Stem cell aging: Implications for tissue regeneration and disease.

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Introduction

Aging is an inevitable biological process characterized by the progressive decline in physiological function and increased susceptibility to age-related diseases. At the cellular level, aging is accompanied by a gradual deterioration in the regenerative capacity and homeostatic balance of tissues, attributed in part to alterations in stem cell function. In this article, we explore the phenomenon of stem cell aging, elucidating the molecular mechanisms underlying cellular decline and its implications for aging-related pathologies [1].

The role of stem cells in tissue homeostasis

Stem cells play a pivotal role in maintaining tissue homeostasis throughout life by replenishing damaged or senescent cells and supporting tissue repair and regeneration. These self-renewing and multipotent cells reside in various tissues and organs, continuously dividing to generate daughter cells that differentiate into specialized cell types. However, with advancing age, the regenerative capacity of stem cells declines, contributing to tissue dysfunction and susceptibility to age-related diseases [2].

Molecular hallmarks of stem cell aging: Stem cell aging is characterized by a multitude of molecular alterations that impair cellular function and regenerative potential [3]. Telomere shortening, a hallmark of cellular aging, limits the proliferative capacity of stem cells by triggering replicative senescence or apoptosis. Moreover, epigenetic modifications, such as changes in DNA methylation patterns and histone modifications, can alter gene expression profiles and impair stem cell function. Dysregulated signaling pathways, including the mTOR pathway and oxidative stress response, further exacerbate stem cell aging by disrupting cellular homeostasis and promoting senescence [4].

Mitochondrial dysfunction and metabolic changes: Mitochondrial dysfunction is a prominent feature of stem cell aging, characterized by impaired mitochondrial biogenesis, increased reactive oxygen species (ROS) production, and compromised ATP synthesis [5]. These metabolic alterations disrupt cellular energy balance and compromise stem cell function, leading to impaired proliferation and differentiation capacities. Additionally, age-related changes in metabolic pathways, such as alterations in glycolysis, fatty acid metabolism, and nutrient sensing, further contribute to stem cell aging and functional decline [6].

The decline in stem cell function associated with aging has profound implications for the development of aging-related pathologies, including neurodegenerative diseases, cardiovascular disorders, and musculoskeletal degeneration [7]. Reduced regenerative capacity and tissue repair mechanisms contribute to the progression of these diseases, exacerbating tissue damage and functional decline. Moreover, age-related changes in the microenvironment, or "stem cell niche," further impair stem cell function and exacerbate tissue dysfunction in aging tissues [8].

Strategies for rejuvenating aging stem cells: Despite the challenges posed by stem cell aging, ongoing research efforts are exploring various strategies to rejuvenate aging stem cells and enhance their regenerative potential [9]. These strategies include interventions targeting telomere maintenance, epigenetic modifications, mitochondrial function, and metabolic pathways. Additionally, advances in stem cell-based therapies, such as the use of young donor cells or genetically engineered stem cells, offer promising avenues for rejuvenating aging tissues and treating age-related pathologies [10].

Conclusion

Stem cell aging represents a fundamental aspect of the aging process, contributing to the decline in tissue function and increased susceptibility to age-related diseases. By unraveling the molecular dynamics underlying stem cell aging, researchers aim to develop innovative strategies for rejuvenating aging stem cells and restoring tissue homeostasis. Ultimately, these efforts hold the potential to mitigate the burden of aging-related pathologies and improve healthspan in aging populations.

References

1. Oh J, Lee YD, Wagers AJ. Stem cell aging: mechanisms, regulators and therapeutic opportunities. Nat Med. 2014;20(8):870-80.
2. Liu L, Rando TA. Manifestations and mechanisms of stem cell aging. Med J Cell Biol. 2011;193(2):257-66.
3. Fehrer C, Lepperdinger G. Mesenchymal stem cell aging. Exp Gerontol. 2005;40(12):926-30.
4. Chen F, Liu Y, Wong NK, et al. Oxidative stress in stem cell aging. Cell Transplant. 2017;26(9):1483-95.
5. Revuelta M, Matheu A. Autophagy in stem cell aging. Aging cell. 2017;16(5):912-5.
6. Lengefeld J, Cheng CW, Maretich P, et al. Cell size is a determinant of stem cell potential during aging. Sci Adv. 2021;7(46):eabk0271.

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7. Ergen AV, Goodell MA. Mechanisms of hematopoietic stem cell aging. Exp Gerontol. 2010;45(4):286-90.
8. Hong X, Campanario S, Ramírez-Pardo I, et al. Stem cell aging in the skeletal muscle: The importance of communication. Ageing Res Rev. 2022;73:101528.
9. Wagner W, Horn P, Bork S, et al. Aging of hematopoietic stem cells is regulated by the stem cell niche. Exp Gerontol. 2008;43(11):974-80.
10. Jasper H. Intestinal stem cell aging: origins and interventions. Annu Rev Physiol. 2020;82:203-26.

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