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Angiology: Open Access

ISSN: 2329-9495

Short Communication

Role of Micro Ribonucleic Acid (RNAs) in Cardiac Fibrosis

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DESCRIPTION

The heart, often described as the conductor of the body's orchestra, maintains the rhythm of life by tirelessly pumping blood throughout our entire lifetime. However, like any intricate musical composition, it can face disruptions that lead to detrimental consequences. One such disruption is cardiac fibrosis, a condition characterized by the excessive deposition of collagen fibers in the heart, ultimately impairing its ability to function properly. In recent years, researchers have discovered that micro Ribonucleic Acid (RNAs), small molecules with significant regulatory potential, play a critical role in the development and progression of cardiac fibrosis.

Cardiac fibrosis is a process whereby the heart's muscular tissue undergoes pathological remodeling, resulting in the accumulation of collagen fibers in the myocardium. This excess collagen stiffens the heart muscle, causing impaired contractility and relaxation, which, over time, can lead to heart failure. It can be triggered by various factors, including chronic inflammation, hypertension, myocardial infarction, and genetic predisposition [1-4].

MicroRNAs, often abbreviated as miRNAs, are short non-coding RNA molecules that regulate gene expression by binding to specific messenger RNAs (mRNAs), either degrading them or inhibiting their translation [5-7]. They are involved in a multitude of cellular processes, including proliferation, apoptosis, and differentiation. In the context of cardiac fibrosis, miRNAs emerge as critical players:

Fibrosis promotion: Several miRNAs, such as miR-21, miR-29, and miR-133, have been found to promote fibrosis by targeting genes that regulate collagen synthesis and fibroblast activation. These miRNAs are upregulated in response to cardiac stressors and contribute to the pathological remodeling of the heart.

Fibrosis inhibition: Conversely, some miRNAs, like miR-29 and miR-30, have been identified as negative regulators of fibrosis. They exert their effects by suppressing the expression of pro-fibrotic genes and thus, counteract the progression of cardiac fibrosis.

Immune modulation: MiRNAs can also influence the inflammatory response within the heart, a key driver of fibrosis. MiR-155, for example, is implicated in promoting inflammation and fibrosis by regulating immune cell function and cytokine production.

Angiogenesis: Adequate blood supply is essential for the heart's health. MiRNAs such as miR-126 and miR-92a regulate angiogenesis, a process crucial for maintaining cardiac tissue integrity. Dysregulation of these miRNAs can contribute to fibrotic changes in the heart.

Epigenetic regulation: MiRNAs can modulate the epigenetic landscape of cardiac cells, affecting their response to pathological stimuli. This epigenetic regulation can either promote or inhibit cardiac fibrosis.

Therapeutic potential

Understanding the role of miRNAs in cardiac fibrosis offers promising avenues for the development of targeted therapies [8-10]. Researchers are exploring various strategies, including:

miRNA replacement therapy: Restoring the expression of miRNAs that inhibit fibrosis could be a viable strategy to counteract cardiac fibrosis.

Antagomirs: These are synthetic molecules designed to inhibit specific miRNAs. Antagomirs targeting pro-fibrotic miRNAs may help slow down or even reverse fibrotic processes.

Nanoparticle-based delivery: Nanoparticle-based systems can deliver miRNA-based therapeutics to cardiac cells, offering a highly targeted approach.

Gene editing: Advancements in gene-editing technologies like Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR)-Cas9 may enable precise manipulation of miRNA expression to control fibrosis.

CONCLUSION

The heart's ability to maintain its rhythm and function depends on a delicate balance of cellular processes. Cardiac fibrosis disrupts this balance, leading to heart failure and other severe

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Received: 09-Aug-2023, Manuscript No. AOA-23-26820; **Editor assigned:** 11-Aug-2023, PreQC No. AOA-23-26820 (PQ); **Reviewed:** 25-Aug-2023, QC No. AOA-23-26820; **Revised:** 01-Sep-2023, Manuscript No. AOA-23-26820 (R); **Published:** 08-Sep-2023, DOI: 10.35841/2329-9495.23.11.382.

Citation: Lisabeth J (2023) Role of Micro Ribonucleic Acid (RNAs) in Cardiac Fibrosis. Angiol Open Access. 11:382.

complications. MicroRNAs, with their intricate regulatory roles in gene expression, have emerged as significant contributors to cardiac fibrosis. As our understanding of the role of miRNAs in this process deepens, the potential for innovative therapies to combat cardiac fibrosis grows stronger. In the future, harnessing the power of microRNAs may help us orchestrate the heart's symphony back to health, ensuring a harmonious beat for years to come.

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