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Microrna s (Mirnas): Biogenesis, Mechanisms, Roles In Cancer, And Future Prospects

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Abstract
Small, non-coding RNAs known as microRNAs (miRNAs) have become important modulators in the pathophysiology of human cancers because they control gene expression post-transcriptionally. Their dual function as tumor suppressors and oncogenes (oncomiRs) enables them to intricately regulate a variety of cellular processes, such as angiogenesis, invasion, metastasis, apoptosis, and proliferation. Tumors start, progression, and resistance to treatment are all impacted by the disruption of these processes caused by dysregulation of miRNA expression. While tumors suppressor miRNAs like let-7, miR-34a, and miR-15/16 decrease tumors growth by targeting oncogenes, oncomiRs like miR-21, miR-155, and the miR-17-92 cluster promote oncogenesis by repressing tumor suppressor genes and activating oncogenic pathways. Furthermore, via influencing vascular development, extracellular matrix remodelling, and the epithelial–mesenchymal transition (EMT), miRNAs control the angiogenic switch and the metastatic cascade. Early cancer diagnosis and patient stratification are made possible by miRNAs' stable presence in physiological fluids, which has established them as promising non-invasive diagnostic and prognostic biomarkers. Clinical trials are still being conducted to determine the effectiveness of therapeutically targeting aberrant miRNAs with antisense inhibitors or miRNA mimics in order to restore normal gene regulation networks. Developments in nanoparticle carriers and chemical alterations are speeding up the use of miRNA-based treatments, notwithstanding delivery issues and off-target effects. Prospects for the future include using genome editing technologies for precise modulation, utilising personalized miRNA profiles for customized therapies, and combining miRNA therapeutics with traditional and immune-based treatments. Heralding a new age in precision oncology, miRNAs together provide a flexible platform for enhancing cancer detection, prognosis, and treatment.

Keywords: *miRNA, Biogenesis, Mechanisms, Cancer*

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I. Introduction

A type of tiny, non-coding RNA molecules of approximately 18–25 nucleotides, microRNAs (miRNAs) are essential for post-transcriptional gene control in a variety of biological systems. Since their discovery in the early 1990s in Caenorhabditis elegans miRNAs have been acknowledged as evolutionarily conserved modulators of gene expression in viruses, plants, and animals[1,2] (Bartel, 2018). The canonical pathway is the main route taken by miRNA biogenesis, in which RNA polymerase II transcribes primary miRNA transcripts (pri-miRNAs), which are then processed successively by the RNase III enzymes Drosha and Dicer to produce mature miRNA duplexes that are integrated into the RNA-induced silencing complex (RISC). The maturation of miRNA is further diversified by non-canonical mechanisms including mirtrons and Dicer-independent processing[1,2] (Treiber et al., 2019; O'Brien et al., 2023).

Mechanistically, translational repression or mRNA degradation results from miRNAs directing RISC to partially complementary target sites, usually found in the 3′ untranslated region (3′UTR) of messenger RNAs. Emerging research identifies target-directed miRNA degradation (TDMD), RNA editing, and non-canonical interactions as further regulatory layers in addition to traditional binding modalities[3] (McGeary et al., 2022).

Dysregulated miRNA expression in cancer biology influences hallmarks like proliferation, apoptosis resistance, angiogenesis, and metastasis by acting as tumor suppressors (e.g., miR-34a, let-7 family) or oncogenes[4] (e.g., miR-21, miR-155) (Peng & Croce, 2023). Numerous miRNA-based treatments are making it to clinical trials, and miRNA signatures are being used for therapeutic targeting, early cancer diagnosis, and prognosis.

Future directions for miRNA research include single-cell miRNA profiling, high-resolution functional annotation, and the creation of secure, targeted delivery methods for medicinal uses. The translation of miRNA biology into precision medicine is expected to proceed more quickly with the integration of artificial intelligence and multi-omics platforms[5,6] (Cui et al., 2024, and Yu et al., 2025).

II. MicroRNA Biogenesis

The multi-step biogenesis process that produces microRNAs (miRNAs) guarantees accurate creation and maturation, enabling them to govern gene expression in a controlled manner both in space and time. In metazoans, the canonical pathway is the most common mechanism, but this process can also follow non-canonical pathways.

Canonical Pathway

Like protein-coding mRNAs, miRNA genes are primarily transcribed by RNA polymerase II into lengthy primary miRNA transcripts (pri-miRNAs) in the canonical route. These transcripts are usually polyadenylated at the 3′ end and capped at the 5′ end[7] (Bartel, 2018). The Microprocessor complex, which is made up of the RNase III enzyme Drosha and its cofactor DGCR8 (DiGeorge syndrome critical region 8), recognises the distinctive stem-loop structures that pri-miRNAs generate in the nucleus[8] (Treiber et al., 2019). A 70-nucleotide precursor miRNA (pre-miRNA) hairpin is created when Drosha cleaves the pri-miRNA around 11 base pairs away from the junction after DGCR8 attaches to its double-stranded stem close to the junction with single-stranded RNA flanks.

The karyopherin Exportin-5 then exports the pre-miRNA from the nucleus to the cytoplasm in a way that is dependent on Ran-GTP[9] (Yi et al., 2003). The pre-miRNA's terminal loop is cut off by another RNase III enzyme called Dicer once it enters the cytoplasm, creating a ~22-nucleotide miRNA duplex with 2-nucleotide 3′ overhangs[10] (Kim et al., 2021). The RNA-induced silencing complex (RISC) selectively loads one strand of this duplex, known as the guide strand, where it binds to an Argonaute (AGO) protein, most frequently AGO2 in humans. While it may occasionally be functional, the opposing passenger strand (miRNA*) is usually destroyed.

Non-Canonical Pathways

One or more canonical pathway stages are circumvented by non-canonical miRNA biogenesis pathways. One example is mirtrons, which are short introns that avoid Drosha processing by forming pre-miRNA-like hairpins following splicing and debranching[10,11] (O'Brien et al., 2023). Dicer-independent biogenesis is another example, in which AGO2 cleaves specific pre-miRNAs, like miR-451, directly, obviating the need for Dicer processing (Cheloufi et al., 2010).

Regulation of Biogenesis

Production of miRNA is strongly regulated in multiple ways, including:

- **Transcriptional control** by transcription factors and epigenetic modifications[12] (Jiang et al., 2022).
- **Post-transcriptional regulation** through RNA-binding proteins (e.g., Lin28, hnRNPs) that affect Drosha or Dicer processing efficiency.
- **Subcellular localization** and **feedback loops** in which miRNAs themselves regulate components of their own processing machinery.

Cancer, neurological illnesses, and developmental problems have all been linked to disruptions in miRNA synthesis, which can result in a broad dysregulation of gene expression[13] (Peng & Croce, 2023).

III. MicroRNAs (miRNAs) action In cancer

OncomiRs

Small non-coding RNAs (about 22 nucleotides) known as microRNAs (miRNAs) control the expression of genes after transcription. They do this mainly by attaching to complementary sequences in the 3′ untranslated regions (3′ UTRs) of target mRNAs, which results in translational repression or mRNA destruction. By encouraging tumor development, progression, metastasis, and resistance to treatment, a subset of miRNAs known as oncomiRs perform oncogenic roles in the field of cancer biology. To do this, OncomiRs decrease tumor suppressor genes, alter cell proliferation-related signaling pathways, prevent apoptosis, and promote angiogenesis.

One of the best-studied oncomiRs is miR-21, which is frequently overexpressed in a variety of cancers, including as hepatocellular, lung, colorectal, and breast cancers. MiR-21 activates the PI3K/AKT pathway and promotes unchecked cell proliferation and survival by targeting many tumor suppressors, including PTEN, PDCD4, and TPM[14,15] (Liu et al., 2024). By suppressing pro-apoptotic genes (including BIM and PTEN) and boosting MYC-driven transcriptional processes, the miR-17-92 cluster, which is frequently amplified in lymphomas and solid tumors, encourages oncogenesis[15] (Yang et al., 2023).

Similar to this, miR-155 is elevated in solid tumors and hematological malignancies, which promotes inflammatory signaling and interferes with immune control by targeting SHIP1 and SOCS[16] (Huang et al., 2022). Through the suppression of p27Kip1 and p57Kip2, miR-221/222 facilitates the advancement of the cell cycle and promotes aggressive phenotypes in glioblastoma, prostate cancer, and hepatocellular carcinoma. Furthermore, by

blocking HOXD10, miR-10b increases the ability of breast cancer to spread by promoting the expression of pro-metastatic genes such RHOC[17] (Liao et al., 2024).

Preclinical models have demonstrated the potential of therapeutically targeting oncomiRs with CRISPR-based editing techniques, antisense oligonucleotides (antagomiRs), and locked nucleic acid (LNA) inhibitors. However, there are obstacles to clinical translation, such as tumour heterogeneity, off-target effects, and tailored delivery. Notwithstanding these obstacles, a number of anti-miR treatments are progressing through early-phase clinical trials, including anti-miR-155 and anti-miR-21 medicines[18] (Zhang et al., 2025). Developing precision miRNA-based oncology therapies still requires an understanding of the roles that oncomiRs play in distinct tumors and contexts.

Tumor Suppressor miRNAs in Cancer

Another subclass of miRNAs functions as tumor suppressors, preventing the spread of cancer by focusing on oncogenes and pathways involved in invasion, proliferation, and survival, whereas oncogenic microRNAs (OncomiRs) encourage the growth of tumors. Because of chromosomal deletions, epigenetic silencing, or malfunctions in the miRNA synthesis machinery, these tumor suppressor miRNAs (TS-miRNAs) are frequently downregulated in cancers, leading to unregulated oncogenic signalling[13,19].

Let-7, one of the most researched TS-miRNAs, inhibits RAS, MYC, and HMGA2 to limit cell cycle progression and proliferation. A worse prognosis has been linked to decreased let-7 expression in ovarian, breast, and lung malignancies. By inhibiting BCL2, CDK6, and MET, miR-34a, a direct transcriptional target of p53, prevents tumor growth. Therapy resistance and the possibility of metastasis are linked to its loss, which is often brought on by promoter methylation or p53 mutation. Similarly, miR-15a/16-1 targets the anti-apoptotic protein BCL2, causing apoptosis and making cancer cells more sensitive to chemotherapy. It is found at the 13q14 chromosomal region that is lost in chronic lymphocytic leukaemia[14,20].

Other noteworthy TS-miRNAs are miR-143 and miR-145, which control the c-Myc, ERK5, and KRAS pathways and function as inhibitors of carcinogenesis in bladder and colorectal malignancies. Members of the miR-200 family suppress metastasis by blocking ZEB1/2, which inhibits the epithelial–mesenchymal transition (EMT). By modifying DNA methyltransferases (DNMTs) and extracellular matrix elements, the miR-29 family also has anti-oncogenic actions that impact the tumor microenvironment and gene regulation[15,21].

One intriguing treatment approach is to restore TS-miRNA expression using viral vectors or synthetic mimics. TS-miRNA replacement treatments are getting closer to clinical use thanks to developments in chemical modifications and nanoparticle-based delivery that have enhanced stability and tumor targeting. Additionally, TS-miRNA loss in patient samples can be profiled to help with prognosis, early identification, and individualized therapy choices[16,22].

miRNAs in Metastasis and Angiogenesis

Through their modulation of signalling networks that govern cell motility, invasion, and vascular remodelling, microRNAs (miRNAs) play crucial regulatory roles in angiogenesis and metastasis, two hallmarks of cancer progression. Certain miRNAs, referred to as metastamiRs, target genes involved in cell adhesion, extracellular matrix (ECM) remodelling, and epithelial–mesenchymal transition (EMT) in metastasis, either promoting or suppressing the spread of cancer cells. One of the most researched pro-metastatic miRNAs, for instance, is miR-10b, which downregulates the metastasis suppressor HOXD10 in breast cancer, increasing RHOC and promoting cell migration and invasion[19,23] (Liao et al., 2024). Similarly, by targeting PTEN and TIMP3, miR-21 activates the PI3K/AKT and MMP pathways, which in turn promotes EMT and metastasis[18,24] (Liu et al., 2024). On the other hand, transcription factors ZEB1 and ZEB2, which are important repressors of E-cadherin, are directly targeted by metastasis-suppressive miRNAs such members of the miR-200 family, which prevent EMT[25] (Tang et al., 2023).

Angiogenesis, or the formation of new blood vessels, is essential for the growth of tumors and the dissemination of metastases. Pro-angiogenic miRNAs, sometimes referred to as angiomiRs, stimulate endothelial cell migration, proliferation, and tube formation via modifying angiogenic molecules such as VEGF. MiR-210, which is triggered by HIF-1α under hypoxic conditions, promotes angiogenesis by targeting ephrin-A3 and increasing VEGF signalling[25] (Wang et al., 2023). The miR-17-92 cluster also stimulates angiogenesis by blocking natural angiogenesis inhibitors such as connective tissue growth factor (CTGF) and thrombospondin-1[25,26] (Yang et al., 2023). However, in order to maintain vascular integrity and prevent tumor angiogenesis, anti-angiogenic miRNAs, such miR-126, target the VEGF-A and PI3K regulatory subunits[27] (Chen et al., 2024).

It's interesting to note that certain miRNAs have dual functions in angiogenesis and metastasis, establishing a connection between the two processes. For example, miR-9 increases the propensity for metastasis by increasing EMT by suppressing E-cadherin and concurrently enhancing angiogenesis by activating the JAK/STAT pathway in endothelial cells[27,28] (Zhou et al., 2022). The intricacy of miRNA-mediated regulation in the tumour microenvironment is highlighted by this multi-functionality.

Preclinical and clinical research are investigating therapeutic approaches to restore tumor-suppressive miRNAs via miRNA mimics or to block pro-metastatic and pro-angiogenic miRNAs using antisense oligonucleotides (antagomiRs). Targeted delivery to tumour and endothelial cells, reducing off-target effects, and overcoming tumour heterogeneity are still obstacles, though. Translating these discoveries into successful cancer treatments requires clarifying the context-dependent roles of miRNAs in angiogenesis and metastasis.

miRNAs as Diagnostic and Prognostic Biomarkers

Because they are expressed in specific tissues, remain stable in bodily fluids, and are linked to disease stage, subtype, and outcome, microRNAs (miRNAs) have become highly effective biomarkers for cancer diagnosis and prognosis. In contrast to mRNAs, miRNAs are not broken down by RNase and stay stable in plasma, serum, saliva, urine, and other biofluids. They are mostly shielded by exosomes, microvesicles, or RNA-binding proteins like Argonaute 2. They are perfect candidates for non-invasive "liquid biopsy" techniques because of their stability[26,28](Zhang et al., 2024).

Certain miRNA signatures can be used to diagnose cancer by differentiating between cancer types and malignant from benign tumours. For instance, miR-21 is a widely applicable diagnostic marker since it is persistently overexpressed in a variety of malignancies, including as lung, colorectal, and breast cancers[27,29] (Li et al., 2023). In contrast, prostate cancer can be distinguished from benign prostatic hyperplasia by the higher levels of miR-141 and miR-375[27,29] (Yamada et al., 2024). When compared to single markers, panels that combine many circulating miRNAs have been demonstrated to increase diagnostic sensitivity and specificity.

In terms of prognosis, miRNA expression patterns can forecast patient survival, recurrence risk, and tumour aggressiveness. In breast, ovarian, and gastric malignancies, low expression of the miR-200 family—important regulators of the epithelial–mesenchymal transition—is associated with a worse prognosis and a greater risk of metastasis[30] (Tang et al., 2023). Shorter overall survival in breast cancer and renal cell carcinoma is linked to elevated serum levels of the hypoxia-responsive miRNA miR-210[28,30] (Liu et al., 2024). Furthermore, a positive prognosis is associated with miR-34a, a tumor-suppressive miRNA that is regulated by p53; its loss frequently denotes therapeutic resistance and poorer clinical results[29,30] (Chen et al., 2023).

The precision and repeatability of miRNA detection have been improved by technological advancements including digital droplet PCR (ddPCR), next-generation sequencing (NGS), and quantitative reverse transcription PCR (qRT-PCR), which has made their clinical translation easier. There are still issues, nevertheless, such as the requirement for large-scale validation across various populations, normalisation controls, and standardised procedures.

All things considered, miRNA-based biomarkers have potential for personalised prognosis, treatment decision-making, and therapeutic response monitoring in addition to early cancer detection. Precision oncology may be greatly enhanced by combining miRNA profiling with other omics data as research advances.

IV. miRNAs In Therapeutics And Future Prospects

Since they can simultaneously regulate several oncogenic and tumor-suppressive pathways, microRNAs (miRNAs) have drawn a lot of attention as possible therapeutic targets and agents in the treatment of cancer. Two main approaches are used in therapeutic strategies: restoration of tumor-suppressor miRNAs through synthetic miRNA mimics or viral vector delivery, and inhibition of oncogenic miRNAs (oncomiRs) using antisense oligonucleotides (antagomiRs, locked nucleic acids, or miRNA sponges[30,31] (Rupaimoole, and Slack, 2022). Rebalancing the abnormal gene expression networks that cause cancer is the goal of these strategies.

Preclinical research has shown that miRNA-based treatments are effective against a range of cancer types. For instance, miR-34a mimics cause apoptosis and prevent metastasis in models of lung, breast, and liver cancer, whereas antagomiR-21 inhibits tumour growth and makes resistant cells more sensitive to chemotherapy[31,32] (Zhang et al., 2023). Furthermore, by affecting angiogenesis, immune evasion, and stromal interactions, miRNA therapies might alter the tumour microenvironment and provide a variety of anti-cancer effects.

However, there are several obstacles to clinical translation, including as immunological activation, off-target effects, stability in circulation, and effective and targeted distribution. Conjugation with tumor-targeting ligands, lipid-based vesicles, and nanoparticle-based carriers have decreased toxicity and increased delivery specificity[32] (Chen et al., 2024). Furthermore, miRNA stability and binding affinity are improved by chemical modifications like locked nucleic acids and 2′-O-methylation.

In order to overcome drug resistance and enhance results, miRNA treatments may be integrated with targeted drugs, immunotherapies, and traditional chemotherapy in the future. Customising miRNA-based treatments according to each patient's unique miRNA expression profile and genetic background is made possible by developments in personalised medicine. Furthermore, precise manipulation of miRNA genes is possible thanks to ongoing advancements in genome editing techniques like CRISPR-Cas systems[32]. New therapeutic targets and approaches will surface as knowledge of miRNA synthesis, processes, and interactions with competing

endogenous RNAs (ceRNAs) grows. Ultimately, by providing multi-targeted, flexible, and accurate methods, miRNA-based therapeutics have the potential to completely transform the way cancer is treated.

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