COLLOQUIA ERUDITA 2026; 1: 71–72

PROCEEDINGS ABSTRACT

Immunosenescence and inflammaging: molecular mechanisms, age-related diseases, and therapeutic interventions

Fatima Abdulkareem Alkhafji and Enass Najem Oubaid

Received: 07 April 2026; Revised: 13 June 2026; Accepted: 28 June 2026; Published: 20 July 2026

Aging is a complex biological process marked by progressive cellular and molecular changes that affect all physiological processes, especially those related to the immune system. Amongst these, increased susceptibility to infections, decreased vaccine efficacy, and a higher incidence of chronic diseases are all consequences of immunosenescence. The latter is defined as "the age-related decline and dysregulation of immune responses". The tissue accumulation of senescent cells that release a variety of pro-inflammatory mediators is collectively referred to as the senescence-associated secretory phenotype (SASP) and is known to be capable of causing chronic low-grade inflammation / "inflammaging". Inflammaging is a key component of the process that accelerates immune cell aging, reduces the efficient clearance of senescent cells, and limits the immune response to novel antigens. This decline of both innate and adaptive immunity is significantly influenced by this ongoing inflammatory state, resulting in delayed immune responses and compromised host defence systems. This review provides an overview of the molecular drivers of immunosenescence and inflammaging, and discusses their roles in age-related diseases and their importance for targeted therapeutic interventions. Immunosenescence is controlled at the molecular level by a number of interrelated molecular signalling pathways. Amongst these, the nuclear factor kappa B (NF-κB) pathway is a major cause of chronic inflammation because it inhibits the senescent cells' ability to undergo apoptosis and stimulates the production of immune mediators. Similarity, immunological dysfunction, and decreased T lymphocyte responsiveness are caused by a dysregulation of the mammalian target of rapamycin (mTOR) pathway, which also affects autophagy and cellular metabolism. Furthermore, by detecting cytosolic DNA and by inducing type I interferons, the activation of the cyclic GMP–AMP synthase / stimulator of interferon genes (cGAS-STING) pathway has become a significant mediator of age-related inflammation. These inflammatory signalling pathways are further amplified by oxidative stress and mitochondrial dysfunction. Significant cellular changes are linked to immunosenescence and include telomere attrition, thymic involution, haematopoietic stem cell dysfunction, and reduced naïve T lymphocyte production; all these impair immune surveillance and response to novel antigens. Reduced B lymphocyte diversity and antibody production are also consequences of this process. Together, these alterations play a key role in the pathophysiology of age-related diseases (such as cancer), severe viral infections (such as the coronavirus disease 2019), cardiovascular diseases, neurodegenerative diseases (such as Alzheimer's disease), and type 2 diabetes mellitus. In fact, immune dysregulation and chronic inflammation

Alkhafji F. A., Oubaid E. N.: Immunosenescence and inflammaging: molecular mechanisms, age-related diseases, and therapeutic interventions. *Colloq. Erud.* 1: 71–72 (2026).
https://doi.org/10.5281/zenodo.21452224

This proceedings abstract is published in *Colloquia Erudita – Volume 1: Proceedings of the 3rd International Babylon Conference on Clinical and Experimental Pharmacological Research (Hillah; June 29–30, 2026)*; a volume of proceedings edited by Rafal J. Al-Saigh, Argyro Kyriakaki, and Apostolos Zarros.

serve as common underlying mechanisms connecting immunosenescence to the aforementioned pathologies. Recent approaches in the field of therapeutics have been targeting the underlying mechanisms of immune aging. Senotherapeutics, being the use of senomorphics in order to alter the secretory phenotype of senescent cells and senolytics that specifically eradicate them, have demonstrated encouraging outcomes in both preclinical and clinical studies. Through the modification of important pathways such as the mTOR and NF-κB ones, pharmaceutical agents like metformin and rapamycin can lower inflammation and boost immunity. Cytokine modulation, immune-based treatments, and lifestyle modifications (including the adoption of regular physical activity, balanced nutrition, and adequate sleep) have all been shown to exert positive effects on immune aging. In conclusion, immunosenescence is a multifaceted process that is forced by intricate molecular pathways and cellular changes, and that has important ramifications for the emergence of age-related diseases. However, the development of targeted therapeutic interventions that are meant to improve immune function, increase longevity, and encourage healthy aging, requires a deeper understanding of these mechanisms and their clinical implications.

Keywords

aging; immunosenescence; inflammaging; senotherapeutics; signalling pathways

Acknowledgements

The authors would like to express their gratitude to the staff and colleagues at the College of Pharmacy of the University of Babylon for their support.

Conflicts of interest statement

None to declare.

Affiliation(s)

College of Pharmacy, University of Babylon, Hillah, Iraq *(FAA)* ▪ Department of Pharmacognosy, College of Pharmacy, University of Babylon, Hillah, Iraq *(ENO)*

Correspondence

Fatima Abdulkareem Alkhafji
College of Pharmacy
University of Babylon
Hillah, Iraq
pha829.fatimah.abdulkareem@student.uobabylon.edu.iq