Dyslipidemia-Induced Cellular Senescence in Atherosclerosis: Mechanisms and Therapeutic Implications

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Atherosclerosis is a prevalent age-related condition characterized by the accumulation of lipid-rich plaques in the walls of arteries. is signi cant global health issue stands as a leading cause of mortality worldwide. Despite its signi cance, the speci c mechanisms behind atherosclerosis remain complex and multifaceted [1]. Recently, emerging evidence has shed light on the role of cellular senescence in various cell types, including endothelial cells (ECs), vascular smooth muscle cells (VSMCs), macrophages, endothelial progenitor cells (EPCs), and adipose-derived mesenchymal stem cells (AMSCs), contributing to the development of atherosclerosis [2]. Both cellular senescence and atherosclerosis share various causative stimuli, with dyslipidemia gaining considerable attention. Dyslipidemia, characterized by elevated plasma levels of atherogenic lipids or lipoproteins, or functional impairment of anti-atherogenic lipids or lipoproteins, plays a pivotal role in promoting cellular senescence and atherosclerosis. is review aims to provide a comprehensive summary of the current evidence regarding dyslipidemia-induced cellular senescence in atherosclerosis. e focus will be on low-density lipoprotein (LDL) and its modi cations, the hydrolysate of triglyceride-rich lipoproteins (TRLs), and high-density lipoprotein (HDL) [3,4]. Moreover, we will discuss potential senescence-related therapeutic strategies for atherosclerosis, with particular emphasis on the anti-atherosclerotic e ects of promising geroprotectors and the anti-senescence e ects of current lipid-lowering drugs. Understanding the interplay between dyslipidemia, cellular senescence, and atherosclerosis could pave the way for innovative approaches to tackle this prevalent and life-threatening condition. Atherosclerosis is a chronic immune-in ammatory disorder, linked to aging and characterized by the accumulation of lipid-rich plaques in arterial walls. Despite advancements in cardiology, atherosclerosis remains the leading cause of mortality worldwide [5,6]. Consequently, monocytes are recruited and cross the endothelial barrier, di erentiating into macrophages to clear accumulated lipids and lipoproteins. However, these macrophages transform into foam cells when overloaded, intensifying atherosclerotic plaque formation and eliciting an in ammatory response through the release of pro-in ammatory factors. In parallel, vascular smooth muscle cells (VSMCs) from the arterial media migrate into the intima, surrounding the in ammatory factors and lipids. ese highly proliferative VSMCs contribute to the stabilization of atherosclerotic plaques by forming a brous cap, but they also secrete various matrix metalloproteinases (MMPs) that promote plaque rupture. In advanced atherosclerotic plaques, VSMCs may adopt a foam-cell-like phenotype, further exacerbating plaque

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senescence, and atherosclerosis, we can pave the way for more targeted and personalized interventions, ultimately reducing the burden of this age-related disorder on global health.

Ack edge e

Not applicable.

C c f I e e

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