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Editorial

Senescent Tumor Cells: The Pacifism or Power Play of Tumor Aggression

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Cellular senescence is an irreversible cell cycle arrest caused by various internal or external stimuli [1,2]. Senescence phenotype was first identified by Hayflick in normal human fibroblasts [3]. Senescence is caused due to exhaustion of cells by continuous replication (replicative senescence) or it can be induced (induced senescence or premature senescence) by various agents including reactive oxygen species, tumor suppressors, oncogenes, radiations etc. Senescent cells are characterized by high accumulation of β-galactosidase, $p16^{INK4A}$, $p21^{Waf1}$, flattened cells morphology, reduced cells proliferation and also the release of senescence associated secretory phenotypes (SASPs), which comprise proinflammatory cytokines and chemokines, extracellular matrix proteins, growth factors, and exosome-like small extracellular vesicles [4-7].

Senescence is the characteristic of both normal cells and cancerous cells. In normal individuals senescence is a replicative phenomenon due to telomere shortening and is considered as a consequence of aging [8]. In cancer patients, the tumor cells' senescence is caused by a plethora of complex mechanisms involving tumor suppressors, oncogenes, therapeutic agents etc. Cancer cells senescence was once considered to be an anti-tumorigenic mechanism restricting proliferation and the invasion of cancer cells, however, the latest research reveals cancer cells senescence as an elusive phenomena adding to tumor progression. For example, senescent tumor cells (STCs) have been shown to be involved in epithelial-mesenchymal transition (EMT), inhibit immune cells infiltration and promote local invasion through the release of cancer-promoting factors [1,4,5]. This pro-malignant behavior of STCs have led to the use of a new class of drugs called senolytics in cancer therapy that specifically target senescent cells. Contrary to this, chemotherapy or radiotherapy are associated with cancer cell senescence and dormancy, which leads to a high rate of cancer relapse thus acting as an anticancer mechanism [9]. However, obscure causative factors and their heterogeneity in various cancers has made it difficult to establish the physiological role of STCs

The debate regarding the deceptive character of senescent cells in cancer is one of the leading research topics in cancer biology these days. Many review and original research articles are being published highlighting this overlooked role of STCs. It is worth mentioning that so far the negative role of STCs has not been identified in all types of cancers and the research on this stringent topic is still at an infant stage and is largely unknown. Moreover, the associated SASPs also vary greatly in cancer type dependent manner. In cancer cells, senescence can be induced by several factors. This diversity in induction may define the pacifism or aggressive nature of STCs. The role of STCs in cancer progression has been overlooked because STCs have been considered as a defense mechanism against cancer rather than a progression. However, recent studies have suggested new insight of cancer promoting effect of STCs. But analysis of large tumor tissue samples of various cancer stages and different cancer types are required to validate these cancer promoting effects. Finally, the senolytic drugs used to combat the STCs also show some off target adverse reactions in normal cells of patients; thus to eliminate these unsolicited effects, more specific target oriented senolytic drugs need to be discovered for cancer therapy.

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