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Perspective

Cellular Senescence: Unraveling the Molecular Mechanisms and Implications in Aging

Isabella Camila*

Department of Geriatrics, Harvard University, Massachusetts, USA

DESCRIPTION

Cellular senescence is a state of irreversible growth arrest that cells enter into as a response to various stresses and signals. It is a fundamental biological process that plays a crucial role in aging, tissue repair, and the development of age-related diseases. This paper delves into the world of cellular senescence, exploring its mechanisms, triggers, functions, and implications in the context of aging and disease.

Functions of Cellular senescence

Definition and characteristics of cellular senescence: Cellular senescence is characterized by a permanent arrest of cell proliferation and the acquisition of a senescent phenotype. Senescent cells undergo morphological and functional changes, such as enlarged and flattened cell shape, altered gene expression patterns, and secretion of a range of molecules collectively known as the Senescence-Associated Secretory Phenotype (SASP). The SASP includes cytokines, growth factors, proteases, and other factors that can have both beneficial and detrimental effects on the surrounding tissue.

Senescence triggers and signaling pathways: Cellular senescence can be triggered by a variety of intrinsic and extrinsic factors. Intrinsic triggers include telomere shortening, DNA damage, oncogene activation, and epigenetic alterations. Extrinsic triggers include oxidative stress, inflammation, and various other stressors. These triggers activate specific signaling pathways, such as the p53-p21 and p16INK4a-Rb pathways, which regulate cell cycle arrest and senescence-associated gene expression.

Role of telomeres in cellular senescence: Telomeres, the protective caps at the ends of chromosomes, play a crucial role in cellular senescence. With each cell division, telomeres shorten due to the end replication problem. Once telomeres reach a critical length, a DNA damage response is activated, leading to cell cycle arrest and cellular senescence. Telomere attrition is considered a hallmark of aging, and the enzyme telomerase, which can elongate telomeres, is involved in regulating cellular lifespan.

Senescence and aging: Cellular senescence has been closely linked to the aging process. Accumulation of senescent cells in tissues is a hallmark of aging, and their presence is associated with tissue dysfunction and age-related pathologies. Senescent cells can disrupt tissue homeostasis, induce chronic inflammation, impair stem cell function, and contribute to the decline in regenerative capacity observed in aging tissues. Strategies targeting senescent cells, such as senolytics, hold promise for delaying aging and preventing age-related diseases.

Senescence and age-related diseases: Cellular senescence has been implicated in the development and progression of various age-related diseases, including cancer, cardiovascular diseases, neurodegenerative disorders, and chronic kidney disease. In some cases, senescent cells can promote tissue repair and suppress tumor formation. However, the persistent presence of senescent cells can also contribute to tissue dysfunction and drive disease pathogenesis. Understanding the role of senescence in these diseases opens up avenues for therapeutic interventions.

Senescence as a therapeutic target: Given the detrimental effects of senescent cells, targeting senescence has emerged as a potential therapeutic strategy. Senolytics are drugs that selectively eliminate senescent cells, thereby reducing the burden of senescent cells in tissues. Preclinical studies have shown that clearing senescent cells can improve tissue function, extend healthspan, and delay the onset of age-related diseases. Clinical trials are underway to evaluate the safety and efficacy of senolytic interventions.

Correspondence to: Isabella Camila, Department of Geriatrics, Harvard University, Massachusetts, USA, E-mail: camila@gmail.com

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