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Poster presentation

The AP-1 binding sites located in the *pol* gene intragenic regulatory region of HIV-1 are important for virus infectivity

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We have previously identified three AP-1 binding sites in the *pol* gene of human immunodeficiency virus type 1 (HIV-1) and shown that short oligonucleotides containing these sites functioned as phorbol ester-inducible enhancers (Van Lint et al., 1991, J. Virol., 65:7066-7072). These sites are located in a region, called fragment 5103, exhibiting a phorbol ester-inducible enhancing activity on the viral thymidine kinase promoter in HeLa cells. In this study, we have further characterized each of the AP-1 binding sites and have shown that transcription factors c-Fos, JunB and JunD interacted *in vitro* with these motifs. For each site, we have identified mutations abolishing AP-1 factor binding without altering the underlying amino acid sequence of the HIV-1 reverse transcriptase. By transient transfection assays, we have demonstrated that the intragenic AP-1 binding sites were entirely responsible for the PMA-dependent transcriptional activity of fragment 5103. Moreover, this PMA-stimulated activity of fragment 5103 was inhibited by a dominant-negative A-Fos mutant provided the AP-1 sites were not mutated. Finally, we have investigated the biological significance of the intragenic AP-1 binding sites in HIV-1 replication and have shown that these sites are important for viral infectivity.