

The HIV-2/SIVSM Vpx protein and an antiviral restriction in primary myeloid cells

Gregory Berger, Xuan N'Guyen, Stéphanie Cordeil, Stéphanie Durand, Ferdinand Roescht, Jean-Luc Darlix, Andrea Cimorelli

► **To cite this version:**

Gregory Berger, Xuan N'Guyen, Stéphanie Cordeil, Stéphanie Durand, Ferdinand Roescht, et al.. The HIV-2/SIVSM Vpx protein and an antiviral restriction in primary myeloid cells. *Frontiers of Retrovirology: Complex retroviruses, retroelements and their hosts*, Sep 2009, Montpellier, France. BioMed Central, 6 (Suppl 2), pp.P12, 2009, Retrovirology. <10.1186/1742-4690-6-S2-P12>. <inserm-00663607>

HAL Id: inserm-00663607

<http://www.hal.inserm.fr/inserm-00663607>

Submitted on 27 Jan 2012

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Poster presentation

Open Access

The HIV-2/SIV_{SM} Vpx protein and an antiviral restriction in primary myeloid cells

Gregory Berger*^{1,2,3}, Xuan Nhi N'guyen^{1,2,3}, Stephanie Cordeil^{1,2,3},
Stephanie Durand^{1,2,3}, Ferdinand Roescht^{1,2,3}, Jean-Luc Darlix^{1,2,3} and
Andrea Cimarelli^{1,2,3}

Address: ¹LaboRetro, Department of Human Virology, Ecole Normale Supérieure de Lyon, Lyon, France, ²INSERM, U758, Lyon, France and ³University of Lyon, Lyon1, IFR128 BioSciences Lyon-Gerland, Lyon-Biopole, Lyon, France

* Corresponding author

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):P12 doi:10.1186/1742-4690-6-S2-P12

This abstract is available from: <http://www.retrovirology.com/content/6/S2/P12>

© 2009 Berger et al; licensee BioMed Central Ltd.

Myeloid cells are key targets of human immunodeficiency virus (HIV) replication *in vivo* and are important players in viral-induced pathogenesis. Albeit with variations depending on their differentiation status, these cells are less susceptible to viral infection than other cell types like primary lymphocytes and established cell lines *ex vivo*. This resistance is obviously exerted at multiple levels, yet a major restriction during lentiviral infection occurs during the early steps of infection, that is, during those steps comprised between viral entry and viral DNA integration.

To date, a single viral non structural protein has been identified as capable of removing this restriction in primary myeloid cells, the Vpx protein. Vpx is coded by members of the SIV_{SM}/HIV-2 lineage, but is absent in HIV-1 and in most of the remaining SIV lineages. We have already shown that Vpx is not only required for the completion of infection by the parental HIV-2 and SIV_{SM} viruses, but also that Vpx exerts a positive effect during the infection of myeloid cells with a number of distantly related lentiviruses, like HIV-1, FIV and EIAV. In all these cases, Vpx promoted the accumulation of complete viral DNA and this accumulation correlated with an increased efficacy of infection.

The effect of Vpx has been linked to the action of an E3 ubiquitin ligase complex, but this action remains controversial and the mechanism and the effects through which

it may be exerted are unclear. In an effort to understand the mechanistic role of Vpx in the removal of the restriction to lentiviral infection existing in myeloid cells, we have performed a wide analysis of the post-translational modifications that over the years have been described for Vpx, ubiquitination, phosphorylation, and so forth and we have tried to correlate these properties to the functionality of Vpx proteins during the early steps of infection. These and other results will be presented here.

Work supported by Sidaction, ANRS and MNERT.