Retrovirology

BioMed Central

Poster presentation

Open Access

Insufficient Tat Production Leads to Latent HIV Infection in Astrocytes

Ashok Chauhan*‡, Suzanne Gartner, Nicole Benoit and Avi Nath

Address: Department of Neurology, Johns Hopkins University, Baltimore, MD 21287

Email: Ashok Chauhan* - achauha1@jhmi.edu

* 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):P23 doi:10.1186/1742-4690-2-S1-P23

Background

In the brain, HIV infects microglia and macrophages productively while astrocytes are infected limitedly. Long life span and low turnover rate of astrocytes make them suitable hosts for viral persistence. Several studies have shown that HIV replication is blocked in astrocytes. We investigated regulation of HIV infection in astrocytes.

Methods

We used human fetal astrocytes (PFA), astrocyte cell line (SVGA) and reporter cells to monitor Tat and Rev activity. HIV IIIB and NL4-3 strains were used to infect astrocytes.

Results

Following HIV infection of SVGA reporter cells, very limited infection was detected. Further, no receptor or co-receptors except CXCR4 were present on astrocytes. To see further if viral replication is blocked in astrocytes, we infected PFA or SVGA cells with VSV pseudotyped NL4-3. High levels of p24 and robust LTR-GFP or LTR-gagGFP activation were seen. VSV-HIV infected reporter cells never lost green fluorescence and green cells were negative despite of continuous low Tat and Rev production. To rule out If Tat and Rev expression were from circular unintegrated HIV-DNA, Alu PCR revealed integration of viral DNA. Further, Tat or TNF-a reactivated the latent HIV in astrocytes and the latent HIV-SVGA cells upon co-culture transmitted the infection to Jurkat cells.

Conclusion

HIV establishes latent infection in astrocytes and HIV latency is due to the low levels of Tat production.