Improved wound healing due to cardiac overexpression RNA m5c modification's emerging role in cardiovascular diseases.

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Abstract

The investigation of RNA changes resulting from epigenetic modifications is known as epitranscriptomics, an emerging field of study. Epigenetic research is advancing, which has aided in managing and understanding human diseases. All RNA functions, including those involved in the pathogenesis of human diseases, are regulated by RNA methylation. Interestingly, there is a strong correlation between RNA m5C methylation and a number of human diseases, including cardiovascular diseases (CVD). M5C regulatory proteins, which function methyltransferases, methyltransferases and RNA-binding proteins, regulate the m5C methylation. Cardiovascular disease is significantly linked to the dysregulated expression of m5C regulatory proteins, which play critical roles in cellular and biological processes. The main focus of this review is on how mitochondrial dysfunction and CVD are related to RNA m5C modification.

Keywords: Post-transcriptional gene, Cardiovascular disease, Pulmonary hypertension.

Introduction

The most common cause of morbidity and mortality worldwide is cardiovascular disease (CVD), and its complicated pathological mechanisms. A number of circulatory system diseases, including pulmonary hypertension, hypertension, vascular calcification, cardiac hypertrophy, cardiac arrhythmias, atherosclerosis, angina pectoris, myocardial infarction, and heart failure, are collectively referred to as cardiovascular diseases (CVDs). When the pulmonary artery pressure exceeds the threshold, it is said to be in a condition known as pulmonary hypertension (PH). Which can result in right heart failure. Mean pulmonary artery pressure less than 25 mmHg is the hemodynamic diagnostic threshold for PH. PH can be a complication or an independent disease, and it has a high morbidity and mortality rate. In its early stages, PH is non-specific and asymptomatic. Dyspnea, exhaustion, a decreased capacity for exercise, syncope, angina pectoris, chest pain, and right heart failure are among the symptoms of the disease as it progresses [1].

A clinical syndrome known as hypertension is characterised by elevated systemic arterial blood pressure (systolic blood pressure of at least 140 mmHg and diastolic blood pressure of at least 90 mmHg), which can result in damage to the kidney, heart, brain, and other organs. The most prevalent chronic illness and the main CVD risk factor is hypertension. Patients with hypertension experience a certain range of blood pressure changes as a result of internal and environmental changes. Early signs of hypertension may be asymptomatic or not noticeable, but later signs can include palpitations, headaches, dizziness, and fatigue. After exhaustion, mental stress, and emotional upheaval, hypertension can develop [2].

A common pathological symptom of atherosclerosis, hypertension, diabetic vascular disease, vascular injury, chronic kidney disease, and ageing is vascular calcification. Increased vascular wall sclerosis and decreased compliance are the main signs of vascular calcification, which can quickly result in myocardial ischemia, left ventricular hypertrophy, and heart failure. Vascular calcification-induced thrombosis and plaque rupture are significant contributors to the high incidence and mortality rates of CVDs. Vascular calcification is a crucial indicator of peripheral vascular disease. Cardiac hypertrophy primarily happens when the myocardium's total volume is increased and its contractility is strengthened due to prolonged myocardial pressure overload. The heart can maintain regular blood flow thanks to cardiac hypertrophy. Myocardial ischemia results when the blood supply from the coronary arteries is unable to keep up with the increased oxygen demand of the myocardium due to cardiac hypertrophy. As a result, myocardial contractility eventually declined. Heart failure and palpitations are two of the main signs and symptoms of cardiac hypertrophy, along with dyspnea, chest pain, exhaustion, wooziness and fainting. Advanced patients' disease is characterised by severe myocardial fibrosis and impaired ventricular systolic function [3].

Heart failure is the inability of the cardiac systolic and/or diastolic functions to completely empty the veins of the heart's

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blood, which ultimately results in venous system blood stasis and inadequate arterial blood perfusion. Heart failure also causes a condition known as cardiac circulation disorder, which shows up as pulmonary and venous congestion. Heart failure is the final stage of the progression of a number of different heart diseases rather than a distinct illness. Left heart failure is where the vast majority. It is advised for patients with CVDs to adopt preventive measures to lower the risk, such as lifestyle modifications. Contrarily, conventional medications like renin-angiotensin blockers, lipid-lowering medications, beta receptor inhibitors, and antithrombotic medications are used to stop the progression of disease. People have been working to stop the emergence and growth of CADs for a very long time. None of these remedies, however, has really addressed the issue of the rising CAD incidence rate. Identifying the potential therapeutic targets for these diseases is the focus of basic scientific research, and it is crucially important to fully comprehend the pathological mechanisms [4].

Nearly every aspect of RNA processing, including nuclear export, RNA translation, splicing, and non-coding RNA processing, is regulated by RNA methylations. The relationship between RNA methylations and fundamental genetic processes is gradually being understood thanks to the accessibility and comprehension of new detection technologies. Methylation of RNAs results in post-transcriptional regulatory mechanisms that can fine-tune gene expression by changing how RNAs interact with other cellular elements. The "writers," "erasers," and "readers" are all involved in RNA methylations. Although many RNA species' structures and functions depend on their methylations, RNA methylations appear to be somewhat dynamic, allowing for fine-tuning of protein-coding genes and cellular functions. Although the coding sequence is unaffected, these modifications have a significant impact on the expression characteristics of transcripts [5].

Conclusion

The new RNA methylation types M1A, m6Am, and m7G. In eukaryotes, M1A is a highly prevalent post-transcriptional modification of tRNAs and rRNAs. The regulation of mRNA translation is impacted by M1A modification. M6Am is a functionally distinct mRNA modification from m6A that has undergone evolutionary conservation. M6Am has a negative impact on the cap-dependent translation of methylated mRNAs but has no effect on mRNA transcription or stability. M7G methylation controls the maturation and processing of 18S rRNA, tRNA stability, miRNA biosynthesis, and biological functions. The relationship between m1A, m6Am, and m7G modifications and CVDs has not been reported because they are novel types of RNA methylations, and further research into their mechanisms and functions is urgently needed. This work focuses on the roles and mechanisms of RNA methylations (m6A, m5C, m1A, m6Am, and m7G) in CVDs and reveals the molecular characteristics, biological functions, and effects on these diseases. This is because RNA methylations have become new research hotspots and their functions in CVDs are gradually emerging.

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