Retrovirology

Poster presentation

CCAAT/Enhancer-binding Protein-mediated Inhibition of HTLV-I Viral Gene Expression

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from 2005 International Meeting of The Institute of Human Virology
Baltimore, USA, 29 August – 2 September 2005

Published: 8 December 2005

Retrovirology 2005, **2**(Suppl 1):P79 doi:10.1186/1742-4690-2-S1-P79

Recently, CCAAT/enhancer-binding protein (C/EBP) transcription factors have been shown to form heterodimers with cAMP-responsive element binding protein 2 (CREB-2), another bZIP transcriptional regulator known to play a critical role in driving basal and Tax-mediated transactivation of the HTLV-1 long terminal repeat (LTR). Herein, we describe that overexpression of C/EBPa and C/EBPb, including the endogenous isoforms liver-enriched activation protein (LAP) and liver-enriched inhibitory protein (LIP) inhibits Tax-mediated transactivation of the HTLV-1 LTR. C/EBP-mediated inhibition was not the result of competition with the viral oncoprotein Tax for recruitment of co-activators CREB-binding protein (CBP)/p300 to the viral promoter. Electrophoretic mobility shift analyses demonstrated that C/EBP proteins derived from U-937 monocytic nuclear extracts directly interacted with the Tax-responsive element 1 repeat III as well as Tax-responsive element 2. However, deletion of these sequences within the context of the full-length LTR did not prevent C/EBP-mediated inhibition. Disruption of C/EBPb/CREB-2 heterodimerization by deletion of the C/EBPb leucine zipper prevented C/EBP inhibition of Tax-mediated transactivation of the LTR. These results suggest that C/EBP binding to Tax was not a requirement for C/EBP-mediated inhibition.