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Oral presentation

HIV-1 LTR Activity is Altered by Recruitment of Sp Transcription Factors During Monocytic Differentiation

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Viral replication, in part, is mediated by interactions between the HIV-1 long terminal repeat (LTR) and a variety of host cell and viral proteins. Basal and activated LTR activity is dependent on interactions between the G/C box array of the HIV-1 LTR and the Sp family of transcription factors. The effect of monocytic differentiation on Sp factor binding and transactivation has been examined with respect to the HIV-1 LTR. Primary monocyte-derived macrophages (MDM), as well as monoblastic (U-937 and THP-1) and myelomonocytic (HL-60) cell lines were utilized in both the absence and presence of chemical differentiating agents to model selected aspects of monocytic differentiation. The binding of Sp1, full-length Sp3, and truncated Sp3 to a high affinity HIV-1 Sp element was examined utilizing electrophoretic mobility shift analyses. Sp1 binding increased relative to the sum of full-length and truncated Sp3 binding following PMA-induced monocytic differentiation in the cell lines. Sp binding ratios obtained with nuclear extracts from PMA-induced cell lines were also shown to correlate with those derived from studies performed with extracts from primary MDMs. This Sp binding phenotype was shown to alter the transcriptional activation generated by the HIV-1 G/C box array.