Lymphadenopathy Associated Virus (LAV) as the Primary Etiologic Agent of AIDS and Therapeutic Perspectives

J. C. CHERMANN, F. BARRE and L. MONTAGNIER

Viral Oncology Unit, Institut Pasteur, Paris, France

A new type of human retrovirus, LAV, first isolated from an homosexual with persistent lymphadenopathy and thereafter from patients with frank AIDS is now considered as the primary cause of AIDS.

Its frequent isolation from all individuals at risk for the disease, its selective tropism for the T4+ subset of lymphocytes, its cytopathic effect on T cells and the prevalence of antibodies against LAV proteins in AIDS and ARC patients as well as in high risk groups strongly supported a causal relationship between the virus and the disease. LAV can be transmitted via blood as it is suggested by documented cases of viral infection in hemophiliacs and in recipients of blood transfusions.

The occurence of AIDS often requires a long incubation period which allows the interplay of cofactors such as viral or bacterial infections and repeated antigenic stimulation. Analogy with the slow retroviruses (lentiviruses) is suggested by some similar aspects of the pathogenicity and by the structure of the AIDS retrovirus itself. They both have unusually large genomes and envelope glycoproteins as well as typical morphology. LAV genome contains two new open reading frames (Q and F) and an exceptionally long env gene. The genetic organisation of LAV is so far unique among retroviruses indicating that this virus is clearly the prototype of a new group of human retroviruses.

The identification of LAV as the causative agent of AIDS has allowed the investigations of compound inhibiting viral replication. One of this compound, HPA-23, is a strong inhibitor of LAV reverse transcriptase and inhibits the viral replication
in vivo in treated patients. Whether such treatment will be enough or should be combined to bone marrow transplant for restoring immune functions remains to be determined.