



MEETING ABSTRACT

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HTLV-1 bZIP factor enhances TGF-beta signaling through p300 coactivator

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From 15th International Conference on Human Retroviruses: HTLV and Related Viruses
Leuven and Gembloux, Belgium. 5-8 June 2011

Adult T-cell leukemia (ATL) is a neoplastic disease caused by Human T-cell leukemia virus type 1 (HTLV-1). ATL cells possess a CD4+CD25+ phenotype, similar to that of regulatory T cells (Treg). The HTLV-1 bZIP factor (HBZ), which is consistently expressed in ATL, has a critical role in the development of ATL and HAM/TSP. In the present study, we found that HBZ enhanced TGF-beta/Smad transcriptional activity in a manner dependent on p300. Co-immunoprecipitation assay confirmed that HBZ interacted with Smad3, and formed a ternary complex with Smad3 and p300. In the presence of HBZ, the interaction between Smad3 and p300 was enhanced. The N-terminal LXXLL motif of HBZ was essential for HBZ-mediated TGF-beta signaling activation, while Smad3 interacted with HBZ through its C-terminal MH2 domain. Furthermore, physiological level of HBZ could rescue the repressed TGF-beta responses by Tax. We also found that HBZ activated transcription of the Foxp3 gene through its Smad site. Our study shows that HBZ enhances TGF-beta signaling while Tax suppresses this pathway, and this enhancing activity leads to transcription of Foxp3 gene.

doi:10.1186/1742-4690-8-S1-A142

Cite this article as: Zhao et al.: HTLV-1 bZIP factor enhances TGF-beta signaling through p300 coactivator. *Retrovirology* 2011 **8**(Suppl 1):A142.

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Published: 6 June 2011

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