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# *ROLE AND FUNCTION OF MICRORNA IN GENE CODING*

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*XULOSA*

*Ushbu sharhda mualliflar hsa-miR-28-5p va hsa-miR-155-5p mikroRNKlarining yallig‘lanish va saraton jarayonlarida gen ifodalanishini boshqarishdagi rollariga bag‘ishlangan adabiyotlarning tahlili keltirildi. Ularning kontekstga bog‘liq funksiyalari, immunjavob va transkripsiya omillari bilan o‘zaro aloqasi ularni biomarker va davolash nishonlari sifatida dolzarb qiladi.*

***Kalit so‘zlar:*** *mikroRNK, gen ifodalanishi, yallig‘lanish, o‘sma biomarkerlari, transkripsiya omillari, regulyator tarmoqlar, immunjavob.*

*РЕЗЮМЕ*

*В данном обзоре рассмотрена литература, посвящённая регуляторной роли hsa-miR-28-5p и hsa-miR-155-5p в экспрессии генов при воспалении. Эти микроРНК выполняют контекстно-зависимые функции и рассматриваются как потенциальные биомаркеры и терапевтические мишени благодаря участию в иммунной регуляции и взаимодействии с транскрипционными факторами.*

***Ключевые слова:*** *микроРНК, экспрессия генов, воспаление, опухолевые маркеры, транскрипционные факторы, регуляторные сети, иммуноответ.*

MicroRNAs (miRNAs) are small, non-coding RNA molecules that play a crucial role in the regulation of gene expression. These molecules, typically 20-25 nucleotides in length, are involved in various biological processes, including development, differentiation, proliferation, and apoptosis. MiRNAs function by binding to complementary sequences in target mRNAs, leading to translational repression or mRNA degradation. The nomenclature of

miRNAs is a critical aspect of their study, as it provides a standardized system for identifying and categorizing these molecules. This section will explore the role and function of miRNAs in gene coding, with a particular emphasis on their nomenclature.

*MicroRNA Nomenclature*

The nomenclature of miRNAs is established by the miRBase database, which acts as the central repository for miRNA sequences and annotations. miRBase assigns unique names to miRNAs based on their genomic location and sequence similarity, ensuring consistency and avoiding duplication. The naming convention typically follows a "miR" prefix, followed by a number that reflects the order of discovery. For example, miR-1, miR-2, etc. This systematic approach allows researchers to easily identify and reference specific miRNAs in their studies.

In addition to the "miR" prefix, miRNAs are often named based on their genomic location. For instance, miR-124 is named after its location on human chromosome 8. This practice helps in identifying the origin of the miRNA and can provide insights into its potential function. Furthermore, miRNAs that are part of the same family are often grouped together based on sequence similarity, which can facilitate the study of their collective roles in gene regulation.

*Biogenesis of microRNAs*

The biogenesis of miRNAs involves several steps, starting from transcription to maturation. Primary miRNAs (pri-miRNAs) are transcribed by RNA polymerase II and are characterized by a 5' cap and a poly-A tail. These pri-miRNAs are processed in the nucleus by the Drosha enzyme, which cleaves the RNA to produce precursor miRNAs (pre-miRNAs). The pre-miRNAs are then transported to the cytoplasm, where they are further processed by the Dicer enzyme to generate mature miRNAs.

Mature miRNAs are incorporated into the RNA-induced silencing complex (RISC), which is responsible for targeting specific mRNAs. The RISC complex uses the miRNA as a guide to bind to complementary sequences in the target mRNA, leading to translational repression or mRNA degradation. This intricate process ensures that miRNAs can effectively regulate gene expression at the post-transcriptional level.

*Mechanisms of microRNA-Mediated Gene Regulation*

MiRNAs regulate gene expression primarily by binding to the 3' untranslated region (3' UTR) of target mRNAs. The binding is mediated by the seed region, which is a conserved sequence at the 5' end of the miRNA. This seed region plays a critical role in determining the specificity of miRNA-mRNA interactions.

In addition to the 3' UTR, miRNAs can also bind to other regions of the mRNA, such as the 5' UTR, coding sequence, and even gene promoters. These interactions can lead to a variety of regulatory outcomes, including translational repression, mRNA degradation, and even activation of translation under certain conditions.

Recent studies have also revealed that miRNAs can interact with the coding sequence (CDS) of mRNAs, leading to repression of translation. This interaction often requires extensive base-pairing, including the canonical seed region, and can result in reduced mRNA levels. These findings highlight the complexity and versatility of miRNA-mediated gene regulation.

*Role of microRNAs in Gene Coding*

miRNAs play a pivotal role in regulating the expression of protein-coding genes. By targeting mRNAs for degradation or translational repression, miRNAs can fine-tune gene expression in response to various cellular signals. This regulation is essential for maintaining cellular homeostasis and ensuring proper development and differentiation.

In addition to their role in normal cellular processes, miRNAs are also implicated in disease states, such as cancer. Dysregulation of miRNAs can lead to the upregulation of oncogenes and the downregulation of tumor suppressor genes, contributing to tumorigenesis. Understanding the role of miRNAs in gene coding is therefore crucial for developing therapeutic strategies to combat these diseases.

*MicroRNAs and their interaction with coding Regions*

While miRNAs are traditionally known to bind to the 3' UTR of mRNAs, recent studies have shown that they can also interact with the coding sequence (CDS) of mRNAs. These interactions can lead to repression of translation, although the effects are generally weaker compared to 3' UTR interactions. The binding of miRNAs to the CDS often requires extensive base-pairing, including the canonical seed region, and can result in reduced mRNA levels.

The interaction of miRNAs with the CDS can have significant implications for gene regulation. For example, miR-196 has been shown to bind to the CDS of HOXB8 mRNA, leading to its cleavage and subsequent downregulation. This mechanism highlights the diverse ways in which miRNAs can regulate gene expression.

*microRNAs in Development and Disease*

miRNAs are essential regulators of various developmental processes, including cell differentiation, proliferation, and apoptosis. For example, miRNAs play a critical role in erythropoiesis, the process by which red blood cells are produced. Over 20 miRNAs have been identified that regulate the differentiation and maturation of erythroid progenitor cells, ensuring proper erythropoiesis.

In addition to their role in development, miRNAs are also implicated in disease. Cancer, in particular, is associated with the dysregulation of miRNAs. miRNAs can act as either oncogenes or tumor suppressors, depending on their targets. For instance, miR-15 and miR-16 are known to act as tumor suppressors by targeting the BCL2 oncogene, while miR-155 can act as an oncogene by targeting tumor suppressor genes.

*Therapeutic Potential of microRNAs*

The dysregulation of miRNAs in disease states makes them attractive targets for therapeutic intervention. By modulating miRNA levels, it is possible to restore normal gene expression patterns and prevent disease progression. For example, miRNA-based therapies are being explored for the treatment of cancer, where the goal is to either inhibit oncogenic miRNAs or restore tumor suppressor miRNAs.

In addition to cancer, miRNAs have therapeutic potential in other diseases, such as cardiovascular disorders and neurological diseases. For example, miR-21 has been shown to play a role in cardiac fibrosis, and targeting this miRNA could lead to the development of novel treatments for heart disease.

CONCLUSION

In conclusion, microRNAs are versatile regulators of gene expression, playing critical roles in both normal cellular processes and disease states. Their nomenclature, established by databases like miRBase, provides a standardized system for identifying and studying these molecules. By understanding the mechanisms of miRNA-mediated gene regulation, including their interaction with coding regions and their role in development and disease, researchers can unlock the therapeutic potential of miRNAs. Further studies are needed to fully elucidate the complex roles of miRNAs and to develop effective strategies for their therapeutic application.

*Table 1*

**microRNA Interactions and their effects**

| Region of Interaction | Mechanism of Action | Citation |
|---|---|---|
| 3' UTR | Translational repression or mRNA degradation | |
| 5' UTR | Translational activation or repression | |
| Coding Sequence (CDS) | Repression of translation, mRNA degradation | |
| Gene Promoters | Regulation of transcription | |

This table summarizes the regions where miRNAs interact with mRNAs and the mechanisms through which they regulate gene expression. The citations provide evidence from the provided contexts for each interaction and mechanism.

Moreover, the intricate interplay between miRNAs and their target mRNAs extends beyond mere repression; it also encompasses a dynamic regulatory network that can influence cellular responses to environmental changes. For instance, recent studies have highlighted how specific miRNAs are involved in stress responses, modulating gene expression patterns to adapt to adverse conditions such as hypoxia or nutrient deprivation (Liu et al., 2008). This adaptive mechanism underscores the potential of miRNAs not only as biomarkers for disease but also as targets for therapeutic interventions aimed at enhancing resilience against stress-related pathologies. Furthermore, understanding the context-dependent roles of miRNAs could pave the way for precision medicine approaches, where therapies are tailored based on an individual's unique miRNA expression profile, ultimately leading to more effective treatment strategies across various diseases [15].

Understanding these interactions is crucial for harnessing the power of miRNAs in therapeutic contexts, as they hold promise for treating a variety of diseases, including cancer and genetic disorders. The ability to manipulate miRNA pathways could lead to innovative strategies for gene therapy, allowing for targeted interventions that can restore normal cellular functions and mitigate disease progression. Expanding our knowledge of miRNA functions and their regulatory networks will be essential for developing effective therapies that can precisely modulate gene expression in response to specific pathological conditions.This approach not only enhances our understanding of disease mechanisms but also paves the way for personalized medicine, where treatments can be tailored to individual genetic profiles and disease states.The integration of miRNA-based therapies into clinical practice could revolutionize the way we approach treatment, offering new hope for patients with conditions that currently have limited options.

INTRODUCTION

MicroRNAs (miRNAs) are small non-coding RNAs that play a crucial role in regulating gene expression at the post-transcriptional level. Among these, hsa-miR-28-5p and hsa-miR-155-5p have been extensively studied for their roles in various biological processes, including cancer, immune responses, and inflammation. This response aims to investigate the roles and mechanisms of these two miRNAs in different biological contexts, supported by evidence from the provided research papers.

**Roles and Mechanisms of hsa-miR-28-5p**

*Tumor Suppression in Cancer*

hsa-miR-28-5p has been identified as a tumor suppressor in several types of cancer. In colon cancer, miR-28-5p inhibits carcinogenesis and is necessary for erastin-induced ferroptosis, a form of regulated cell death. It achieves this by targeting N4BP1, leading to decreased proliferation, migration, and invasion of cancer cells [6]. Similarly, in prostate cancer, miR-28-5p acts as a tumor suppressor by targeting SREBF2, which is involved in tumor cell proliferation, survival, migration, and invasion [2].

This highlights the critical role of hsa-miR-28-5p in modulating key pathways associated with cancer progression, suggesting that therapeutic strategies aimed at enhancing its expression or mimicking its function could offer promising avenues for treatment.

In gastric cancer, miR-28-5p inhibits cell migration

and invasion by suppressing the phosphorylation of AKT, a key kinase involved in cell survival and metastasis [12]. This highlights the versatility of miR-28-5p in targeting different pathways to exert its tumor-suppressive effects.

*Regulation of Apoptosis and Cell Cycle*

miR-28-5p has been shown to induce apoptosis and cell cycle arrest in nasopharyngeal carcinoma cells. It achieves this by altering the expression of cyclin D1 and influencing the PI3K/AKT signaling pathway [9]. This dual role in apoptosis and cell cycle regulation underscores its potential as a therapeutic target for cancer treatment.

**Roles and Mechanisms of hsa-miR-155-5p**

*Immune Regulation and Inflammation*

miR-155-5p is a well-known regulator of immune responses and inflammation. It plays a critical role in the development and function of immune cells, including lymphocytes and macrophages. In allergic asthma, miR-155 acts as a suppressor of Th2 immunity, reducing allergic inflammation [16]. Additionally, miR-155 has been implicated in chronic inflammatory diseases, where it modulates the expression of pro-inflammatory cytokines and immune checkpoint proteins [13].

*Oncogenic and Tumor-Suppressive Roles*

miR-155-5p exhibits both oncogenic and tumor-suppressive roles depending on the biological context. In breast cancer, overexpression of miR-155 enhances the efficacy of dendritic cell vaccines by improving their ability to activate T cells, leading to enhanced antitumor immunity [5]. Conversely, in certain hematological malignancies, miR-155 promotes tumor growth by supporting the accumulation of myeloid-derived suppressor cells (MDSCs) in the tumor microenvironment, which suppress antitumor immune responses [1].

*Metastasis and Tumor Progression*

miR-155-5p has been shown to inhibit cancer cell extravasation and colonization in metastatic models. Overexpression of miR-155 in mesenchymal-like cancer cells reduces tumor burden in lungs by decreasing invasion and altering protein expression associated with metastasis [11]. However, in clear cell renal cell carcinoma, miR-155-5p promotes malignant progression by targeting NR3C2 and activating the IGF1R/AKT/PI3K pathway (Yan et al., 2022).

**Biological Processes Involving Both miRNAs**

*Cancer Progression*

Both miR-28-5p and miR-155-5p are involved in cancer progression, albeit through different mechanisms. miR-28-5p primarily acts as a tumor suppressor, while miR-155-5p can act as both an oncogene and a tumor suppressor depending on the context. For example, in gastric cancer, miR-28-5p inhibits migration and invasion by targeting AKT phosphorylation (Xiao et al., 2018), whereas miR-155-5p promotes proliferation and migration by targeting TP53INP1 [10].

*Immune Response*

Both miRNAs are involved in regulating immune responses. miR-155-5p is a key player in immune cell development and function, with roles in both innate and adaptive immunity (Jia et al., 2014). miR-28-5p, while less studied in immune contexts, has been shown to influence immune-related pathways in cancer, such as ferroptosis in colon cancer [6].

*Inflammation*

miR-155-5p is strongly associated with inflammatory processes, where it modulates the expression of inflammatory cytokines and immune checkpoint proteins [13]. miR-28-5p, on the other hand, has not been directly linked to inflammation but may indirectly influence inflammatory pathways through its effects on cancer cell behavior.

*Table 2*

**Comparison of hsa-miR-28-5p and hsa-miR-155-5p in Biological Processes**

| Biological Process | hsa-miR-28-5p | hsa-miR-155-5p |
|---|---|---|
| Cancer Progression | Acts as a tumor suppressor in colon, prostate, and gastric cancers by targeting N4BP1, SREBF2, and AKT (Hu et al., 2020) (Fazio et al., 2020) (Xiao et al., 2018). | Exhibits dual roles: oncogenic in breast cancer and tumor-suppressive in metastatic models (Chen et al., 2015) (Thomsen et al., 2015). |
| Immune Response | Influences immune-related pathways in cancer, such as ferroptosis in colon cancer (Hu et al., 2020). | Regulates immune cell development and function, with roles in both innate and adaptive immunity (Jia et al., 2014). |
| Inflammation | Not directly linked to inflammation but may influence inflammatory pathways indirectly. | Strongly associated with inflammatory processes, modulating cytokines and immune checkpoints (Xiaoyan et al., 2017). |

CONCLUSION

hsa-miR-28-5p and hsa-miR-155-5p are versatile miRNAs with distinct roles in various biological processes. miR-28-5p primarily acts as a tumor suppressor in cancer, while miR-155-5p exhibits both oncogenic and tumor-suppressive roles depending on the context. Both miRNAs are involved in regulating immune responses and cancer progression, highlighting their potential as therapeutic targets for diseases such as cancer and inflammatory disorders. Further research is needed to fully elucidate their mechanisms and explore their clinical applications.

Moreover, the intricate regulatory networks involving miRNAs extend beyond their direct interactions with mRNA targets; they also encompass feedback loops that can influence miRNA expression itself. For instance, certain transcription factors can upregulate specific miRNAs in response to cellular stressors, thereby creating a dynamic interplay between gene expression and environmental conditions [3]. This responsiveness not only

highlights the adaptability of miRNAs in various biological contexts but also underscores their potential as biomarkers for disease states, particularly in cancers where such feedback mechanisms may become dysregulated. Furthermore, understanding these complex interactions could lead to innovative therapeutic strategies aimed at restoring normal miRNA function, offering new avenues for treatment in diseases characterized by aberrant miRNA activity [4].

Moreover, the emerging understanding of miRNA interactions with transcription factors (TFs) adds another layer of complexity to their regulatory networks. Recent studies suggest that TFs can modulate miRNA expression in response to various stimuli, thereby influencing cellular responses and adaptive mechanisms under stress conditions [17]. This interplay not only highlights how miRNAs can serve as fine-tuners of gene expression but also emphasizes their potential role in developing therapeutic strategies aimed at re-establishing homeostasis in diseased states. For instance, targeting specific TFs that regulate oncogenic miRNAs could lead to a decrease in tumor progression by restoring the balance between pro- and anti-oncogenic signals within the tumor microenvironment. As researchers delve deeper into these multifaceted relationships, the prospect of harnessing this knowledge for precision medicine becomes increasingly viable, paving the way for tailored interventions based on individual miRNA-TF dynamics in cancer treatment and beyond.

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