



HAL
open science

Localization and sub-cellular shuttling of HTLV-1 Tax with the microRNA machinery

Rachel van Duyne, Irene Guendel, Aarthi Narayanan, Kylee Kehn-Hall,
Elizabeth Jaworski, Jessica Roman, William Coley, Zachary Klase, Anastas
Popratiloff, Renaud Mahieux, et al.

► **To cite this version:**

Rachel van Duyne, Irene Guendel, Aarthi Narayanan, Kylee Kehn-Hall, Elizabeth Jaworski, et al..
Localization and sub-cellular shuttling of HTLV-1 Tax with the microRNA machinery. *Retrovirology*,
2014, 11 (Suppl 1), pp.O73. inserm-00924968

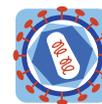
HAL Id: inserm-00924968

<https://inserm.hal.science/inserm-00924968>

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.



ORAL PRESENTATION

Open Access

Localization and sub-cellular shuttling of HTLV-1 Tax with the microRNA machinery

Rachel Van Duyne^{1,2}, Irene Guendel¹, Aarthi Narayanan¹, Kylene Kehn-Hall¹, Elizabeth Jaworski¹, Jessica Roman¹, William Coley², Zachary Klase³, Anastas Popratiloff⁴, Renaud Mahieux⁵, Fatah Kashanchi^{1,2*}

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

The innate ability of the human cell to silence endogenous retroviruses through RNA sequences encoding microRNAs suggests that the cellular RNAi machinery is a major means by which the host mounts a defense response against retroviruses. Indeed, cellular miRNAs target and hybridize to specific sequences of both HTLV-1 and HIV-1 viral transcripts. However, the virus itself contains various mechanisms that assist in the evasion of viral inhibition through control of the cellular RNAi pathway. Retroviruses can hijack components of the RNAi pathway, in some cases to produce novel viral miRNAs that can either assist in active infection or promote a latent state. Here, we show that HTLV-1 Tax contributes to the dysregulation of the RNAi pathway by altering the expression of key components. A survey of uninfected and HTLV-1 infected cells revealed that Drosha is present at lower levels in all HTLV-1 infected cell lines and infected primary cells, while other components such as DGCR8 were not dramatically altered. We show co-localization of Tax and Drosha in the nucleus *in vitro* as well as co-immunoprecipitation in the presence of proteasome inhibitors, indicating that Tax interacts with Drosha and may target it to specific areas of the cell, namely, the proteasome. In the presence of Tax we observed a prevention of primary miRNA cleavage by Drosha. Finally, the changes in cellular miRNA expression in HTLV-1 infected cells can be mimicked by the add back of Drosha or the addition of antagomiRs against the cellular miRNAs which are down-regulated by the virus.

Authors' details

¹School of Systems Biology, National Center for Biodefense & Infectious Diseases, George Mason University, Manassas, Virginia, USA. ²Department of Microbiology, Immunology, & Tropical Medicine, The George Washington University Medical Center, Washington, D.C., USA. ³Molecular Virology Section, Laboratory of Molecular Microbiology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland, USA. ⁴Department of Anatomy and Regenerative Biology, The George Washington University, Washington, D.C., USA. ⁵Retroviral Oncogenesis Team, INSERM-U758 Virologie Humaine, Lyon, France.

Published: 7 January 2014

doi:10.1186/1742-4690-11-S1-O73

Cite this article as: Van Duyne *et al.*: Localization and sub-cellular shuttling of HTLV-1 Tax with the microRNA machinery. *Retrovirology* 2014 **11**(Suppl 1):O73.

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



* Correspondence: fkashanc@gmu.edu

¹School of Systems Biology, National Center for Biodefense & Infectious Diseases, George Mason University, Manassas, Virginia, USA
Full list of author information is available at the end of the article