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REVIEW

Aging and Geriatric Dentistry

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ABSTRACT

Five theories shed lights on the potential mechanisms of aging: somatic mutations, telomere loss, mitochondrial defects, and accumulation of altered proteins inside proteasomes. The existence of a program of aging is not yet identified, but overlaps with a program for risks of death. On the other hand, organisms are programmed for survival, which ultimately fails. This failure results in aging, notabily, focusing on alterations of specific genes. Irregular examinations, dysfunctions, insufficient use of fluoride, and removable partial dentures, are favoring the formation of caries and periodontal pathologies. Oral lesions are due to local trauma, related gingival recession, and formation of pockets. They are associated to insufficient removal of food/plaque. Epithelial thinning, and reduction of extracellular matrix components, lead to plications and foldings of the mucosal surface, and subsequently to bacterial colonization. Geriatric dentistry (or gerodontology) is an increasing field of dentistry, mostly associated with the growing percentage of patients over 80+ years.

1. What is Aging: a Summary of Aging Theories

Aging is characterized by deteriorations, increasing risks of death. Geriatric dentistry includes the diagnosis, prevention of caries and periodontal diseases and treatment, oral mucosal pathologies, alveolar bone resorption, development of malignant tumors, and salivary dysfunctions.

Theories aiming to clarify the aging phenomenon include :

(1) the Somatic Mutation Theory, establishing a relationship between longevity and DNA repair.

(2) The Telomere Loss Theory, the telomers becoming shorter after each cell division.

(3) The Mitochondrial Theory implicating accumulation of mitochondrial DNA.

(4) Altered Proteins Theory and waste accumulation, focusing on proteasomes.

(5) Network Theories of aging : The multiplicity and complexity of aging mechanisms is now recognized [1].

The conclusions on the existence of a program of aging are far from being elucidated, facing the increased risks of death. Inversely, it is clear that organisms are programmed for survival. However, this program ultimately fails, and this disfunction clarify the mechanisms of aging [1]. Genes involved in autophagy have provided some insights into senescence and death [3].

With aging, a progressive decline is marked in multiple cells and tissues [4]. After a limited number of cell

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divisions cultured human cells stop dividing according to replicative senescence. The loss of telomere repeats contributes to human aging. Mutations of telomeres lead to progressive failures. Telomere length is acting as a trait that is sufficient to mediate the degenerative defects of aging [2].

Deleterious free radical are also implicated are major contributor to the aging process [5]. Search for a single gene has been replaced by the view that aging is a complex, multifactorial process [6]. Evolutionary, molecular, cellular, and systemic theories have been postulated.

1.1 The Programmed Theories

There are five characteristics of aging in mammal tissues [7]: (1) Increased mortality with age. (2) Changes in biochemical composition in tissues. Increased lipofuscin and cross-linking in ECM. (3) Progressive decrease in physiological capacity. (4) Reduced ability to respond to environmental stimuli, and (5) Increased susceptibility and vulnerability to disease.

Alltogether they implies three sub-categories [8].

(1) Programmed Longevity. Aging result of a sequential switching of genes,

(2) Endocrine Theory. Biological clocks act through hormones and control aging.

(3) Immunological Theory. The immune system is declining. This leads to an increased vulnerability.

Two biomarkers of aging accumulate : lipofuscin and an increased cross-linking in extra-cellular matrix molecules. Pigment is formed by oxidative polymerization of mitochondrial lipids. Large deposits of melanin produced by free radical reactions are associated with detrimental changes. The free radical theory of aging (FRTA), and simultaneously discovery of the involvement of free radicals are producing changes together with a decrease in biological capacities, The susceptibility and vulnerability to diseases is increased. The ALE (average life expectancy) is reaching plateau values, less than the maximum of elderly above 85 years.

1.2 Causes and Theories of Aging

The concepts behind the stochastic theorie are the build-up of ‘‘damages’’, whereas the genetic theories consider aging as part of the lifespan. The genetic and stochastic theories are not mutually exclusive. Damaged proteins are broken down by proteases, however, when proteases are inhibited, damaged proteins accumulate.

Several investigators have suggested the essentiality of various membranes in maintaining intracellular homeostasis and deterioration of membrane integrity. This is the underlying cause of the aging process: (1) vesicle-dependent lysosomal pathway and (2) the ubiquitin/proteasome pathway. The proteasome is a non-lysosomal threonine type protease. Ubiquitin is crucial for the degradation of many cytosolic, nuclear and endoplasmic reticulum proteins.

Aged cells stored damaged or altered proteins when compared to young cells. One of the hypothesis is the decrease with age of proteasome activity. Accumulation of altered proteins explain the increased protein alterations, and the decreased protein degradation or the combination of both.. Age-related alterations in lysosomes regulate the accumulation of lipofuscin.

1.3 Autophagy

Autophagosomes characterize autophagy. Subsequently merging with lysosomes they form an autophagolysosome where degradation occurs and used for the renewal of synthesis of molecules. The vesicles fuse with secondary lysosomes, and are enriched by proteases Two enzymes modulate the formation of the autophagic compartments.

1.4 Causes of Cellular Senescence

Normal human cells did not proliferate indefinitely in culture. These cells have a finite replicative life span, and undergo replicative senescence. The number of divisions that cells complete upon reaching the end of their replicative life span has been termed the Hayflick limit.

Many senescent cells display a senescence-associated secretory phenotype (SASP), which may explain the role of cellular senescence in the aging process.. These factors include IL-6 and IL-8, a variety of MCPs (monocyte chemoattractant proteins), MIPs (macrophage inflammatory proteins), and proteins regulating granulocyte/macrophage colony–stimulating factor.

Macromolecules such as nucleic acids, lipids, sugars, and proteins are susceptible to free radical attack. The body does possess some natural antioxidants in the form of enzymes, which help to curb the build-up of these free radicals. Cellular death rates would be greatly increased without these enzymes. Subsequently if this was the case, life expectancies would decrease.

2. Aging and Oral Health

In 1900, 3.1 million people, or 4 percent of the population, were 65 years or older. By 2005, the number had increased to 34.3 million people, or 12.4 percent of the population. For older than 65 years, the percentage is going from 16% in 2000, to 25% in 2030 and 29% in 2050. Effects of aging on oral hard and soft tissues are summa-

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rized in this second part [9].

The elderly are at risk of chronic diseases, including infections, benign mucosal lesions and oral cancer. Frequent conditions are xerostomia (sensation of dry mouth) and oral candidiasis, appearing as acute pseudo-membranous,, and/or angular cheilitis.. Many systemic conditions, are typical in the elderly. Socio-economic factors (traveling costs, educational background) interfere with the maintenance of a functioning dentition and a healthy oral cavity. The aging population is growing and older adults have more teeth and oral problems than did previous cohorts.

2.1 Diseases of Oral Tissues

Dental tissues : severe enamel wear exposes dentin. Cementum gradually thickens. Pulpal calcification and external root resorptions contribute to a decreased pulp volume.

Elderly patients are more susceptible to root caries due to inadequate oral hygiene, salivary gland dysfunction, insufficient use of fluoride and removable partial dentures, traping plaque around the teeth, local trauma, exposure of cementum/dentin at the cervical junction, periodontal pockets, and insufficient removal of food/plaque between the teeth contributes to the onset of oral pathologies. Endodontic considerations in the elderly reduce the potential infection, namely in narrow root canals, that are difficult to find, enlarge and fill.

Periodontal tissues diseases are initiated by gingivitis, becoming periodontitis. Atrophic Bone Loss reduces mostly alveolar bone, basal bone being unaffected.

2.2 Oral and Pharyngeal Mucosa

Ageing affects oral tissues and pathologies that are integral components of general health. Oral diseases cause difficulties in speaking, mastication, swallowing, in addition to aesthetical considerations and facial alterations. Strategies should be adopted including the management of oral conditions, which are necessary for re-establishing effective masticatory function. Oral health is affecting the quality of life.

With elders, tongue dorsum shows reddening, and atrophy of the papillae. Tongue may be completely smooth or lobulated. These changes bring about an altered taste and decreased appetite. Increased varicosities at the ventral surface are common.

A lifelong history of oral mucosal trauma, can modify the clinical aspect. Covered by keratinocytes, other types of cells are also found, including Langerhans cells (LC), lymphocytes and Merkel cells. Alterations in the distribution of macrophages in gene trascription pattern was significantly increased. Histologically, epithelial thinning, increased fibrotic connective tissues, reduction of LC density have been reported. They are displaying long and branched cytoplasmic processes. In older patients, the LC network is deteriorated and the reduction of this network is age-related.

Leukoplakia is associated with an increased risk of cancer. Most of the oral carcinomas are squamous carcinomas in the lower lip, tongue, gingivae and floor of the mouth. Potential malignant lesions: Tobacco and alcohol use are responsible for up to 75% of oral cancers. The human papillomavirus, influence the immunosuppression, and is implicated in this types of cancer. With aging, epithelium becomes thinner, with a reduced elasticity. Gingival recession occurs, with a parallel decrease of cells producing collagen fibers, vascularization and simultaneously a decrease in alveolar bone density.

2.3 Aging Salivary Glands (xerostomia)

This involves degenerative alterations, sialadenitis, sialolithiasis, and hypofunction. Xerostomia is associated with complaints of dry mouth and hyposialia. The prevalence of hyposalivation in older people may be explained by the increased incidence of medication, such as diuretics or daily aspirin.

The volume of the connective tissue and intralobular ducts is increased, whereas acinar cells are decreasing. Saliva becomes thicker. Medications like anti-hypertensives, anti-psychotics, and anxiolytics lead to xerostomia,. The absence of protective influence of saliva in the oral cavity intensify the predisposition to oral disease. Financial constraints and lack of family transportation making easier the access to dental services. The burning mouth syndrome (BMS): Several conditions lead to ‘secondary’ BMS: allergic reactions, galvanism, parafunctional habits, and salivary gland dysfunctions. Moreover, hormonal disorders correlate to menopause, diabetes and nutritional deficiencies. Systemic conditions influence the prevalence, onset, and severity of BMS..

3. Conclusions

Elderly people lose manual dexterity, and they are likely to have poor oral hygiene. Elderly people receives medical treatments that result in the consumption of approximately 25% of the national total of the drugs prescripted. Medications cause a reduction in salivary flow as a side effect. Elderly people are deficient in salivary flow, combined with diminished oral hygiene practices. This lead to plaque accumulation on the tooth and denture surfaces, therefore to caries, periodontal diseases and prosthetic

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problems [10].

The phenomenon of aging is not yet elucidated, but the consequences of aging are better identified, leading to adapted gerodontological therapies. Caries and periodontal diseases, infection due to bacterial invasion, early stages of cancer and related tumors, radiotherapy inducing xerostomiae, burning mouth syndrome, are the fundamental targets of gerodontology. This is an increasing field of dental practice, mostly associated with the growing group of patients over 80+ years.

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