Mediators of Inflammation

Hindawi

Special Issue on

Cellular Senescence and Inflammaging in Age-Related Diseases

CALL FOR PAPERS

The connection between cellular senescence and inflammaging has recently emerged as a causal mechanism of ageing process. This interplay constitutes the favourable substrate for the development of all age-related diseases (ARDs), including cardiovascular diseases (CVDs), type 2 diabetes mellitus (T2DM), musculoskeletal disorders, various types of cancer, and neurodegenerative diseases.

The growing evidence suggests that senescent cells can exert a proinflammaging effect when the rate of accumulation increases, that is, during aging. Upon ageing, they, indeed, acquire a proinflammatory secretory phenotype (SASP), which contributes to spreading inflammaging at the systemic level. In turn, this feeds to a vicious circle that accelerates both tissue dysfunction and onset of all ARDs. Accordingly, SASP of senescent cells has been suggested to contribute to onset of CVDs, insulin resistance, T2DM, and osteoporosis and to senescence of adult stem/progenitor (ASC/APC) cells (i.e., hematopoietic stem cells) associated with tissue dysfunction, immunosenescence, hematologic malignancies, and anaemia. SASP can also stimulate development and progression of several forms of cancer. However, the characterization of SASP components released by different types of senescent cells is not yet complete, as well as the biological functions of various secretomes associated with senescence in different tissue microenvironments.

Although cellular senescence occurs chronically during "normal" ageing, unhealthy lifestyle choices, such as smoking, nonregular exercise, sucrose and/or fat rich diets, and exposure to external stressors (e.g., radiations, pollutants, and chemotherapy), can accelerate the accumulation of senescent cells, therefore promoting onset of ARDs.

Recently, there is increasing evidence that the clearance of senescent cells in animal models attenuates the progression of age-related disorders. This is leading strongly to supporting the hypothesis that senescent cell clearance, reprogramming of senescent cells (epigenetic modulation), or the modulation of proinflammatory pathways related to the acquisition of SASP (i.e., NLRP3 inflammasome, TLR-2/-4, Notch, NF-Kb pathway, insulin/IGF-pathways, and mTOR) may be pursued as potential strategies for combating ARDs and expanding the health span of humans.

The purpose of this special issue is to publish high-quality research papers as well as review articles addressing recent advances in this field. Original, high-quality contributions that are not yet published or that are not currently under review by other journals or peer-reviewed conferences are sought.

Potential topics include but are not limited to the following:

- Senescence of adult stem/progenitor cells and related mechanisms: inflammaging of niches and effects
- Senescence of normal cells (including endothelial cells, immune cells, fibroblasts, and preadipocytes) and related biological effects, that is, onset of CVDs, T2DM, musculoskeletal disorders, various types of cancer, and neurodegenerative diseases
- Mechanisms and pathways related to SASP and inflammaging evocation in different microenvironments
- Inflammatory markers related to cellular senescence and their diagnostic/prognostic relevance in human ARDs
- Strategies and interventions to improve the clearance of senescent cells and/or to reduce the systemic spreading of SASP
- Futuristic clinical settings appropriate to test new senescence or SASP-inflammaging modifying drugs in humans

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/mi/csiin/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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