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Ageing - a biological phenomenon.

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Abstract : Ageing is slow, progressive, symmetrical & biochemical changes that take place at cellular, tissue & organ level. Such alterations decrease the organizing ability to face environmental stress, resulting into an increase in the eventuality of "death". Ageing is due to the malfunctioning of body systems. Most organisms start to age sometimes just after they acquire the ability to reproduce. Signs of ageing are visible in almost all body systems from circulatory to urinogenital system. Changes also include physiological or functional & cellular changes. Many diseases are age associated like osteoporosis, sleeping disorders Parkinson's disease etc.

Kind of diet & its quantity also substantially affect the rate of ageing. Several theories have been put forth to explain the cytological basis of ageing process which have been discussed in this paper.

Key words: Ageing, Intelligence, free radicals, Masking, Diseases, life style, Antecedents, biological phenomena.

INTRODUCTION

Outlines
1. Signs of ageing
2. Theories of ageing
3. Age associated diseases
4. Ageing An overview
5. Masking ageing
6. Diet & ageing (4th part)

Given a chance, all of us would like to stay forever young. However, ageing is an inevitable truth, a unique feature of life cycle of all multi cellular organisms. Ageing decreases an organisms ability to face environmental stress & increases the likelihood of dying. Ageing & diseases sometimes co-exist.

SIGNS OF AGEING

(A) Functional changes (Physiological)

i) Pumping capacity of heart decreases

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ii) Oxygen carrying capacity of blood decreases.
iii) Blood supply to the brain & kidney is reduced.
iv) Production of new erythrocytes from the bone marrow declines.
v) Number of kidney tubules and taste bud reduce.
vi) Blood volume decreases.
vii) Vital capacity & pulmonary ventilation decline.

B. Cellular changes –

i) Chromosomal aberration occurs.
ii) Rate of protein synthesis decreases, quantity of defective protein increases.
iii) Condensation of chromatin takes place due to loss of water from cytoplasm *(Nuclear Pyknosis)*
iv) Larger accumulation of pigment like lipofuschin (formed by degradation of fat) in the cell.
v) Changes in the cellular enzymes.
vi) Cell volume decreases.
vii) Semi permeability of the membrane decreases due to accumulation of calcium in peripheral part of the cytoplasm.

viii) The activities of the endocrine organs like thyroids, gonads, adrenals (Cortex and medulla) decreases.

ix) *Collagen*, an extra cellular protein constituting about 30% of total protein of body, plays an important part in ageing. In young age, it is filterable, permeable & easily soluble. With the increasing age, it becomes rigid & insoluble & get deposited in the skin & bones. This deposition in extra cellular region may hinder the flow of nutrients & waste products to & from the cell.

x) Vitamin C (Ascorbic acid) is required for collagen synthesis. That is the reason, why its requirement increases in old age. Since it is involved in metabolic processes for the maintenance of bones, teeth & cartilage. Gradual decrease in solubility of collagen causes starvation of skin cells & also decreases their metabolic activity. This change causes wrinkling of skin in old age.

THEORIES OF AGEING

1. Rate of living theory - In 1928 Pearl, an American biologist said that the duration of life varies inversely with the rate of energy expenditure.
2. Somatic mutation theory-Hungarian physicist Leo szilard in 1956 stated that genetic mutations of DNA accumulate with time, ultimately resulting in "miscopying" & functional failure.
3. Immunity theory- Australian Immunologist Burnet said that due to decreased activity of the immunological system of an organism, ageing occurs.
4. Cross Linkage Theory John Bjarksten (1968) said that an alteration occurred in structural proteins causing them to develop inter & intra – modular cross links with other proteins. Such progressive cross linking in the body was responsible for the charges that occurred during ageing.
5. Neuro – endocnine theory – Vladimin Dilman of Peter Institute of endocrinology at St. Petersburg, Russia in 1983 proposed that ageing is due to loss of receptor sensitivity to feedback inhibition with time, resulting in to a progressive shifting of homeostasis & alterations of hormone levels & their effects with time.
6. Genetic theory- Genes regulate ageing. Studies on some nematodes & fruit fly (*Drosophila melancgaster*) have shown that genes influence ageing. Cells are genetically programmed to reproduce a limited no. of times before they ultimately die (*Apoptosis*). Hence it can be said that ageing is programmed cell death.
7. Free radical theory of ageing (FRTA) One of most accepted theories, proposed by D. Harman of Nebraska Medical centre, postulates that ageing results from an accumulation of changes caused by reactions in the body initiated by highly reactive molecules known as "Free radicals". Such changes are also believed to be cause of disease development.
8. Mitochondrial theory of ageing Harman (1972) (further refined & developed by Jaine Miquel in 1980), said that all cell types were equally subject to mitochondrial DNA mitochondrial at damage with ageing, Miquel further correlated MTA with FRTA & said that it is primarily "fixed post mitotic" (FPM Cells) cells, that MTA applies, MTA is said to be the specific development of FRTA.

FPM cells are those that no longer divide after early childhood & are therefore, irreplaceable (primarily brain, heart & skeletal muscle cells).

Free radical is a molecule having an unpaired electron or free electron that is capable of independent existence. All free radicals are extremely reactive & short lived.

Free radicals try to get an electron & in the process of acquiring the electron attach themselves to another molecule, thereby modifying it biochemically. Free radicals are capable of damaging any bimolecule including proteins, sugars, fatty acids & nuclear acids.

(**French paradox** People consuming moderate levels of wine, specially red wine have lower incidences of heart diseases. Red wine contains high level of polyphenoles with antioxidative properties inacting Free radicals. Polyphenols are formed in plants only, abundant in grapes & rich in tea, apples, onions & peanuts).

Free radicals are produced endogenously as well as exogenously by Air pollution including Industrial waste & cigarette smoke & radiation along with trace metals like lead, Mercury etc. are exogenous, sources of free radicals. FRs are mainly produced by four mechanisms :-

i) During production of ATP, when O_2 is changed to H_2O_2 in mitochondria.

ii) Phagocytic activities of WBC.

iii) In the perioxisomes during metabolism of fatty acids (peroxisomer).

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iv) During defence against toxic chemicals.

Age associated diseases –

i) Alzheimer's disease -loss of memory causing disturbance in balanced behavior.
ii) Dementia mental deterioration.
iii) Osteoporosis, Osteo-arthritis thickening of collagen fibers reduce elasticity of bone making them more brittle.
iv) Sleeping disorders.
v) *Atherosclerosis, Arteriosclerosis* Hardening of arteries causing High BP (Hypertension), Haemorrhage, thrombosis, strokes etc.
vi) Parkinson's disease A Progressive nervous disease associated with destruction of brain cells.
vii) Benign Prostatic *Hyperplasia* Enlargement of prostate gland leading to compression of urerhra and obstruction of flow of urine.

Diet & ageing - Kind of diet & its amount affects the rate of ageing. Diet having less fat contents & more of natural antioxidants protect from ageing & related diseases & sufferings.

Eating less leads to healthy life span, as it decreases metabolic rate in the body slowing down the mitochondrial action resulting in a decline in ROS production. It also increases the antioxide concentration in the body, protecting

Free radicals & ageing –

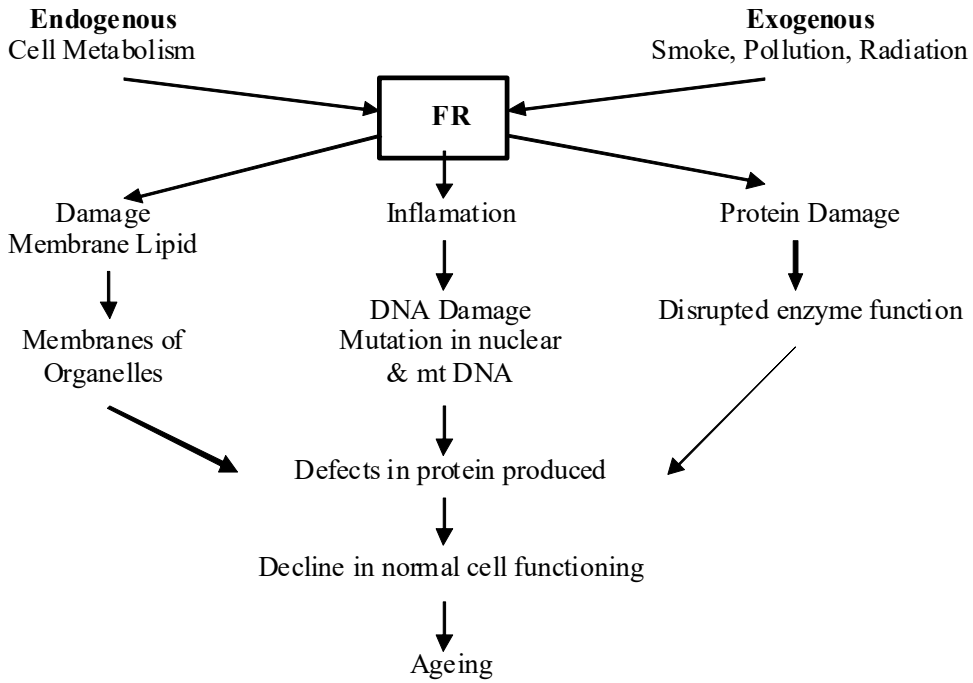

Fig. Free radicals & Ageing

transplantat therapy.

Apoptosis

Apoptosis (vide Genetic theory) has been defined by Richter (1998) as an evolutionary conserved form of physiologic cell death important for tissue development & homeostasis. It is a highly regulated and controlled process that avoids inflammation and damage to the surrounding tissues (Steller, 1995)

Apoptosis was discoverd by Vogt (1842)but actual word Apoptosis was coined by Kerr *et al*. 1972. It is an important process seen during embryonic development, metamorphosis, renewal of tissues & harmone induced atrophy Kerr, *et al*, (2000) and maintains balance between division & death called homeostasis, which is necessary for defence and aging (senescence).

The axact functioning of Apoptosis is still a mystry yet it is marked by nuclear condensation, cell shrinkage bleb formation and absence of inflammatory responses of the affected cells. In all probability oxidative stress Ca^{2+}, proteases, nucleases and mitochondria are participants of Apoptosis where as NO (nitric oxide independent of its redox state), NO^-, NO^+ and NO (nitric monoxide residues) nitrosonium ion and nitroxyl anions are mediators of Apoptosis Sahay *et al* (2007)[3] (A review on Apoptosis by Sahay *et al* 2007 may be consulted).

Sahay *et al* (2007)[3] opines that Apoptosis is controlled by specific genes (enzymes get activated to induce Apoptosis, it has been studied in *C.elegans* where CED3 & CED4 are expressed in dying cells and where Ca^{2+} dependents endonuclease is activated to fragment DNA into 180 bp pieces. Neverthless certain mechanisms protect neighbouring cells as well. In this protectin mechanism CED-9 (in *C.elegans)* & Bcl_2 (in mammals) are involved.

The authors further say that 4 components are required to cause cell death. (i) CED3/ caspases (ii) CED-9 /Bcl-2 (iii) CED-4 & (iv) mitochandria. CED-4 is controlled by Bcl-2 and induce apoptosis after caspase activation. over expression of CED-4 in mamalian cells may result into death of the cell but this gets blocked by over expression of Bcl-x (a homologue of CED-9) or by caspase inhibitors (vide page 30 of the said article.)

REFERENCE

1. **Science Reporter Book by Dr. S.T.** Rizvi University of Allahabad.
2. **Rashmi Jha,** University of Allahabad
3. **Sahay, Umapati, Deepa Prasad and R.P.Singh,** 2007. Apoptosis a review. Anusandhan. **IX(14)**: 1-29.

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