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Role of Sp1 binding site and TAR sequence in the onset or development of PML

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PML is a demyelinating disease of SNC caused by JCV. The non coding control region (NCCR) of JCV archetype (CY strain) is divided into six regions: A-F containing binding sites for cell factors involved in viral transcription. The different variants of JCV are characterized by rearranged NCCR with deletions and enhancements of these binding sites giving rise to variants that could promote viral gene expression and could be more suitable for the onset or development of PML. We analysed NCCR structures, found in CSF of HIV positive and negative subjects both with PML and we focused on Sp1 binding sites (box B and D) and up-TAR sequence (box C). It is known that Sp1 activates JCV early promoter and can contribute in maintaining methylation-free CpG islands in active genes, while up-TAR sequence is important for HIV-1 Tat stimulation of JCV late promoter. Our results show that in HIV-positive subjects all NCCR structures were characterized by enhancements of up-TAR element, demonstrating the possible synergism between HIV-1 and JCV in the CNS, whereas in HIV-negative subjects both Sp1 binding sites were always retained.

Therefore we can hypothesize that both Sp1 binding sites could be necessary for JCV to complete its replication cycle in absence of HIV coinfection.