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Effects of Human Ageing on Periodontal Tissues

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1. Introduction

Aging and death are two natural consequences to which the human individuals are subject after their birth. Improvement in both social living conditions and health care has led to a greater life span in the world [1], resulting in an increase in periodontal disease expectancy among the dentate elderly [2]. Although moderate loss of both alveolar bone and periodontal attachment is common in the elderly, severe periodontitis, defined as periodontal attachment loss of 6 mm or more and radiographic bone loss of 50% or more involving at least one tooth, is not a natural consequence of ageing. Some loss of periodontal attachment and alveolar bone may be expected in older persons, but age alone in healthy adults does not lead to a critical loss of periodontal support [3].

Human ageing induces histophysiological and clinical alterations in oral tissues [4]. These alterations must be understood to differentiate pathological conditions from the altered physiology of oral tissues resulting from ageing [5]. Some studies in humans and animals have demonstrated that alterations in periodontium dynamics occur with age [6,7].

In spite of the fact that the periodontal disease severity is known to be associated with age, functional changes in periodontal tissue cells during the ageing process have not been well characterized [8]. It is important to define how cellular ageing affects the progression of periodontal diseases associated with ageing [6]. The understanding of the influence of human aging in the dynamic of the inflammatory process in patients with periodontal disease may help in the treatment planning of these patients.

2. Age-dependent changes of the periodontal tissues

The tissues that support the teeth are called the periodontium, which consists of gingiva, periodontal ligament, cementum, and alveolar bone. Anatomical and functional changes in periodontal tissues have been reported as being associated with the ageing process [9].

Gingiva, a tissue exposed to the oral cavity, is histologically composed by epithelium and connective tissues. Changes in the human oral epithelium caused by ageing are related to a thinning of the epithelium and diminished keratinization. Conflicting results have been reported regarding the shape of the retepegs. A flattening of retepegs and an increase in the height of the epithelial ridges associated with ageing were both demonstrated. In a morphological 3-dimensional study of the epithelium-connective tissue interface, connective

tissue ridges were observed to be more prevalent in young individuals whereas connective tissue papillae were predominant in old individuals. The change from ridges to papillae involves the formation of epithelial cross-ridges with advanced age [9].

Furthermore, it has been shown that the number of cellular elements decreases as age increases. The fibroblasts are the main cells in the synthesis of periodontal connective tissue. There are phenotypic subpopulations of fibroblasts with different functions in the synthesis and maintenance of extracellular matrix constituents [10]. *In vivo* and *in vitro* studies have shown functional and structural alterations in fibroblasts associated with ageing [6, 11-13].

Gingival fibroblasts (GF) may be constantly affected by oral bacteria and their products, such as the lipopolysaccharides (LPS), present in their cell walls. The LPS induces GF to release some inflammatory cytokines such as prostaglandin E_2 (PGE_2), interleukin (IL)-1 β, and plasminogen activator (PA) [6, 14]. The influence of these inflammatory mediators on both GF and periodontal ligament fibroblasts (PLF) might account for the severity of periodontal disease [6].

Quantitative differences in protein synthesis were found in young and old gingival fibroblasts *in vitro*. The collagen production decreased more than 5-fold as a function of increasing donor age [15]. Old fibroblasts also presented an increased rate of collagen intracellular phagocytosis, which can affect the balance between synthesis and degradation of collagen in the connective tissue [12]. The ageing process in GF causes an increase in DNA structure metilation of collagen alpha 1 gene, followed by a reduction in mRNA levels and collagen type I synthesis [13]. Alterations in the composition of extracellular matrix proteoglycans secreted by GF *in vitro* were also observed. The proteoglycans secreted by old fibroblasts might increase the rates of heparan sulfate and reduce chondroitin sulfate when compared to those secreted by young fibroblasts [16].

The periodontal ligament, which is a soft connective tissue, anchors the tooth into the alveolar bone and functions as a cushion between hard tissues to mitigate the occlusal force. It is basically constituted by fibroblasts, cementoblasts, osteoblasts, osteoclasts, Malassez epithelial rests and collagen matrix (Sharpey´s fibers). The periodontal ligament cells are involved in the repair of alveolar bone, cementum and periodontal ligament itself, being able to differentiate into osteblasts, cementoblasts and fibroblasts [17]. With age, the fiber and cellular contents decrease and the structure of the periodontal ligament becomes irregular. Periodontal ligament fibroblasts (PLF) are constantly subject to mechanical stress caused by occlusal forces. Cultured PLF were observed to produce a large amount of PGE_2, IL-1β, and PA in response to mechanical stress [6].

The ageing process might induce a significant reduction in chemotaxy, motility, and proliferation rate of periodontal ligament cells. The chemotaxy and differentiation of osteoclasts from the periodontal ligament induced by devitalized osseous matrix might be influenced by donor's age. A reduction in osteoblast chemotaxy and lower rate of osteoclast differentiation in the cells of elderly donors were observed [18].

The cells of the periodontal ligament from the elderly showed lower rates of chemotaxy and proliferation than those of the periodontal ligament from young patients [19]. The reduced ability of senescent cells to express the *c-fos* ligant might be associated with the low rates of chemotaxy and proliferation of these cells [20]. The expression of osteocalcin in fibroblasts from the periodontal ligament is either reduced or ceased in senescent fibroblasts. This reduction may be directly related to the cell's difficulty in progressing in the cellular cycle (G1-S) and accomplishing cell respiration [21].

Cementum is a calcified connective tissue covering the roots of teeth. Its formation is a continuous process which occurs throughout the life of humans and animals. With age, the cementum increases in width. It has been demonstrated that there is a tendency towards greater cemental apposition in the apical region of the teeth. Collagen fibers are embedded in the cementum during its formation [9]. Ageing and death of cells are common characteristics of the life cycle of the cementocytes. This might be due to a rapid reduction in the accessibility of nutritive substances and poor elimination of waste products of the cementocytes. In general, cementum is cellular-except at the root apices and in the furcation areas of multirooted teeth. With age, cementum becomes acellular. Although remodeling of cementum occurs infrequently, resorption at the cementum surface followed by cementum apposition is often observed and, with age, this might result in irregular cementum surfaces [9, 22].

Both the alveolar bone and the periodontal ligament serve as support to the teeth. It is well known that bone formation steadily declines with age, leading to a significant reduction in bone mass [23]. The alveolar bone has high plasticity and under physiological conditions it is preserved by the equilibrium between osteoblastic and osteoclastic activities. These cells are directly or indirectly influenced by the parathyroid hormone (PTH), vitamin D metabolites, calcitonin, estrogen, plasmatic concentration of calcium and phosphate, neurotransmitters, growth factors, and local cytokines [24].

The reduction in bone formation might be due to a decrease in osteoblast-proliferating precursors or to decreased synthesis and secretion of essential bone matrix proteins [6, 23]. The extracellular matrix surrounding osteoblasts has been shown to play an important role in bone metabolism. A possible dysfunction of this matrix might occur concomitantly with the ageing process [6].

Oxygen-free radicals have been reported to cause cellular damage and, consequently, contribute to the ageing process [25, 26]. In an *in vitro* study, oxygen radical-treated fibronectin (FN) was found to inhibit bone nodule formation by osteoblasts when compared to intact FN. This finding suggested that intact FN plays an important role in osteoblast activity and that FN damaged by oxygen radicals during the ageing process might be related to less bone formation [6].

3. Systemic ageing and periodontium

Some alterations in the organism endocrine profile influence the osseous metabolism with age. Vitamin D deficiency is a common phenomenon in persons living in elderly homes [27]. The low levels of calcium resulting from vitamin D deficiency associated with renal insufficiency might lead to secondary hyperparathyroidism [28]. The high levels of PTH resulting from secondary hyperparathyroidism act in the mobilization of osseous calcium, which might cause mineralization problems, bone fractures, and a reduction in osseous density [29]. With regard to the periodontium, osteopenia and osteoporosis are considered important risk factors for alveolar bone loss in the presence of periodontal diseases [30, 31].

Female patients with osteoporosis or osteopenia presented greater levels of alveolar bone loss, when compared to patients with normal mineral osseous density [32, 33]. Estrogen hormonal reposition therapy in patients with osteoporosis might reduce gingival inflammation and alveolar bone loss in relation to patients with untreated osteoporosis [34, 35]. In a randomized double-blind study, patients with osteoporosis treated with estrogen reposition showed an increase in alveolar bone levels, when compared to the placebo group [36].

The supplement ingestion of calcium (1000-1200 mg/day) and vitamin D (400-600 IU/day) in elderly patients with osteoporosis has been tested. After 5 years of treatment, the results showed a reduction in tooth loss in patients who ingested calcium and vitamin D, when compared to the placebo group [37]. Studies evaluating periodontal status and hormonal reposition therapies, indicated for prevention and treatment of osteoporosis, have reported the reduction in alveolar bone loss and tooth loss as secondary benefits. It is worth emphasizing that some risks in hormonal reposition therapy have been recently reported; for example, an increase in the incidence of breast cancer, thromboembolic diseases, and myocardial infarction has been found [38].

4. Immunosenescence and periodontal cells interaction

Immunosenescence refers to the gradual deterioration of the immune system caused by natural aging. It involves the host's capacity to respond to infections and the development of long-term immune memory. This is not a random deteriorative phenomenon; rather it appears to repeat inversely an evolutionary pattern. Most of the parameters affected by immunosenescence appear to be under genetic control. Immunosenescence can also be sometimes envisaged as the result of the continuous challenge of the unavoidable exposure of the organism to a variety of antigens such as viruses and bacteria [39].

Aging is a complex, continuous, and slow process that gradually involves most if not all organs of the organism, causing their abnormal functioning of in both qualitative and quantitative terms, as well as morphological or structural changes. So, senescence is not represented by a pre-established moment, but consists of a long-lasting preparation of the organism for a morpho-functional involution, which itself is a normal part of the biological cycle [40]. In this context, periodontal tissues also are included.

The immune system also undergoes age-related modifications leading to structural changes in the lymphoid organs, and functional impairment of some types of immunocompetent cells. The most evident changes in the immune system occur in the thymus, a specialized organ of the immune system. The thymus is a primary lymphoid organ, but also an endocrine gland, responsible for T-cell production and maturation [41].

The thymus is the largest and most active during the neonatal and pre-adolescent periods. By the early teens, the thymus begins to atrophy and thymic stroma is replaced by adipose tissue. Nevertheless, residual T lymphopoiesis continues throughout adult life. Thymus involution in humans is observed until the age of 70 years [42]. The consequences of thymic involution in the peripheral pool of T-cells are still a matter of controversy. Thus, whereas some authors report no significant decline in the total number of T-cells, but considerable shifts in the ratios of activated/memory T-cells [41], others claim that the age-related changes in the thymus and T-cells are quantitative, not qualitative [43]. In any case, it remains to be clarified whether changes in the immune system with age have something to do with survival [44], although the stage of being elderly is associated with an increase in infections, tumors, and other diseases related to a decline in the immune function [45].

T-lymphocytes are considered thymus-dependent cells of fundamental importance in the immune response. Reductions in peripheral blood T-lymphocytes, mitotic agents, anti-CD3, and monoclonal antibodies are the main alterations in the senescent phenotypes of T-lymphocytes. The proliferative phase alterations in T-lymphocytes may be induced by the reduced secretion of interleukin-2 (IL-2) and reduced expression of its high-affinity receptors. The reduced expression patterns of IL-2 and IL-2R in peripheral monocytes of

elderly patients have been reported as influencing the proliferative response of T-lymphocytes [46]. IL-2 is produced by helper T-cells and plays an important role in the proliferation and differentiation of virgin T-cells into effector T-cells (this term is used frequently for populations of T-cells with cytolytic activities and for T-helper cells, which secrete cytokines and activate directly other immune cells, to distinguish them from another class of T-cells known as regulatory T-cells) [47].

Ageing-related immunological alterations in the leukocyte subpopulations [48] and B-lymphocyte subpopulations [49] have been reported. The reduction in the peripheral blood population of B-lymphocytes is associated with the decrease in the production of high-specificity antibodies as well as in the avidity of antigen-antibody legation [50]. In establishment of long-term memory of immune response, the maturation of antigen-induced $CD4^+$ T-cells, which are postthymic human cell, results in the terminally differentiated $CD45RA^+$. An increase in $CD45RA^+$ memory cell circulation in relation to $CD45RO^+$ virgin cells disturbing the response to new antigens has been noted. Other changes in immune senescence include a decline in macrophage, neutrophil, and natural killer function with ageing [51].

A more rapid and severe development of gingivitis as well as changes in inflammatory response induced by gingivitis in elderly patients have been reported [52]. A greater presence of alpha 2-macroglobulin, IgG3, and B-lymphocytes in the crevicular fluid, and a reduction in polymorphonuclear leukocytes (PMN) have also been observed in the elderly [53].

Periodontal ligament cells from the elderly showed an increase in the production of plasminogen activator (PA) [54], prostaglandin E2 (PGE2), interleukin-1β (IL-1β) [6, 54] and interleukin-6 (IL-6) [55] when compared to younger cells.

Lipopolysaccharide (LPS), responsible for bacterial cytotoxicity, is a component of the gram negative bacterial cell wall. It induces the activation of transcription factors in lymphocytes, promoting regions of DNA which activate genes that contribute to the adaptive response and secretion of pro-inflammatory cytokines [56].

PA is a serine protease that acts in the activation of plasmatic plasminogen into plasmin and it is secreted by many cell types, including periodontal fibroblasts. The activity of PA and the expression of tissue plasminogen activator (tPA) mRNA in fibroblasts from the periodontal ligament *in vitro* submitted to mechanical tension were evaluated. The cells from elderly individuals presented greater activity of PA and greater expression of tPA mRNA when compared to those from young individuals [7]. The action of PA is involved in physiological and pathological mechanisms of periodontium, including host-microbiota interaction, PMN migration, and proliferation and migration of both epithelial cells and fibroblasts [57]. Analysis of PA distribution in the periodontium showed that, in healthy periodontium, PA is expressed in the superficial cells of the junctional epithelium. However, in patients with periodontitis, PA is expressed in all the epithelium lining of the periodontal pocket. The alteration in the pattern of PA distribution, according to periodontal status, suggests that PA is involved in the periodontal homeostasis [58].

The levels of tPA and the plasminogen activator inhibitor-2 (PAI-2) are greater in gingival crevicular fluid of patients with periodontitis than in periodontally healthy patients [59]. Patients with high levels of alveolar bone loss presented higher levels of tPA and PAI-2 than those with low levels of alveolar bone loss. A greater release of PA induced by ageing might affect the gingival fibroblasts and the periodontal ligament, and aggravate the inflammation process and the degradation of the extracellular matrix from periodontal tissues in the elderly [7, 53].

Fibroblasts from old mice (20 months) demonstrated a significant increase in the synthesis of PGE2 and IL-1β when compared to the fibroblasts from young mice (6 weeks) [54, 60]. An increase in the production of PGE2 and Cox 2 mRNA stimulated by LPS and mechanical stress, respectively, was also observed in periodontal ligament cells from elderly donors [61, 62].

PGE2 is produced by the metabolism of arachidonic acid through the cyclooxygenase (COX) pathway and has a recognized role in the inflammatory process, through vascular dilatation, increased in vascular permeability, and sensibilization of nociceptors to the stimulus of histamine and bradykinin. PGE2 may have an indirect effect on alveolar bone resorption by the sensibility of osteoclasts to the action of other cytokines involved in this process [63]. In patients with periodontitis, high levels of PGE2 were observed to be related to the severity of periodontal disease and the increase in alveolar bone loss [64, 65]. The greater production of PGE2 by old periodontal ligament cells might account for the greater rate of alveolar bone resorption in elderly patients [54, 66].

In vitro studies have demonstrated an increase in the synthesis of IL-1β and in the expression of IL-1β mRNA in fibroblasts from human elderly donors and in fibroblasts of old mice, stimulated by mechanical stress [60, 66]. Human gingival fibroblasts from elderly donors presented greater production of IL-6 by the stimulus of LPS from Campylobacter rectus when compared to young donors [55]. IL-1β is the most active cytokine in the process of bone resorption, being 15 times more potent than IL-1α and 1000 times more potent than TNF-β [67]. Patients with severe periodontal disease (periodontal pocket >6mm) presented with a rate of IL-1β twice times higher than that observed for patients with moderate (<4mm) and intermediate (4-6 mm) periodontal disease [67]. In the same way, another study demonstrated that patients with periodontal bone loss had more IL-1β in the gingival fluid when compared to periodontal patients without bone loss ($P<0.0001$) [68]. The levels of IL-1α, IL-1β, plaque accumulation, gingival fluid, and gingival inflammation in young adults (20-22 years) and elderly (61-65 years) were compared with experimental periodontal disease. Levels of IL-1β in the gingival fluid were significantly higher in elderly, with a progressive increase until the 21st day of oral hygiene suspension, while levels of IL-1α were similar for both groups. In the elderly there was also an increase in plaque accumulation, gingival fluid, and clinical signs of inflammation [69].

The pattern of IL-1β and IL-6 secretion in periodontal disease in menopausal patients was analyzed [34]. The results demonstrated that menopausal patients that were not in hormone reposition therapy presented higher rates of IL-1β ($p < 0.0004$) and IL-6 ($p>0.05$) than patients under hormonal treatment. IL-6 has an important role in the osseous lyses in periodontitis. It acts stimulating the growth and proliferation of osteoclast precursors and there is evidence that it is an extracellular messenger signaling osteoblast resorption for the osteoclast [55]**.** A greater concentration of IL-1β e IL-8 in patients with estrogen deficiency compared to untreated patients without estrogen deficiency was also observed [70]. The hypothesis that the greater liberation of IL-1β by older periodontal ligament cells may represent an important factor for the greater rate of alveolar bone resorption in elderly patients has been proposed [54, 60, 66].

Periodontal ligament fibroblasts also express osteoblastic phenotypes, such as the production of bone-like matrix proteins, high alkaline phosphatase activity, and the formation of calcified nodules [71]. In addition, human periodontal ligament cells express and secrete osteoprotegerin (OPG) [72] and receptor activator of NF-kappa B ligand (RANKL) such as osteoblasts, suggesting that the periodontal ligament plays a role in alveolar bone metabolism [73].

RANKL, a member of tumor necrosis factor (TNF) ligand family, is expressed on osteoblast/stromal cell membranes. This ligand binds to the receptor activator of NF-kappa B (RANK), which is a receptor on the membrane of osteoclasts and mononuclear pre-osteoclasts, and induces osteoclast differentiation and activity [74, 75]. In contrast, OPG is known to inhibit osteoclast differentiation and activity by interrupting the interaction between RANKL and its receptor, RANK, by binding to RANKL as a decoy receptor with higher affinity than RANK [76].

RANKL expressed on gingival or synovial fibroblasts may direct macrophages present in connective tissue and monocytes recruited from blood to differentiate into osteoclasts, which leads to bone destruction in periodontitis or arthritis. RANKL is expressed also on activated T-cells [73, 75]. RANKL promoted the survival of mature dendritic cells and enhances the ability of these cells to stimulate T-cell proliferation in a mixed leukocyte reaction. In addition, RANKL induced the production of proinflammatory cytokines (IL-1 and IL-6) and cytokines that direct differentiation of T- cells, such as IL-12 and IL-15 from dendritic cells [77].

In periodontitis, LPS, which is a combination of lipid and polysaccharide, is a component of the outer membrane of gram-negative bacteria [56]. It has been reported that toll-like-receptor-4 (TLR4), a protein that in humans is encoded by the TLR4 gene and it detect lipopolysaccharide from Gram-negative bacteria, is essential for the response to LPS [78]. Human periodontal ligament fibroblasts express TLR4, suggesting that LPS directly acts on these cells. LPS interacts with endotoxin and CD14 (endotoxin receptor) to present LPS to TLR-4, which activates inflammatory gene expression through NF-kappa-B. CD14 is a critical receptor for LPS because monoclonal antibodies directed against CD14 can inhibit the biological effects induced both *in vitro* and *in vivo* by LPS [79].

LPS induces inflammatory cytokines, such as interleukin-1 beta (IL-1β) and tumor necrosis factor-alpha (TNF-α), in macrophages and neutrophilic leukocytes that infiltrate areas infected with bacteria, and in periodontal ligament fibroblasts [80]. These cytokines are thought to play an important role in the pathogenesis of periodontitis because they cause inflammation and destruction of periodontal tissue and resorption of alveolar bone by various biological mechanisms. IL-1 and TNF-a induce bone resorption by acting both directly and indirectly on osteoclasts [81].

The effects of LPS on OPG and RANKL expression in human periodontal ligament fibroblasts (HPLF) were reported. These suggest that LPS stimulates both OPG and RANKL expression in HPLF by up-regulating IL-1β and TNF-α [82].

All of these studies related to the dynamics of the inflammatory process allow a better understanding of the interaction of immunosenescence on cells periodontal. However, continuous research on this subject should be held.

5. Ageing as a risk factor for periodontal disease

The age-related changes in the periodontal tissues show that increasing age could potentially be a risk factor for periodontal disease [83]. Some moderate loss of periodontal attachment and alveolar bone is associated with age, but age alone in a healthy adult does not lead to a critical loss of periodontal support. Although moderate loss of alveolar bone and periodontal attachment is common in the elderly, severe periodontitis is not a natural consequence of ageing [3, 83]. Clinical attachment level and bone loss are irreversible measures of prior disease experience. Cross-sectional studies measuring disease experience demonstrated more attachment loss and alveolar bone loss among older age groups than other groups [3].

Longitudinal studies addressing potential relationships between age and attachment loss or bone loss showed statistically significant relationships between age and incidence of periodontal disease [84-86]. However, this age-associated increase in risk may not be linear, since some studies show no significant differences within age groups above 65 years [87-91].
The main issue related of this fact is the magnitude of any increase in risk. Studies that demonstrate statistically significant associations do not necessarily indicated that these will lead to severe clinical outcome for older adults [92]. A 28-year follow-up study reported an odds ratio of 10.4 for people aged 36-50 years compared with people aged 5-15 years. While this result is comparable in magnitude with other clinically important risk factors (smoking odds ratio in the same study was 14), it corresponds to a mean increase in clinical attachment level of only 1.34 mm over 28 years [85].
This level of increased risk probably is not sufficient, alone, to cause tooth loss. Consequently, periodontal disease may be considered as time-associated, and ageing itself appears to be responsible for some attachment and bone loss, it is of a magnitude that is unlikely to have a clinical significance [83, 92]. This fact is influenced by multiple factors that have been found to be associated with the prevalence and incidence of the periodontal disease [88].

6. Clinical Implications on ageing

Periodontal disease is not thought to be the major cause of tooth loss among adults, the loss of teeth in epidemiological studies usually induce an underestimate of periodontal disease incidence, since attachment loss tends to be greater in teeth that are extracted (whether or not the extraction is due to periodontal disease), and such teeth cannot be measured in follow-up studies [92].
Although longitudinal studies showed moderate levels of attachment loss in a high percentage of middle-aged and elderly subjects, severe loss is confined to a minority. Approximately one-fifth of older patients have experienced more generalized severe loss. The rate is higher in the oldest subjects [92]. The habitual observation in elderly populations is some loss of periodontal attachment and alveolar bone, but age alone in a healthy adult does not lead to a critical loss of periodontal support [3].
The most important clinical conclusion to draw from the longitudinal studies concerning the effects of aging is that increased age poses some increased risk for periodontal loss, but the amount of loss due to age alone is probably consistent with “successful aging” rather than accelerated pathological processes [92]. Really, it is probably appropriate to view the aging-periodontitis association as a rationale for redefining appropriate endpoints of periodontal therapy, such that the objective of treatment is to maintain a functioning dentition rather than a perfect level of periodontal attachment. Models for decision making regarding periodontal treatment needs in elderly have been proposed. Age-related thresholds could be used to decide on appropriate levels of therapy [92, 93].
Recognition of the dental needs of this special category of the population compels us to bear the responsibility of treating them now and in the future. One of the major criteria of successful ageing is to maintain a natural, healthy, functional dentition throughout life, including all the social and biological benefits, such as aesthetics and comfort, and the ability to chew, taste and speak. However, the oral health of elderly people is far from optimal. The demand for treatment is much lower than the need. In the future the elderly will retain their natural dentition and more teeth per individual will be present [94]. This fact can result in an increase in periodontal disease expectancy among the dentate elderly [2].

Certainly, in a short time, the trends for oral health care will be change. The new elderly populations will be more critical and more demanding for oral health care services than current elderly population. The dental profession must be aware of these trends which should be reflected in undergraduate and postgraduate dental education [94]. In worldwide, some countries recognized the Geriatric Dentistry as a new specialty into the dentistry [95] or included specific dental programs for elderly treatment and research [96]. If as an specific specialty or developed by a established specialty, such as the Periodontics, periodontal care services for elderly people must show proficient dental professionals, including knowledge about the ageing interactions on periodontal treatment.

7. Conclusions

In conclusion, ageing alone leads to no critical loss of the periodontal attachment in the healthy elderly. The effects of ageing human on periodontal tissues were based on biomolecular changes of the cells of periodontium that exacerbate bone loss in elderly patients with periodontitis [83].

These effects may be associated with: 1) alterations in differentiation and proliferation of osteoblasts and osteoclasts; 2) an increase in periodontal cell response to the oral microbiota and mechanical stress leading to the secretion of cytokines involved in bone loss; and 3) systemic endocrine alterations in the elderly [83].

8. References

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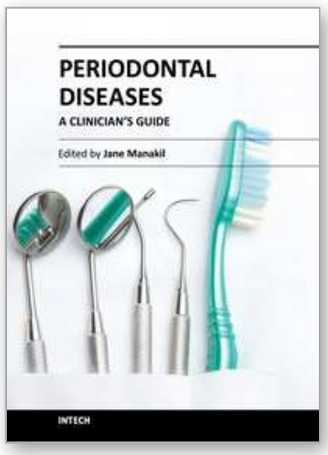

Periodontal Diseases - A Clinician's Guide
Edited by Dr. Jane Manakil

ISBN 978-953-307-818-2
Hard cover, 368 pages
Publisher InTech
Published online 03, February, 2012
Published in print edition February, 2012

"Periodontal diseases" is a web-based resource intended to reach the contemporary practitioners as well as educators and students in the field of periodontology. It is fully searchable and designed to enhance the learning experience. Within the book a description is presented of the current concepts presenting the complex interactions of microbial fingerprint, multiple genotypes, and host modulations. In addition, an overview is given of the clinical outcome of the disease's progression, as influenced by the epigenetic factors. Emerging concepts on periodontitis as a risk factor for various systemic diseases and as a bilateral modulating factor have been elucidated in detail as well.

How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Eduardo Hebling (2012). Effects of Human Ageing on Periodontal Tissues, Periodontal Diseases - A Clinician's Guide, Dr. Jane Manakil (Ed.), ISBN: 978-953-307-818-2, InTech, Available from: http://www.intechopen.com/books/periodontal-diseases-a-clinician-s-guide/effects-of-human-ageing-on-periodontal-tissues

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