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Short Communication

Replicative Senescence in Human Ageing

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DESCRIPTION

Replicative senescence is thought to start as a result of telomere erosion. Recent research, however, indicates that numerous other triggers that work independently of telomeres may be able to cause a senescent phenotype. Furthermore, whether cells are of rat or human origin, as well as the species of origin of the cells, affects telomere-dependent replicative senescence. Moreover, the process through which cells enter replicative senescence may differ depending on the tissue of origin. We divide cellular senescence into two categories in this study-intrinsic and extrinsic senescence-and highlight the variations between human and mouse cells while also talking about the functions of the p53 and pRb tumour suppressor pathways in cellular senescence.

Adaptive immunity relies heavily on clonal proliferation, which is why the immune system must take replicative senescence, the stringent limit in the proliferative ability of normal human somatic cells, into consideration. In cell culture, $CD8^+$ T lymphocytes that undergo repeated cycles of antigen-driven proliferation ultimately reach the replicative senescence end stage, which is marked by irreversible cell-cycle arrest and a dangerously low telomere length. Senescent $CD8^+$ T cell cultures additionally display resistance to apoptosis, a permanent loss of CD28 expression, changed cytokine profiles, a diminished capacity to react to stress, and a number of functional alterations. Human immunodeficiency virus infection in younger people causes the accumulation of cells with identical traits, indicating that replicative senescence is not just a phenomenon of cell culture but also occurs *in vivo*. It's interesting to note that osteoporotic fractures and low antibody responses to vaccines are both associated with older people's large percentage of $CD8^+$ T cells with replicative senescence features. Furthermore, $CD8^+$ $CD28^-$ T cells increase in certain cancer patients. Human diploid fibroblasts experience replicative senescence mostly as a result of cell cycle arrest at the G1/S border. Although post-transcriptional mechanisms may also be involved, senescent arrest resembles a process of terminal differentiation that seems to entail the repression of genes that promote proliferation and the reciprocal new expression of genes that suppress it.

The general cellular decline of biological ageing and its significant opposite manifestation, the infrequent escape of cells from senescence leading to immortalization and oncogenesis, will be clarified with the identification of contributing genes and elucidation of their mechanisms of action.

A limited number of population doublings in culture causes human diploid fibroblast cells to stop growing. We measured the degree of oxidative DNA damage in senescent IMR-90 human fibroblast cells by employing indicators such as 8-oxoguanine excised from DNA and 8-oxo-2'-deoxyguanosine in DNA.

Four times more 8-oxoguanine is excreted from DNA each day by senescent cells than by early-passage youthful cells. In senescent cells compared to young cells, the steady-state level of 8-oxo-2'-deoxyguanosine in DNA is around 35% higher. Protein carbonyl measurements reveal that senescent cells didn't seem to have increased protein oxidation. We compared the replicative life span of cells cultured at the O_2 concentration of air (20%) to cells cultured at a more physiological O_2 concentration (3%) to limit the amount of oxidative damage. Mesenchymal Stem Cells (MSCs) are being used in regenerative medicine to create cell treatments for a variety of disorders linked to ageing. A large number of cells are required for effective therapy, necessitating considerable *ex vivo* cell growth. The limited proliferative capacity of MSCs suggests that long-term culture elicits ongoing alterations in MSCs. As a result, a sizeable number of cells may experience senescence. The phenotypic characterisation of senescent human MSCs (hMSCs) and any potential functional changes they may undergo are presented here.

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