Retrovirology

BioMed Central

Oral presentation

Integration of HIV-1 Caused STAT3-Associated B Cell Lymphoma in an AIDS Patient

Harutaka Katano*‡[1], Yuko Sato[1], Satomi Hoshino[2], Shigeo Mori[3], Tetsutaro Sata[1], Micheal D Weiden[2] and Yoshihiko Hoshino[2]

Address: [1]Department of Pathology, National Institute of Infectious Diseases, Tokyo 162-8640, Japan, [2]Division of Pulmonary and Critical Care Medicine, New York University School of Medicine, NY 10016, USA and [3]Department of Pathology, Teikyo University School of Medicine, Tokyo 173-8605, Japan

Email: Harutaka Katano* - katano@nih.go.jp

* Corresponding author ‡Presenting author

from 2005 International Meeting of The Institute of Human Virology
Baltimore, USA, 29 August – 2 September 2005

Published: 8 December 2005

Retrovirology 2005, **2**(Suppl 1):S53 doi:10.1186/1742-4690-2-S1-S53

B cell lymphomas remain a significant cause of morbidity in AIDS but the pathophysiology of this disease is unclear. We report a case of B cell lymphoma in which HIV-1 integrated into the host genome. The lymphoma cells with anaplastic large cell morphology formed multiple nodular lesions in the lung of a homosexual AIDS patient. The lymphoma cells did not express KSHV-LANA and EBV-EBER or HIV-1 p24 but did express high levels of nuclear localized STAT3. The provirus had a 5'LTR deletion and the 3'LTR was inserted just before the first coding exon of STAT3. Reporter gene assay demonstrated that the 3'LTR had a strong promoter activity especially when co-transfected with HIV Tat. These data suggest HIV-1 integration resulted in induction of STAT3 and possibly promoted lymphoma formation. This suggests that HIV-1 insertional mutagenesis may be associated with some cases of AIDS lymphoma.