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EMBARGOED FOR RELEASE:

7:00 AM (PDT), WEDNESDAY, MAY 9

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UC-SAN FRANCISCO STUDIES CONFIRM FRENCH FINDING OF RETROVIRUS IN AIDS AND DEMONSTRATE A RETROVIRUS COULD BE PASSED THROUGH BLOOD CLOTTING FACTOR

TORONTO -- A UC-San Francisco virologist today reported results of two studies that add further support to the theory that a retrovirus plays a major role in acquired immune deficiency syndrome.

A study by Jay Levy, MD, UCSF associate professor of medicine and an investigator in the Cancer Research Institute, confirms findings by French scientists that a retrovirus -- lymphadenopathy associated virus (LAV) -- could be the primary cause of AIDS. So far, Levy has found evidence of LAV or a LAV-like agent in blood cells of 28 of 50 AIDS patients in San Francisco. In three of the 28, Levy's laboratory has direct proof of the similarity to LAV.

The study was reported here today at a joint meeting of the American Society of Clinical Oncology and the American Association for Cancer Research.

Last year, researchers (F. Barre-Sinoussi, L. Montagnier, J.C. Chermann) at the Pasteur Institute in Paris discovered LAV in blood of AIDS and pre-AIDS patients in France. The suspected virus grows preferentially in certain lymphocytes (white blood cells), which become damaged or diminished in AIDS patients. The loss of these cells appears to be responsible for their inability to fight off opportunistic infections and cancer. Levy's research group found retroviral enzymes and proteins in white blood cells, as well as antibodies to LAV, which indicate infection, in over 50 percent of the patients studied. Evidence of the virus was not detected in any of 21 healthy controls. For the study, Levy used reagents supplied by the Pasteur Institute researchers to compare his isolates with theirs. Electron micrographs also have confirmed the similarities, Levy reported.

The relationship of the UCSF LAV-like agent and LAV to the HTLV-III retrovirus, recently described by Robert Gallo of the National Cancer Institute, is not yet known, says Levy. Recent reports, however, indicate strong

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similarities between LAV and HTLV-III. Although they are major steps in AIDS research, Levy cautioned that these new findings do not yet establish absolute proof that either virus or a similar one is the cause of AIDS. He is conducting experiments, infecting animals with the UCSF isolates, in an attempt to determine whether or not they are responsible for AIDS.

In a separate presentation at the ASCO and AACR meetings, Levy today reported results showing retroviruses can survive procedures previously used to produce the clotting factor (Factor VIII) used by hemophiliacs. Levy, working with researchers from Cutter Laboratories in Berkeley (Milton M. Mozen, PhD, and George Mitra, PhD), tested the stability of a mouse retrovirus, resembling LAV, through the clotting factor production process. The process did not substantially inactivate the retrovirus, Levy reported.

This is counter to previous beliefs that a retrovirus could not survive such a process, and the study possibly explains why some 30 hemophiliacs in the United States have developed AIDS. The factor, used by hemophiliacs to prevent uncontrollable bleeding, is usually prepared from blood contributed by thousands of donors. The study by Levy and his coworkers did indicate that the heating procedure used now by Cutter eliminates the retrovirus effectively.

The process is now being used by manufacturers of Factor VIII to further minimize the chances of hemophiliacs contracting AIDS, says Levy.

Taken together, Levy's studies -- indicating presence of LAV or a LAV-like agent in the blood of AIDS patients in San Francisco and demonstration that a retrovirus can be transmitted in blood clotting factors -- lend further support to the theory that a retrovirus is involved in AIDS.

Levy's research is supported by grants from the state of California and the National Institutes of Health. His UCSF research team includes Susan Kramer, PhD, Lyndon Oshiro, PhD, Anthony Hoffman, Jill Landis and Joni Shimabukoro.

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