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Review Article

Pro-Atherogenic and Anti-Atherogenic Role of MicroRNAs in Pathobiology of Atherosclerosis

Misbah Hussain[1,2]*

[1]*Department of Biotechnology, University of Sargodha, Sargodha, Pakistan*
[2]*Institute of Cardiovascular and Medical Sciences, University of Glasgow, UK*

ABSTRACT

MicroRNAs (miRNAs) are small non-coding RNA molecules that play a crucial role in gene regulation. This review focuses on the recent advancements in understanding the involvement of miRNAs in atherosclerosis. Atherosclerosis, a major cause of cardiovascular diseases, involves the accumulation of lipid-laden macrophages and immunological cells, leading to plaque formation and artery blockage. This review details the intricate pathogenesis of atherosclerosis, from endothelial dysfunction to the development of atherosclerotic plaques and plaque rupture. The role of miRNAs in atherosclerosis is highlighted, with a focus on both pro-atherogenic and anti-atherogenic miRNAs. Pro-atherogenic miRNAs, such as miRNA-155, miRNA-200b, and miRNA-33, contribute to inflammation, lipid accumulation, and vascular damage. Conversely, anti-atherogenic miRNAs, including miRNA-218-5p, miRNA-98, miRNA-126, and miRNA-223, exhibit protective effects by regulating inflammation, endothelial function, and plaque stability. Recent studies from 2023 are specifically addressed, emphasizing the dynamic landscape of miRNA research in atherosclerosis. The review explores potential therapeutic interventions targeting miRNAs for the treatment of atherosclerosis, providing insights into the promising role of miRNA-based therapies in cardiovascular health.

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INTRODUCTION

MicroRNAs are small non-coding RNA molecules usually coded from different parts of genome. Most commonly they are transcribed from intron however, they can be coded by exons of other genes (Boyd, 2008). Initially, microRNAs (miRNA) are transcribed as primary-miRNA which are then processed by Drosha and Dicer and converted to miRNA duplex which are incorporated to miRNA-induced silencing complex (miRISC) where Argonaut direct miRNA maturation and help in binding to its target mRNA (usually on 3` UTR region) (Liu *et al*., 2023). MiRNA can travel as free circulatory miRNA and/or encapsulated in extracellular vesicles (EV) (Creemers *et al*., 2012; O'Brien *et al*., 2020). Various cellular proteins help in selective sorting of miRNAs in their respective EVs for nearby and distant targets (Groot and Lee, 2020). Studies have also found that endogenous circulatory miRNA are highly stable and escape RNase present in circulation. Now these circulatory miRNAs are targeted to find biomarkers and therapies for cardiovascular diseases. A recent study have found enhanced expression of circulatory miRNA-126-5p, miRNA-126-3p, miRNA-181b-5p, and miRNA-29b-3p in serum of coronary artery disease patients (Ekedi *et al*. 2023). While EV encapsulated miRNA-155 is found to be a culprit for lipid accumulation and atherosclerosis progression (Wang *et al*. 2023). These miRNAs help in cell communication and control expression of ~60% of human genes.

MiRNAs were discovered as unique regulatory components in *C. elegans* in 1993. These small regulatory molecules play a significantly important role in gene regulation and influence the translation of transcriptome (Engels and Hutvagner, 2006). Increased understanding of RNA-interference and microRNA (miRNA) mediated gene regulation revealed that miRNA exhibit their gene regulatory role by either mRNA degradation or translational repression resulting in downregulation of target gene. In rare cases, miRNA can upregulate the target genes (Engels and Hutvagner, 2006; Nilsen, 2007). MiRNA expression is highly cell specific, and studies of human cells found that some miRNAs are unique to a

* Corresponding author: misbah.hussain@uos.edu.pk, misbahussain@yahoo.com
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single class of cells (Ludwig *et al*. 2016) Owing to their cell and/or tissue specificity, miRNAs are presented as important modulators of physiological and/or pathological cell signaling pathways. In 2006, for first time, Van Rooji *et al*. (2006) explained the role of miRNA in development of cardiovascular diseases (CVDs) (Van Rooij *et al*., 2006) which evoked further studies to delineate the regulatory role of miRNA in pathophysiology of CVDs.

CVDs are second major cause of deaths worldwide with an annual death rate of ~17 million (WHO, 2021). In-spite of huge morbidity and mortality rate, it is tricky to treat CVDs due to their complex etiology and pathology. CVDs include various pathologies like atherosclerosis, coronary artery disease, cerebrovascular diseases, heart failure, congenital heart diseases and venous diseases. Scientists are continuously trying to find new potential drug targets to improve the treatment strategies and reduce the burden of CVDs. 32% of all cause mortalities are caused by atherosclerosis (Nedkoff *et al*., 2023). Atherosclerosis is an inflammatory disease characterized by the development of atherosclerotic plaque in coronary arteries, which supply blood to hearts muscles. Obstruction in these arteries owing to development of atherosclerotic plaque causes coronary artery disease (CAD). CAD can leads to myocardial infarction due to rupturing of atherosclerotic plaque and complete blockage of blood flow (Bergström *et al*., 2021).

PATHOGENESIS OF ATHEROSCLEROSIS

Atherosclerosis starts with the endothelial dysfunction and proceed with accumulation of immunological cells (monocytes and macrophages) and lipid molecules. Activation of atherogenic proteins damage endothelial cells and allow entry of low-density lipoproteins (LDL) into the intima (innermost layer of blood vessels), where proteoglycans (major component of extracellular matrix) bind to Apolipoprotein B-100 (Apo B-100, a surface protein of LDL) and help in LDL accumulation in sub-endothelial region. Different stimuli activate endothelial cells to express surface adhesion molecules which recruit monocytes from blood and transmigrate them to intima where they are matured to macrophages. In intima, LDL is oxidized (Ox-LDL) by cellular and enzymatically produced reactive oxygen species (ROS) and endocytosed by macrophages forming foam cells. Apoptotic cell death of these lipid laden macrophages release lipids and cellular debris establishing necrotic core. Macrophage secreted inflammatory factors stimulate smooth muscle cell proliferation and migration from media to intima where they form fibrous cap which is very important for the stability of atherosclerotic plaque. Growing necrotic core size and thinning of fibrous cap results in plaque rupture which subsequently form thrombus and block artery (Lusis, 2000; Skeoch and Bruce, 2015) (Fig. 1).

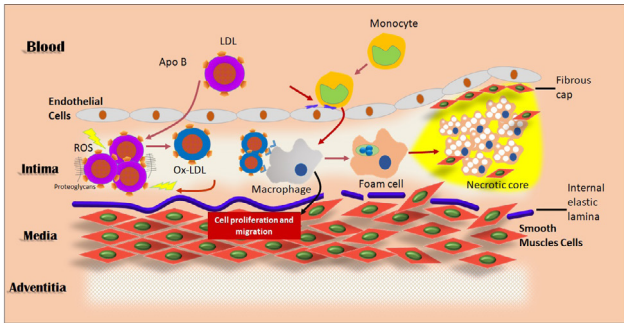

Fig. 1. Pathogenesis of Atherosclerosis.

microRNA AND ATHEROSCLEROSIS

Scientist are trying hard to find the risk factors and potential therapeutic agents for treatment of atherosclerosis. Recently, microRNA(miRNA), either circulatory or encapsulated in EVs, are highlighted for their predominant role in regulating atherosclerosis related genes (Blaser *et al*., 2023). This is also raising hope for using anti-miRNA therapeutics for improving cardiac health. Comprehensive review articles on the role of microRNA in atherosclerosis have been published in previous years by various researchers (Sun *et al*., 2013; Natarelli and Schober, 2015; Schober and Weber, 2016; Churov *et al*., 2019; Hajibabaie *et al*., 2020; Tabaei and Tabaee, 2021; Li *et al*., 2023). These manuscripts are summarizing the data of previous years. Even review articles published on said topic in 2023 are discussing the data majorly published in 2022 and previous years (Li *et al*., 2023; Sidorkiewicz, 2023). Current review article is mainly focusing on the recent studies reported in 2023. PubMed and Google scholar were searched for microRNA and atherosclerosis related article. PubMed retrieved 130 publications with following search query [(microRNA (Title/Abstract) AND atherosclerosis (Title/Abstract)]. Among these, 27 were review articles and four manuscripts were discussing the potential drugs for treating atherosclerosis by modulating microRNAs (Li *et al*., 2023; Yang *et al*., 2023; Zhang *et al*., 2023; Zhu *et al*., 2023). Some of the manuscripts were about other pathologies or discussing the delivery systems. Figure 2 shows the pro-atherosclerotic and anti-atherosclerotics miRNAs.

PRO-ATHEROGENIC microRNAS

Previous studies have concluded that miRNA-206

plays an anti-atherosclerotic role by down-regulating the atherogenic proteins in vascular smooth muscle cells and macrophages (Xing *et al*., 2017; Hu *et al*., 2019; Wang and Bai, 2021). Contrary to these studies, Cimen *et al*. (2023) reported that miRNA-206 promotes atherosclerosis by interacting with CXCR4 (a chemokine receptor) gene and repressing its expression in advanced atherosclerotic lesions. Vascular cell specific down-regulation of miRNA-206 up-regulates the CXCR4 expression and ameliorates the atherosclerosis in mice model.

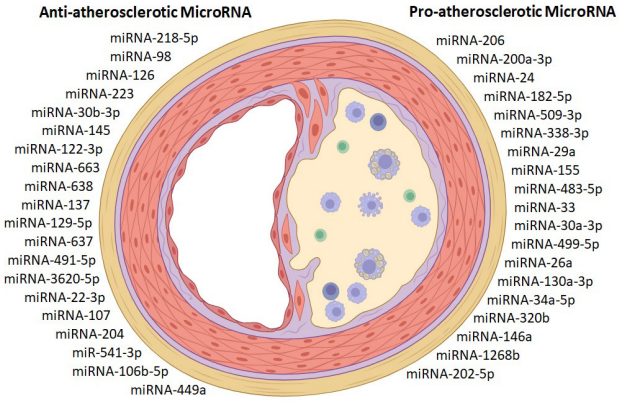

Fig. 2. Pro-atherogenic and anti-atherogenic MicroRNAs

Endothelial dysfunction is first step towards the development of atherosclerosis. Endothelial dysfunction is characterized by impaired vasorelaxation owing to decreased production of nitric oxide (NO) by endothelial nitric oxide synthase (eNOS). Endothelial dysfunction further start a series of atherosclerotic events including increased endothelial permeability, release of adhesion molecules, recruitment of monocytes and lipoproteins to the sub-intimal region, phenotype switching of vascular smooth muscle cells (VSMC) and foam cell formation by macrophages.

Majority of the previous studies report beneficial role of microRNA (miRNA) in regulating NO release from eNOS (Sun *et al*., 2012; Yang *et al*., 2017), however, some studies have reported that miRNA-200b induce endothelial dysfunction by deregulating NO release from eNOS (Bi *et al*., 2015; Janaszak-Jasiecka *et al*., 2018). Another family member of miRNA-200, miRNA-200a-3p, also promote endothelial dysnsfunction by inducing pyroptosis of H_2O_2 treated human aortic endothelial cells (HAEC) by activating SIRT1/NF-κB/NLRP3 pathway (Liu *et al*., 2023). MiRNA-24 induce endothelial dysfunction by down-regulating the PI3K/AKT/eNOS signaling pathway (Yang *et al*., 2023). MiRNA-182-5p mediate proinflammatory effects in endothelial cells by upregulating MYD88 (myeloid differentiation factor 88) and activating NF-κB/NLRP3 signaling pathway during sleep deprivation (Li *et al*., 2023). MiRNA-509-3p induce apoptosis in mouse aortic endothelial cells under oxidative stress conditions (Zhang *et al*., 2023). The similar apoptotic effect in HAEC was induced by miRNA-338-3p, acting downstream to circ_0026218 and targeting SIRT6 (Yang *et al*., 2023). miRNA-29a also promotes inflammation driven apoptosis in human umbilical vein endothelial cells (HUVEC) by down-regulating PI3K/AKT/Bcl-2 axis (Zhang *et al*., 2023). Nicotine induced miRNA-155 rich extracellular vesicles from monocytes trigger endothelial dysfunction by modulating the TIMP3, BCL2, BCL6, MCL1. MicRNA-155 also activates NF-κB mediated vascular inflammatory response leading to vascular damage (Wang *et al*., 2023). Anti-miRNA-155 nanotherapy efficiently downregulated miRNA-155 in endothelial cells of atherosclerotic lesion and helped in inhibiting inflammation (Liu *et al*., 2023).

An *in-vitro* experiments conducted by Zhu *et al*. (2023) revealed critical role of miRNA-483-5p in inducing endothelial dysfunction and impairing endothelial autophagy by modulating the expression of TIMP-2 gene. Upregulation of this miRNA in response to oxidized-low density lipoprotein (ox-LDL; an indicator of oxidative stress) also increased the expression of proinflammtory cytokines, interleukin 6, interleukin 1β, intercellular adhesion molecule 1(ICAM-1) and vascular cell adhesion molecule 1(VCAM-1). Increased expression of ICAM-1 and VCAM-1 stimulate recruitment and transmigration of monocytes to sub-intimal region (Habas and Shang, 2018), where these monocytes mature into macrophages and start formation of foam cells by taking up ox-LDL.

MiRNA-33, encoded from introns of sterol regulatory element-binding protein (SREBP)-1 and SREBP-2 genes, affect the lipid metabolism and increase risk of atherosclerosis (Tanashyan *et al*., 2023) by post-transcriptionally regulating ABCA1 and ABCG1 genes which are mainly involved in reverse cholesterol transport and high-density lipoprotein (HDL; a.k.a good cholesterol) synthesis (Horie *et al*., 2012; Huang *et al*., 2023). A recent study by Montaño-Samaniego *et al*. (2023) showed that hepatocyte (main hub of lipid metabolism) specific down-regulation of miRNA-33 by antimiR-33 sponge can help in reducing the onset of atherosclerosis. Even precise inhibition of miRNA-33 in pro-inflammatory endothelial cells can reverse atherogenic changes by increasing the expression of ABCA1 and Apo-AI (HDL specific surface protein) (Huang *et al*., 2023). Doxorubicin, a well-known drug used for cancer treatment, also uses miRNA-33 as linking piece of puzzle for causing cardiac toxicity by impairing lipid metabolism (Zhu *et al*., 2023). Another

potential drug, Astaxanthin, is also reported to promote reverse cholesterol efflux in macrophages by modulating CircTPP2/miR-3073b-5p/ABCA1 Pathway (Zhang *et al*., 2023). MiRNA-30a-3p also trigger atherosclerosis in non-alcoholic fatty liver disease (NAFLD) patients by ameliorating the expression of *ABCA1* gene and abolishing the cholesterol efflux (Chen *et al*., 2023).

Initiation of these proinflmmatory pathways also trigger phenotype switching of VSMC from contractile to proliferatory phenotype leading to proliferation and migration of VSMC to sub-intimal region. Sheng *et al*. (2023) observed an increased expression of miRNA-499-5p in ox-LDL treated mice aortic tissues and VSMC. Experiments proved that miRNA-499-5p induce VSMC proliferation and migration by targeting *SOX6* gene. MiRNA-499-5p/*SOX6* axis leads to overexpression of PCNA, cyclin D1, and matrix metalloproteinase (MMP2) and down-regulates the expression of p21, an inhibitor of VSMC proliferation. Increased level of serum miRNA-499-5p and miRNA-26a were also observed in patients of acute myocardial infarction (which is a consequence of atherosclerosis plaque rupturing) and coronary artery disease, respectively (Abdel-Hamed *et al*., 2023; Coban *et al*., 2023). Contrary to the pathological role of miRNA-499-5p in atherosclerosis and myocardial infarction, interaction between miRNA-499-5p and *SOX6* gene play beneficial role in arterial fibrillation by suppressing the proliferation, migration and invasion of atrial fibroblasts due to activation of transforming growth factor β (TGF- β) induced Smad2 signaling (Han *et al*., 2023; Zhao *et al*., 2023).

MiRNA-130a-3p also promotes atherosclerosis by disturbing the plasma lipid balance. This miRNA binds with *LDLR* (low density lipoprotein receptor) gene and down-regulates its expression resulting in increased concentration of circulatory LDL, which is a risk factor for atherosclerosis (Xu *et al*., 2023). A recent study by Xu *et al*. (2023) have shown that Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors down-regulate the production of miRNA-130a-3p in hepatocytes and play a positive role in lipid metabolism to alleviate atherosclerosis in coronary arteries. This finding is in-line with the study of Sun *et al*. Thus, it could be said that PCSK9 inhibitors paly anti-atherosclerotic role by hindering the binding between miRNA-130-3p and *LDLR* gene.

MiRNA-34a-5p also increases lipid uptake in macrophages by targeting *MDM4* gene (Kong *et al*., 2023). Ox-LDL (an indicator of oxidative stress) stimulated macrophages also release miRNA-320b enriched exosomes which induce VSMC viability, invasion and phenotype switching (Ren *et al*., 2024).

Differentiation of VSMC from stem cells is an important physiological mechanism in blood vessels. Recent study by Zhang *et al*. (2023) reported that along with other differentiation promoting factors, miRNA-146a also induce VSMC differentiation from embryonic stem cells by targeting Kruppel-like factor 4 (KLF4) and increasing the expression of VSMC specific markers genes (SMαA, SM22, SMMHC and h1-calponin) (Zhang *et al*., 2023). Expression of these VSMC specific markers decreases with the phenotype switching from contractile to proliferatory form (Bennett *et al*., 2016). Overexpression of Lectin-like oxidized low-density lipoprotein receptor-1 (LOX-1) in hepatocytes protects from phenotype switching of VSMC by up-regulating ALOX15-SRBI/ABCA1 axis (Zhang *et al*., 2023). Liposome based delivery of exogenous miRNA-146a to HAEC, aortic VSMC and macrophages reduce inflammatory responses by decreasing adhesion of monocytes, reducing ox-LDL uptake and foam cell formation (Ho *et al*., 2023).

miRNA-1268b acts downstream to circSCRG1 (a circulatory RNA) and promotes ox-LDL induced angiogenesis by increasing the expression of nuclear receptor subfamily 4 group A member 1 (NR4A1) (Yuan *et al*., 2023). Oxidative stress induced angiogenesis causes plaque instability and increases the chances of plaque rupture (Camaré *et al*., 2017). It is also experimentally proved that miRNA-202-5p induce atherosclerotic plaque formation with less collagen and thin fibrous cap by targeting Bcl-2 in macrophages (Xu *et al*., 2023).

ANTI-ATHEROGENIC microRNAS

MiRNA-218-5p, miRNA-98 plays a protective role in atherosclerosis by regulating inflammation and endothelial dysfunction (Chen *et al*., 2023; Yu *et al*., 2023). MiRNA 126 and miRNA-223 are associated with increased carotid plaque stability (Theofilis *et al*., 2023; Zhu *et al*., 2023). MiRNA-30b-3p is mostly expressed in vascular smooth muscle cells (Zhang *et al*., 2023) and plays an important role in ameliorating atherosclerosis induced myocardial injury by down-regulating the Brd4 gene. These protective effects of miRNA-30b-3p are ameliorated by taurine upregulated gene 1 (TUG1; long non-coding RNA) which inhibit its binding to Brd4 gene (Li *et al*., 2023). This microRNA also targets Rho-related spiral coil 2 containing protein kinase (RHO2) in patients of arteriosclerosis obliteran and reduce the disease progression by inhibiting vascular smooth muscle cell proliferation, migration and phenotype switching (Zhang *et al*., 2023). MiRNA-145 and miRNA-122-3p also regulates phenotype switching of VSMCs and reduce atherosclerosis (Wu *et al*., 2023; Zhang *et al*., 2023). Precise delivery of miRNA-145 to

VSMC *in-vitro* and *in-vivo* promotes contractile phenotype of VSMCs and reduce atherosclerotic plaque area by 48% (Wu *et al*., 2023). A single dose of miRNA-145 micelle provides long term efficacy against atherosclerosis (Chin *et al*., 2023). Proliferation and migration of VSMCs under oxidative stress is also inhibited by miRNA-663/HMGA2, circ_0007478/miRNA-638/ROCK2 and miRNA-137/TRPC3 signaling pathways (Deng and Li, 2023; Guan *et al*., 2023; Li *et al*., 2023). On the other side, MiRNA-129-5p decreases proliferation of endothelial cells by targeting fibroblast growth factor 2 (FGF-2) (Garmy-Susini *et al*., 2004; Li *et al*., 2023).

Human umbilical vein endothelial cells (HUVEC) and human aortic endothelial cells (HAEC) are commonly used in cardiovascular research. A study by Lau *et al*. (2021) has proved that both venous or arterial endothelial cells are equally suitable for cardiovascular research and no significant differences were found in membrane integrity and biochemical molecules produced by these cells (Lau *et al*., 2021). At physiological state, expression level of miRNA-21, miRNA-126, let-7a, miRNA-23a, miRNA-221, miRNA-125b, miRNA-26a, miRNA-29a, miRNA-16, and miRNA-100 is also almost same in both cell types (An *et al*., 2023). However, two independent studies published by Ge *et al*. (2023) and Zhang *et al*. (2023) showed differences in the upstream and downstream regulation of same miRNA (miRNA-637) in ox-LDL induced HUVEC and HAEC. In HUVEC, ox-LDL increase the expression of LINC00346 which act as sponge for miRNA-637 and increase vascular injury. Overexpression of miRNA-637 helps in mitigating vascular endothelial injury by modulating the expression of NLRP1 (Ge *et al*., 2023). While in ox-LDL induced HAEC, miRNA-637 promotes cell proliferation and angiogenesis and inhibited inflammation and cell apoptosis by modulating the expression of *TRAF6* by acting downstream to Circ-0003575 (Zhang *et al*., 2023). Ox-LDL induced HUVEC also down-regulate the expression of miRNA-491-5p by up-regulating LINC002381. miRNA-491-5p mitigate the vascular injury, cell apoptosis and inflammation by suppressing the transcription factor 7 (TCF7) (Zhu *et al*., 2023). MiRNA-3620-5p also reduce oxidative stress induced endothelial cell dysfunction by binding to the 3` untranslated region (UTR) of MAOB (monoamine Oxidase, a regulator of oxidative stress in endothelial cells) gene.

MiRNA-126 is another protective microRNA, it suppress the foam cell formation by reducing the phygocytosis of ox-LDL by macrophages. Furthermore, it also promotes the switching of M1 macrophage (proinflammatory) to M2 macrophage (anti-inflammatory) (Shou *et al*., 2023). Macrophage M2 polarization was also promoted by miRNA-22-3p by targeting *JAK1* gene resulting in reduced expression of downstream protein, NLRP3 (Bian *et al*., 2023).

MiRNA-107 also reduce plaque size and increase plaque stability by down-regulating the expression of mitochondrial dynamics protein of 51 kd (MiD51) and hypoxia-inducible factor-1a (HIF-1a) (Ren *et al*. 2023). miRNA-223-3p reduces necroptosis of foamy macrophages in unstable nacrotic cores of larger size by targeting Ripk3 (receptor-interacting protein kinase 3) gene (Jia *et al*., 2024). Increased expression of miRNA-204 in macrophages also inhibit the pathological atherosclerotic changes induced by cyclosporine-A, by targeting ApoB II gene and down-regulating the expression of CD-36 in nucleus (Su *et al*., 2023).

Most of the miRNAs either decrease ApoB II (surface protein of LDL) or increase ApoAI (surface protein of HDL) to improve lipid metabolism and ameliorating atherosclerosis. However, miR-541-3p play dual function; 1) decrease apoB II and 2) increase apoA1 expression by targeting mRNA of Znf101 (increase ApoB expression) and Casz1 (decrease ApoAI expression) (Ansari *et al*., 2023). MiRNA-106b-5p also reduces lipid deposition (in sub-intimal region), atherosclerotic plaque number, and size in ApoE knockdown mice by acting in PVT1(long non-coding RNA)/miRNA-106b-5p/ACSL4 axis (Zhang *et al*., 2023). miRNA-449a also help in reducing the burden of atherosclerosis in elderly patients by repressing the accumulation of senescent cells during aging (Noureddine *et al*., 2023).

CONCLUSION

In conclusion, the intricate regulatory role of microRNAs (miRNAs) in atherosclerosis has emerged as a dynamic and promising area of research. Recent studies from 2023 underscore the ongoing advancements in miRNA research, focusing on their specific roles and potential applications in cardiovascular health. The exploration of miRNA-based interventions, including anti-miRNA therapeutics and delivery systems, opens avenues for innovative strategies to address the complexities of atherosclerosis. Overall, the evolving landscape of miRNA research in atherosclerosis offers new possibilities for refining treatment strategies and reducing the global burden of cardiovascular diseases. Future studies are likely to uncover additional intricacies of miRNA involvement in atherosclerosis, paving the way for personalized and targeted therapeutic approaches in cardiovascular medicine.

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Statement of conflict of interest

The authors have declared no conflict of interest.

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