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GUEST EDITORIAL

The Etiology and Pathogenesis of Periodontitis Revisited

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The science of periodontology has yielded significant insights into this common disease of man. The field of anaerobic microbiology is arguably developed to its current level by periodontal researchers and our treatment strategies are based on our understanding of the infectious nature of the disease. Yet, we are in the midst of a major paradigm shift in periodontology. We have come to understand that while the etiology of periodontitis is bacteria, the pathogenesis is inflammatory. Our understanding of regulation of inflammation in periodontitis is far from complete; however, as our understanding of periodontal inflammation increases, our current understanding of the microbiology of periodontitis becomes less clear. While we think we know that bacteria initiate the disease, the role of specific bacteria is still unknown. We cannot cultivate most of the bacteria associated with the disease and new data would suggest that we don't really understand the relationship of the biofilm to inflammation. A review of the literature will reveal that our knowledge of the microbiology of periodontitis is based on large cross-sectional and association studies. Periodontitis is seen as the direct consequence of bacterial invasion and is regarded as an infectious disease. It is however, not possible to draw cause-and-effect inferences from these studies; the implication of the "red complex" as pathogens is an "association".

New work in medicine and other studies in periodontology have introduced a new idea in biofilm biology. That is, the host inflammatory response dictates the composition of the biofilm[1,2]. What this means is that the inflammatory response changes the microenvironment of the biofilm and selects for specific organisms. One might then argue that *P.g. and T.f.* overgrow in the periodontal pocket *because* of inflammation; the implication is that the inflammation precedes the overgrowth. What then initiates the disease? Could it be early, gram-positive colonizers that elicit a profound inflammatory response in the susceptible host?

In a recently published study by Tanner, et al.[3], the onset on periodontitis was studied longitudinally in an effort to identify organisms that preceded attachment loss. No organisms were identified as being predictive of future attachment loss; gingival inflammation was the only predictor of future attachment loss in this study. Interestingly, once attachment loss was observed, deeper pockets were associated with the overgrowth of *P.g.*, which is in concert with all previously published cross-sectional studies. In other studies by Hasturk, et al.[2] in an animal periodontitis model, pharmacologic control of inflammation resulted in the spontaneous disappearance of *P.g.* from the periodontal lesions. Taken together, these data bring into question the initiators of disease. Are "traditional" periodontal pathogens the initiators of disease or do they result from disease?

This is not a solely academic argument. Understanding the pathogenesis of a disease has direct impact on treatment strategies. We treat periodontitis as an infectious disease; most periodontists would agree that our success is limited. The implication of the paradigm shift outlined above is that periodontitis is an inflammatory disease. Should the primary target of pharmacotherapy be the inflammation, rather than the bacteria? Emerging evidence suggests that the host inflammatory response might dictate the composition of the biofilm and the emergence of pathogens. The question to be investigated is whether dampening of the inflammatory response in certain individuals susceptible to periodontitis will prevent development of disease.

1- Finlay BB, Medzhitov R. Host-microbe interactions: fulfilling a niche. Cell Host Microbe. 2007;15(1):3-4.

2- Hasturk H, Kantarci A, Goguet-Surmenian E, Blackwood A, Andry C, Serhan CN, et al. Resolvin E1 regulates inflammation at the cellular and tissue level and restores tissue homeostasis in vivo. J Immunol. 2007;179 (10):7021-9.

3- Tanner ACR, Kent R Jr., Kanasi E, Lu SC, Paster BJ, Sonis ST, et al. Clinical characteristics and microbiota of progressing slight chronic periodontitis in adults. J Clin Periodontol. 2007;(34):917-30.