsars: One by light, two by radio

When the first radio pulsar with a period in milliseconds (PSR 1937+214) was discovered (SN: 12/4/82, p. 357), many astronomers reasoned that it represented a new class of pulsar for which a new theory was needed. It pulses 20 times as fast as the next fastest and its pulse rate is slowing down at a rate far slower than ordinary (SN: 1/1/83, p. 4). Its fast pulse rate also led astronomers to look for pulses from it in visible light, as the two next fastest radio pulsars (out of more than 300) do have light pulses. Now, light pulses from PSR 1937+214 have been found. At the same time a second pulsar with radio pulses in the millisecond range has been found, so maybe there is a distinct class. (PSR 1937+214 used to be PSR 1937+215; the numbers are the coordinates of its location, and a more accurate determination changed them.)

R. N. Manchester, B. A. Peterson and P. T. Wallace, working at the Anglo-Australian Telescope in Siding Spring, Australia, report that they detected visible light pulses from PSR 1937+214 on two successive nights, April 20 and 21. The pulses have the same period, 1.6 milliseconds, as the radio pulses. They were detected through an aperture centered on a red star that S. B. Djorovski of the University of California at Berkeley had suggested as an optical identification for the pulsar. The Anglo-Australian group accepts that identification. The amplitude of the pulses is about one percent of the total brightness of that red star.

Carlton E. Pennypacker of the Lawrence Berkeley Laboratory, a member of an American group looking for such pulses with the Multiple Mirror Telescope in Arizona, says that his group has seen no pulses, but their equipment cannot distinguish pulses smaller than three percent of the red star's light, so the two results are consistent with one another. What is not consistent is that the American group has seen spectral lines of carbon monoxide in the red star's light and so they suggest that it is not the pulsar, but an ordinary red giant that happens to be in the same field of view.

V. Boriakoff of Cornell University in Ithaca, N.Y., R. Buccheri of the National Research Center in Palermo, Sicily, and F. Fauci of the University of Palermo report a radio pulsar with a period of 6.13369 ± 0.000019 milliseconds at right ascension 19 hours 53 minutes 25.7 seconds. It is thus in the same general part of the sky as PSR 1937+214, and it may correspond to a gamma-ray source, 2C G065+1. Its pulse period is increasing at $0.58 \times 10^{-15}$ seconds per second.

The six-millisecond pulsar is in a binary star system with an orbital period of about 120 days. Some theorists have suggested that millisecond pulsars should be formed in binary systems (SN: 1/1/83, p. 4). PSR 1937+214 is not now in a binary system, but there is evidence that it once was. *—D. E. Thomsen*