

Transcriptomic analyses reveal that the cellular Gem protein promotes HTLV-1 infected cell migration and viral transmission

Sébastien Chevalier, Cynthia Pise-Masison, Antoine Gessain, Renaud Mahieux

► **To cite this version:**

Sébastien Chevalier, Cynthia Pise-Masison, Antoine Gessain, Renaud Mahieux. Transcriptomic analyses reveal that the cellular Gem protein promotes HTLV-1 infected cell migration and viral transmission. *Retrovirology*, BioMed Central, 2014, 11 (Suppl 1), pp.O64. <inserm-00924961>

HAL Id: inserm-00924961

<http://www.hal.inserm.fr/inserm-00924961>

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

Transcriptomic analyses reveal that the cellular Gem protein promotes HTLV-1 infected cell migration and viral transmission

Sébastien A Chevalier^{1*}, Cynthia A Pise-Masison², Antoine Gessain³, Renaud Mahieux¹

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

In a previous study, we used gene expression microarrays and functional assays to identify cellular genes whose expression profiles were similarly affected by Tax proteins from all three HTLV subtypes (HTLV-1, HTLV-2 and HTLV-3). We found forty-eight genes up-regulated by all three Tax proteins (Chevalier *et al*, *Plos One*, 2012). Among those, *Gem*, which encodes a member of the Ras GTP-binding proteins superfamily, was strongly up-regulated. Herein, we first show that *Gem* expression is strongly up-regulated at the protein level not only in Tax-expressing cells, but also in all tested HTLV-infected cell lines and in primary uncultured T lymphocytes isolated from TSP/HAM patients. We then demonstrate that Tax activates transcription from the Gem promoter through the recruitment of CREB and CBP/p300 onto a cAMP Responsive Element (CRE). Gem protein has been shown to regulate reorganization of the cell cytoskeleton. Since efficient transmission of HTLV-1 from infected to uninfected T cells is mediated by cell-cell contacts, whose formation relies on cytoskeletal reorganization, we investigated the impact of Gem expression on cell migration and formation of cell-cell contacts. Our results show that Gem-overexpressing T lymphocytes display an increased spontaneous migration, while Gem-knocked down HTLV-infected cell lines show a strong reduction in their ability to migrate. We also observe that Gem enhances conjugate formation between infected and non-infected T lymphocytes. Altogether, our results indicate that Gem could be essential for the cell-to-cell spread of HTLV.

Authors' details

¹Oncogenèse Rétrovirale, label "Ligue Nationale Contre le Cancer", CIRI, LabEx ECOFECT, INSERM U1111 - CNRS UMR 5308, Ecole Normale Supérieure - Université Lyon 1, Lyon, Cedex 07, France. ²Animal Models and Retroviral Vaccine Section, Vaccine Branch, CCR, National Cancer Institute, National Institutes of Health, Bethesda, MD, USA. ³Epidémiologie et Physiopathologie des Virus Oncogènes, CNRS UMR 3569, Pasteur Institute, Paris, Cedex 15, France.

Published: 7 January 2014

doi:10.1186/1742-4690-11-S1-O64

Cite this article as: Chevalier *et al*: Transcriptomic analyses reveal that the cellular Gem protein promotes HTLV-1 infected cell migration and viral transmission. *Retrovirology* 2014 **11**(Suppl 1):O64.

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: sebastien.chevalier@ens-lyon.fr

¹Oncogenèse Rétrovirale, label "Ligue Nationale Contre le Cancer", CIRI, LabEx ECOFECT, INSERM U1111 - CNRS UMR 5308, Ecole Normale Supérieure - Université Lyon 1, Lyon, Cedex 07, France

Full list of author information is available at the end of the article

