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Editorial

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Cellular Senescence, Anti-Aging Therapies and Inflammaging

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Introduction

Aging is a complex biological process characterized by the gradual decline of cellular and tissue function, increased susceptibility to disease, and reduced regenerative capacity. Cellular senescence, a state in which cells permanently lose the ability to divide, plays a central role in aging and age-related disorders. While senescence prevents the proliferation of damaged cells and reduces cancer risk, the accumulation of senescent cells contributes to chronic inflammation, tissue dysfunction, and what is termed "inflammaging." Understanding the mechanisms of senescence and its relationship to inflammaging has paved the way for the development of novel anti-aging therapies aimed at promoting healthspan and mitigating age-related decline [1,2].

Discussion

Cellular senescence can be triggered by various stressors, including DNA damage, oxidative stress, telomere shortening, and oncogene activation. Senescent cells adopt a distinctive phenotype, often characterized by the senescence-associated secretory phenotype (SASP), which involves the secretion of pro-inflammatory cytokines, chemokines, growth factors, and proteases. While SASP signals recruit immune cells to clear damaged cells, chronic accumulation of senescent cells leads to persistent low-grade inflammation, tissue remodeling, and functional decline—hallmarks of inflammaging. This process is implicated in numerous age-related conditions, including cardiovascular disease, osteoarthritis, neurodegenerative disorders, and metabolic dysfunction [3,4].

Anti-aging therapies targeting cellular senescence aim to reduce its detrimental effects while preserving its tumor-suppressive benefits. Senolytics are a class of drugs that selectively eliminate senescent cells, thereby reducing SASP-driven inflammation and restoring tissue function in preclinical models. Another strategy involves senomorphics, which modulate the secretory profile of senescent cells to mitigate inflammation without killing the cells. Additionally, lifestyle interventions, such as regular exercise, calorie restriction, and antioxidant-rich diets, have been shown to slow senescence accumulation and reduce inflammaging. Emerging approaches, including gene editing and immune-mediated clearance of senescent cells, hold promise for more precise interventions in the future [5].

The translation of these therapies from bench to bedside requires careful evaluation of efficacy, safety, and long-term outcomes, as the complete removal of senescent cells may have unintended consequences. Moreover, personalized approaches considering individual aging trajectories and genetic predispositions are likely to enhance therapeutic success.

Conclusion

Cellular senescence is a double-edged sword in aging: protective against cancer yet contributing to chronic inflammation and tissue dysfunction through inflammaging. Targeting senescent cells and their pro-inflammatory secretions represents a promising avenue for anti-aging therapies aimed at extending healthspan. By integrating pharmacological, genetic, and lifestyle strategies, it may be possible to mitigate age-related functional decline and promote healthy aging, ultimately improving quality of life in the elderly population.

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