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Commentary

Role of MicroRNAs in Osteoporosis Pathogenesis and Therapeutic Targeting

Sneha Reddy*

Department of Molecular Biology, University of Hyderabad, Hyderabad, India.

ABOVE THE STUDY

Osteoporosis is a prevalent metabolic bone disorder characterized by reduced bone mass and microarchitectural deterioration, leading to increased fracture risk. The disease arises from an imbalance between bone formation by osteoblasts and bone resorption by osteoclasts. In recent years, microRNAs (miRNAs), a class of small non-coding RNAs, have emerged as critical regulators of gene expression in bone biology. Their involvement in osteoblast and osteoclast differentiation has positioned them as key players in the pathogenesis of osteoporosis and as promising targets for therapeutic intervention.

MiRNAs function by binding to complementary sequences on target messenger RNAs (mRNAs), leading to their degradation or translational repression. This post-transcriptional regulation allows miRNAs to fine-tune complex cellular processes, including those involved in bone remodeling. In osteoporosis, dysregulation of specific miRNAs disrupts the delicate balance between bone formation and resorption. For instance, miRNAs such as miR-21, miR-29, and miR-335 have been shown to promote osteoblast differentiation by targeting inhibitors of osteogenesis, thereby enhancing bone formation. Conversely, miRNAs like miR-133 and miR-204 inhibit osteoblast activity by suppressing key transcription factors such as Runx2, contributing to decreased bone formation.

In addition to their effects on osteoblasts, miRNAs also regulate osteoclastogenesis. Certain miRNAs, including miR-148a and miR-223, promote osteoclast differentiation and activity, thereby increasing bone resorption. Others, such as miR-155, act as negative regulators of osteoclastogenesis. The dual role of miRNAs in modulating both osteoblast and osteoclast function underscores their importance in maintaining skeletal homeostasis. Dysregulation of these miRNAs can shift the balance toward bone loss, a hallmark of osteoporosis.

The expression of miRNAs is influenced by various factors associated with osteoporosis, including aging, hormonal changes, and inflammatory signals. Estrogen deficiency, a major contributor to postmenopausal osteoporosis, has been shown to alter the expression of several bone-related miRNAs. For example, decreased estrogen levels can upregulate miRNAs that promote osteoclast activity while downregulating those that support osteoblast function. Similarly, chronic inflammation, often observed in aging populations, can modulate miRNA expression through cytokine signaling pathways, further exacerbating bone loss.

One of the most promising aspects of miRNA research in osteoporosis is their potential as diagnostic biomarkers. Circulating miRNAs are stable in body fluids such as blood and serum, making them attractive candidates for non-invasive diagnosis and disease monitoring. Specific miRNA signatures have been associated with low bone mineral density and increased fracture risk, suggesting their utility in early detection and prognosis of osteoporosis.

From a therapeutic perspective, targeting miRNAs offers a novel strategy for modulating bone remodeling. Approaches such as miRNA mimics and antagomirs (anti-miRNA oligonucleotides) can be used to restore normal miRNA function. For example, delivering miRNA mimics that promote osteoblast differentiation or inhibiting miRNAs that enhance osteoclast activity could help rebalance bone remodeling in osteoporotic patients. Advances in nanotechnology have facilitated the development of targeted delivery systems that improve the stability and specificity of miRNA-based therapies, reducing off-target effects.

Despite these advances, several challenges hinder the clinical translation of miRNA-based therapies. These include issues related to delivery efficiency, immune responses, and long-term safety. Additionally, the pleiotropic nature of miRNAs, where a single miRNA can target multiple genes, raises concerns about unintended effects on other biological processes. Therefore, a comprehensive understanding of miRNA networks and their context-specific functions is essential before their widespread clinical application.

In conclusion, microRNAs play a pivotal role in the regulation of bone remodeling and are deeply implicated in the pathogenesis of osteoporosis. Their ability to modulate both osteoblast and osteoclast activity makes them attractive candidates for diagnostic and therapeutic applications. While

Correspondence to: Sneha Reddy. Department of Molecular Biology, University of Hyderabad, Hyderabad, India. E-mail: sneha.reddy.lab@gmail.com

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challenges remain, ongoing research continues to uncover the potential of miRNAs as innovative tools in the fight against osteoporosis, paving the way for more precise and effective treatment strategies.