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ORAL PRESENTATION

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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

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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.

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* 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

