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Stress, cell senescence and organismal ageing

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Highlights

1. Cellular senescence is the irreversible arrest of normally dividing cells.
2. Telomere shortening is the mechanism driving senescence in human fibroblasts.
3. Olivier Toussaint and others have shown that stress plays a major role in the induction of senescence *in vitro*.
4. Senescent cells can cause tissue disruption and/or degeneration
5. Pharmacological approaches targeting senescent cells are being developed

Abstract

Cellular senescence was first described by Hayflick and Moorhead in the 1960s as the irreversible arrest of cells following prolonged cultivation. Telomere shortening is the key mechanism driving replicative senescence in human fibroblasts. Later, pioneering work by Olivier Toussaint and others showed that stress plays a major role in the induction of senescence *in vitro*, a phenomenon known as stress-induced premature senescence or SIPS. It is also now widely accepted that senescence plays a role *in vivo*. An emerging body of evidence from animal models, and particularly mice, has demonstrated an important role for senescence in several processes such as embryonic development, wound healing, tumour suppression and ageing. However, mostly due to a lack of availability of tissues and specific markers, less is known about the importance of cell senescence in humans. In this review, we summarize some of the key findings in the field of senescence, stress-induced senescence and telomeres. We focus particularly on the role of telomere dysfunction and senescence during the ageing process as well