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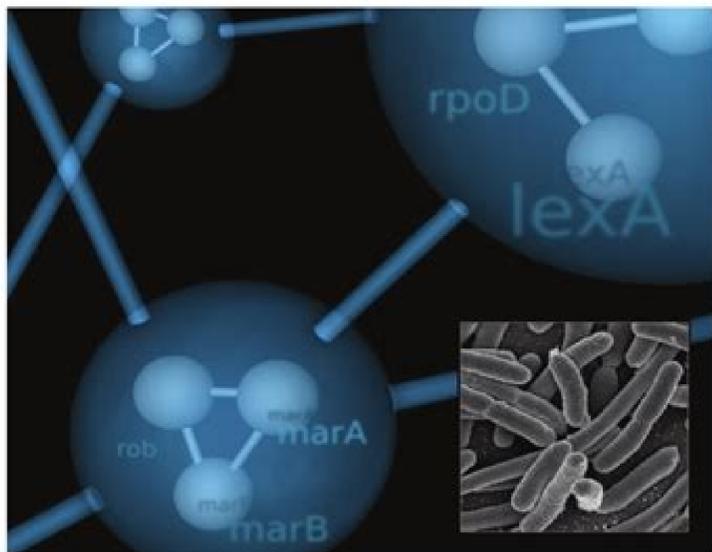
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Regulation of HIV-1 gene transcription: from lymphocytes to microglial cells

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Abstract: Transcription is a crucial step for human immunodeficiency virus type 1 (HIV-1) expression in all infected host cells, from T lymphocytes, thymocytes, monocytes, macrophages, and dendritic cells in the immune system up to microglial cells in the central nervous system. To maximize its replication, HIV-1 adapts transcription of its integrated proviral genome by ideally exploiting the specific cellular environment and by forcing cellular stimulatory events and impairing transcriptional inhibition. Multiple cell type-specific interplays between cellular and viral factors perform the challenge for the virus to leave latency and actively replicate in a great diversity of cells, despite the variability of its long terminal repeat region in different HIV strains. Knowledge about the molecular mechanisms underlying transcriptional regulatory events helps in the search for therapeutic agents that target the step of transcription in anti-HIV strategies. *J. Leukoc. Biol.* 74: 736–749; 2003.

Key Words: cellular specificity · LTR · Tat · acetylation · monocytes

INTRODUCTION

Human immunodeficiency virus type 1 (HIV-1) replication is regulated by multiple events occurring at different steps of the viral life cycle [1]. After fusion-mediated entry within host cells, uncoating, reverse transcription of the RNA genome and nuclear entry of the preintegration complex, the proviral DNA is integrated into the host cell genome. Transcription of the HIV-1 provirus is then regulated by an interplay between a combination of distinct viral and cellular transcription factors with binding sites present in the HIV-1 long terminal repeat (LTR) [for review, see refs. [2–4]]. The LTR is divided in functional regions designated by transactivation response element (TAR), core, enhancer, and modulatory elements. The TAR region binds the viral transactivator Tat and the core region contains the initiator (Inr), the TATA box, and three Sp1-binding sites. While the enhancer element binds nuclear factor (NF)- κ B and NF of activated T cells (NFAT) transcription factors, the modulatory region harbors numerous target sequences for a variety of cellular transcription factors such as NF- κ B-interleukin (IL)-6, cyclic AMP (cAMP) response element-binding protein (CREB), Ets, and nuclear hormone receptors.

These host-cell factors regulate LTR-driven transcription not only by direct binding to their target DNA sequence but also by indirect binding via DNA-bound proteins. Extensive studies have described how cis- and trans-acting elements regulate LTR-driven transcription, but few have focused on the ability of the viral genome to adjust its transcriptional mechanism to each specific cell type.

This review highlights the extraordinary capacity of HIV to adapt its transcriptional strategy to different cellular environments. We describe the mechanisms underlying the cell type-specific transcription directed by the HIV-1 LTR in T4 lymphocytes, thymocytes, monocytes, macrophages, and microglial cells. In addition, we present LTR variations in different HIV-1 isolates. New advances suggest that the transcription step of the viral life cycle could be used as a target for an anti-HIV strategy.

CELLULAR SPECIFICITY OF HIV-1 GENE TRANSCRIPTION

When the HIV-1 provirus is integrated in the host genome, early-phase transcription is regulated by cellular transcription factors and results in the production of early viral gene products. The late phase of transcription is under the control of Tat, which potently enhances gene expression by a direct binding to TAR-RNA and association with cyclin T1 (Cyt1), which recruits the cyclin-dependent kinase 9 (cdk9). Formation of this positive transcription-elongation factor b (P-TEFb) complex leads to the phosphorylation of the C-terminal domain of RNA polymerase II (RNAPII) and efficient elongation. These two steps of proviral transcription occur in various cell types, which indicate that distinct combinations of factors mediate transcription in a cell type-specific manner.

REGULATION OF HIV-1 TRANSCRIPTION BY CELLULAR TRANSCRIPTION FACTORS

The following studies illustrate the high complexity and the cell-type specificity of the early phase of HIV-1 gene transcription.

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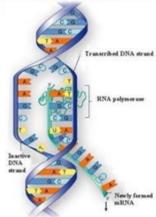


Deciphering principles of transcription regulation in eukaryotic genomes

Transcription regulation is a complex process involving a wide variety of transcription factors and regulatory elements. The study of these mechanisms is essential for understanding gene expression and cellular differentiation. This review discusses the principles of transcription regulation in eukaryotic genomes, focusing on the role of transcription factors and the organization of regulatory elements.

Introduction
 Transcription is the first step in the central dogma of molecular biology, where genetic information is transferred from DNA to RNA. This process is tightly regulated by a variety of transcription factors and regulatory elements. The study of these mechanisms is essential for understanding gene expression and cellular differentiation. This review discusses the principles of transcription regulation in eukaryotic genomes, focusing on the role of transcription factors and the organization of regulatory elements.

OVERVIEW OF TRANSCRIPTION



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