

Medical Biology

By

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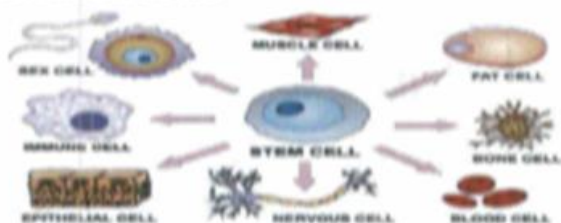
أ.م.د . خالدة ابراهيم نوئيل : Academic Profile

**CELL DIFFERENTIATION,
PROLIFERATION, RENEWAL,
DEATH
& CELL SENESCENCE**

Difference between cell proliferation and cell differentiation:

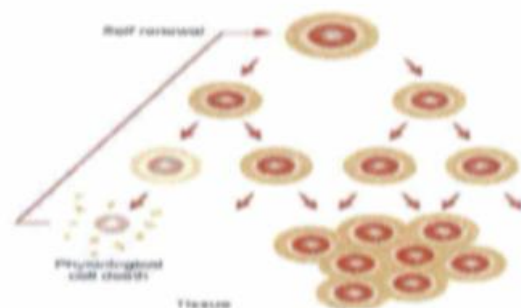
Cell differentiation

It is the process by which unspecialized cells acquire specialized structural and/or functional features that characterize the specialized cells.



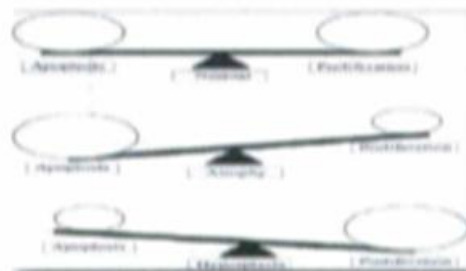
Cell proliferation

Increase in the number of cells by division.

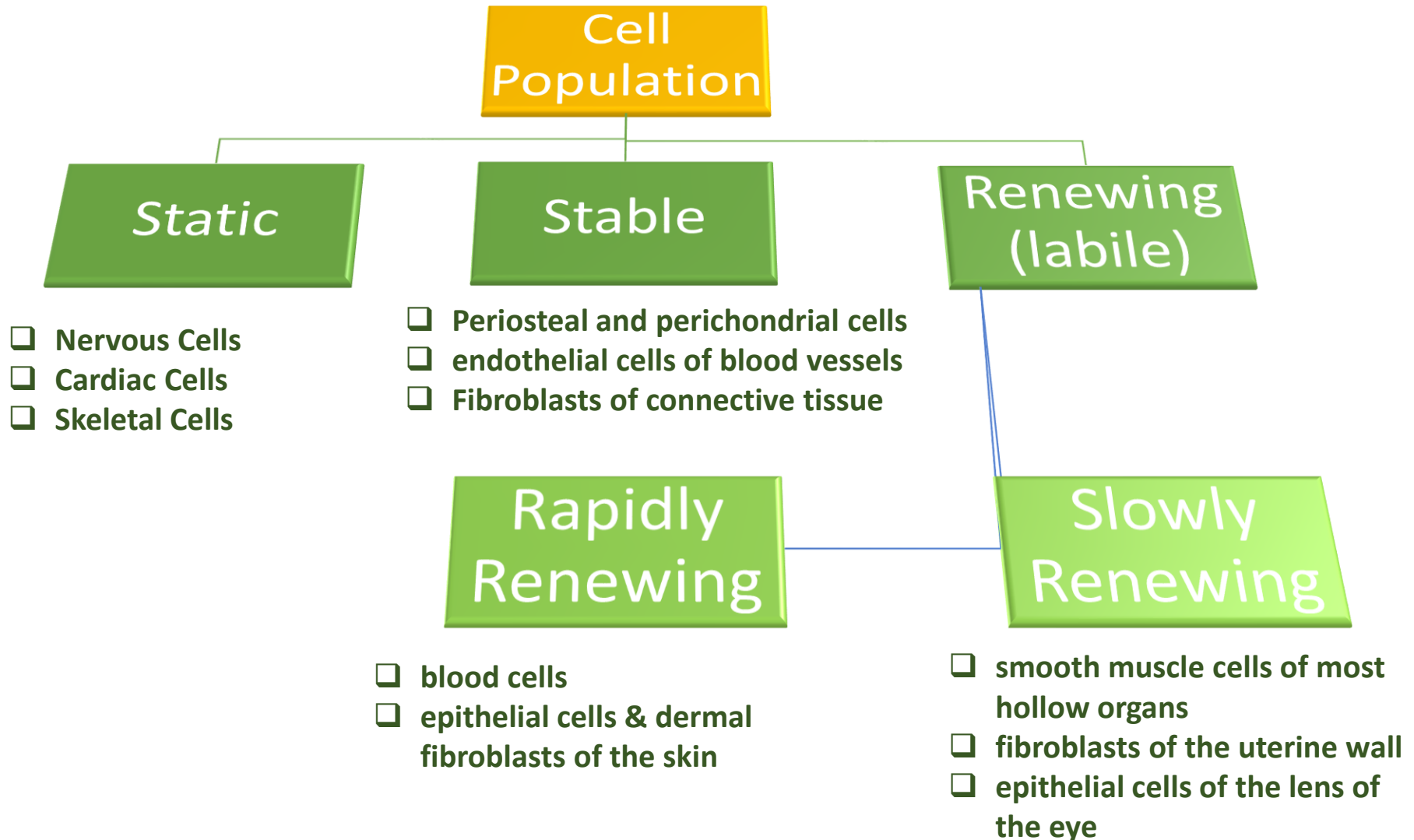


Cell proliferation & differentiation:

- Early development: Rapid proliferation of embryonic cells, which then differentiate to produce the many specialized types of cells that make up the organs.
- As cells differentiate: The rate of proliferation decreases, and many cells are arrested in the G0 stage.
- Cell proliferation is balanced with cell death to maintain a constant number of cells.



CELL RENEWAL



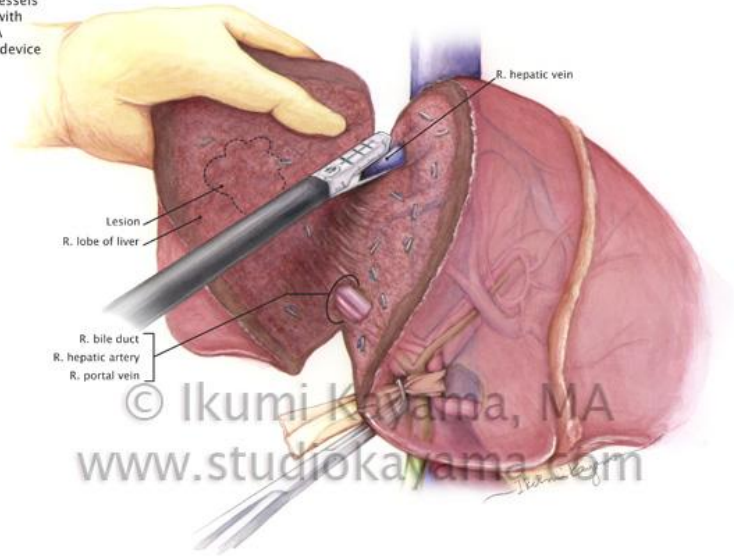
RESERVE

STEM CELLS



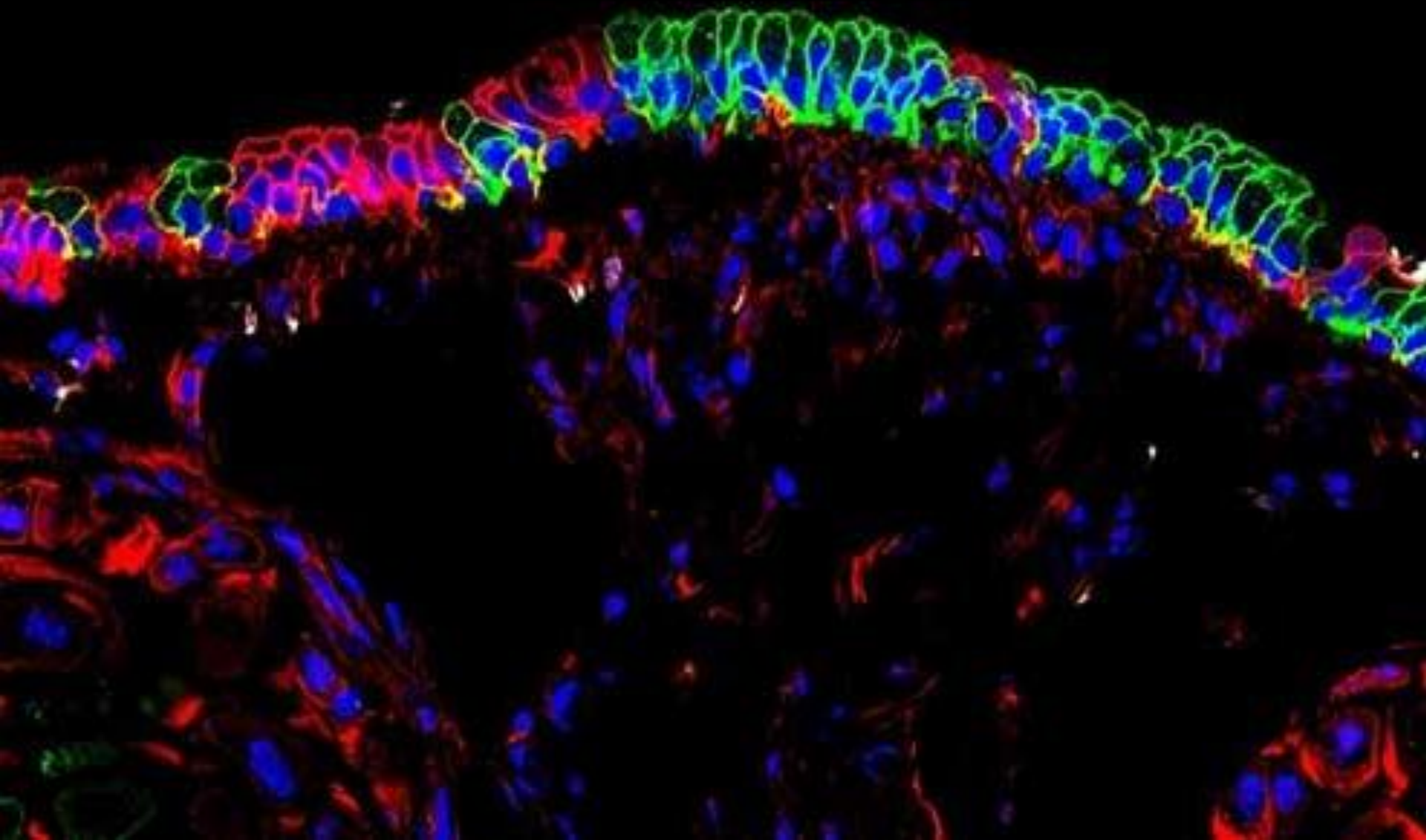
Wound Healing

6 Larger vessels divided with Endo-GIA stapling device



Replacement of removed tissue

Green colored cells are reserve stem cells



CELL DIVISION



CELL DEATH



HOMEOSTASIS



CELL ACCUMULATION DISORDERS:

- cancer
- lupus erythematosus
- glomerulonephritis
- viral infections



CELL LOSS DISORDERS:

- AIDS
- Alzheimer's disease
- Parkinson's disease
- aplastic anemia
- myocardial infarction

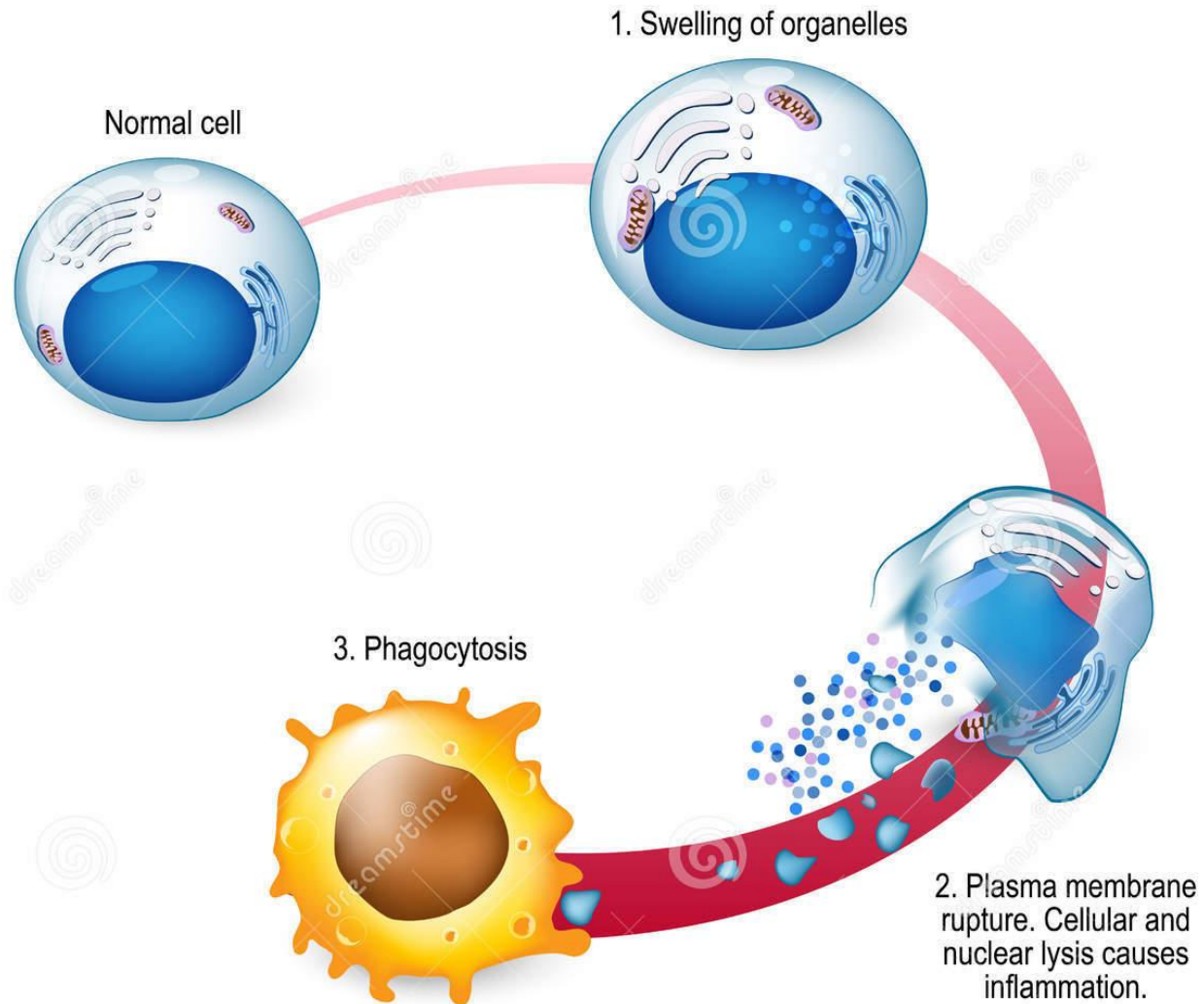
Cell death

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graph TD; A[Cell death] --- B[Apoptosis]; A --- C[Necrosis]
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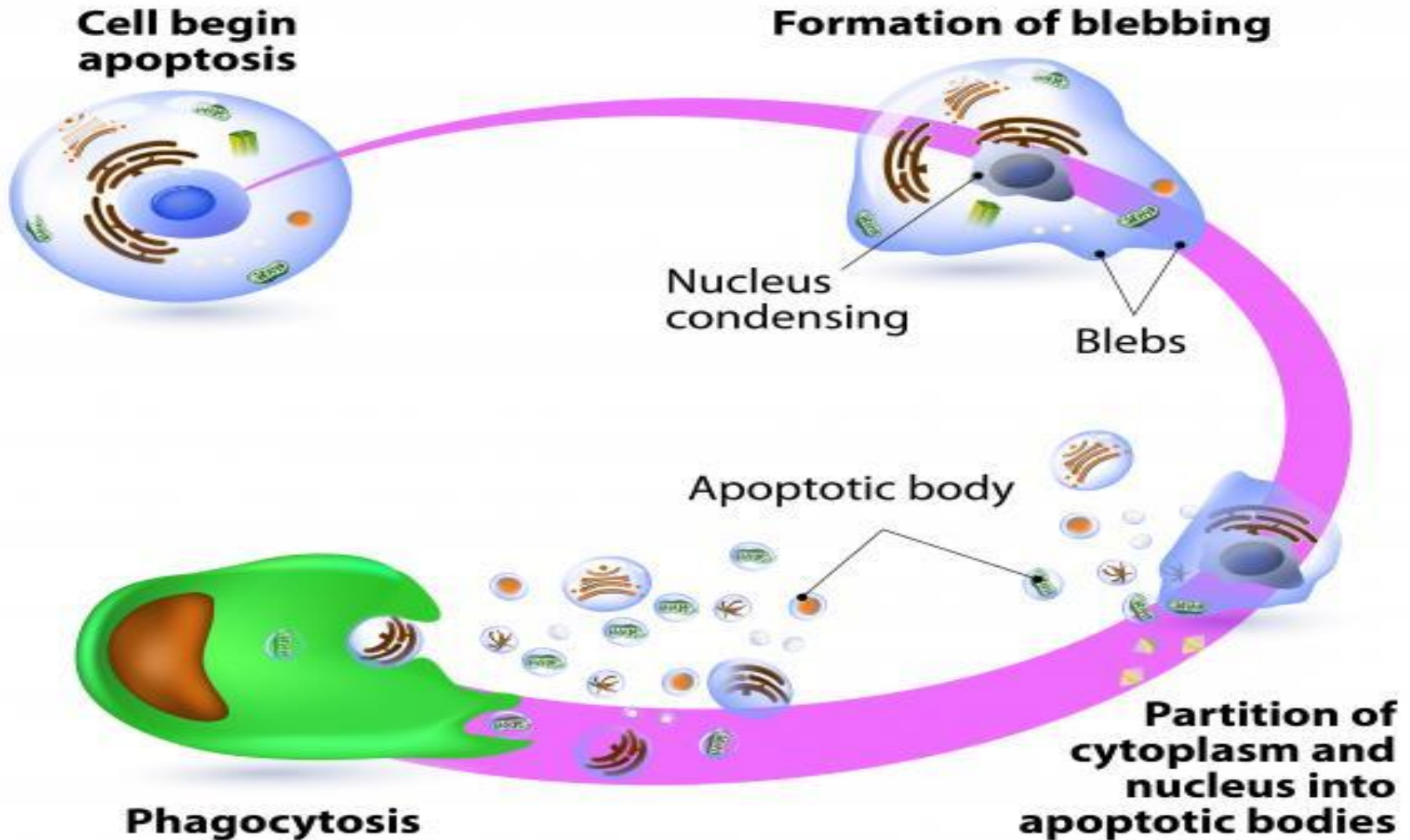
Apoptosis

Necrosis

NECROSIS



APOPTOSIS

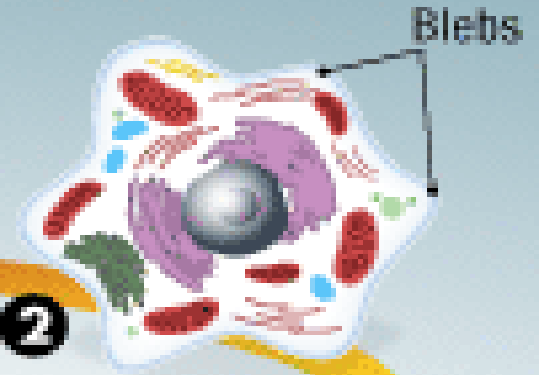


Apoptosis Process

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1 Cells damaged, stressed or triggered by body signals, begin apoptosis.



2 The cell begins to shrink and form blebs. Proteins are activated to break down cellular components.



3 Enzymes break down the nucleus and the cell emits signals to attract macrophages.



4 The cell breaks into several smaller pieces containing the cell components and destroyed nucleus.



5 Macrophages recognize the cell parts and remove them from the body.

Regulation of Apoptosis

- ⊕ External activators: free radicals, oxidants, and UV and ionizing radiation.
- ⊕ Internal activators: oncogenes (tumor forming gene), tumor suppressors (such as p53), nutrient-deprivation antimetabolites and mitotic catastrophe.
- ⊕ Apoptosis inhibited by signals from other cells and the surrounding environment via so-called survival factors. These include growth factors, hormones such as estrogen and androgens, and interactions with extracellular matrix proteins.

Necrosis

Apoptosis

1. Type

Pathological.

Physiological.

2. Cell membrane

Damage with loss of its integrity.

Change of some characters without loss of its integrity.

3. Organelles

Broken down.

Intact.

4. Proteins

Denatured or coagulated.

Broken down of DNA with hypercondensation of chromatin.

5. Apoptotic bodies

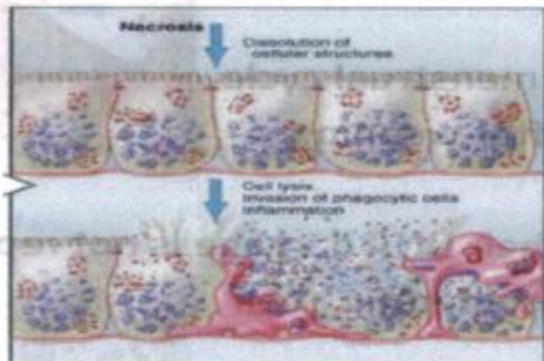
Absent

Present

6. Inflammation

Present

Absent

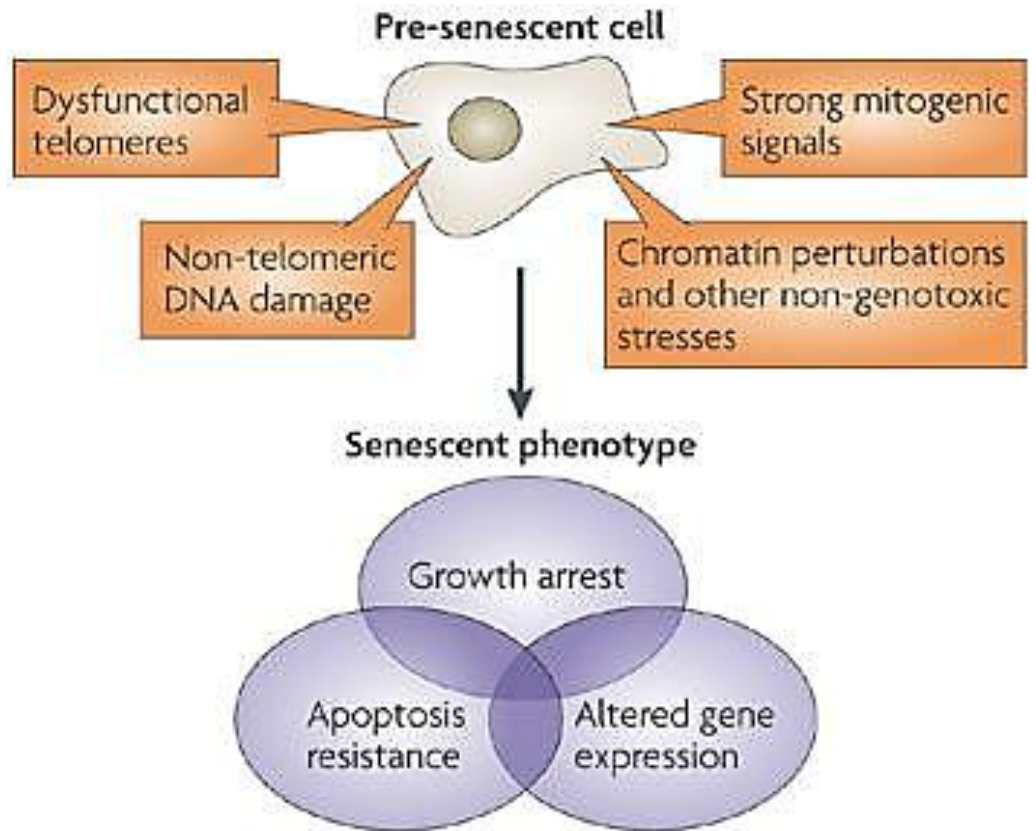


AGEING & CELLULAR SENESCENCE

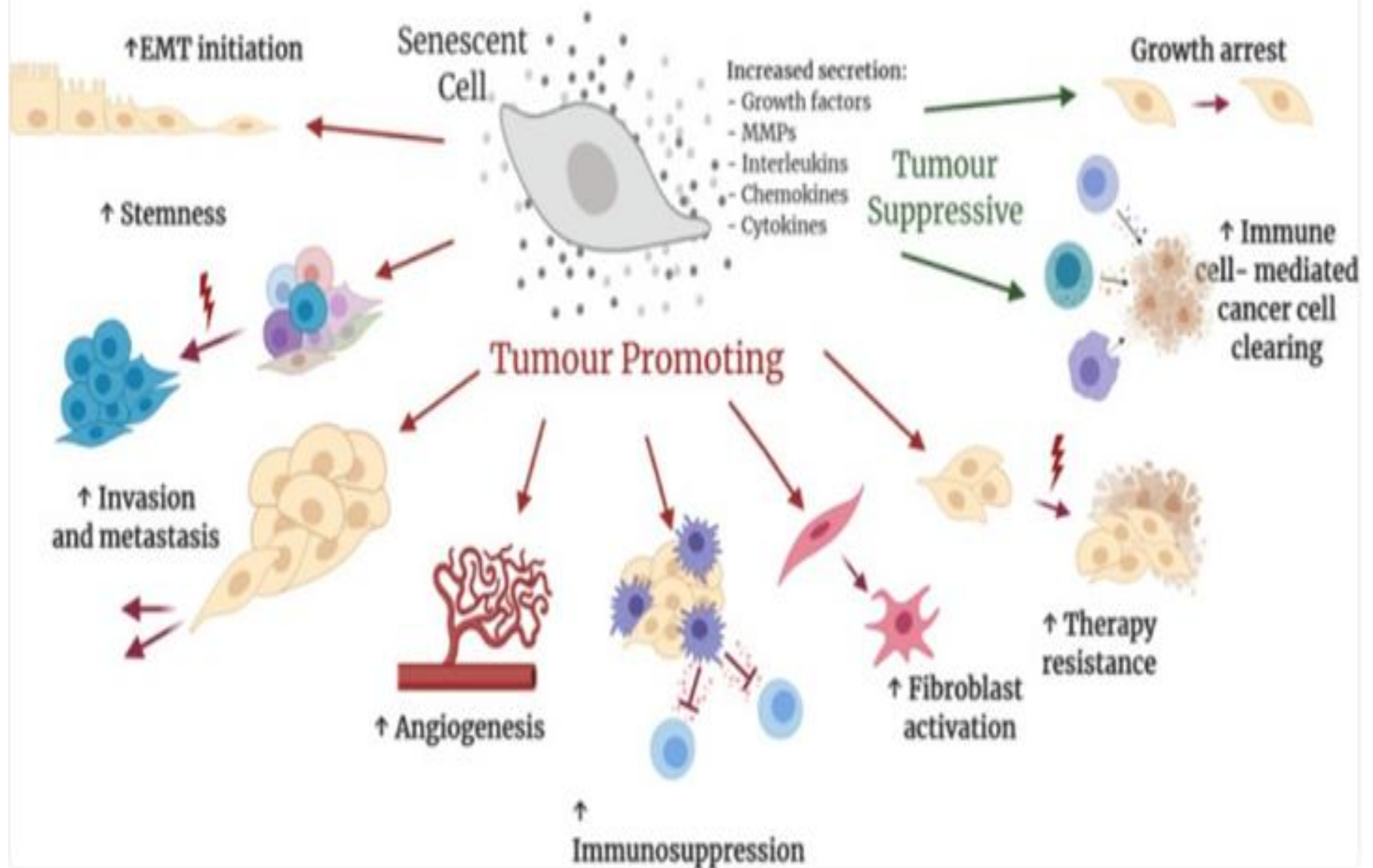
⊕→ Ageing is a universal feature of biological organisms, defined by a gradual decline over time in cell and tissue function that often, but not always, decreases the longevity of an individual.

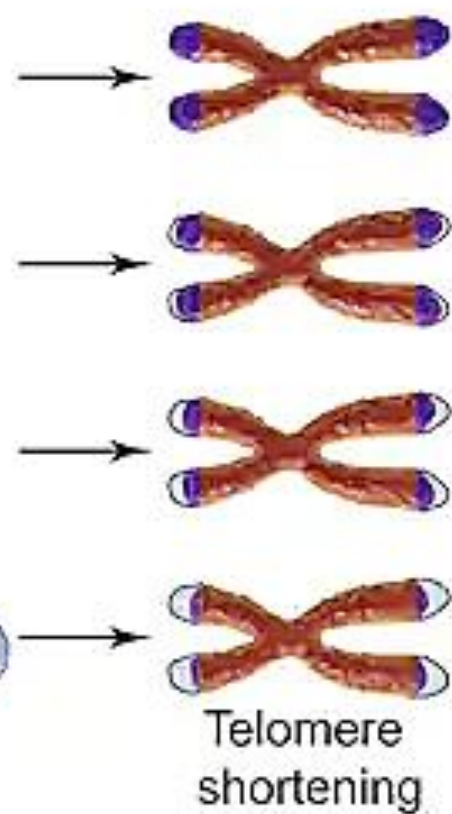
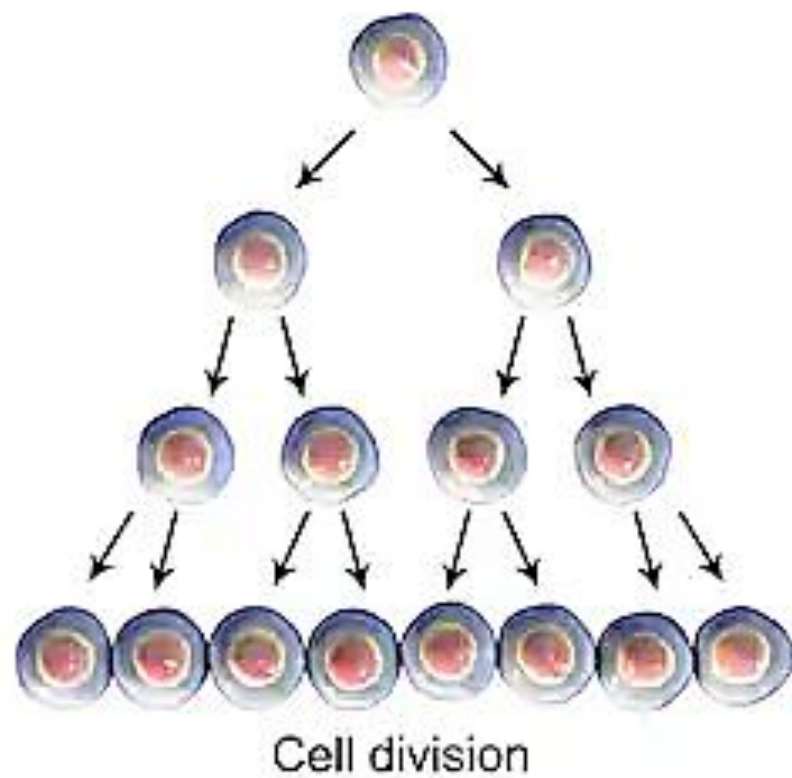
⊕→ Cellular senescence is defined by an irreversible arrest in cell proliferation when cells experience DNA damage at telomeres and a decrease in mitogenic signaling.

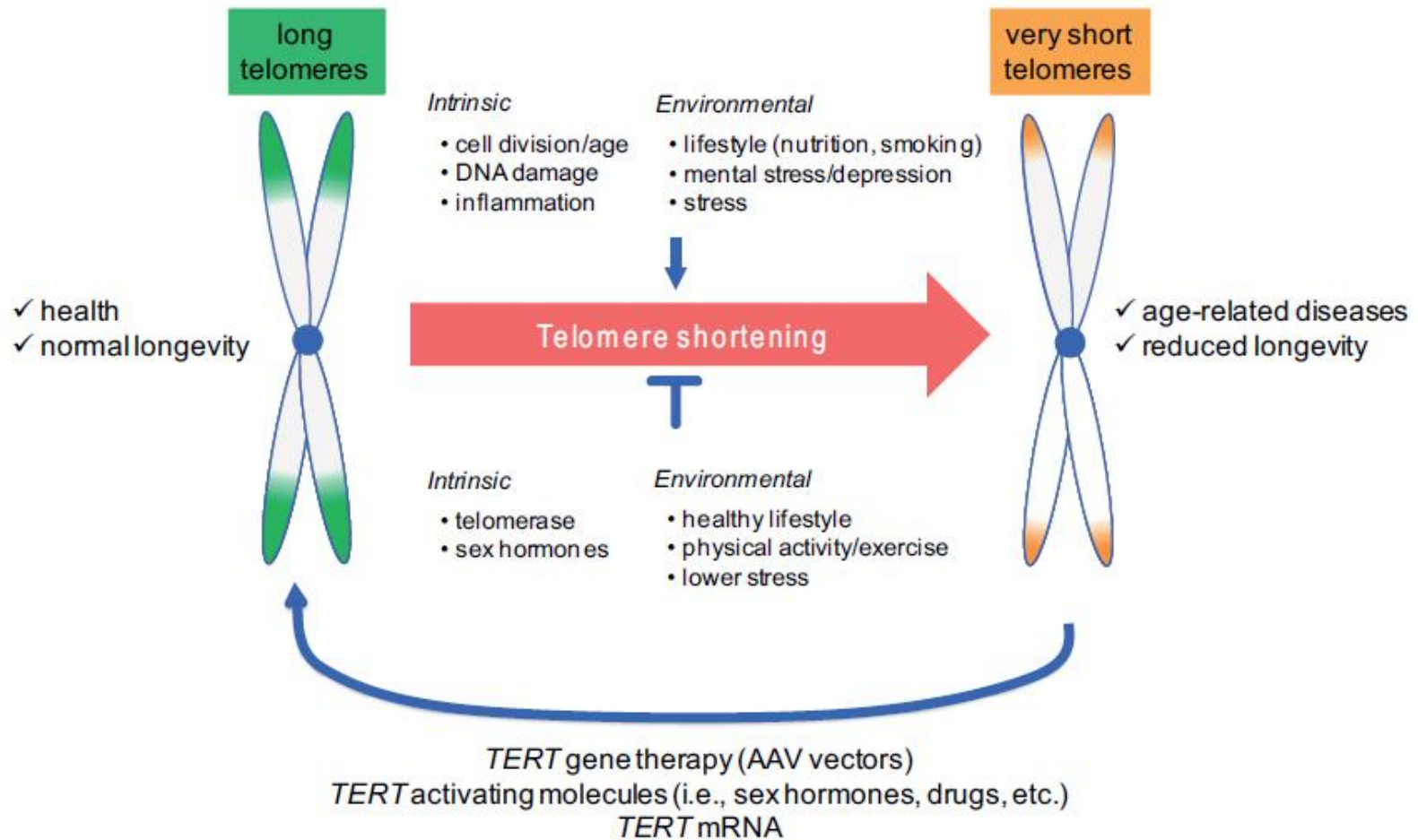
CELL SENESCENCE



Senescence-associated secretory phenotype (SASP)







Thank
You

