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Natalia Loaiza, Marco Demaria

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Cellular senescence and tumor promotion: is aging the key?

Natalia Loaiza[1], Marco Demaria[1,2*]

[1]University Medical Center Groningen (UMCG), Groningen, The Netherlands; [2]European Research Institute for the Biology of Aging (ERIBA), Groningen, The Netherlands;

[*]Correspondence to: m.demaria@umcg.nl

Abstract

The senescence response is a potent tumor suppressor mechanism characterized by an irreversible growth arrest in response to potentially oncogenic signals to prevent proliferation of damaged cells. Late in life some of the features of senescent cells seem to mediate the development of age-related pathologies, including cancer. In the present review, we present a summary of the current knowledge regarding the causes, effector pathways and cellular features of senescence. We also discuss how the senescence response, initially a tumor suppressor mechanism, turns into a tumor promoter apparently as a consequence of aging. We argue that three age-related phenomena—senescence-associated secretory phenotype (SASP) dysregulation, decline in the immune system function and genomic instability—could contribute, independently or synergistically, to deteriorate the efficacy of the senescence response in stopping cancer. As a consequence, senescent cells could be considered premalignant cells, and targeting senescent cells could be a preventive and therapeutic strategy against cancer.