Ageing or senescence

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Ageing can be defined as a Process occurring in all members of a population after maturity, it involves progressive decline in vital capacities of the organism, terminating in death. ageing processes progressive and not reversible under physiological conditions. These are widespread, both in non-living and living systems, which have attained certain levels of complexity and Organisation. in living system ageing occurs at all levels- from macromolecules to the intact animal.

Ageing at cellular level

Ageing follows cessation of growth. At the cellular level ageing can be studied on the basis of three processes:

1. possible decline in the final efficiency of non-dividing highly specialised cells, such as neurons and muscle cells.
2. Progressive stiffening with age of the structural proteins- such as collagens.
3. Limitation imposed on cell division as revealed by the studies on fibroblast producing collagen and fibrin.

Mechanism of cell ageing

The probability that animals age because some of the more important cell populations lose their proliferative capacity is very unlikely. It could be suggested that normal cells have a finite capacity for replication and this finite limit is rarely reached in *vitro* but is, of course, demonstrable *in vitro*. The functional losses that occur in cells prior to their loss of division capacity produce age changes in animals much before their normal cells have reached the limit of their ability to divide.

In1963, L. Orgel put forward a hypothesis suggesting that cellular ageing results from impaired specificity of the translation step in protein synthesis. This hypothesis exerted an impact on experimental gerontology and it is gratifying to know that Orgel's predictions have been confirmed to a great extent.

Theories of ageing

Many modern theories concerned with biochemical mechanism of cellular ageing Centre upon the possible role of the various types of nucleic acid

and complex nucleoprotein structure in this ageing process. If after a certain time a cell loses its ability to maintain homeostasis, it is logical to assume that this is due to the failure of nucleic acid functions. Some of the possibilities of this failure are being described here.

Quantitative changes in nucleic acid

Loss of DNA or RNA per cell or organ could explain declining functional efficiency with increasing age, although it has never been possible to demonstrate loss of DNA per cell. Cellular RNA content is highly dependent on the prevailing functional state as well as on various physiological factors and circadian rhythm, and therefore is not a suitable parameter of the ageing process. loss of DNA per organ is far too small to explain declining functional organic efficiency. Histological findings point to progressive loss with age of certain irreparable types of cells.

Changes in Information content

One of the best-known theories of ageing is the maturation theory which explains the ageing process to progressive accumulation of gene mutation in somatic cells. detection of somatic mutation is not an easy proposition the theory had to be abandoned. Mutation is to bring about the synthesis of faulty messenger RNA and in turn faulty proteins which are unable to fulfil their biological function or can do so only Imperfectly. More recent theories of ageing try to attempt to explain the occurrence of faulty protein as a consequence not of gene mutation but of impaired specificity of the enzymes involved in the translation mechanism as suggested by Orgel.

In the complex mechanism of protein synthesis there are many ways in which information content can be changed, and the method of Investigation that has been employed in recent years are equally numerous. This makes it more difficult to compare the results of many workers.

However, the following mechanism may be considered:

1. It is assumed that in ageing cell replacement of defective molecules of metabolic DNA gradually becomes impossible and the defective molecules therefore accumulate. When enough faulty DNA molecules are present in the cell. functional impairments follow.
2. According to another suggestion, ageing is attributed to the loss of non- repetitive information. The highly repetitive DNA sequence in the genomes of the eukaryotes would not only represent a certain evolutionary reserve and a way of reinforcing the functional expression of the genetic information but would also be a form of

protection for this information against random molecular deterioration which accumulated during ontogeny.

3.It has been reported that the number of methyl groups in DNA decreases with age, thus information content of DNA is modified affecting protein synthesis.

Changes in protection regulatory mechanism

According to Orgel's hypothesis the ability of cells to produce functional proteins depends not only on the correct specification of the primary structure of the polypeptide chain in the genes, but also on the competence of the total protein synthesizing mechanism.

Free radical Theory of ageing

The ageing process may be divided into two categories of cumulative degradative changes:

1. more widespread damage produced by a variety of means such as autoimmune reactions ionizing radiations and smog that are subject to inhibition to a greater or lesser degree.
2. Alterations in the so-called biological clocks- changes that can be altered a little, determining the potential maximum life span of an individual.

   D. Haman postulated that the ageing process is probably due to cumulative degradative changes brought about by free radical reactions, ubiquitous in living systems.

   Thus, decreasing the level of deleterious free radical reaction in an organism main result in a decreased rate of biological degradation.

   Free radical reactions give rise to a variety of products. Reactions are almost invariably reversible. In the biological system molecular oxygen is always present, which participate in free radical reactions involving oxidation of organic compounds.

   The rate of free radical reactions involving molecular oxygen is enhanced by catalyst such as copper, iron, manganese, and inhibited by antioxidants such as vitamin E,2- mercaptoethylamine and butylated hydroxytoluene, which are capable of removing intermediate free radicals.

   The compounds are expected to minimise deleterious effect of free radicals' stanzas vary in their tendency to be involved in free radical reactions with molecular reactions the polyunsaturated fatty acid

react more rapidly than the saturated ones and tyrosine is more reactive than phenylalanine.

If free radical Reaction contribute to the degradation of biological system then it is possible that dietary considerations might influence the lifespan studies have shown that consumption of unsaturated fat cells decrease the average lifespan in mice Vitamin E is a natural lipid antioxidant and it has a modest beneficial effect on the life span as it decreases the rate of free radical formation.

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