## Title
HTLV-1 bZIP factor suppresses c-Fos transcription and impairs T cell activation

## Author(s)
Kawatsuki, Akihiro; Yasunaga, Jun-ichiro; Matsuoka, Masao

## Citation
Retrovirology (2014), 11(Suppl 1)

## Issue Date
2014-01-07

## URL
http://hdl.handle.net/2433/180649

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## Type
Journal Article

## Textversion
publisher
HTLV-1 bZIP factor suppresses c-Fos transcription and impairs T cell activation

Akihiro Kawatsuki*, Jun-ichiro Yasunaga, Masao Matsuoka

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

HTLV-1 bZIP factor (HBZ) is responsible for the suppressed c-fos transcription in ATL cells. The results of reporter assay implied the roles of LXXLL motif of HBZ on the suppression of c-fos. HBZ has been reported to suppress AP-1 and NFAT signaling pathways through the direct interaction with c-Jun and NFATc2, respectively. We found c-Fos overexpression impairs the suppressive effects of HBZ on AP-1 and NFAT, suggesting that HBZ overcomes the inhibitory effects of c-Fos by suppressing its transcription. HBZ is known to bind to c-Jun instead of c-Fos. Suppressed transcription of c-fos facilitates HBZ to interact with c-Jun, and enhances suppressive effect of HBZ on AP-1 pathway. AP-1 and NFAT signaling pathways are activated by T-cell receptor (TCR) stimulation, leading to T cell activation. We found that TCR stimulation induces the c-fos up-regulation and the expression of the activation marker CD69 on CD4+ T cells of wild type mice, but not of HBZ transgenic mice, indicating that the activation of the signaling pathways initiated from TCR are suppressed by HBZ. We hypothesize that c-fos suppression by HBZ may contribute to impaired activation of T cells and pathogenesis of HTLV-1 infection.

Published: 7 January 2014

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