

Interplay between HIV-1 replication and RNAi effectors

Christine Chable-Bessia, Oussama Meziane, Daniel Latreille, Robinson Triboulet, Stéphane Emiliani, Olivier Schwartz, Yamina Bennasser, Monsef Benkirane

▶ **To cite this version:**

Christine Chable-Bessia, Oussama Meziane, Daniel Latreille, Robinson Triboulet, Stéphane Emiliani, et al.. Interplay between HIV-1 replication and RNAi effectors. Frontiers of Retrovirology: Complex retroviruses, retroelements and their hosts, Sep 2009, Montpellier, France. pp.O13, 10.1186/1742-4690-6-S2-O13 . inserm-00663606

HAL Id: inserm-00663606

https://inserm.hal.science/inserm-00663606

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.

Retrovirology

BioMed Central

Oral presentation

Open Access

Interplay between HIV-1 replication and RNAi effectors

Christine Chable-Bessia[1], Meziane Oussama[1], Latreille Daniel[1], Triboulet Robinson[1], Emiliani Stéphane[2], Schwartz Olivier[3], Bennasser Yamina*[1] and Benkirane Monsef[1]

Address: [1]Institut de Génétique Humaine CNRS UPR1142. Montpellier, France, [2]Institut Cochin, INSERM U567, CNRS UPR8104, Paris, France and [3]Institut Pasteur, URA CNRS 3015, Paris Cedex 15, 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):O13 doi:10.1186/1742-4690-6-S2-O13

This abstract is available from: http://www.retrovirology.com/content/6/S2/O13

© 2009 Chable-Bessia et al; licensee BioMed Central Ltd.

RNA silencing involving small non coding RNA is a mechanism for gene regulation as well as an innate host cell defence mechanism against viruses. miRNA genes are most often transcribed by RNApolII, and the resulting primary (pri)-miRNA is processed in the nucleus by the RNAse type III Drosha to produce precursor (pre)-miRNA. Pre-miRNAs are then exported to the cytoplasm by Exportin-5 and processed into miRNA/miRNA* (guide/passenger) duplexes through the action of the cytoplasmic type III RNAse Dicer. miRNA/miRNA* is incorporated into the RNA-Induced Silencing Complex (RISC) where miRNA* is degraded, with miRNA serving as a guide for its mRNA target. miRNA-armed RISC targets specific mRNA to inhibit its translation or induce its degradation. Accumulating evidence suggests that the miRNA pathway also controls the replication of both RNA and DNA viruses. We have recently provided evidence for a physiological role of the miRNA-silencing machinery in controlling HIV-1 replication and latency. Type III RNAses Dicer and Drosha, responsible for miRNA processing, inhibited virus replication in both PBMCs from HIV-1 infected donors and in latently infected cells. Additionally, cellular miRNAs can target HIV-1 mRNA to induce latency. Finally, HIV-1 actively regulates the expression of cellular miRNA which regulate virus replication. We will present further evidence and discuss the involvement of miRNA effectors and cellular miRNA in both activation and repression of HIV-1 replication.