ROSEMARIE E. WALCHUCK
EDITOR

PERIODONTITIS

SYMPTOMS, TREATMENT AND PREVENTION

Nova Biomedical

Public Health
— in the —
21st Century

NOVA

PUBLIC HEALTH IN THE 21ST CENTURY

PERIODONTITIS

SYMPTOMS, TREATMENT AND PREVENTION

ROSEMARIE E. WALCHUCK
EDITOR

Nova Science Publishers, Inc.
New York

Copyright © 2010 by Nova Science Publishers, Inc.

All rights reserved. No part of this book may be reproduced, stored in a retrieval system or transmitted in any form or by any means: electronic, electrostatic, magnetic, tape, mechanical photocopying, recording or otherwise without the written permission of the Publisher.

For permission to use material from this book please contact us:
Telephone 631-231-7269; Fax 631-231-8175
Web Site: http://www.novapublishers.com

NOTICE TO THE READER

The Publisher has taken reasonable care in the preparation of this book, but makes no expressed or implied warranty of any kind and assumes no responsibility for any errors or omissions. No liability is assumed for incidental or consequential damages in connection with or arising out of information contained in this book. The Publisher shall not be liable for any special, consequential, or exemplary damages resulting, in whole or in part, from the readers' use of, or reliance upon, this material.

Independent verification should be sought for any data, advice or recommendations contained in this book. In addition, no responsibility is assumed by the publisher for any injury and/or damage to persons or property arising from any methods, products, instructions, ideas or otherwise contained in this publication.

This publication is designed to provide accurate and authoritative information with regard to the subject matter covered herein. It is sold with the clear understanding that the Publisher is not engaged in rendering legal or any other professional services. If legal or any other expert assistance is required, the services of a competent person should be sought. FROM A DECLARATION OF PARTICIPANTS JOINTLY ADOPTED BY A COMMITTEE OF THE AMERICAN BAR ASSOCIATION AND A COMMITTEE OF PUBLISHERS.

LIBRARY OF CONGRESS CATALOGING-IN-PUBLICATION DATA

Periodontitis : symptoms, treatment, and prevention / editor, Rosemarie E. Walchuck.
p. ; cm.
Includes bibliographical references and index.
ISBN 978-1-61668-836-3 (hardcover)
1.Periodontitis. 2. Periodontitis--Treatment. 3. Periodontitis--Prevention. I. Walchuck, Rosemarie E.
[DNLM: 1. Periodontitis. WU 242 P4895 2010]
RK450.P4P47 2010
617.6'32--dc22

2010014101

Published by Nova Science Publishers, Inc. ✣ New York

PUBLIC HEALTH IN THE 21ST CENTURY

PERIODONTITIS

SYMPTOMS, TREATMENT AND PREVENTION

PUBLIC HEALTH IN THE 21ST CENTURY

Additional books in this series can be found on Nova's website at:

https://www.novapublishers.com/catalog/index.php?cPath=23_29&seriesp=Public+Health+in+the+21st+Century

Additional E-books in this series can be found on Nova's website at:

https://www.novapublishers.com/catalog/index.php?cPath=23_29&seriespe=Public+Health+in+the+21st+Century

此为试读,需要完整PDF请访问: www.ertongbook.com

PREFACE

Periodontitis refers to a number of inflammatory diseases affecting the periodontium, that is, the tissues that surround and support the teeth. Periodontitis involves progressive loss of the alveolar bone around the teeth, and if left untreated, can lead to the loosening and subsequent loss of teeth. Periodontitis is caused by microorganisms that adhere to and grow on the tooth's surfaces, along with an overly aggressive immune response against these microorganisms. This book reviews research on periodontitis including the role of the TH17 pathway in the progression of periodontal disease; an outline of the risk factors relating to the most prevalent chronic periodontal diseases and others.

Chapter 1 - Chronic periodontal diseases include a group of inflammatory diseases that affect periodontal supporting tissues of the teeth and encompass destructive and nondestructive conditions. Periodontal diseases are multifactorial and the role of dental biofilm in their initiation is primary. However, whether dental biofilm affects a particular subject, what form the disease takes and how it progresses, are all dependent of a wide variety of factors. Therefore, the objective of this chapter is to outline the risk factors described for the most prevalent chronic periodontal diseases (plaque induced gingivitis and chronic periodontitis) and to explain some basic concepts related to the current understanding of the role of these risk factors based on *in vitro*, animal and human studies. The review will focus on the factors that may be associated with a direct increase in the likelihood of occurrence of disease or an increase in its severity. The following factors will be discussed: 1) host characteristics, such as age, gender and race; 2) social and behavioral factors (socioeconomic status, cigarette smoking and emotional stress); 3) systemic factors, e.g. diabetes mellitus and osteoporosis; 4) genetic factors; 5) tooth-level factors (root grooves, tooth position, caries, occlusal discrepancies, iatrogenic restorations, root abnormalities and periodontal parameters); and 6) the microbial composition of dental biofilm. Finally, this chapter will also present literature-based evidence on predictive factors associated with patients and tooth susceptibility for recurrence of periodontitis after the end of the active periodontal therapy and will examine the use of some prognostic models which may be useful for clinicians in the identification high-risk groups of patients.

Chapter 2 - The goals of periodontal therapy according to the American Academy of Periodontology are to alter or get rid of the microbial etiology and causative risk factors for periodontitis, thus arresting the progression of disease and preserving the dentition in a state of health, comfort, and function with appropriate esthetics; and to prevent the recurrence of periodontitis. In addition, regeneration of the periodontal attachment apparatus, where

indicated, may be attempted. Mechanical debridement of the pocket has shown to significantly reduce the risk of tooth loss, slow down the rate of periodontal disease progression and improve gingival health.

Chapter 3 - The oral cavity is a warm, moist environment, in which a number of microorganisms colonize and live in harmony as a community, a so-called biofilm. In this environment, antimicrobial peptides may play a critical role in maintaining normal oral health and controlling innate and acquired immune systems in response to continuous microbial challenges in periodontal disease. Two major families of antimicrobial peptides, found in the oral cavity, are defensin and cathelicidin. Members of the defensin family are cysteine-rich peptides, synthesized by plants, insects, and mammals. These peptides vary in length and in the number of disulfide bonds, and have a beta-sheet structure. In the oral cavity, four alpha-defensins are synthesized and stored in neutrophil granules, which are converted into active peptides by proteolytic processing, while three human beta-defensins (hBDs), hBD-1, hBD-2, and hBD-3, are predominantly produced by oral epithelial cells. The only member of the cathelicidin family found in humans is LL-37, an alpha-helical peptide that contains 37 amino acids and begins with two leucines at its NH_3-terminus. LL-37 is derived from enzymatic cleavage of a precursor peptide, namely, human cationic antimicrobial peptide-18. Clinically, differential expression of antimicrobial peptides has been reported in specific types of periodontal disease, and their presence has been shown in saliva and gingival crevicular fluid. Current evidence suggests that alpha-defensins, beta-defensins, and LL-37 have distinct, but overlapping, roles in antimicrobial and pro-inflammatory activities. Several studies have shown antimicrobial activities of hBD-2, hBD-3, and LL-37 against several periodontal pathogens, suggesting their potential role as antimicrobial agents for periodontal disease. The clinical significance of antimicrobial peptides in periodontal disease has recently been demonstrated in morbus Kostmann syndrome, a severe congenital neutropenia, in which chronic periodontal infection in young patients, resulting from a deficiency of neutrophil-derived antimicrobial peptides, causes early tooth loss. Although researchers initially focused their attention on antimicrobial activities, it is now becoming evident that defensins and LL-37 are multifunctional molecules that mediate various host immune responses, and may thus represent essential molecules of innate immunity in periodontal disease. In this chapter, basic knowledge and the clinical importance of antimicrobial peptides in periodontal disease will be discussed in detail.

Chapter 4 - This comprehensive review highlights a detailed overview related to devising a periodontal prognosis. A precise predictability of the results of a disease is profound and crucial for proper treatment planning. Since the understanding of periodontal disease has progressed to include the influence of risk factors, assigning a prognosis has become more perplexing to the clinician. Various factors that influence the overall and individual tooth prognosis have been enumerated. The classification systems required to assign a prognosis has also been included. The potential adverse influences of both local and systemic factors have also been discussed. An experienced clinician should analyze all these factors, along with the patients attitude towards dental therapy, prior to arriving at a judgment for a single tooth or teeth. With newer trends in treatment modalities, patients can seek better options for treatment, thus improving the long term prognosis.

Chapter 5 - Background: Plaque-induced periodontitis is gingival inflammation at sites undergoing loss of connective tissue, apical migration of junctional epithelium and loss of alveolar bone. Non-surgical treatment of plaque-induced periodontitis typically involves

removal of biofilm conducted through mechanical scaling and root planing (SRP) procedures. The antibiotic minocycline hydrochloride, delivered as a sustained-release product used for professional subgingival administration into periodontal pockets, has been shown to be beneficial as an adjunct to conventional SRP. Use of chlorhexidine rinse is also a typical adjunct therapy to SRP procedures for chemical control of supragingival plaque. Lidocaine (2.5%) and prilocaine (2.5%) provides localized anesthesia for SRP. The objective of this study is to develop and use bioluminescent recombinants of oral streptococci in determining the potential antibacterial activity of minocycline hydrochloride used either alone or in combination with the anesthetic lidocaine/prilocaine, or with the antiseptic chlorhexidine.

Methods: Recombinant plasmids containing the bioluminescence-generating *lux* gene from *Photorhabdus luminescens* were transformed into the oral bacterium *Streptococcus mutans*, strains UA159 and ATCC 25175. Transformants were verified as *S. mutans* derivatives by selection and growth on mitis salivarius agar supplemented with bacitracin, in addition to an antibody test directed specifically against *S. mutans* cell wall proteins and polymerase chain reaction experiments targeting sequences in the *S. mutans* glucosyltransferase (*gtf*) gene. *S. mutans* transformants were then subjected to growth analysis for comparison of absorbance and bioluminescence activity. Minocycline hydrochloride and lidocaine/prilocaine, or minocycline hydrochloride and chlorhexidine, were used in combination to determine the potential interactive effects of these agents on the antibacterial activity of minocycline hydrochloride.

Results: Using two distinct *S. mutans* transformants representing both strains UA159 and ATCC 25175, showed rapid and pronounced bacteriostatic activity when using high doses of minocycline hydrochloride ($\geq$1 μg/ml), which were statistically distinct from untreated cultures ($p=0.000058$) when measured at the peak of metabolic activity. Reduced bacteriostatic activity was seen using lower doses. When lidocaine/prilocaine at doses >100 μg/ml is used in conjunction with minocycline hydrochloride, also shown was an additive antibacterial effect. The *S. mutans* transformant strain UA159, when treated with chlorhexidine (0.01%) in conjunction with either high (1 μg/ml) or low (0.1 μg/ml) doses of minocycline hydrochloride, displayed reduced levels of cell mass accumulation, as measured by absorbance, that were additive when both antimicrobial agents were deployed. Bioluminescence determinations, which are a direct measure of metabolic activity and an indirect measure of cell number when cells are in logarithmic stage of growth, displayed similar reductions when cultures were treated with minocycline hydrochloride and chlorhexidine used singularly or in combination.

Conclusions: The *S. mutans* lux transformants serve as sensitive biosensors in the determination of antimicrobial activity, and can rapidly monitor inhibition of bacterial metabolism. It was concluded that the anesthetic lidocaine/prilocaine does not interfere with the potent bacteriostatic activity of minocycline hydrochloride, and actually has an additive antibacterial effect. The potent bacteriostatic activity of minocycline hydrochloride can also be complemented with the addition of chlorhexidine. The application of the *lux* biosensor system in the assessment of minocycline hydrochloride and lidocaine/prilocaine, or minocycline hydrochloride and chlorhexidine, represents its first use in examining antimicrobial drug interactions in periodontology.

Chapter 6 - Periodontitis is a chronic inflammatory disease which destroys the tooth-supporting tissues. This disease is initiated by bacteria; in particular, faculative anaerobic

Gram-negative microorganisms. Several types of these pathogens initiate periodontal disease, and the host response determines the disease progression and ultimate tissue damage. The early periodontal lesion (gingivitis) is characterized by the presence of large numbers of T cells and macrophages within the gingiva, while the presence of beta (B) and plasma cells characterize the advanced lesion. These phenomena suggest that a shift in the type of host response occurs during the progression of periodontal disease. However, there is little specific information available concerning the characteristics of this shift.

Chapter 7 - Oral epithelia represent the first physical and chemical barrier against bacterial invasion and colonization of the underlying tissues. This protection results from the production of epithelial innate immune responses, including the secretion of cationic antimicrobial peptides with a large spectrum of activity against pathogenic microorganisms. Among these antimicrobial cationic peptides, ß-defensin 2 (hBD-2) is expressed in the gingival epithelia upon stimulation by microorganisms or inflammatory mediators such as interleukin-1β or tumour necrosis factor-α. The aim of the present study was to investigate the effect of AV119, a patented blend of two sugars from avocado, on the induction of hBD-2 in two epithelial cell lines and a primo-culture of gingival epithelial cells. Culture supernatant from epithelial cells treated with AV119 was also evaluated for its antimicrobial activity against the periodontopathogen *Porphyromonas gingivalis*. Cell ELISA assays revealed that AV119 induces the production of hBD-2 by all the epithelial cells tested. Minimal Inhibition Concentration assay also showed that the culture supernatant of epithelial cells treated with AV119 possesses antibacterial activity. In conclusion, our data revealed that AV119 component, through hBD2 induction and antibacterial activity, could be considered for potential use in the control of oral mucosal infections and reduction of microbial tissue invasion during periodontitis.

Chapter 8 - Cheilitis granulomatosa is characterized by the non-inflammatory swelling of the lips, and is considered as the incomplete expression of the Melkersson-Rosenthal syndrome, which consists of the triad of recurrent orofacial swelling, relapsing facial paralysis, and fissuring of the tongue. Rapid improvement after the treatment of periodontitis was first reported in 1961 by Kawamura et al in Japan, and 46 such cases have been reported since then in the Japanese literature. A typical case of cheilitis granulomatosa can be excperienced. The swollen lip showed marked improvement following the treatment of apical periodontitis. A 57-year-old woman presented with a swelling of the lower lip for the period of two months. Skin biopsy of the lip disclosed non-caseous giant cell granuloma. Neither facial nerve palsy nor fissuring of the tongue was present, excluding the diagnosis of Melkersson-Rosenthal syndrome. Patch testing for metal allergy was negative for all dental metallic ions, except for only mild irritation reaction for Zinc ion. The patient was first treated with topical corticosteroid ointment and oral tranilast, which inhibits the release of chemical mediators from leukocytes, for 4 months. Although the treatment was ineffective, rapid and remarkable improvement of the swelling was noted soon after the treatment of apical periodontitis. Thus, it is highly likely that the periodontitis was the cause of cheilitis granulomatosa in this case. In this article, such 46 cases are reviewed in the Japanese literature.

Chapter 9 - Periodontitis is a complex disease which is associated with multiple factors, including host immune responses, and genetic, behavioral and environmental factors. It is generally accepted that genetic polymorphisms can modulate host immune responses to bacterial challenge, hence influencing subjects' susceptibility to periodontitis. Genetic

association with periodontal disease experience has been a subject of interest for more than a decade. With the completion of Human Genome Project, genetic association studies emerged in many fields of research including research into periodontitis, one of the most common human diseases. This chapter summarizes past and current research approaches with respect to periodontal disease experience and genetic polymorphisms, and suggests anticipated directions of future studies.

Chapter 10 - Coronary heart disease (CHD) shares a number of features with chronic periodontitis (CP) including risk factors such as smoking and diabetes; an aetiopathogenesis implicating a number of microbial species, as well as chronic inflammation. However, the link between these two conditions remains unclear. The prevalence of CHD increases with age and is higher in males than females. CP is a chronic inflammatory condition which destroys the supporting tissues of teeth and also increases in prevalence with age. Immune responses against heat shock proteins (HSP) can be cross-reactive among bacterial and human antigens. There is evidence that microbial HSP65 and human HSP60 is involved in periodontal disease and CHD and may therefore provide a mechanistic link between CP and CHD. The aim of this study is to investigate immune responses to the human HSP60 and microbial HSP65 in patients with CP and CHD and relate these to the level of inflammation. Serum samples was collected from 100 male subjects divided into 4 groups, each matched for age: (a) Healthy control group with minimal gingivitis, (b) CP, (c) CHD with gingivitis (d) CHD with CP. ELISA was used to determine the levels of serum anti-HSP and C-reactive protein (CRP) in the 4 groups. Peripheral blood mononuclear cells were also isolated from these 4 groups and stimulated with HSPs. Significant lymphoproliferation was seen in CHD with or without CP when stimulated with human HSP60. CRP and serum anti-human HSP60 IgG were elevated in the patients groups compared to the healthy control group, but not serum anti-microbial HSP 65 IgG,. In view of the potential confounding effects of smoking in CP and CHD, a group of current smokers (n=24) were also recruited to investigate whether smoking affects HSP immune responses.There was no significant difference in HSP-induced lymphoproliferation between smokers and non-smokers in any of the four groups. There was a significant correlation between CRP and lymphoproliferative responses to Human HSP60.

This study shows that serum anti-human HSP60 IgG and serum CRP are raised in CHD with or without CP. In CHD with or without CP, serum CRP levels correlated significantly with human HSP60-induced lymphoproliferation, but not with anti-HSP antibody levels.

Chapter 11 - Morbidity and mortality from oral cancer are high and this has not improved in decades in spite of extensive research. A significant portion of research is concentrated on chemoprevention. However, advances in this field have not translated into a visible change in mortality and morbidity. In addition, existing chemoprevention strategies have two important obstacles: toxicity and reversal of the effects after cessation of treatment. Chronic infection and inflammation have been linked to carcinogenesis in a few organs. For oral cancer, substantial evidence has accumulated for the role of *human papillomavirus* (HPV). However, the development of an effective preventive vaccine strategy for oral cancer is still years away and the target population is largely unexplored. Therefore, safe and practical additional approaches are necessary to change the status quo of oral cancer. Periodontitis is a chronic oral infection caused by inflammatory reactions in response to gram negative anaerobic bacteria in the endogenous dental plaque. It leads to irreversible destruction of tissues around teeth clinically detectable as periodontal pockets and alveolar bone loss. Periodontal pockets have been suggested as reservoirs of HPV. Chronic proliferation and ulceration of the pocket

epithelium may help HPV's initial infection and persistence. Our preliminary results from existing data at Roswell Park Cancer Institute suggest a robust independent association between the history of periodontitis and incident oral cancer. Our next step is to test the synergy between periodontitis and HPV for the risk of oral cancer. If this is true, it will translate to practical and safe prevention and treatment strategies. This chapter will review the evidence supporting the association between chronic periodontitis and oral cancer as well as HPV-periodontitis synergy.

此为试读,需要完整PDF请访问: www.ertongbook.com

CONTENTS

In: Periodontitis Symptoms, Treatment and Prevention ISBN: 978-1-61668-836-3
Editor: Rosemarie E. Walchuck, pp. 1-33 ©2010 Nova Science Publishers, Inc.

Chapter 1

RISK FACTORS FOR CHRONIC PERIODONTAL DISEASES

Daniela da Silva Feitosa[1], Mauro Pedrine Santamaria[1], Márcio Zaffalon Casati[2], Enilson Antonio Sallum[3], Francisco Humberto Nociti Júnior[3] and Sérgio de Toledo[3]

DDS, MS, PhD - Department of Prosthodontics and Periodontics – Division of Periodontics, Piracicaba Dental School, University of Campinas.[1]
Associate Professor - Department of Prosthodontics and Periodontics - Division of Periodontics, Piracicaba Dental School, University of Campinas.[2]
Professor - Department of Prosthodontics and Periodontics - Division of Periodontics, Piracicaba Dental School, University of Campinas, Brazil[3]

ABSTRACT

Chronic periodontal diseases include a group of inflammatory diseases that affect periodontal supporting tissues of the teeth and encompass destructive and nondestructive conditions. Periodontal diseases are multifactorial and the role of dental biofilm in their initiation is primary. However, whether dental biofilm affects a particular subject, what form the disease takes and how it progresses, are all dependent of a wide variety of factors. Therefore, the objective of this chapter is to outline the risk factors described for the most prevalent chronic periodontal diseases (plaque induced gingivitis and chronic periodontitis) and to explain some basic concepts related to the current understanding of the role of these risk factors based on *in vitro*, animal and human studies. The review will focus on the factors that may be associated with a direct increase in the likelihood of occurrence of disease or an increase in its severity. The following factors will be discussed: 1) host characteristics, such as age, gender and race; 2) social and behavioral factors (socioeconomic status, cigarette smoking and emotional stress); 3) systemic factors, e.g. diabetes mellitus and osteoporosis; 4) genetic factors; 5) tooth-level factors (root grooves, tooth position, caries, occlusal discrepancies, iatrogenic restorations, root abnormalities and periodontal parameters); and 6) the microbial composition of dental biofilm. Finally, this chapter will also present literature-based evidence on predictive factors associated with patients and tooth susceptibility for recurrence of periodontitis

after the end of the active periodontal therapy and will examine the use of some prognostic models which may be useful for clinicians in the identification high-risk groups of patients.

INTRODUCTION

Chronic periodontal diseases include a group of inflammatory diseases that affect the periodontal supporting tissues of teeth and encompass destructive and nondestructive conditions [12]. The term chronic periodontal diseases will refer, in this chapter, to both plaque-induced gingivitis and chronic periodontitis. Plaque induced gingivitis is the inflammation of the soft tissues without apical migration of the junctional epithelium [32]. In addition, chronic periodontitis, the most frequent form of periodontitis, results in inflammation of the supporting tissues of the teeth, progressive attachment and bone loss at a slow rate, characterized by pocket formation and/or gingival recession [34]. Cross-sectional epidemiologic studies from many countries have shown that gingivitis is highly prevalent in the primary and permanent dentitions of children [7] and affects many adults [5]. Further, chronic periodontitis is also a common entity worldwide [6]. Therefore, a knowledge of the factors that may influence the transition from health to disease and of the progression of the disease through various stages of severity are important in the development of effective strategies of prevention and treatment.

Gingivitis has already been established as a consequence of dental biofilm accumulation. It is produced as the result of a general increase in the number of microorganisms and a change in the composition of the flora associated with the increasing age of the dental biofilm [99]. Several studies show that periodontitis is preceded by gingivitis and, although the accumulation and duration of microbial dental plaque biofilm will predictably lead to the development of inflammation in the nearby gingival tissues, the duration of onset and the intensity of the inflammatory process vary considerably from person to person, as well as between teeth. Albandar et al. (1998) [4] studied a periodontally high-risk group comprising 156 young subjects that were examined twice during a period of six years to study the relationship between the presence of gingival inflammation (gingival bleeding) and the occurrence of attachment loss. They found that 9.3% of sites that had gingival bleeding and 0-2 mm of attachment loss at baseline showed a longitudinal attachment loss of ≥ 3 mm over 6 years, whereas only 4.8% of sites with no gingival bleeding at baseline showed a corresponding attachment loss. Hence, 90.7% of sites with gingival bleeding at baseline did not show any clinical attachment loss during the study period. This study showed that not all sites with gingival inflammation developed periodontitis during the study period. Thus, predisposition to periodontitis development varies significantly and may possibly be influenced by other factors. However, defining the factors involved in initiation and progression of chronic periodontitis is a more complex issue.

Chronic periodontitis is a multi-factorial disease. While the role of bacteria is primary, a number of host-related factors have been hypothesized as influencing its diverse clinical presentation and rate of progression [72]. Loe et al. (1986) [100], in a longitudinal study, evaluated a Sri Lanka population never exposed to any programs or incidents related to the prevention and treatment of dental diseases. This population did not practice any conventional oral hygiene measures. Three subpopulations were identified: 1) individuals with rapid

progression of periodontal disease (8%); 2) individuals with moderate progression (81%); and 3) a group who exhibited no progression (11%). When another longitudinal study was made comprising a sample of middle-class Norwegian men who had the benefit of a comprehensive health care program, a group that represented an extreme condition of periodontal maintenance when compared to the Sri Lanka population, two subpopulations (moderate disease and no disease) were found, despite the severity of attachment loss [164]. These studies illustrate significant differences in the pattern and rates of attachment loss among individuals, even when they receive regular and adequate professional and personal health care. Based on the evidence above, the identification of factors involved in the initiation and progression rate of chronic periodontal diseases has been the focus of considerable research in recent times.

Chronic inflammatory periodontal diseases have several etiological factors for which a plausible biological model of effect exists. The term risk factors is commonly used and it refers to an aspect of personal behaviors or lifestyle, an environmental exposure, or an inborn or inherited characteristic, which on the basis of epidemiological evidence is known to be associated with a health-related condition [99]. The presence of a risk factor implies a direct increase in the likelihood of a disease occurring [95]. Prospective longitudinal studies, and in particular clinical trials, provide the most powerful evidence for the existence, and the amount, of risk. However, in most cases these types of studies are not easily conducted. For this reason, most evidence for the existence of possible risk factors for periodontal diseases comes from cross-sectional studies. Although the identification of risk factors for disease is unfeasible using cross-sectional studies, when a proper study design is employed, these studies can provide valuable information on the presence or absence of an association between the variables under study and the occurrence of periodontal diseases. In order to make a distinction between the results of the different types of studies, it is customary to refer to significant effects assessed in cross-sectional studies as associations, whereas effects disclosed using case-control studies and prospective studies have been referred to as risk determinants, risk indicators, or risk markers [8].

Usually, the overview of factors associated with chronic periodontal diseases is systematically presented as host characteristics, social and behavioral factors, systemic factors, genetic factors, tooth-level factors and microbial factors [126]. In addition to the investigation of these factors at the onset of chronic periodontal diseases, longitudinal studies of patients treated for periodontitis try to determine the patient's susceptibility to disease recurrence [64, 96]. As a result, the prognostic factors (disease predictors), defined as characteristics related to the progression of preexisting disease [133], have been the subject of much discussion. The identification of groups and individuals at risk for disease progression during maintenance therapy still represents one of the greatest challenges in the management of periodontal patients. Thus, prognostic models aimed at identifying high-risk individuals or teeth in a clinical setting have been described [56, 91]. A question remains about the safety of these models routinely used to help clinicians in decision-making.

HOST CHARACTERISTICS

Age

Several epidemiological studies have clearly demonstrated an increase in the prevalence (percentage of persons), extent (percentage of teeth per person) and severity of periodontal attachment loss with increasing age [6, 9]. Papapanou et al. (1988) [132] examined full-mouth radiographs from 531 dentate individuals aged 25-75 years and found that bone loss increased with age. Moreover, two large epidemiological studies estimated the prevalence and extent of periodontal diseases in the United States using data from the National Health and Nutrition Examination Survey (NHANES) in the years 1985 to 1986 and 1988 to 1994 [6, 23]. It was demonstrated that 48.6% of persons 35 to 44 years old and 77.3% of those 55 to 64 years old had $\geq$ 3 mm attachment loss in the first survey. The same trend was observed in the second study, in which 48.5% for the 40 to 49 year old cohort and 74.8% for the 60 to 69 year old group had $\geq$ 3 mm attachment loss. Regarding the healing of periodontal tissues following periodontal therapy, Lindhe et al. (1985) [97] evaluated 62 patients and reported that, although age did not seem to have a significant effect on the results of periodontal treatment, there was a tendency for younger patients to have a shallower probing depth and gain more periodontal attachment than older patients.

With increasing age, people have to cope with a lifelong antigenic burden encompassing several decades of evolutionary unpredicted antigenic exposure, with a major impact on survival and frailty. In fact, there is a peculiar chronic inflammatory status characterizing aging, which has been denominated by Franceschi et al. (2000) [57] as inflamm-aging, and which is considered a random process detrimental for longevity, leading to long-term tissue damage, and related to a wide range of age-related diseases, including neurodegeneration, atherosclerosis, diabetes and osteoporosis among others, which share an inflammatory pathogenesis. It may therefore be speculated that this phenomenon may also affect the periodontium, in which after a lifetime's challenge by oral periodontopathogenic bacteria and their virulence factors, periodontal tissues may develop an intense subclinical inflammatory process, but also lead to healing/regeneration outcomes after periodontal therapy [15]. In vitro studies have clearly demonstrated an age-related decrease in the proliferation of periodontal ligament cells [15, 166]. Further, aging is able to modulate the expression of genes reported to participate in periodontal homeostasis (e.g. cytokines, metalloproteinases and their inhibitors and bone-related genes) by periodontal ligament cells [14, 15]. It is important to remember the role of periodontal ligament cells on periodontal health and disease because of their ability to proliferate, migrate and synthesize several components of the periodontium and also participate in the protective host mechanism that prevents periodontitis or impedes its progression [60]. Little information on the influence of aging on the periodontium is provided by animal studies. It has been documented that the periodontal ligament presented decreased cell density and collagen synthesis, and also a decreased number of cells in the osteogenic layer of the alveolar bone has also been reported [135, 165].

Despite the well-documented loss of attachment with increasing age and the rationale behind the association, the question as to what extent aging affects periodontal homeostasis is still a controversial issue in the periodontal literature. A number of arguments have been used

此为试读,需要完整PDF请访问: www.ertongbook.com