Senolytics and senescent cells

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The study deals with anti-ageing therapy and the role of senolytics in eliminating senescent cells, delaying the deterioration of organs and preventing age-related diseases.

As the human body ages, increasing numbers of cells enter the state of senescence. Senescent cells do not divide or support the tissue they are part of. Instead of this they emit a wide range of potentially harmful chemical signals, which encourage other nearby cells to also enter the same senescent state.

Senescent cells normally destroy themselves via a genetically programmed process called apoptosis, and they can also be removed by the immune system.

However, the immune system weakens with age and many senescent cells escape this process and build up. Senescent cells cause many problems: increase levels of inflammation, impair tissue function, contribute to osteoarthritis and atherosclerosis and eventually raise the risk of cancer. Over the past few years experiments have confirmed that senescent cells accumulate in ageing organs and their elimination can alleviate or even prevent certain diseases. Relevant studies have shown that by removing senescent cells it is possible to give a boost to some of the tissue's natural repair mechanisms and stimulate new tissue production. This anti-ageing phenomenon has been an unexpected turn in the study of senescent cells, a common, non-dividing cell type first described more than fifty years ago. When a cell enters senescence — and almost all cells have the potential to do so — it stops producing copies of itself, begins to belch out hundreds of proteins, and cranks up anti-death pathways full blast. A senescent cell is in its twilight: not quite dead, but not dividing as it did at its peak. Senescent cells depend on protective mechanisms to survive in their undead state. Scientists have discovered and identified six signaling pathways that prevent cell death, which senescent cells activate to survive. To date, 14 senolytics have been described in the literature, including small molecules, antibodies and a peptide that activates a cell-death pathway and can restore thick hair and physical fitness of ageing mice. So far, each senolytic kills a particular type of senescent cell. Targeting different diseases of ageing, therefore, will require multiple types of senolytics.

Successful results from mouse studies have already attracted several biotechnology and pharmaceutical companies into this field, which are currently testing senolytics, trying to kill senescent cells in the hope of delaying or preventing the destroying effects of aging.

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