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Review Article

Role of Non-Coding Rna in Cardiovascular Diseases

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Abstract

Non-coding RNAs (ncRNAs), including microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), are crucial regulators of gene expression and cellular function, with significant implications for cardiovascular diseases (CVDs) [1, 5]. These molecules influence a wide range of processes, including heart development, cardiomyocyte differentiation, cardiac remodeling, and vascular homeostasis. This review provides a detailed summary of their roles in these processes, highlighting their potential as diagnostic and therapeutic targets for CVDs. Understanding the mechanisms through which ncRNAs operate offers the possibility of innovative therapeutic strategies and underscores their importance in cardiovascular research.

Kew Words: miRNA; lncRNA; cardiovascular diseases; myocardial infarction; cardiac hypertrophy; heart failure; atherosclerosis; arrhythmia; regeneration; apoptosis; drug delivery; therapeutic targets; RNA-based therapy

Introduction

Cardiovascular diseases (CVDs) remain a leading global health challenge, contributing significantly to morbidity and mortality worldwide [11, 13, 14, 77, 78]. Despite advances in medical treatments and interventions, the complex molecular mechanisms underlying CVDs are not fully understood. Non-coding RNAs (ncRNAs) have emerged as key regulators in cardiovascular biology, offering new insights into disease pathogenesis and potential therapeutic interventions [5, 10, 73]. The exploration of ncRNAs represents a promising frontier in cardiovascular research, as they provide a deeper understanding of the molecular pathways involved in CVDs. Their ability to fine-tune gene expression makes them ideal candidates for therapeutic targeting. As we delve into the roles of ncRNAs, it becomes evident that they are not merely passive participants but active modulators of cardiovascular processes, which can be harnessed for therapeutic benefit.

MicroRNAs in Cardiovascular Biology:

MicroRNAs (miRNAs) are small, single-stranded non-coding RNA molecules, typically 22 nucleotides in length, that negatively regulate gene expression at the post-transcriptional level [1, 59]. They bind to complementary sequences in the 3'-untranslated region (3'-UTR) of messenger RNAs (mRNAs), leading to mRNA degradation or translational repression [1, 59]. MiRNAs participate in various cellular processes within the cardiovascular system, including cardiomyocyte growth, contractility, angiogenesis, and the development and maintenance of cardiac rhythm [1, 2, 3, 4, 5]. These processes are critical for normal heart function and adaptability to various stressors. Altered miRNA expression profiles have been observed in the blood of patients with various CVDs, making them attractive candidates for non-invasive biomarkers for disease diagnosis and prognosis [1, 6, 7, 43, 44]. Specific miRNAs, such as miR-1, miR-133, and miR-195, have been shown to play critical roles in heart development and disease [1]. For instance, miR-1 is essential for cardiomyocyte proliferation, while miR-133 is involved in maintaining cardiac rhythm. Furthermore, the identification of circulating miRNAs as biomarkers has the potential to revolutionize early detection and monitoring of cardiovascular conditions, enabling clinicians to tailor interventions more effectively.

Long non-coding RNAs in Cardiovascular Disease:

Long non-coding RNAs (lncRNAs) are transcripts longer than 200 nucleotides that do not code for proteins but play significant roles in CVDs [5, 6, 7]. These molecules regulate gene expression and cellular signaling through diverse mechanisms, including chromatin remodeling, transcriptional regulation, and post-transcriptional processing [5, 8, 41, 42]. lncRNAs can function as signals, decoys, guides, and scaffolds, influencing mRNA stability, translation, and protein localization [5, 55, 56]. This multifunctionality allows lncRNAs to integrate various signaling pathways and cellular contexts. They also participate in sponging miRNAs, thus regulating miRNA activity and target gene expression [5, 50]. Specific lncRNAs, such as MALAT1, MIAT, and HOTAIR, have been implicated in various CVDs, including myocardial infarction, heart failure, and atherosclerosis [5, 118, 119, 117]. For example, MALAT1 has been shown to regulate cell proliferation and apoptosis in cardiomyocytes, impacting the overall health of cardiac tissue. The versatility of lncRNAs in regulating gene expression underscores their importance in maintaining cardiovascular homeostasis and their potential as therapeutic targets.

ncRNAs in Heart Development and Regeneration:

Both miRNAs and lncRNAs are essential for cardiac development and regeneration [1, 124, 50, 52]. Specific miRNAs, such as miR-199 and miR-590, have been shown to promote cardiomyocyte proliferation and heart regeneration after injury [1, 23, 47]. These findings highlight the regenerative potential of miRNAs, particularly in the context of myocardial

infarction, where timely cellular proliferation is crucial for restoring heart function. lncRNAs, such as Braveheart (Bvht), play critical roles in cardiac lineage commitment and mesoderm isolation by interacting with chromatin-modifying complexes [66]. This indicates that lncRNAs are not only involved in adult cardiac physiology but also play foundational roles in embryonic development. Manipulating ncRNA expression shows therapeutic potential for promoting cardiac repair and rejuvenation after myocardial infarction [1, 50, 52, 53, 54].

For instance, overexpression of specific miRNAs or delivery of lncRNA-targeting molecules can enhance cardiomyocyte survival, promote angiogenesis, and reduce fibrosis in the infarcted heart. This regenerative capability highlights the importance of ncRNAs as potential therapeutic agents in cardiovascular medicine, offering hope for innovative treatments that harness the body's innate healing processes.

ncRNAs in CVD Pathophysiology:

ncRNAs are intertwined in various cardiovascular conditions, including myocardial infarction (MI), heart failure (HF), cardiac hypertrophy, arrhythmias, and atherosclerosis [5, 101, 113, 115, 116, 117, 118, 119, 120, 121, 122, 123].

- **Myocardial Infarction (MI):** In the context of MI, specific lncRNAs, such as CAIF, can inhibit autophagy and attenuate MI by blocking p53-mediated myocardin transcription [101]. The miR-15 family supports cardiomyocyte survival post-MI [1, 34]. Downregulation of miR-29 after MI results in scar formation due to increased collagen and extracellular matrix protein expression. These findings suggest that targeting these ncRNAs could improve outcomes in MI patients by enhancing the heart's ability to heal.
- **Heart Failure (HF):** Dysregulation of miRNAs, such as miR-765 and miR-25, contributes to HF by disrupting calcium handling and promoting inflammation [1, 35]. lncRNAs, including OIP5-AS1, have been shown to exacerbate HF in a sex-specific manner [104]. Increased expression of miR-30d has been shown to protect cardiomyocytes from inflammation and cell death in the context of heart failure, indicating potential therapeutic strategies involving miR-30d modulation. Understanding these pathways could lead to targeted therapies that mitigate HF progression and improve patient quality of life.
- **Cardiac Hypertrophy:** lncRNAs, such as Ahit and Chast, have been implicated in the pathogenesis of cardiac hypertrophy by modulating signaling pathways and gene expression [113, 78]. The lncRNA CHRF regulates cardiac hypertrophy by targeting miR-489 [115]. Insights into these regulatory mechanisms open avenues for targeting lncRNAs in the treatment of hypertrophic cardiomyopathy, potentially halting or reversing pathological remodeling of the heart.
- **Arrhythmias:** miRNAs, such as miR-1 and miR-133, play an important role in the pathophysiology of arrhythmias by affecting cardiac conduction and electrical activity [31, 32]. Their precise regulation is crucial for maintaining normal cardiac rhythm and preventing arrhythmic events. Abnormal expression of these miRNAs can lead to disrupted electrical signaling, increasing the risk of life-threatening arrhythmias.
- **Atherosclerosis:** In atherosclerosis, miRNAs regulate endothelial and smooth muscle cell function, influencing inflammation, lipid metabolism, and remodeling [1, 13, 46, 45, 4, 34]. lncRNAs can also modulate macrophage activation and cholesterol homeostasis in atherosclerotic plaques. For example, miR-33a and miR-122 have been described as regulators of lipid homeostasis [40, 41], illustrating the pivotal role of ncRNAs in lipid metabolism and atherosclerotic progression. Targeting these ncRNAs could provide new strategies for managing atherosclerosis and its complications.

Therapeutic Strategies Targeting ncRNAs in CVDs:

Emerging therapeutic strategies targeting ncRNAs in CVDs include:

- **miRNA Mimics:** Small, chemically synthesized double-stranded RNAs that imitate endogenous miRNAs and induce gene silencing [1]. These mimics can restore normal miRNA function in cases where endogenous levels are reduced, potentially reversing disease processes.
- **AntagomiRs:** Synthetic RNA molecules designed to silence aberrantly expressed miRNAs by binding to them and inhibiting their actions [1]. This approach can potentially reverse pathological conditions associated with dysregulated miRNA expression, offering a targeted method to address specific CVDs.
- **miRNA Sponges:** Transcripts containing multiple complementary regions for miRNAs, resulting in their sequestration and reduced activity [1]. By sponging specific miRNAs, these constructs can modulate gene expression and impact disease outcomes, providing a novel mechanism for therapeutic intervention.
- **siRNAs and ASOs:** Short interfering RNAs (siRNAs) and antisense oligonucleotides (ASOs) are utilized to target and degrade specific lncRNAs [60, 61]. These strategies allow for precise modulation of lncRNA expression, which could be crucial for therapeutic interventions aimed at correcting gene expression dysregulation.

Delivery Systems:

Innovative delivery systems, such as lentiviral vectors, liposomes, and exosomes, are being explored to enhance the efficacy and specificity of ncRNA-based therapies [69, 70, 71, 72, 73]. Effective delivery mechanisms are essential to ensure that therapeutic ncRNAs reach their targets in the cardiovascular system while minimizing off-target effects. Advances in nanotechnology and biomaterials are paving the way for more efficient delivery systems that can improve the bioavailability and stability of ncRNA therapeutics.

Limitations and Future Directions:

Despite advances, challenges remain in translating ncRNA-based therapies to the clinic, including species-specific differences, off-target effects, immunogenicity, and drug delivery [5, 98, 99, 100]. Further research is needed to fully harness the therapeutic potential of ncRNAs and develop safe and effective treatments for CVDs [5, 91, 92, 93, 94, 95, 96, 97].

Future Research Directions:

Future research should focus on:

- Identifying novel ncRNAs involved in CVD pathogenesis, which could lead to new biomarkers and therapeutic targets. The discovery of previously uncharacterized ncRNAs may unlock new avenues for treatment.
- Elucidating the precise mechanisms of ncRNA action in different cardiovascular cell types, enhancing our understanding of their roles in health and disease. This knowledge could facilitate the development of cell-type-specific therapies.
- Developing more specific and efficient delivery systems for ncRNA-based therapeutics, which is critical for improving clinical outcomes. Innovations in delivery methods could enhance therapeutic efficacy while reducing side effects.
- Conducting clinical trials to evaluate the safety and efficacy of ncRNA-based therapies for CVDs, paving the way for their integration into standard medical practice. These trials are essential for establishing the clinical relevance of ncRNA-based interventions.

Conclusion:

Non-coding RNAs are crucial regulators in cardiovascular biology and offer potential therapeutic avenues for treating CVDs [1, 64, 76, 99]. A deeper understanding of their mechanisms and improved delivery strategies are essential for realizing their clinical potential [5, 83, 84, 85, 86, 87, 88, 89, 90]. The development of ncRNA-based therapies holds great promise for improving the treatment and prevention of CVDs. By advancing our knowledge of these regulatory molecules, we can unlock new strategies for combating cardiovascular diseases and improving patient outcomes. The future of cardiovascular medicine may well hinge on our ability to manipulate these powerful regulators of gene expression.

References:

1. Wojciechowska A, Braniewska A, Kozar-Kamińska K. MicroRNA in cardiovascular biology and disease. *Adv Clin Exp Med*. 2017;26(5):865–874.
2. MiRNAs play a role in regulating various biological processes including embryogenesis, cell proliferation and differentiation, apoptosis or tumorigenesis.
3. In the cardiovascular system, miRNAs control cardiomyocyte growth and contractility, the development and maintenance of cardiac rhythm, plaque formation, lipid metabolism and angiogenesis.
4. Altered miRNA expression can be found in the blood of patients with various cardiovascular diseases, which makes them attractive candidates for noninvasive biomarkers.
5. Unlike transcriptional regulators, which have a turn-on-and-off function in controlling gene expression, the varied profiles of miRNAs appear to fine-tune the level of protein expression to changes in environmental conditions.
6. If two pre-miRNAs that are located at different sites in the genome lead to an identical mature miRNA, the miRNA is annotated with an additional hyphen and number, e.g. miR-194-1 or miR-194-2.
7. Two different miRNAs that originate from the same precursor are named according to their location on the hairpin: miR-17-5p (5’ arm) or miR-17-3p (3’ arm), or based on their level of expression: miR-123 or miR-123*.
8. MiRNA sponges are another approach to reducing miRNA levels.
9. Cyclin D2 controls cardiomyocyte proliferation by acting on the phosphorylation of retinoblastoma protein in the G1 phase of the cell cycle.
10. In rodents, β-MHC (a slow ATPase) expression occurs during embryonic development, α-MHC (a fast ATP-ase) after birth, while Myh7b expression occurs at both stages.
11. Deletion of miR-208a results in ectopic expression of fast skeletal muscle genes and impaired postnatal stress response.
12. Overexpression of miR-195 results in VSDs and ventricular hypoplasia.
13. MiR-15b controls ATP level in cardiomyocytes by targeting Arl2, a component of the ADP/ATP exchanger in mitochondria.
14. Deletion of miR17~92 leads to VSDs and lung hypoplasia, and consequently to death.
15. Overexpression of miR-195 has been shown to impair myocardial regeneration and cause massive cardiac fibrosis.
16. Upregulation of miR-133 diminishes its regenerative potential, whereas decreasing the level of miR-133 with specific miRNA sponges promotes regeneration.
17. MiR-199 and miR-590 have been shown to promote regeneration of the myocardium and to improve cardiac function.
18. Moreover, transplanting stem cells overexpressing miR-1 into the infarcted zone increases cardiomyocyte differentiation, promotes regeneration and improves cardiac function.
19. Restoration of miR-24 to physiological levels by specific miRNA mimics attenuates apoptosis and decreases scar size.
20. Restoration of physiological miR-199 levels inhibits HIF-1α expression and its stabilization of p53, a tumor supressor responsible for sustaining the genome integrity, which leads to a reduction in apoptosis.
21. The upregulation of miR-499 reduces apoptosis and infarct size, while miR-499 knockdown has the opposite effect.
22. Deletion of miR-214 increases injury and mortality following myocardial infarction.
23. Deletion of miR-208a protects the heart from pathological remodeling under stress conditions.
24. Specific antagomiR-mediated inhibition of miR-21 blocks this cascade and results in a reduction in both hypertrophy and fibrosis.
25. MiR-765 contributes to increased protein phosphatase 1 (PP-1) activity and the subsequent dephosphorylation of key calcium cycling proteins by silencing its endogenous inhibitor-1.
26. Anti-miR-25 delivery restores cardiac function and improves survival.
27. MiR-24 regulates calcium homeostasis through Junctophilin-2 repression, which results in decreased efficiency of excitation-contraction (E-C) coupling in cardiomyocytes.
28. It protects cardiomyocytes from TNF-α-elicited inflammation and cell death by targeting the MAP4K4 protein or, possibly via other indirect pathways, resulting in beneficial cardiac remodeling.
29. Administration of miR-126 mimics ameliorates microvascular density, improves RV function and diminishes fibrosis, whereas antagomiR-mediated miR-126 downregulation exacerbates RV failure.
30. Pharmacological inhibition of miR-146a or 16K PRL attenuates ErbB4 downregulation and improves cardiac function.
31. The arrhythmogenic properties of miR-1 include repression of GJA1 and KCNJ2, which encode the connexin43 and Kir2.1 subunits of the IK1 channel, respectively.
32. Age-associated low levels of miR-1 and miR-133 contribute to the overexpression of HCN2 and HCN4, and this results in abnormal cardiac electrical activity.
33. Inhibition of miR-155 in Chinese hamster ovary cells resulted in upregulation of AGTR1 and ERK1/2 activation.
34. Downregulation of miR-23a with a specific antagomiR prevents cardiac hypertrophy.
35. MiR-10a controls a pro-inflammatory EC phenotype by regulating the expression of adhesion molecules, and miR-10a expression is decreased in atherosusceptible regions of the aorta.
36. MiR-145 controls neointimal lesion formation by silencing Kruppel-like factor 5 (KLF5) and its downstream molecule, myocardin.
37. MiR-145 diminishes expression of matrix genes, while smooth muscle differentiation genes remain unaffected.
38. MiR-143 and miR-145 target hexokinase II (HKII) and integrin β 8 (ITGβ8) genes, respectively.
39. Administering EC-derived EVs enriched with miR-143/145 results in a reduction in atherosclerotic lesion formation in the aortas of apolipoprotein E-deficient mice.
40. Downregulation of miR-122 results in decreased levels of both HDL and LDL cholesterol.
41. MiR-33a targets ATP-binding cassette transporter A1 (ABCA1) and inhibits cellular cholesterol export.
42. Delivering the antisense inhibitor of miR-122 reduces the number of viral copies without evidence of treatment resistance.
43. MRX34, which mimics miR-34, has recently (at the time of writing) entered phase I clinical trials for the treatment of primary liver cancer.

44. The Human Genome Project started in 2001 and was 99% completed in 2004
45. RNA transcripts arose from at least 76% of the human genome and these RNA molecules likely had biological functions
46. there are more than 16,000 currently identified long noncoding RNAs (lncRNAs)
47. lncRNAs are defined as RNAs that do not code for proteins and are longer than 200 nucleotides in length
48. lncRNAs perform multiple functions through various mechanisms
49. lncRNAs can regulate mRNA splicing by binding to proteins that modulate mRNA turnover, and/or stabilize and translate mRNA transcripts by binding to target RNA
50. lncRNAs also play a role in sponging miRNA, with sequestration of microRNAs, and serve as competing endogenous RNA (ceRNA) to regulate mRNA expression
51. lncRNAs contribute to both human disease and genetic traits.
52. CVD remains a leading cause of morbidity and mortality worldwide
53. the prevalence of CVD in adults aged 20 years or older was 49.2%
54. WHO estimates that 17.9 million people died from CVD, which represents 32% of all deaths worldwide, and 37% of non-communicable diseases among those under 70 years of age in 2019
55. an estimated 19.05 million people died in 2020 from CVD, an 18.71% increase from 2010
56. Myocardial infarction (MI) occurs when the myocardium is deprived of oxygen for a prolonged period of time
57. surveys conducted by the Atherosclerosis Risk in Communities (ARIC) study between 2005 and 2014, there are 605,000 new cases of MI each year and 200,000 cases of recurrent MI.
58. the MI incidence rate is higher in men than in women with an age of first onset of 65.5 years in men and 72.0 years in women
59. Men often have more demanding occupations and employ distinct stress management techniques compared to women.
60. men with hypertension, high BMI (body mass index), and type II diabetes experience higher rates of MI
61. the gender differences in MI risk tend to reduce as individuals age
62. Snhg1 regulates cardiomyocyte proliferation after MI by upregulating PI3K/Akt signaling and promoting an angiogenic response through the induction of VEGFA gene expression
63. Carl regulates mitochondrial dynamics through the Carl/miR-539/Phb2 signaling axis
64. Chrf appears to limit myocardial I/R injury by preventing Atg7 from being inhibited by miR-182-5p
65. Sirt1 is known to suppress oxidative stress and apoptosis during MI; Ampk is involved in energy metabolism which aids cell survival following ischemic injury; and Pgc1-α is responsible for energy homeostasis, oxidative metabolism, and cardiac mitochondrial function.
66. the Hotair/miR-206/Fn1 axis is similarly thought to prevent apoptosis following AMI
67. Meg3 mechanistically decoys p53 by interfering with p53's ability to bind to the promoter of matrix metallopeptidase 2 (Mmp2) and thus allowing the cell to upregulate Mmp2 expression
68. identification of Wisper represents crucial step toward anti-fibrotic therapeutic approaches and diagnosis
69. Malat1 appears to cause cardiac fibrosis by limiting miR-145-dependent suppression of TGF-β1
70. Miat activates Furin/Tgf-β1 by inhibiting miR-24 as a ceRNA, and that this resulted in cardiac fibrosis
71. Cardiomyopathy manifests as myocardial or electrical dysfunction within the heart
72. cardiomyopathy and myocarditis caused 0.37 million deaths in 2020 and affected 6.11 million people globally during that year
73. eight cases out of approximately 100,000 are diagnosed with cardiomyopathy each year and classically fall into three types of cardiomyopathies: hypertropic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), and restrictive cardiomyopathy (RCM).
74. 30–60% of HCM patients have sarcomere variants
75. Mhrt functions as cardio-protective because it inhibits the development of myocardial hypertrophy
76. Ahit binding to suppressor of zeste 12 protein homolog (SUZ12), a component of the polycomb repressor complex 2 (PRC2) complexes, and this event blocks the regulation of myocyte enhancer factor 2A (Mef2A) in cis
77. Chaer reduces H3K27 trimethylation of genes linked to pathological changes by binding to enhancer of zeste 2 polycomb repressive complex 2 subunits (EZH2), a methyltransferase subunit of PRC2
78. Chast functions as a signaling molecule. Overexpression of Chast results in an increased cardiomyocyte size and expression of hypertrophic markers.
79. Chrf also increases the myeloid differentiation primary response gene 88 (Myd88) protein by sponging the miRNA miR-489
80. Plscr4 represses cardiac hypertrophy through the miR-214/Mfn2 axis
81. Decreased expression of ZNF593-AS results in loss of cardiac contractile function because ZNF593-AS guides for heterogeneous nuclear ribonucleoproteins C1/C2 (HNRNPC) to stabilize the ryanodine receptor 2 (RYR2) mRNA transcript.
82. the H19/miR-675/PA2G4 axis appears to significantly regulate doxorubicin-induced DCM
83. Cardiomyocytes increase by 1% per year at the age of 25, and this decreases to 0.45% by age 75
84. Infarction-induced heart failure results in a 25% reduction in left ventricle cardiomyocytes and leads to the death of approximately up to 1 billion myocardial cells
85. ECRAR elevates ECRAR expression by binding within the ECRAR promoter
86. Sirt1-as also inhibits apoptosis and decreases cardiomyocyte size due to Sirt1-as binding, and consequential stabilization of the Sirt1transcript.
87. NR_045363 appears to positively control cardiomyocyte mitotic activity and proliferation by binding miR-216a and activating JAK2-STAT3 phosphorylation
88. lncDACH1 binds to inorganic pyrophosphatase 1 (PPA1) and restricts its dephosphorylation activity and also regulates yes1 associated transcriptional regulator (YAP1) signaling by increasing YAP1 phosphorylation and decreasing its nuclear translocation by binding PP1A
89. CRRL appears to act as a ceRNA by sequestering miR-119a-3p
90. Bvht plays an important role in the differentiation of nascent mesoderm to cardiomyocytes by inhibiting mesoderm posterior bHLH transcription factor 1 (MesP1) through interaction with SUZ12, leading to cardiac lineage commitment
91. Linc1405 acts as a scaffold that drives cardiac differentiation by regulating the MesP1 gene
92. Moshe suppresses cardiomyocyte differentiation by downregulating secondary heart field genes.
93. lentiviral vectors have a packing capacity of up to 8 kb and can infect both dividing and non-dividing cells
94. adenovirus vectors have a packing capacity of up to 5 kb and can transduce both dividing and non-dividing cells similar to lentiviral vectors
95. liposomes reduce immunogenicity and toxicity by not integrating into the genome, and can provide short-term,

transient, and high-level expression of transported materials, including nucleic acids, small molecules, and proteins.

96. exosomes contain DNA, mRNA, miRNA, and lncRNA, and facilitate communication between cells
97. exosomes regulate cell-to-cell communication and these characteristics suggest that EVs can be used for lncRNA delivery
98. CVD and especially AMI remain the leading cause of mortality and morbidity in western countries
99. reperfusion often induces reperfusion injury which is thought to be mediated by increased oxygen free radicals

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