Periodontal Disease
Frontiers of Oral Biology

Vol. 15

Series Editor

Paul Sharpe  London
Periodontal Disease

Volume Editors

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26 figures, 16 in color, and 6 tables, 2012
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Foreword

Our understanding of the etiopathology of periodontal disease has changed greatly over the last decade through greater appreciation of the complexity found when examining both the host and pathogens. The species diversity within the microbial biofilm and the huge layered complexity of the innate, inflammatory and adaptive immune responses to this biofilm warrant study and discussion. The current volume comprises reviews from renowned experts in these fields, and aims to present a readable overview of this exciting and pertinent subject matter.

The volume opens with a review by Zijnge et al. of the subgingival biofilm structure, utilizing combined histology, confocal scanning fluorescent microscopy and fluorescent in situ hybridization to localize the most abundant species from different phyla and the species associated with periodontitis. The authors provide new insights into the structure and composition of subgingival biofilms and the nature of the extracellular matrix. They describe ‘subgingival’ biofilms produced in vitro that may be useful in future therapeutics testing and modeling of host-pathogen interactions.

Diaz then discusses the microbial diversity of the subgingival biofilm and describes a complex human subgingival environmental niche where microorganisms from the three domains of life meet to form diverse biofilm communities existing in close proximity to the host. Bacteria constitute the most abundant, diverse and ultimately well-studied component of these communities, with about 500 bacterial taxa reported to occur in this niche. Cultivation and molecular approaches continue to reveal the breadth and depth of subgingival biofilm diversity as part of an effort to understand the subgingival microbiome, the collection of microorganisms that inhabit the gingival crevices. This review presents a summary of current understanding of subgingival microbial diversity and an overview of experimental models used to dissect the functional characteristics of subgingival communities.

Innate cellular responses to the periodontal biofilm are critical to our understanding of the host response in periodontal disease, and Benakanakere and Kinane address this subject. In response to infection, the host’s resources comprise the innate, inflammatory and adaptive immune systems, whose role is to provide the appropriate response to the offending microorganisms. In some cases, this will be little or no response – i.e. when ‘commensals’ are encountered – and in other cases it is a gradated
response depending very much on the host's own determination of the pathogenic nature of the microbial insult: and herein lies the root of variation in host responses that govern individual susceptibility.

As discussed in depth in the articles of Diaz and Zijnge et al., gingival inflammation originates from responses to multiple microorganisms that comprise the biofilm rather than to just one highly virulent species. An understanding of the interaction of structural and defensive host cells with the biofilm is pivotal in understanding periodontal disease etiology and to developing tailored therapeutics. Thus, this chapter addresses the main structural cell exposed to the biofilm, i.e. epithelial cells, and the subsequent chapter by Scott and Krauss addresses the neutrophil response to biofilms. Neutrophils or polymorphonuclear leukocytes (PMN) are the most abundant leukocytes whose primary purpose as antimicrobial professional phagocytes is to kill extracellular pathogens. PMN and macrophages are phagocytic cell types which, along with other cells, effectively link the innate and adaptive arms of the immune response, and help promote inflammatory resolution and tissue healing. This review covers the innate and inflammatory functions of PMN and describes their importance in the integrity of the periodontium in health and disease.

Gorr addresses the role of antimicrobial proteins and peptides (AMP) in the host response to biofilm bacteria. AMP are an early component of the host-response produced by salivary glands, oral epithelial cells and neutrophils. Over 45 AMP have been identified in the oral cavity. All are found in saliva and several are also present in the gingival crevicular fluid. This review considers the differential regulation of AMP expression in periodontal disease and suggests that AMP panels may have a role in oral fluid diagnosis of periodontal disease and in monitoring treatment outcome.

Having addressed fundamental aspects of the etiopathogenesis, Apatzidou now considers modern approaches to non-surgical biofilm management. In contrast to microorganisms growing in a planktonic state, the inhabitants of a biofilm are effectively protected within this dense structure from host defense mechanisms and from therapeutic agents including antimicrobials. The mechanical removal of the microbial biofilm and the establishment of meticulous plaque control measures comprise the key elements for the success of non-surgical periodontal treatment. Apatzidou considers controversies and modern trends in non-surgical periodontal therapy, such as full-mouth approaches and power-driven instrumentation. As outcome variables, clinical, microbiological and immunological aspects are considered following different treatment protocols, in addition to cost-effectiveness.

Continuing in the therapeutic vein, Mombelli addresses advances in antibiotic use in the management of periodontal disease. Antibiotics are generally an efficient means of treating bacterial infections, and therefore an obvious candidate in the treatment of periodontal diseases. Systemically and locally administered antimicrobial agents of all kinds have been evaluated in multiple clinical trials. The vast majority of studies have tested antibiotics as adjuncts to non-surgical debridement. No regime has demonstrated superiority over systemically administered amoxicillin and metronidazole.
in the treatment of any clinically or microbiologically defined variant of periodontal disease. The author argues that in light of the excellent results of a combination therapy with well-established drugs, clinical trials should compare newly proposed protocols for periodontal therapy to a positive control.

Traditional treatments have focused on repairing the damage induced by periodontal disease; however, clinicians have begun to utilize regenerative techniques to rebuild bone, cementum and the periodontal ligament. Kao and Fiorellini describe the potential of barrier membranes with bone grafts and pharmacologically based efforts to foster selective cell repopulation and the regrowth of osseous structures.

To close the therapeutic contributions and the volume itself, Hasturk et al. propose a paradigm shift in the pharmacological approach to periodontal disease management. They argue that while it is clear that the inflammatory response is a major determinant in susceptibility to periodontitis, our understanding of the relationship of the causal agents in periodontitis to the pathogenesis is not as clear – and therefore neither is the rationale for therapies based on this. The discovery of new families of lipid mediators of resolution of inflammation, the lipoxins and EPA- and DHA-derived chemical mediators, and the resolvins and protectins opens new avenues in the design of resolution-targeted therapies to control the unwanted side effects of excessive inflammation. The novel protective and therapeutic actions of pro-resolution lipid mediators following microbial challenge are mediated by regulation of the inflammatory response that has a direct impact on the organization of the biofilm (plaque) and could suggest a paradigm shift in clinical periodontal therapeutics.

All of these valuable and insightful contributions make this volume a timely update on the etiopathogenesis of periodontal disease and its relevance to modern periodontal therapeutics.

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