Post-transcriptional regulation of human viral infection: a mechanistic perspective.

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Introduction

Post-transcriptional regulation refers to the various processes that occur after a gene is transcribed into RNA, but before the protein is synthesized. This includes splicing, mRNA stability, translation initiation and RNA modification. In the case of viruses, post-transcriptional regulation plays a crucial role in viral gene expression and replication. Viral gene expression is regulated by a complex interplay of viral and host factors. Viruses have evolved numerous mechanisms to manipulate host cellular machinery and co-opt it for their own replication. Post-transcriptional regulation of viral gene expression is one such mechanism by which viruses control the synthesis of their proteins. This allows the virus to precisely regulate the timing and amount of viral protein expression, and evade host immune responses.

Splicing of viral RNA is a key post-transcriptional regulatory mechanism that allows for the generation of multiple proteins from a single viral gene. Viruses such as HIV and influenza produce multiple mRNA isoforms through alternative splicing, allowing for the synthesis of different proteins from a single viral gene [1]. Splicing is regulated by specific cis-acting sequences present in the viral RNA, as well as by host splicing factors that are recruited by the virus. For example, the HIV-1 protein that binds to a specific RNA sequence to recruit the host splicing factor, U1 snRNP, which promotes the efficient splicing of viral RNA.

Another important mechanism of post-transcriptional regulation is the stability of viral mRNA. The stability of mRNA is determined by the length of the poly(A) tail and the presence of specific RNA-binding proteins that bind to the mRNA and protect it from degradation. Viruses have evolved various strategies to manipulate the stability of their mRNA. For example, the hepatitis B virus (HBV) destabilizes its mRNA by recruiting host proteins that promote mRNA decay. On the other hand, the Kaposi's sarcoma-associated herpes virus (KSHV) stabilizes its mRNA by encoding viral proteins that bind to the mRNA and protect it from degradation.

Translation initiation is another critical step in post-transcriptional regulation that controls the rate of protein synthesis. The initiation of translation requires the assembly of the ribosome on the mRNA and the recruitment of specific initiation factors. Viruses have evolved numerous mechanisms to manipulate the initiation of translation to control the synthesis of viral proteins [2]. For example, the hepatitis C virus (HCV) uses an internal ribosome entry site (IRES) to recruit the ribosome and initiate translation in a cap-independent manner. Similarly, the human cytomegalovirus (HCMV) encodes a viral protein, pUL38, which recruits the host translation initiation factor eIF3 to promote the efficient translation of viral mRNA. Finally, RNA modification is another important mechanism of post-transcriptional regulation that can affect the stability and translation of viral mRNA. RNA modification refers to the addition or removal of chemical groups to the RNA molecule, which can alter its structure and function. Viruses such as HIV and HCV modify their RNA by adding a methyl group to the 5' end of the RNA, which enhances translation initiation and stabilizes the RNA.

In addition to the above mechanisms, viruses also encode specific proteins that can directly regulate post-transcriptional processes. For example, the HIV-1 protein Rev binds to a specific RNA sequence to promote the export of viral RNA from the nucleus to the cytoplasm, where it can be translated into protein. Similarly, the HCMV protein pUL69 inhibits the splicing of viral RNA, allowing for the synthesis of a specific viral protein [3]. Post-transcriptional regulation of viral gene expression refers to the mechanisms by which the expression of viral genes is controlled at the level of RNA processing, stability, and translation. This regulation plays a critical role in determining the outcome of viral infection, including viral replication, immune evasion, and pathogenesis. One important mechanism of post-transcriptional regulation is alternative splicing. Viruses can utilize alternative splicing to generate multiple isoforms of their mRNAs from a single gene. This allows viruses to increase the diversity of their gene products and potentially evade host immune responses. For example, the human cytomegalovirus (HCMV) encodes multiple spliced variants of its major immediate early gene (IE1) that have different functions and can modulate the host immune response.

RNA editing is another mechanism of post-transcriptional regulation that allows viruses to modify their RNA sequences after transcription. This can result in changes to the viral genome and the production of different protein isoforms. For example, the hepatitis B virus (HBV) undergoes RNA editing to generate a truncated form of its surface antigen that is associated with viral persistence and immune evasion [4,5].

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In addition, viruses can regulate the stability of their mRNAs through interactions with host RNA-binding proteins and microRNAs. These interactions can affect the stability and translation of viral mRNAs and ultimately determine the level of viral gene expression. For example, the human immunodeficiency virus (HIV) encodes the Rev protein, which binds to a specific RNA sequence in the viral mRNA and promotes its nuclear export. This ensures that the viral mRNA is available for translation and increases the efficiency of viral gene expression. Finally, viruses can also modulate the translation of their mRNAs through interactions with host translation factors and ribosomes. This can affect the efficiency of protein synthesis and ultimately the level of viral gene expression. For example, the hepatitis C virus (HCV) encodes an internal ribosome entry site (IRES) in its mRNA that allows for cap-independent translation initiation. This allows the virus to bypass the host translation machinery and efficiently translate its mRNA.

In conclusion, post-transcriptional regulation of viral gene expression is a complex and highly regulated process that involves multiple mechanisms to control RNA processing, stability, and translation. Understanding these mechanisms is critical for developing strategies to control viral infections and limit their pathogenic effects.

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