

## Ageing

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Ageing has been defined as the decline in the biological organ function with time leading to death. It is one of the major risk factors for common diseases (diabetes, Alzheimer's, cancer, and heart disease) and takes place at various stages as we become less fit mentally and physically. Research has been going on regarding the mean and maximal longevity to provide us with evidence on how age can be operated. There are factors that can affect ageing such as vitamin supplement, physical exercises as well as living a healthy lifestyle leading to longevity. This article reviews the molecular hallmarks behind ageing biologically. These nine hallmarks can be put into three categories as shown in the figure 1.

Genomic instability	
Telomere attrition	
Epigenetic alterations	Primary hallmarks Causes of damage
Loss of proteostasis	
Deregulated nutrient sensing	
Mitochondrial dysfunction	Antagonistic hallmarks Responses to damage
Cellular senescence	
Stem cell exhaustion	
Altered intercellular communication	Integrative hallmarks Culprits of the phenotype

**Figure 1:** Primary hallmark will cause damage to the cellular role; Antagonistic hallmark will respond to the damage and lastly the integrative hallmark will be the causes of the phenotype, leading to clinical effects of ageing. It is important to know many of these hallmarks can be of other connected disease processes.

There are factors that have been connected to changes in ageing, as the body gets exposed to radiation, nutrition and social isolation called as impairment-inducing system of ageing. This system can be influenced by instability of genes caused by mutagens (chemicals and UV exposure). Hence, as the cell gets older, it is prone to stored genetic instability. This will lead to an exposure to formation of cancer and other disorders related to ageing. Both telomere dysfunction and gene instability play an important role in ageing at a cellular level. Studies have also shown how telomere shortening have been in connection with many ageing diseases (coronary heart disease, osteoarthritis, and atrial fibrillation). The gene expression can be changed without affecting the DNA sequence also known as epigenetic alterations. The epigenetic system involves DNA methylation (predictor of human age in GWAS), remodeling of the chromatin (includes chemical reactions) and non-coding RNA (play a role in regulation and silencing of the gene). Lastly, cellular mechanisms that are involved in enabling the homeostasis of the whole complement of proteins is called loss of proteostasis. Its main function is to avoid misfolding and accumulation of damaged polypeptides that may endanger the cell. The misfolding of the cell and accumulation will cause the age-related disease in the body such as Parkinson's, cataracts, and Alzheimer's. As we age, the cells lose its efficacy to maintain homeostasis.

There are opposed responses to molecular and cellular damage in the form of using nutrients and fluids for growth and maintenance of the body functions. Mitochondrial disruption can lead to undesirable effects in humans as they are exposed to reactive oxygen species. Senescent cells are a process which undergoes

phenotypic modifications when the cell stops dividing. Recently, these cells have shown a connection with ageing and age-related diseases.

The last hallmark leads to phenotypical changes of aging. Clinical signs have shown stem cell exhaustion and modified intercellular communication in rate-limiting steps. They show a change of communication in cell and organ systems. Stem cells also have a vital role in ageing and have many features in ageing. Changes have also taken place at intercellular level leading to neurohormonal changes.

Understanding ageing can help to give rise to new techniques of managing surgical disease in the elders. It is important to know how ageing affects organ systems and leads to dysfunction of the human body. Biomarkers can help to predict a person's life expectancy and to determine the biological age.

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