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Perspective

Regulation of Cellular Senescence

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DESCRIPTION

Cellular health is regulated at various points throughout the cell. As chromosome structure and organisation, transcriptional regulation, nuclear export and import begin in the nucleus and move outward to control protein translation and quality, organelle recycling by autophagy, maintenance of cytoskeletal structure and maintenance of the extracellular matrix and extracellular signalling. Cellular ageing is usually understood to be the gradual loss of cellular function and eventual cell death caused by a cellular response to stress and other cellular abnormalities. Cellular ageing has been incredibly instructive, shedding light on cancer, differentiation and cell-cycle control. The term "cellular senescence" which was first used to describe cells cultivated in cell culture refers to a permanent cell cycle stop. Senescence is thought to be a highly dynamic, multi-step process and during this time senescent cells' characteristics are constantly changing and diversifying in a context-dependent way. Numerous cellular and molecular modifications as well as distinctive phenotypic alterations including a persistent proliferative arrest that is insensitive to mitogenic stimuli are connected to it. Senescent cells exhibit substantial changes in gene expression, altered metabolic activity and a complicated senescence-associated secretory phenotype while still alive.

Several cell-biological processes control cellular health. Various cellular ageing processes are coordinated by conserved gene-regulatory networks to preserve cellular health. Because cellular health is regulated at many different scales from the molecular to the cellular and in every area of the cell the processes that govern cellular health are interconnected poor protein quality results in defective organelles which in turn cause increased ROS (Reactive Oxygen Species) which in turn results in even lower protein quality. Given the connections between nutritional availability, longevity and reproduction it is likely that longevity pathways evolved to connect somatic health to postponed reproduction in times of poor nutrient supply. Ageing's mechanisms whether replicative or chronological are poorly understood through research using several genetic model species twenty genes that can increase an organism's longevity have been discovered. Although older cells are more prone to apoptosis not all senescent cells necessarily undergo quick self-destruction. When a cell undergoes apoptosis it releases proteins that induce the cell to lyse by neatly packing up all of its internal components.

Depending on the severity of the damage and the surrounding physiological conditions, DNA damage initiates the DNA repair process, apoptosis or senescence. Chronic ATM (Ataxia Telangiectasia and Rad3 related) and ATR (Ataxia Telangiectasia and Rad3 related) kinase signalling which ultimately causes cell cycle arrest and senescence through activation of the p53/p21 and p16/pRb pathways are characteristics of senescent cells. A small portion of the genetic code is lost throughout each replication cycle, every time a cell divides and as the cell ages. Additionally, the telomere caps decrease the likelihood of DNA damage or improper replication increases as a cell ages and its telomeres shorten.

CONCLUSION

Senescence can be activated both during development and during tissue remodelling. Additionally senescent cell temporary induction is seen during wound healing and aids in the healing process. Senescence may also act as a stress response that is protective. Senescence, for instance is best known for being a powerful anticancer process. Senescent cells on the other hand can build up with time. Senescent cells restrict metabolic tissue regeneration and alter tissue architecture and sterile inflammation. Age-related disorders are brought on by this alteration of the metabolic equilibrium.

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