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Using Senescent Cells as a Target to Slow the Course of Cardiovascular Disease

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Introduction

The most prevalent disease to rise with increasing life expectancy is cardiovascular disease (CVD). The lack of success of the majority of well-known pharmacological treatments for age-related cardiovascular disease (CVD) suggests that novel strategies for treating these conditions are required. Senescent cardiovascular cells, which have an irreversible cell cycle arrest and a distinct senescence-associated secretory phenotype, have been found to accumulate in aging or diseased cardiovascular systems, raising the possibility that they impair cardiovascular function. The evidence that senescent cells are involved in cardiovascular aging, the onset and progression of cardiovascular disease (CVD), and the molecular mechanisms that underlie cardiovascular cell senescence are discussed in this review. We also discuss immune cell-mediated efferocytosis and toxicity, as well as small-molecule-drug-mediated apoptosis, as promising and precisely targeted therapies for the prevention and treatment of cardiovascular disease [1].

Human life expectancy is significantly increasing as a result of improved vaccine use and medical care, as well as improved water, food, hygiene, housing, and lifestyle. According to projections, the proportion of the global population under the age of 65 will rise from 13% in 2010 to 19% in 2030, while the proportion of the population under the age of 85 will rise from 0.03% in 2010 to 1.4% in 2030. According to Niccoli and Partridge, advanced age is the most important unchangeable risk factor for chronic diseases that kill people, such as cardiovascular disease (CVD), cancer, and neurodegenerative diseases CVD is the most prevalent of these, and its prevalence is expected to rise globally as populations continue to age. CVD is the main source of death in the old. In any case, the components basic improvement old enough related CVD are to a great extent obscure. Despite continued viability and metabolic activity, cellular senescence, a state of permanent cell-cycle arrest, occurs in diseased cardiovascular tissues and is strongly associated with cardiovascular ageing [2].

Description

Ageing, on the other hand, is characterized by progressive functional decline; senescence is the opposite. Ageing occurs at the tissue or organ level, whereas senescence typically occurs at the cellular level. Cell senescence drives tissue maturing and is likewise not the same as cell peacefulness portrayed by reversible cell cycle capture. In the pathophysiology of CVD, cell senescence and quiescence have distinct characteristics and functions. Numerous cardiovascular diseases, including atherosclerosis, are strongly triggered or exacerbated by senescent cardiovascular cells, according to growing evidence, stiffening of the arteries, aneurysms of the aorta, (re) stenosis, and myocardial fibrosis, and cardiovascular breakdown. The distinct characteristics of senescent cardiovascular cells, the underlying molecular mechanisms of cardiovascular cell senescence, and the emerging roles of senescent vascular cells in the onset and progression of cardiovascular disease are the subjects of this discussion [3].

The term "cardiac cell senescence" refers to a condition in which cells remain metabolically active but enter an irreversible and permanent cell cycle arrest. Vascular cell senescence can be sparked by a variety of harmful factors, such as radiation, oxidative stress, and shorter telomeres, damage to DNA, malfunctioning mitochondria, abnormal metabolism, and gene mutation. According to Bennett et al., there are two types of vascular cell senescence: The first is replicative senescence, which occurs when multiple cell divisions result in an irreversible stoppage of cell proliferation. The second is stress-induced premature senescence (SIPS), which is a stable cell cycle arrest without any detectable telomere loss or dysfunction and is typically triggered by distinct endogenous or exogenous stresses. Mitotic cells typically employ cell senescence as a strategy to prevent dysregulated cell division [2].

Post-mitotic cells, such as mature cardiomyocytes and adipocytes, also undergo cell senescence, according to new research. Post-mitotic cardiomyocyte senescence is generally triggered by DNA damage in the telomere regions. P53 enlistment intervenes the senescence of post-mitotic adipocyte. Upregulation of favorable to senescence factor p21 triggers cell senescence in post-mitotic dopaminergic neurons. Cardiovascular cell senescence is imperative for the upkeep of cardiovascular tissue homeostasis during undeveloped turn of events, tissue recovery, and wound mending. However, the pathogenesis of age-related cardiovascular disease (CVD) has been linked to the persistent accumulation of senescent cells in cardiovascular tissues, which impairs cardiovascular function. Conversely, a lack of nutrients or growth factors is typically the cause of cardiovascular cell quiescence with reversible cell cycle arrest [3].

Proliferating and quiescent cells, as well as senescent cardiovascular cells, typically differ significantly from non-senescent cardiovascular cells. There are a number of morphological and molecular characteristics of senescent cardiovascular cells that could be useful as therapeutic targets and markers. Senescent cardiovascular cells by and large presentation a trademark leveled and extended morphology, increased activity of beta-galactosidase associated with senescence (SA-gal), telomere wearing down, and amassing of cyclin-subordinate kinase inhibitor p16ink4a or p21. The senescence-associated secretory phenotype, or SASP, is the most prominent characteristic of senescent cardiovascular cells. Pro-inflammatory cytokines (like IL-6 and IL-8) and growth factors (like vascular endothelial growth factor [VEGF] and platelet-derived growth factor AA [PDGF-AA]) are secreted by senescent vascular cells, matrix metalloproteinases (MMPs), and chemokines The SASP of senescent vascular cells enables them to communicate with other cells and the microenvironment, as well as to encourage the senescence of nearby cells, tissue regeneration, and embryonic development [4].

Numerous studies have examined EC quiescence. Endothelium quiescence is typically induced by Notch signaling (Harrington et al., In order to maintain the tricarboxylic acid cycle for redox homeostasis by regenerating the reduced form of nicotinamide adenine dinucleotide phosphate (NADPH), Notch1-mediated carnitine palmitoyltransferase 1A (CPT1A) is elevated to levels three to four times higher than in proliferating ECs. Additionally, whereas

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FoxO1 activation does not cause EC senescence or apoptosis, forkhead box O1 (FoxO1) activation increases EC quiescence by lowering Myc protein levels and thereby inhibiting glycolysis. Additionally, pulmonary artery smooth muscle cell quiescence is mediated by FoxO1 activation. In EC-specific CPT1A-deleted mice, supplementation with acetate, which is metabolized into acetyl-coenzyme A, restores endothelial quiescence and combats oxidative stress-mediated EC dysfunction, providing opportunities for therapy [5].

Conclusion

A healthy cardiovascular system requires senescent cardiovascular cells to maintain homeostasis. Cardiovascular cell senescence is regulated in vivo and in vitro by a number of intricate molecular pathways. The onset and progression of a variety of cardiovascular diseases (CVDs) as well as cardiovascular aging have been linked, according to emerging evidence. CVD treatment and prevention strategies are being developed using senolytics and senescence immunotherapy. As of now, there are no exceptionally particular markers for senescent cardiovascular cells in vivo. Non-invasive spatiotemporal identification and quantification of individual senescent cardiovascular cells in vivo remains challenging. Effective CVD treatments have not been developed due to any of these circumstances. A thorough comprehension of the senescence biology of each of the major cell types that contribute to the pathogenesis of CVD will be necessary for the development of novel therapeutic approaches to target senescent cardiovascular cells and reduce significant clinical consequences like MI or stroke. Only D+Q has been tested in a clinical setting thus far, and there are currently no clinical trials testing whether senolytics can protect against cardiovascular diseases.

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