Retrovirology

BioMed Central

Oral presentation

Characterization of C/EBP Binding Sites Downstream of the Transcriptional Start Site in the HIV-1 LTR

Yujie Liu, Michael Nonnemacher*‡, Aikaterini Alexaki and Brian Wigdahl

Address: Department of Microbiology and Immunology and Institute for Molecular Medicine and Infectious Disease, Drexel University College of Medicine, Philadelphia, PA

Email: Michael Nonnemacher* - mrn25@drexel.edu

* Corresponding author ‡Presenting author

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):S155 doi:10.1186/1742-4690-2-S1-S155

Previous studies have shown that at least one upstream CCAAT enhancer binding protein (C/EBP) site was necessary for HIV-1 LTR activity in cells of the monocyte/macrophage lineage. However, no investigation has been performed to date on C/EBP sites downstream (DS) of the start of transcription. Analyses of 115 clade B LTRs indicated there are three potential C/EBP sites within the downstream LTR region. Electrophoretic mobility shift (EMS) analyses demonstrated one of the three sites (DS3) was able to bind members of the C/EBP family. Analyses of clade A, C, and D LTRs indicated this site was highly conserved among different clades, suggesting the presence of a functionally important cis-acting element. In comparison to the clade B consensus DS3 element, EMS analysis demonstrated the DS3 7A variant exhibited a relative high affinity for C/EBP factors, while the 3C and 7G configurations exhibited lower affinities. Additional studies demonstrated specific DS3 variants exhibited differences in relative affinity for full-length and truncated C/EBPb. Transient transfection studies utilizing parental LTRs derived from LAI, YU.2, and 89.6 molecular clones containing the DS3 7A, 3C, or 7G variants exhibited altered LTR activity compared to their parental strains. These results have suggested that DS3 plays a role in regulating HIV-1 transcription.