

Antisense protein of HTLV-2 (APH-2) associates with PML nuclear bodies: molecular determinants and functional implications

Chloé Journo, Jocelyn Turpin, Estelle Douceron, Anaïs Oliva, Renaud Mahieux

► To cite this version:

Chloé Journo, Jocelyn Turpin, Estelle Douceron, Anaïs Oliva, Renaud Mahieux. Antisense protein of HTLV-2 (APH-2) associates with PML nuclear bodies: molecular determinants and functional implications. *Retrovirology*, BioMed Central, 2014, 11 (Suppl 1), pp.P100. <inserm-00924963>

HAL Id: inserm-00924963

<http://www.hal.inserm.fr/inserm-00924963>

Submitted on 7 Jan 2014

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

Antisense protein of HTLV-2 (APH-2) associates with PML nuclear bodies: molecular determinants and functional implications

Chloé Journo^{*}, Jocelyn Turpin, Estelle Douceron, Anaïs Oliva, Renaud Mahieux

From 16th International Conference on Human Retroviruses: HTLV and Related Viruses
Montreal, Canada. 26-30 June 2013

Antisense Protein of HTLV-2 (APH-2) was described in 2009. APH-2 mRNA is expressed *in vivo* in most HTLV-2 carriers. In recent years, several laboratories have searched for similarities and/or differences between APH-2 and the antisense protein of HTLV-1, HBZ. Similarly to HBZ, APH-2 negatively regulates HTLV-2 transcription. However, it does not promote cell proliferation. *In vivo*, APH-2 localizes in discrete nuclear domains distinct from nucleoli. We therefore characterized APH-2 subcellular localization, in order to decipher the determinants of such localization and to correlate it or not with APH-2 functions. We first identify APH-2-containing nuclear domains as PML nuclear bodies (PML-NB). PML-NB are modulators of a number of cellular processes ranging from transcription regulation to cell proliferation and death. We show that both an *in silico*-identified nuclear localization signal and the carboxy-terminal LXXLL motif contribute to APH-2 targeting to PML-NB. Covalent modification of APH-2 by SUMO-1 and non-covalent interaction between APH-2 and SUMO-1-modified cellular partners have also been investigated as mechanisms of APH-2 targeting to PML-NB. Our results further demonstrate that APH-2 association with PML-NB is critical for its ability to inhibit viral transcription. This association also leads to a striking decrease in APH-2 stability, suggesting that APH-2 might be active but also targeted to degradation in PML-NB. Finally, we show that APH-2 localization in PML-NB leads to PML-NB clustering and correlates with a decrease in cell proliferation. Altogether, our study sheds new light on the links between the subcellular localization of APH-2 and its cellular functions.

Published: 7 January 2014

doi:10.1186/1742-4690-11-S1-P100

Cite this article as: Journo *et al.*: Antisense protein of HTLV-2 (APH-2) associates with PML nuclear bodies: molecular determinants and functional implications. *Retrovirology* 2014 **11**(Suppl 1):P100.

* Correspondence: chloe.journo@ens-lyon.fr

Oncogenèse Rétrovirale, Equipe Labellisée Ligue Nationale Contre le Cancer, CIRI, INSERM U1111-CNRS UMR5308, Université Lyon 1, Ecole Normale Supérieure de Lyon, LabEx ECOFECT, Lyon, France

Submit your next manuscript to BioMed Central and take full advantage of:

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution

Submit your manuscript at
www.biomedcentral.com/submit

