

Barré-Sinoussi F, Chermann J C, Rey F, Nugeyre M T, Chamaret S, Gruest J, Dauguet C, Axler-Blin C, Vézinet-Brun F, Rouzioux C, Rozenbaum W & Montagnier L. Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS). *Science* 220:868-71, 1983.
[Dépt. Virologie, Inst. Pasteur; Lab. Cent.-Virologie, Hôp. Claude Bernard; and Dépt. Santé Publique et Médecine Tropicale, Hôp. La Pitié-Salpêtrière, Paris, France]

This paper describes the isolation of the lymphadenopathy AIDS virus, obtained from a homosexual male presenting with lymphadenopathy. Antibodies to this virus were found in two patients' sera. This new retrovirus, not related to HTLV I, was T lymphotropic and produced a decline of lymphocyte proliferation (demonstrating a cytopathic effect). [The *SC1*® indicates that this paper has been cited in over 835 publications.]

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In 1983, when we published our first evidence for the role of a new retrovirus in AIDS and associated symptoms, the viral etiology of AIDS was already acknowledged by researchers in the field. However, the idea that our viral isolate was a new retrovirus that might be the cause of the disease was not very well accepted until it was confirmed one year later by other laboratories.

For several years, our laboratory had been involved in researching animal retroviruses and the control of retrovirus expression. In 1982 we were studying the presence of retrovirus related to mouse mammary tumor virus or M.MTV-related sequences in lymphocytes of patients with breast cancer.¹ Thus, in January 1983, when we decided to look for a retrovirus as a possible cause of AIDS, our labo-

ratory was technically well placed to carry out such a study.

For our first attempt, we looked for a retrovirus produced by T lymphocytes, which were known to be affected by the disease. But we did not start with a preconceived idea about which retrovirus, if any, we would find. In order to be in the best situation for such an attempt and in collaboration with clinicians, we decided to study a man at risk for the disease, but who was not immunosuppressed; in such a case, the target cells for the etiological agent should still be present. We added antihuman interferon serum to the target-cell cultures since, in earlier studies, we had shown control of retrovirus production by endogenous interferon produced by cells.² A retrovirus was detected in our T-lymphocyte cultures' supernatant as early as two weeks after the beginning of the experiment, and, surprisingly, the virus-producing cell culture was dying. At that time, we thought that this virus might be a new one, so we were very eager to propagate it and not to lose it. However, we were rapidly successful in the infection of normal T cells from a healthy donor and from cord blood. This allowed us to study this virus in more detail, to confirm its new characteristics and to develop serologic tests.

Since the first discovery, this new retrovirus, named Lymphadenopathy Associated Virus (LAV) and also Human T-Lymphotropic Virus type III (HTLV III) and ARV (AIDS related virus) by others, has been recognized by the scientific community as the etiological agent of AIDS. This virus is also called Human Immune Deficiency Virus (HIV).

Thus, this paper is highly cited because it provided evidence for the role of a new human retrovirus in AIDS. It is satisfying to see researchers recognizing the contributions of our group to the field in which we continue to work.³⁻⁵

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