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Opinion Article

Significance of a Highly Pathogenic Lentivirus: HIV-1

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DESCRIPTION

Human Immunodeficiency Virus type 1 (HIV-1) is a highly pathogenic lentivirus that requires provirus genome transcription to complete the viral life cycle and produce progeny virions. Much has been known about the transcriptional control of the HIV-1 genome in infected cells since the initial genomic investigation of HIV-1 in 1985. HIV-1 transcription is dependent on a diverse and complicated interaction of host cell transcription factors with the viral long terminal repeat (LTR) promoter. The LTR regulatory elements interact with constitutive and inducible transcription factors to drive the formation of a stable transcription complex that promotes several rounds of transcription by RNA Polymerase II (RNAPII). However without the virally encoded trans-activator protein Tat, which promotes HIV-1 transcription elongation by interacting with a stem-loop RNA element (TAR) generated at the extreme 5′ end of all viral transcripts, the bulk of these transcripts end prematurely. The initiation-elongation complex is joined by a cellular kinase that changes the elongation characteristics of RNAPII as it passes through TAR as a result of the Tat-TAR interaction. This study highlights the significant contributions that human lentivirus gene regulation has made to our overall understanding of the transcription process, summarizing our present knowledge and understanding of the regulation of HIV-1 transcription in infected cells.

HIV-1 is a highly pathogenic lentivirus that causes Acquired Immune Deficiency Syndrome (AIDS). HIV-1 infects CD4-positive T cells and macrophages indefinitely by inserting a DNA copy of its genome into the host cell chromosome. Following the incorporation of the DNA provirus, RNA Polymerase II (RNAPII)-mediated genomic length transcription is required to complete the viral life cycle and create progeny virions. The 5′ Long Terminal Repeat (LTR) controls HIV-1 genome transcription, which is dependent on host cell transcription factors binding to an array of DNA cis-regulatory sites in the LTR promoter.

According to transcriptional analyses of the HIV-1 genome, the 636-base pair HIV-1 LTR promoter can be divided into four functional domains: an upstream enhancer element with two adjacent binding sites for the inducible transcriptional activator Nuclear Factor kappa B (NF-B); an upstream regulatory region with elements; and a basal core promoter with three tandem Sp1 binding sites. These components work together to control the amount of HIV-1 transcription in a certain cell type. The LTR encodes a unique RNA element, the Trans-Activation Response (TAR), which is situated directly downstream of the transcription start site, in addition to the different DNA elements.

TAR generates a 59-nucleotide RNA stem-loop structure that serves as a target for the virally encoded trans-activator protein Tat at the extreme 5′ end of all HIV-1 transcripts. Tat is a viral regulatory protein that is produced from multiply spliced viral mRNAs early in infection. Tat enhances the amount of full-length transcripts *via* binding to TAR. Tat appears to be essential for the fast increase in genomic length transcription required for the change from a dormant to an active viral infection.

CONCLUSION

HIV-1 transcription is strictly controlled by a large number of host cell transcription factors, two virally encoded regulatory proteins, and a complex interaction between these cellular and viral factors. These components' intracellular concentrations and activity, as well as their DNA-protein and protein-protein cooperative interactions on the HIV-1 promoter, ultimately dictate the unique expression pattern of HIV-1 in every given cell type or in response to any given cellular activation signal. This vast array of regulatory elements was most likely acquired by HIV-1 to ensure its expression in a wide range of cell types and in response to a wide range of cell activation indicators.

Although HIV-1 is one of the most extensively researched transcription units in biology, its intriguing regulatory mechanisms continue to draw researchers to it. In fact, HIV-1 has developed into a crucial research tool for understanding both the specifics of lentivirus transcription as well as transcriptional mechanisms in general. Therefore, clarification of the specific mechanism of Tat function will be necessary for a more thorough knowledge of HIV-1 transcription.

Also, a more thorough explanation of the host cell transcription factors involved in HIV-1 transcription and their particular function in the remodelling of the HIV-1 promoter's chromatin will be necessary. Further molecular research on the regulation of HIV-1 transcription will be essential for the future hope given the significance of HIV-1 as a human pathogen.

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