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Cardiovascular Diseases and MicroRNAs

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Abstract

Even though there has been significant advancement in this area over the past 20 years, heart failure and coronary artery disease (CAD) still have high death rates worldwide. A novel diagnostic method and a therapy plan are still needed in the clinical setting to reduce the prevalence of CAD. In order to post transcriptionally silence genes, microRNAs (miRNAs), highly conserved noncoding short RNA molecules, bind to corresponding messenger RNA sequences and control a significant portion of the genome. Specific miRNAs have been found to have a role in every step of atherosclerosis, from endothelial dysfunction to plaque rupture, according to recent studies. These results imply that miRNAs may serve as biomarkers for CAD early diagnosis and as treatment targets. The involvement of miRNAs in each stage of atherosclerosis is highlighted in the current review, and its prospects are also covered.

Using both in vitro and in vivo human and mouse models, the current research highlights the antioxidative and antiatherogenic effects of pomegranate polyphenols on serum lipoproteins and on arterial macrophages (two important components of the atherosclerotic lesion). The production of foam cells, a sign of early atherogenesis, and the build-up of cholesterol and oxidised lipids in macrophages were both significantly decreased by pomegranate juice and its by-products. This attenuated the development of atherosclerosis and the subsequent cardiovascular events.

Keywords: Cardiovascular diseases; miRNAs; Pathogenesis; Biomarker; Therapeutics; Cardiac hypertrophy; Coronary heart disease; Hypertension

Introduction

The main cause of death in the developed world is cardiovascular disease (CVD), particularly coronary artery disease (CAD). The underlying cause of coronary artery disease (CAD) is atherosclerosis, which is known to be promoted by a number of diseases, including smoking, hypertension, diabetes mellitus, and dyslipidemia. These coronary risk factors are typically present in CAD patients for a long time. In other words, atherosclerosis' last stage is CAD. Over the past 20 years, significant effort has been made to regulate these factors in order to reduce the occurrence of CAD [1].

Clinical settings have advanced greatly; the prevalence of CAD is reduced by antiplatelet medications, statins, and angiotensin converting enzyme inhibitors, angiotensin receptor blockers, and medications for diabetic mellitus. Additionally, individuals with acute myocardial infarction benefit greatly from primary percutaneous coronary intervention in terms of their prognosis (AMI). However, the globe still faces serious issues with CAD, particularly AMI, and heart failure brought on by ischemic cardiomyopathy, thus finding new ways to improve existing therapeutic techniques is still important [2].

Noncoding RNAs (ncRNAs), which do not contribute to protein coding and have received a lot of attention in recent years, may play a significant role in the emergence of a variety of illnesses, including cardiovascular disease. MicroRNAs (miRNAs, miR), which are brief ncRNAs with a 22-nucleotide RNA sequence, are the most well-studied ncRNA molecules [3]. The leading cause of death worldwide is cardiovascular disease (CVD), which includes hypertension, coronary heart disease (CHD), cerebrovascular disease, and heart failure. The prevalence of all CVD nearly doubled from 271 million to 523 million cases between 1990 and 2019, while the number of CVD deaths climbed gradually from 12.1 million to 18.6 million. In both the 50–74 and 75–and-older age categories in 2019, ischemic heart disease was among the leading causes of disability adjusted life years (DALYs). By 2030, the World Health Organization (WHO) projects that over 23.6 million individuals would pass away from CVDs. Prostate cancer and benign prostatic hyperplasia (BPH) are illnesses connected to ageing [4].

About 50% of men over the age of 50 have BPH, and that number rises to 80% by the time they are 80 years old or older. Prostate cancer cases grew from 940,000 in 2007 to 1.3 million in 2017, and from 1990 to 2017, the age-standardized incidence of prostate cancer in China increased by 2.75 percent. Aging unquestionably has a significant impact in prostatic illnesses and CVDs (such as hypertension and CHD) (prostate cancer and BPH). A nuclear RNase III called Drosha converts primary miRNAs (pri-miRNAs), which are then translated into miRNA precursors (pre-miRNAs), which are encoded in the genomes of nucleated cells. Pre-miRNAs are processed by Drosha before being exported to the cytosol and processed by the RNase III Dicer, which ultimately produces miRNAs. RNA-induced silencing complexes contain Argonate 2 (Ago 2), a critical component of mature miRNAs in rib nucleoprotein (RNP) complexes (RISC) [5].

MiRNAs bind to the 3′ UTR of the target mRNA to control messenger RNA (mRNA) gene expression at the posttranscriptional level through transcript degradation or translational repression. More than 2,500 human miRNAs have been identified as of this writing. The permissive binding requirement of each miRNA to mRNA, on the other hand, includes dozens to hundreds of target mRNAs, which suggests that an mRNA is controlled by several miRNAs [6].

Methods and Materials

The HMDD database (version 3.0), which is a database of carefully vetted experimental evidence for connections between human

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miRNAs and diseases, is where the human miRNA-disease association dataset was obtained. All relationships in the HMDD are categorised into six groups: Circulation, Tissue, Genetics, Epigenetics, Target, and Other. For further analysis, we have retrieved associations for records with evidence codes "tissue expression down" or "tissue expression up" in the class "Tissue" that provide unambiguous information about tissue expression regulation. MeSH was used to standardise disease nomenclature, and pre-miRNA miRNA levels were evaluated [7]. We first used a vector made up of the miRNAs connected to each disease to define it. The value of each dimension indicates how strongly a disease and a certain miRNA are related [8].

Semantic similarity and vector-based similarity are the two main metrics used to assess illness similarities. A known disease classification system is used in the calculation of disease semantic similarity, which makes it unsuitable for identifying new disease associations. Based on information on associations between disease and other elements such genes, medications, phenotypes, microorganisms, and miRNAs, vector-based disease similarity is calculated. The fact that a disease is often represented as a series of binary (present/absent) observations of miRNAs, missing weights and directions, is a shortcoming of existing approaches for assessing miRNA-based disease similarity. The simplest solution to this issue is to use the cosine of two disease vectors as a similarity metric. Although the directions may be similar but the weights may differ, it tends to overstate the links between well-studied diseases and recently discovered disorders. The Tanimoto coefficient is a better way to calculate similarity that is derived from cosine similarity and additionally takes into account the norms of the two vectors. The Tanimoto coefficient decreases as the difference between the norms of two vectors increases. In order to lessen the impact of research imbalances, we adopt the Tanimoto coefficient as a similarity measurement in the current study [9].

Discussion

Since miRNAs can control how proteins are expressed, they are of great interest in understanding and treating cardiovascular disease (CVD). Currently, systemic anti-miR administration is the primary method of treatment, raising concerns about off-target consequences such platelet activation (79). Future initiatives should focus on examining local delivery or cell-type-specific techniques. In order to deliver miRNAs to specific cell types in the cardiovascular system, it may be possible to use adeno-associated viral vectors, nanoparticle-bound anti-miRs, or miRNA mimics [10].

The majority of preclinical research has been devoted to pinpointing pathways within a particular tissue or cell type. To prevent advancing too rapidly towards a clinical evaluation, caution must be taken. Due to miRNAs' ubiquitous expression and strong context- and cell-type-dependent control of protein expression, adverse effects of miRNA therapies can be unpredictable. Therefore, carefully assessing the impact on the entire body is necessary when targeting specific miRNAs. A cautious approach to developing miRNA medicines could impede progress toward clinical application but prevent gene therapy-like setbacks for miRNA therapeutics. MiRNAs have enormous potential, which merits the use of caution before extensive clinical investigations for CVD.

Circulating miRNAs are proposed as new biomarkers for disease because of their variable expression across disease phenotypes. Circulating miRNAs may be particularly important in the context of CVD due to their platelet origin. Cardiovascular risk may be associated with platelet reactivity; however there is no widely acknowledged biomarker for this. Establishing the clinical relevance of miRNA biomarkers will require more mechanistic research and validation in bigger cohorts [11-14].

Conclusion

MicroRNAs have been found to play a key role in regulating cellular homeostasis as well as stem cell self-renewal and differentiation, which in turn affects lineage commitment. They do this by repressing specific gene patterns. The majority of single miR knockouts do not impair embryonic development, either as a result of compensation for family members or because of their restricted function as regulators of cell activity rather than decision-making. According to the information shown above, certain miRNAs may be involved in the behaviour and differentiation of certain cardiovascular cell lineages, such as vascular smooth muscle, endothelial cells, and cardiomyocytes. Additionally, since altered expression profiles in circulating progenitor or resident tissue stem/progenitor cells may compromise the endogenous capacity for cardiovascular repair, we may be able to alter the functional properties of compromised patient-derived cells via miRNA therapeutics before using them for cell transplantation in myocardial repair.

Numerous miRNAs play critical functions in controlling cell proliferation, differentiation, and death, which suggests that they may act as novel biomarkers and potential therapeutic targets. However, more research needs be done on these prospective uses of miRNA-based medicine. More research is needed to understand the intricate relationships between certain miRNAs and their target genes during CAD, as a miRNA often targets many genes and one gene may be targeted by multiple miRNAs. Investigating the incredibly intricate miRNA network is quite difficult. We think that figuring out how miRNAs work will open up new possibilities for CAD treatment and diagnosis.

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