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▶ **To cite this version:**

Ambroise Marçais, Katia Hanssens, Lucy Cook, Thomas Mercher, Philippe Gaulard, et al.. Recurrent TET2 mutations in adult T cell leukemia. Retrovirology, 2014, 11 (Suppl 1), pp.P119. inserm-00924970

HAL Id: inserm-00924970

https://inserm.hal.science/inserm-00924970

Submitted on 7 Jan 2014

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RETROVIROLOGY

POSTER PRESENTATION Open Access

Recurrent TET2 mutations in adult T cell leukemia

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

Deregulation of DNA methylation has been recently identified in malignant hematologic diseases such as inactivation of the Ten-ElevenTranslocation 2 (TET2) gene by haplo-insufficiency. Inactivating mutations of TET2 were first described in myeloid disorders and more recently in peripheral T-cell lymphomas especially those that are harboring T follicular helper features like angio-immunoblastic T cell lymphoma. In order to determine new oncogenic pathways in Adult T cell leukemia/lymphoma (ATLL) that could cooperate with viral oncogenic proteins, we investigated the presence of TET2 coding sequence mutations and their clinical relevance in a retrospective cohort of 49 ATL patients. We identify inactivating mutations of TET2 gene in 10 patients of 49 analyzed (20%). Of the 37 patients with aggressive forms (acute and lymphoma), 9 (24%) had TET2 mutations as only one (8%) of the 12 with indolent forms had a TET2 mutation. This last patient had nevertheless a poor outcome and died four years from the diagnosis from relapse. In addition, five patients show the same recurrent point mutation, which conducts to the loss of coding sequence in one allele and lead to haplo-insufficiency. Analysis of different clinical cases suggests that TET2 mutation could be acquired at different steps of the T cell oncogenesis and could even in some cases precede HTLV-1 infection. Analysis of viral integration is still ongoing. Here, we show that loss of TET2 is frequently associated with ATLL and seems to more frequent in aggressive forms.

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Published: 7 January 2014

doi:10.1186/1742-4690-11-S1-P119
Cite this article as: Marçais *et al.*: Recurrent TET2 mutations in adult T cell leukemia. *Retrovirology* 2014 **11**(Suppl 1):P119.

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