Answer: A

1. Senescent cells are common in [skin](https://en.wikipedia.org/wiki/Skin) and [adipose tissue](https://en.wikipedia.org/wiki/Adipose_tissue).

Senescent cells are usually larger than non-senescent cells.

Transformation of a dividing cell into a non-dividing senescent cell is a slow process that can take up to six weeks.

The [secretome](https://en.wikipedia.org/wiki/Secretome" \o "Secretome) of senescent [cells](https://en.wikipedia.org/wiki/Cell_(biology)) is very complex.

2. **Senescence-associated secretory phenotype** (**SASP**) is  [phenotype](https://en.wikipedia.org/wiki/Phenotype) associated with [senescent cells](https://en.wikipedia.org/wiki/Cellular_senescence) wherein those cells secrete high levels of [inflammatory cytokines](https://en.wikipedia.org/wiki/Inflammatory_cytokine), [immune](https://en.wikipedia.org/wiki/Immune) modulators, [growth factors](https://en.wikipedia.org/wiki/Growth_factor), and [proteases](https://en.wikipedia.org/wiki/Protease).

Senescent cells appear in the process of embryonic development and use **SASP** to act as a primary signal that triggers macrophage-mediated cell removal, which is necessary for the proper development of individual embryonic structures.

On one hand, **senescent cells** are thought to mediate tissue development when **they** form in the embryo, and also to promote tissue regeneration and wound repair in later life.

3. A **stem**-**cell niche** is an area of a tissue that provides a specific microenvironment, in which **stem cells** are present in an undifferentiated and self-renewable state.

**Cells** of **the stem**-**cell niche** interact with **the stem cells** to maintain them or promote **their** differentiation.

4.  Therapeutic strategies that safely interfere with the detrimental effects of cellular senescence, such as the selective elimination of senescent cells (SNCs) or the disruption of the SNC secretome, are gaining significant attention, with several programmes now nearing human clinical studies.

5. daf-2 mutants survive longer in a decrepit state because of a beneficial trait. They are resistant to colonization of the digestive tract by dietary bacteria, a condition that leads to premature death in the wildtype and prevents their manifestation of decrepitude. If bacterial colonization is prevented, then daf-2 mutants lead both chronologically and proportionately healthier lives relative to the wild-type.

6. Reactive oxygen species produced through peroxisomes are a main contributing factor to cell oxidative pressure, which is supposed to significantly boost up growing old and cellular death consistent with the free radical idea of getting old.

7. Within the TOR pathway, mTOR protein kinase is located in two discrete complexes, both includes protein additives, which phosphorylates different substrates. Rapamycin, is a molecule which inhibits any such complexes can increase lifespan.

8. With recent findings that deletion of the mTOR substrate S6K1 or publicity of mice to the mTOR inhibitor rapamycin bring about lifespan extension.

9. Pathophysiology. The widely known version of bronchiectasis is cole's 'vicious cycle hypothesis'. It is proposed that an environmental insult, on a history of genetic susceptibility or disorder in host defence, unleashes a sequence of occasions main to progressive bronchial wall destruction and dilatation.

10. Severe studies have tested that senescent cells, which might be characterized by means of sustained cell cycle arrest and manufacturing of senescence-related secretory phenotype, gather with age and at sites of age-related illnesses at some point of the body, wherein they actively promote tissue deterioration. Cells with functions of senescence had been detected inside the context of brain aging and neurodegenerative sickness, suggesting that they will additionally promote disorder.

B

1. Mainly in metabolism and aging, the mammalian sirtuin protein family (SIRT1–SIRT7) regulates mitochondrial function and enhances stem cell survival. Sirtuins deacetylate histones and several transcriptional regulators in the nucleus in cellular compartments.

2. Reactive oxygen species (ROS) and nitric oxide (NO) are important regulators in HSC aging. The accumulation of ROS in aged cells induces FOXO depletion, NF-jB activation, p38-mTOR activation, telomere shortening, DNA damage, and mitochondrial dysfunction. Insulin and IGF activate the PI3K-Akt signaling pathway and phosphorylate FOXO, followed by inhibition of the expression of the anti-oxidant N-acetyl-L-cysteine. After oxidative stress, HSCs increase NO levels, which results in loss of self-renewal, abnormal proliferation, and malignancy.

3. Lysosomal dysfunction is implicated in several aging-related neurodegenerative diseases including Alzheimer’s, Parkinson’s, amyotrophic lateral sclerosis/frontotemporal dementia, and Huntington’s.

4. The **stem cell** theory of **aging** postulates that the **aging** process is the result of the inability of various types of **stem cells** to continue to replenish the tissues of an organism with functional differentiated **cells** capable of maintaining that tissue's (or organ's) original function.

5. Age-related changes in systemic factors, intracellular signalling pathways, and cell cycle controlling molecules also seem to be implicated in HSC self-renewal and differentiation diversity. TGF-b1 enhances myeloid differentiation rather than lymphoid differentiation, and the chromatin regulator Satb1, which is induced during lymphoid differentiation, decrease in aged HSCs.

6. The well‐established abilities of peroxisomes to oxidize fatty acids to acetyl‐coenzyme A (CoA) and to use it then in anaplerotic reactions to replenish tricarboxylic acid (TCA) cycle intermediates destined for mitochondria are integrated into several longevity regulation pathways within a cell.

C

1. Mutations in the lamin A gene (LMNA) as the genetic cause of the segmental premature aging disease.

Activation of a cryptic splice site generates an aberrant form of lamin A, called progerin, which is constitutively lipid-modified

 Activation of p53, deregulation of the somatotrophic axis, and attrition of adult stem cells are some of the hallmarks of aging whose alteration can also be linked to the cause of HGPS.

The vast majority of HGPS cases are caused by a single-base substitution (GGC > GGT) which does not cause an amino acid change (G608G) but results in deletion of 150 nucleotides in exon 11 causing an alternatively spliced truncated variant of lamin A mRNA and an in-frame deletion of 50 amino acids near the carboxy terminus, lead to changes in the nuclear architecture.

2. Damaged and misfolded proteins accumulate with age, impairing cell function and tissue homeostasis.

The accumulation of damaged proteins contributes to multiple age-related diseases such as Alzheimer’s, Parkinson’s or Huntington’s disease.

Damaged proteins are degraded by the ubiquitin–proteasome system or through autophagy-lysosome, key components of the proteostasis network.