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# Exploring the Functions of Differentially Regulated miRNAs in Breast Cancer

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## Abstract

MicroRNAs (miRNAs) are small non-coding RNAs that play crucial roles in gene regulation and have emerged as significant players in various cancers, including breast cancer. Differentially regulated miRNAs in breast cancer have been extensively studied due to their potential as diagnostic and prognostic biomarkers, as well as therapeutic targets. This article aims to provide an overview of the functions of differentially regulated miRNAs in breast cancer, highlighting their roles in tumor initiation, progression, metastasis and drug resistance. Understanding the complex regulatory networks involving miRNAs in breast cancer could pave the way for the development of innovative therapeutic strategies and personalized treatment approaches.

**Keywords:** Breast cancer • Diagnostic • Metastasis • Cancers

## Introduction

Breast cancer is one of the most prevalent malignancies affecting women worldwide, with significant morbidity and mortality rates. Despite advances in diagnosis and treatment, the heterogeneity of breast cancer presents challenges in patient management and therapeutic efficacy. MicroRNAs (miRNAs) have emerged as critical regulators of gene expression, exerting control over various cellular processes implicated in cancer development and progression. Dysregulation of miRNA expression is a common feature of breast cancer and contributes to its pathogenesis through the modulation of key signaling pathways and regulatory network [1].

Numerous studies have identified distinct miRNA expression profiles associated with different subtypes of breast cancer, including luminal, HER2-enriched and Triple-Negative Breast Cancer (TNBC). These differentially expressed miRNAs play pivotal roles in the molecular mechanisms underlying breast cancer development and progression. For instance, miR-21, miR-155 and miR-210 are frequently upregulated in breast cancer and are associated with aggressive tumor behavior, metastasis and poor prognosis. Conversely, downregulation of tumor-suppressive miRNAs such as miR-34a, miR-200 family and let-7 family members is commonly observed and is linked to enhanced tumor growth, invasion and therapy resistance.

## Literature Review

Differentially regulated miRNAs influence various aspects of tumor initiation and progression in breast cancer. They can target key oncogenes or tumor suppressors, thereby modulating critical cellular processes such as cell proliferation, apoptosis and cell cycle regulation. For example, miR-21 promotes breast cancer cell proliferation and survival by targeting tumor suppressors PTEN and PDCD4. Similarly, miR-155 enhances tumor growth and metastasis by suppressing the expression of FOXO3a and TP53INP1, leading to increased cell proliferation and invasion [2]. EMT is a crucial process in cancer metastasis, enabling epithelial cancer cells to acquire mesenchymal properties and invade surrounding tissues. Differentially regulated miRNAs play essential roles in regulating EMT-associated pathways and promoting metastasis in breast cancer. For instance, miR-200 family members (miR-200a, miR-200b, miR-200c, miR-141, miR-429) inhibit EMT by targeting ZEB1 and ZEB2, thereby maintaining epithelial characteristics and suppressing metastatic potential. Conversely, miR-10b promotes breast cancer metastasis by targeting HOXD10, a negative regulator of EMT, leading to enhanced invasive behavior and metastatic colonization.

## Discussion

Angiogenesis, the formation of new blood vessels, is critical for tumor growth and metastasis. Differentially regulated miRNAs participate in the regulation of angiogenic pathways in breast cancer by targeting pro-angiogenic factors or anti-angiogenic inhibitors. For example, miR-126 inhibits angiogenesis by targeting VEGF-A and PIK3R2, thereby suppressing tumor growth and metastasis. Conversely, miR-210 promotes angiogenesis by targeting EFNA3 and HOXA9, leading to increased vascularization and tumor progression. Resistance to chemotherapy and targeted therapies poses a significant challenge in the management of breast cancer patients. Differentially regulated miRNAs contribute to drug resistance through various mechanisms, including the modulation of drug efflux transporters, DNA repair pathways and anti-apoptotic signaling. For instance, miR-21 confers resistance to chemotherapy by targeting PTEN and APAF1, thereby promoting cell survival and reducing drug-induced apoptosis. Similarly, miR-221/222 promotes resistance to hormonal therapy by targeting p27 and ERα, leading to enhanced proliferation and hormone independence [3-6].

## Conclusion

Differentially regulated miRNAs play multifaceted roles in breast cancer, influencing tumor initiation, progression, metastasis and drug resistance. Understanding the complex regulatory networks involving miRNAs in breast cancer is crucial for the development of novel diagnostic biomarkers and targeted therapeutic strategies. Further research efforts aimed at elucidating the functions of specific miRNAs and their downstream effectors could lead to the identification of promising therapeutic targets and personalized treatment approaches for breast cancer patients.

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## Conflict of Interest

There are no conflicts of interest by author.

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