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MicroRNAs: Dual Role in Viral Infections and Therapeutics

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Introduction

MicroRNAs (miRNAs) exert a complex and often opposing influence on viral infections, demonstrating a dual role that significantly impacts the host-pathogen interaction. These small non-coding RNA molecules can act as potent antiviral effectors, targeting viral genomes or host factors essential for viral replication to restrict viral spread. Conversely, viruses have evolved sophisticated mechanisms to exploit host miRNAs or encode their own to promote their replication, evade host immunity, and establish persistent infections. This intricate molecular interplay profoundly shapes the outcome of viral pathogenesis, influencing disease severity, chronicity, and the host's ability to mount an effective defense. Understanding these multifaceted roles is crucial for deciphering the dynamics of viral infections and for developing novel therapeutic interventions. The ability of miRNAs to fine-tune gene expression at a post-transcriptional level makes them pivotal regulators in this complex biological arena. Their involvement spans from the initial stages of viral entry and replication to the modulation of the host's innate and adaptive immune responses. This delicate balance can tip the scales in favor of either the host or the virus, determining the ultimate clinical manifestation of the infection.

Viruses are masters of molecular manipulation, and their strategies often involve actively hijacking the host cell's miRNA machinery to their own advantage. This manipulation can manifest in various ways, including the downregulation of cellular miRNAs that would normally act to suppress viral replication, thereby removing a key host defense mechanism. Simultaneously, viruses may upregulate specific host miRNAs that facilitate different stages of their life cycle, such as promoting viral entry into new cells, aiding in viral assembly, or assisting in the release of new virions. This viral-driven reprogramming of the host miRNA landscape is a critical strategy employed by many viruses to ensure their persistence within the host and to effectively evade the immune system's detection and clearance mechanisms. This adaptive capacity highlights the remarkable evolutionary arms race between viruses and their hosts.

As a vital component of the host's innate immune response to viral infections, host-encoded miRNAs play a crucial role in defending against invading pathogens. These cellular miRNAs can directly target viral RNA molecules, leading to their degradation, or they can inhibit the function of host proteins that are essential for viral replication. By interfering with these critical viral processes, host miRNAs act as intrinsic antiviral effectors, limiting the spread of the virus throughout the host. However, the delicate balance of miRNA expression can be disrupted during infection, and dysregulation of these host miRNAs can significantly impair the effectiveness of the antiviral immune response, potentially leading to more severe disease outcomes. The intricate regulatory networks involving miRNAs underscore their importance in maintaining cellular homeostasis and host defense.

Beyond their roles in host defense, the study of specific viral miRNAs has unveiled their sophisticated mechanisms for manipulating host gene expression. Viruses, particularly those with large genomes like herpesviruses and retroviruses such as HIV, often encode their own unique miRNAs. These viral miRNAs are designed to precisely modulate host cellular processes, including immune responses and metabolic pathways, in a manner that promotes viral latency or replication and effectively suppresses host defense pathways. The discovery of these viral-encoded miRNAs has opened up new avenues for understanding viral pathogenesis and has identified these viral miRNAs as promising targets for the development of novel antiviral therapies. Their specific targeting of host machinery makes them attractive candidates for therapeutic intervention.

The dysregulation of cellular miRNAs has been increasingly implicated in the pathogenesis of a wide range of viral diseases, underscoring their broad impact on viral infections. Conditions such as chronic hepatitis C virus (HCV) infection and influenza have been shown to be influenced by altered miRNA profiles within infected cells. Specific cellular miRNAs can exhibit characteristics of oncomiRs, promoting cellular transformation and contributing to the development of cancer, or they can function as tumor suppressor-like molecules, although their role in viral infections is complex and context-dependent. This dysregulation can significantly influence host cell transformation and disease progression, particularly in the context of chronic viral infections where persistent inflammation and cellular stress are common. Understanding these alterations is key to comprehending the long-term consequences of viral infections.

The dynamic interplay between host miRNAs and viral replication is a critical factor in the establishment and maintenance of persistent infections. Viruses have evolved intricate strategies to overcome or evade the antiviral actions of host miRNAs. Some viruses have developed sophisticated mechanisms to counteract miRNA-mediated antiviral responses, such as by altering the expression of miRNA processing machinery or by producing molecules that can sequester or inhibit host miRNAs. These evasion tactics contribute significantly to the virus's ability to establish chronic infections, evade host immunity, and persist within the host for extended periods, often leading to long-term health complications and disease. The evolutionary pressure to evade host defenses drives viral adaptation.

The development of miRNA-based therapeutics for viral infections represents an exciting and rapidly advancing area of research. These innovative strategies aim to harness the regulatory power of miRNAs to combat viral diseases. Two primary approaches are being explored: the use of miRNA mimics, which are synthetic RNA molecules designed to restore the function of downregulated antiviral miRNAs, and the development of anti-miRs, which are complementary molecules designed to inhibit the activity of viral-promoting or host-dysregulated miRNAs. These therapeutic approaches hold significant promise for treating a diverse range of viral diseases, offering a novel modality beyond traditional antiviral drugs. Their specificity offers potential advantages.

Host miRNAs can function as direct antiviral effectors by specifically targeting viral RNA genomes or essential viral replication proteins. For example, research has

demonstrated that certain host miRNAs can effectively restrict the replication of significant viral families, including flaviviruses (such as Dengue and Zika viruses) and coronaviruses (including SARS-CoV-2). These miRNAs achieve this antiviral effect through various mechanisms, such as promoting the degradation of viral RNA or inhibiting the synthesis of vital viral proteins, thereby halting or significantly slowing down viral proliferation. This direct targeting highlights their importance in intrinsic antiviral immunity.

Viruses often encode their own small RNA molecules, known as viral miRNAs (vmiRNAs), which play multifaceted roles in modulating host-pathogen interactions. These vmiRNAs function by interfering with host cellular processes, including the regulation of immune responses and metabolic pathways, in a manner that is highly beneficial for viral replication and pathogenesis. By altering host cell functions, vmiRNAs can suppress antiviral immunity, promote viral persistence, and facilitate immune evasion. Consequently, a thorough understanding of these viral miRNAs is paramount for the development of effective and targeted antiviral strategies that specifically counteract their detrimental effects on the host.

The host cell's sophisticated miRNA machinery can be ingeniously hijacked by viruses to precisely fine-tune the expression of host genes that are critical for mounting an effective antiviral defense. This viral manipulation can involve altering the expression of key components of the innate immune system, such as interferon-stimulated genes (ISGs) and cytokine signaling pathways, which are vital for initiating and coordinating the antiviral response. Furthermore, viruses can interfere with host apoptotic processes, preventing programmed cell death that would otherwise eliminate infected cells, ultimately favoring viral propagation and survival. This reprogramming of host defense mechanisms is a hallmark of viral adaptation.

Description

MicroRNAs (miRNAs) play a critical dual role in viral infections, acting both to restrict viral replication by targeting viral genomes or host factors essential for the virus, and conversely, being exploited by viruses to promote their own replication or evade host immunity. This intricate interplay significantly shapes the outcome of viral pathogenesis, influencing disease severity and chronicity. Understanding these molecular mechanisms offers avenues for novel therapeutic strategies [1].

Viruses actively manipulate host miRNAs to their advantage, often downregulating cellular miRNAs that would normally suppress viral replication or upregulating specific miRNAs that facilitate viral entry, assembly, or release. This viral-driven miRNA reprogramming is a key strategy for viral persistence and immune evasion [2].

Host-encoded miRNAs are a crucial component of the innate immune response to viral infections. They can directly target viral RNA or host proteins involved in viral replication, thereby limiting viral spread. Dysregulation of these host miRNAs during infection can impair antiviral immunity [3].

The study of specific viral miRNAs, such as those encoded by herpesviruses or HIV, reveals their sophisticated mechanisms for modulating host gene expression to promote viral latency or replication and suppress host defense pathways. These viral miRNAs represent promising targets for antiviral therapies [4].

Dysregulation of cellular miRNAs has been implicated in the pathogenesis of various viral diseases, including hepatitis C virus (HCV) and influenza. Specific miRNAs can act as oncomiRs or tumor suppressor-like molecules, influencing host cell transformation and disease progression in the context of chronic viral infections [5].

The interplay between host miRNAs and viral replication is highly dynamic and can lead to the establishment of persistent infections. Some viruses have evolved mechanisms to evade miRNA-mediated antiviral responses, contributing to their ability to establish chronic infections [6].

The development of miRNA-based therapeutics for viral infections is an active area of research. Strategies include using miRNA mimics to restore antiviral functions or anti-miRs to inhibit viral-promoting miRNAs. These approaches hold promise for treating a range of viral diseases [7].

Specific host miRNAs can directly target viral RNA genomes or proteins essential for viral replication. For instance, certain miRNAs have been shown to restrict the replication of flaviviruses and coronaviruses by degrading viral RNA or inhibiting viral protein synthesis [8].

Viruses often encode their own miRNAs that function to modulate host cellular processes, including immune responses and metabolic pathways, thereby promoting viral replication and pathogenesis. Understanding these viral miRNAs is key to developing targeted antiviral strategies [9].

The host cell's miRNA machinery can be hijacked by viruses to fine-tune the expression of host genes critical for antiviral defense. This includes manipulating the expression of interferon-stimulated genes, cytokine signaling pathways, and apoptotic processes, ultimately favoring viral propagation [10].

Conclusion

MicroRNAs (miRNAs) play a dual role in viral infections. They can act as antiviral agents by targeting viral components or host factors, restricting viral replication. Conversely, viruses exploit host miRNAs or encode their own to promote replication and evade immune responses. This complex interplay influences disease severity and chronicity. Host miRNAs are crucial for innate immunity, directly targeting viral RNA or proteins. However, their dysregulation can weaken antiviral defense. Viruses manipulate host miRNAs to downregulate antiviral responses and upregulate factors that aid their life cycle, facilitating persistence and immune evasion. Specific viral miRNAs are adept at modulating host gene expression to promote latency or replication and suppress immunity. Dysregulation of cellular miRNAs is linked to the pathogenesis of various viral diseases, affecting host cell transformation and disease progression. The dynamic interaction between host and viral miRNAs can lead to persistent infections, with viruses evolving mechanisms to evade miRNA-mediated immunity. Emerging miRNA-based therapeutics, using mimics or anti-miRs, offer promising new strategies for treating viral infections by restoring antiviral functions or inhibiting viral-promoting miRNAs. Understanding these intricate miRNA-mediated mechanisms is vital for developing effective antiviral therapies.

Acknowledgement

None.

Conflict of Interest

None.

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