RETROVIROLOGY

MEETING ABSTRACT Open Access

HTLV-3 and HTLV-4 antisense proteins activate JunB-, c-Jun- and JunD-dependent transcription

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

Background

The antisense transcript-encoded HBZ protein acts upon viral and cellular gene expression through its modulation of the activation of transcription factors such as Jun family members. A possible link between HBZ and ATL development has also been highlighted. HTLV-3 and HTLV-4 are two newly discovered human retroviruses closely related to HTLV-1; no known diseases have yet been associated to these viruses. Based on previous in silico analyses, our aim was to test for the presence of antisense transcripts in these new viruses and to functionally assess these viral proteins.

Results

By RT-PCR analyses, HTLV-3 and HTLV-4 were shown to produce spliced antisense transcripts termed APH (Antisense Protein of HTLV)-3 and APH-4. Confocal microscopy analyses of cells expressing Myc- or GFP-tagged APH-3 and APH-4 showed distinct localization but partial colocalization with HBZ to the nucleus and the nucleoli. Using LTR-luciferase constructs, we demonstrated that APH-3 and APH-4 inhibited Tax1 and Tax3 transactivation of respective LTRs. In transfection experiments with a collagenase promoter-driven luciferase reporter construct, we showed that APH-3 and APH-4 modulated JunB- and c-Jun-dependent transcription differently from HBZ. Deletion mutants indicated that this upregulation of the transactivation potential of Jun factors was mediated through the atypical bZIP domain of APH-3 and APH-4.

Conclusion

We show that HTLV-3 and HTLV-4 express new viral proteins, APH-3 and APH-4, which modulate Jun-dependent transcription differently from HBZ. These data underscore the importance of our study on APH-3 and APH-4 to help in better understanding the role of HBZ in the development of ATL.

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Published: 6 June 2011

doi:10.1186/1742-4690-8-S1-A141
Cite this article as: Larocque *et al.*: HTLV-3 and HTLV-4 antisense proteins activate JunB-, c-Jun- and JunD-dependent transcription. *Retrovirology* 2011 **8**(Suppl 1):A141.

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