Retrovirology

BioMed Central

Poster presentation

Do monocytes use the novel adipocytokine Visfatin/NAMPT/PBEF1 to flip the HIV coreceptor switch?

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from Frontiers of Retrovirology: Complex retroviruses, retroelements and their hosts
Montpellier, France. 21-23 September 2009

Published: 24 September 2009

Retrovirology 2009, **6**(Suppl 2):P88 doi:10.1186/1742-4690-6-S2-P88

This abstract is available from: http://www.retrovirology.com/content/6/S2/P88

The Human Immunodeficiency Virus (HIV) coreceptor switch, which entails the change in preferential coreceptor usage of the virus for CCR5 to CXCR4 in ~50% of all HIV subtype B infected patients, is an important determinant in the pathogenesis of HIV infection. However, the mechanisms underlying this switch are poorly understood, and prognostic markers for this switch are unknown. Here, we describe the upregulation of the novel adipocytokine visfatin (NAMPT) in monocytes of therapy-naïve HIV patients, which is reversed during antiretroviral therapy. Induction of visfatin was observed both at the mRNA and protein level and was mirrored by an increase in plasma visfatin in therapy-naïve HIV patients. Visfatin expression correlates with the viral load, and high visfatin expression appears to be associated with the dominance of CXCR4-using HIV in the plasma. We show that visfatin is capable of selectively reducing the infectivity of CCR5-using clones in primary cells (macrophages, resting PBMC) in vitro, while at the same time remaining indifferent to or even favouring infection by CXCR4-using virus. As such, visfatin may play an important contributing role in the development of the HIV coreceptor switch by mounting a selective pressure against CCR5-using and in favour of CXCR4-using viruses.