Early Adopters' Edge: Re-Thinking the Significance of Periodontal Disease

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Author's note: I want to thank Dr. Michael Cohen for inviting me to present at the Seattle Study Club's 2009 Symposium. Such a beautiful week in Cancun, such gracious hosts and hostesses, and what an honor to present to such an esteemed group of practitioners!

Over the last several decades much has been learned about an emerging base of evidence supporting interrelationships between periodontal disease and inflammatory driven disease states/conditions such as diabetes, atherosclerosis-induced diseases, bacterial pneumonia, adverse pregnancy outcomes, osteoporosis, rheumatoid arthritis, Alzheimer's disease, and chronic kidney disease/end stage renal disease, among others. Studying this evolving body of knowledge called periodontal-systemic science has absorbed most my professional life over the last few years. Along with an increasing number of researchers and academicians inside and outside of dentistry, I believe the emerging evidence of periodontal-systemic relationships has now reached a tipping point which can no longer be ignored. Furthermore, integrating credible findings of this research into clinical dentistry and dental hygiene can no longer be considered optional. Given the dialogues I had with course participants after my presentations in Cancun, it looks like there are a lot of SSC members that are thinking along these same lines.

The slow pace at which health care disseminates change has been documented for centuries. In a 2003 article written for the *Journal of the American Medical Association*, Berwick eloquently described the opportunity cost associated with failure to recognize and adopt new scientific knowledge within healthcare: "Health care is among the best endowed of all industries in the richness of its science base…yet, an enormous amount of that scientific knowledge remains unused."[1] The emergence of statin therapy is one of the most poignant examples of this slow rate of knowledge diffusion. It took over a decade for the National Cholesterol Education Program to promote understanding of the dangers of high blood cholesterol as a major cause of coronary heart disease. Further, the adoption curve associated with the acceptance of statins to lower cholesterol was relatively prolonged.[2] (The first adult treatment panel report, the *Report on the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults*, was published in 1988;[3] but it was almost 15 years later that a clinical advisory statement[4] was published that promoted statins as promising in the reduction of risk related to coronary heart disease.) Unfortunately, the clinical adoption of emerging research related to periodontal-systemic links may follow this slow pace of knowledge diffusion. And, unfortunately, like the opportunity cost associated with delayed acceptance of statin drugs, the potential for intervention of periodontal disease to decrease the risk for systemic injury may not be fully recognized for some time in mainstream dentistry.

What we already know about periodontal-systemic relationships

No one can argue that there is still a tremendous amount of investigation necessary to establish cause-and-effect relationships between periodontal disease, systemic injury and the biological pathways linking the two. We also understand how important it is to ensure that statements we make to patients about the risk periodontal disease may pose to systemic consequences are based on research that is validated by scientific evidence. So the question becomes, what is truly valid information that gives us direction for clinical practice? Here's what we do know—researchers are in agreement that evidence of a cascade of events which link periodontal disease to inflammatory driven disease states is at least moderately strong. On the following page are a number of statements related to the role of periodontal disease in increasing the risk for an inflammatory driven disease state which are in alignment with the opinions of the scientific community.

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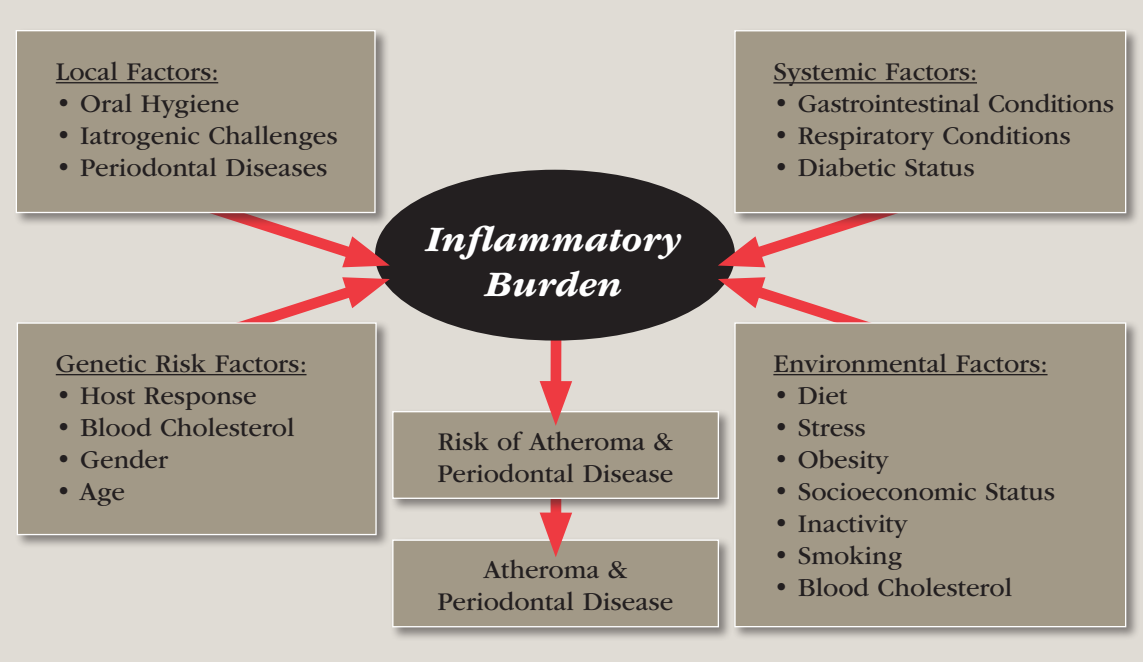

Figure 1. Concept of the cumulative inflammatory burden.[19]

1. Sustained inflammation is the driving force behind most chronic diseases/conditions.

2. Whether a person develops periodontal disease, and/or another chronic disease, is dictated largely by that person's susceptibility.

3. Inflammation from periodontal disease is not confined to the oral cavity.

4. Infection, including periodontal disease, has the potential to initiate the inflammatory response.

5. Chronic diseases are usually not caused by a single bacterial exposure, rather chronic disease states are the result of multiple bacterial exposures/episodes, like periodontal disease, over time.

6. Periodontal disease, like other bacterial exposures, increases the risk for a chronic disease state.

7. Periodontal disease amplifies the sequelae of a chronic condition.

8. Periodontal disease may make it more difficult to treat or change the trajectory of a chronic disease state.

9. Cumulative bacterial burden in conjunction with already existing traditional risk factors (i.e., smoking, obesity) often tip the scale from health to disease. Periodontal infection contributes to the cumulative bacterial burden, and may be implicated in tipping this scale.

10. Prevention or treatment of periodontal disease will at least reduce the cumulative pathogen and systemic inflammatory burden.

Figure 1 illustrates the concept of cumulative inflammatory burden. This concept draws from evidence which suggests that the greater the total systemic inflammatory burden, the greater the risk of a number of disease states including atheroma formation and periodontal disease. Preventing or eliminating periodontal disease, as a modifiable risk factor, decreases, albeit to an unknown extent, the inflammatory burden.

The plausibility of orally derived bacteria being associated with an increased risk for systemic disease

It is well established that given the high vascularity which accompanies inflamed soft tissue, the interface between the subgingival biofilm and the ulcerated epithelial lining of a periodontal pocket provides a very accessible portal for a number of periodontal pathogens to gain entry to the vasculature tree. The surface area of the epithelium potentially exposed to virulent, gram negative bacteria associated with the subgingival biofilm in chronic periodontitis has been estimated to range between from 8 cm^2 to 20 cm^2.[5] Medical professionals understand the

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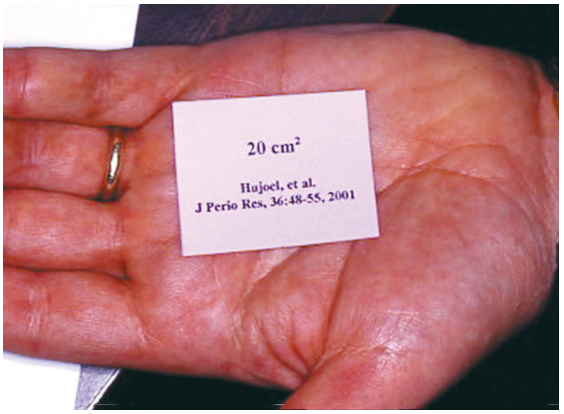

Figure 2. The surface area of the epithelium potentially exposed to virulent, gram negative bacteria associated with the subgingival biofilm in chronic periodontitis is roughly the size of an adult palm.[5] (Courtesy of Dr. Charles Cobb.)

significance of a lesion of gram negative infection of this magnitude. When confronted with this information and a photograph of the size of this lesion (depicted in Figure 2), I often see a lot of jaw drops in a room of physicians and nurses. Any other place on the human body, this level of infection would be a real concern to most healthcare providers. Unfortunately, this lesion is hidden subgingivally, but nonetheless, it is a chronic wound, and a constant reservoir for dissemination of numerous pathogens which are also very adept at evading the host defenses.

There is strong and compelling evidence that at this juncture, short-term recurrent bacteremias or chronic low-grade exposures from oral origin may induce systemic consequences.[6] Tissue fragility accompanies periodontal disease. Subsequently, mastication, tooth brushing, and professional dental procedures, stimulate a portal of entry for bacteria and other cellular matter to gain access to the circulatory system.[7] Evidence also suggests that orally derived bacteremia increases as periodontal disease progresses.[8] These well substantiated findings provide a highly plausible explanation for the potential of periodontally related bacteremia increasing the risk for systemic dissemination of oral bacteria. Accompanying this is the potential for metastatic injury related to the effects of the circulating toxins of periodontal pathogens, and the metastatic inflammation caused by the immunologic response to the pathogens and their toxins.[9,10,11]

The concept of "systemic periodontitis"

Several years ago, Beck and Offenbacher[12] coined the term "systemic periodontitis" to better describe the threat that periodontal disease may pose to systemic health. What these researchers were proposing was that periodontal disease actually represents an exposure event that contributes not to tooth morbidity, but systemic pathology. They proposed that, "periodontal disease must be thought of as a disease process that is an exposure for a systemic disease or condition (outcome) rather than the outcome itself."[12]

In considering the concept of periodontal disease as an exposure event for greater systemic sequelae, I have proposed that periodontal disease must be included in a risk continuum that begins with bacterial challenge of periodontium, eventuating in whole body consequences that include increased risk for atherosclerosis-induced diseases (i.e., coronary heart disease and stroke), complications of diabetes, adverse pregnancy outcomes, respiratory diseases, neurodegenerative diseases, among others. (Figure 3)

Why wait to intercept periodontal disease?

It has been estimated that 60 million people in the United States have moderate to severe periodontal disease.[13] Moderate to severe periodontal disease has been defined as greater than 3 mm of clinical attachment loss and greater than 5 mm of pocket depth.[14,15,16] (It was 1924 when Orban established that 1.8 mm is the average depth of a healthy sulcus, and that 3 mm was the boundary of health.[17])

In an average general dental practice consisting of 2000 patients, it has been estimated that 33-37% or 500-555 adult patients (over the age of 21) have moderate to severe chronic periodontitis.[13] Further, data related to treatment trends indicate that in 1980 only 2.5% of insurance codes were related to treatment of periodontal disease. This clearly does not align with the true prevalence of periodontal disease within the United States. When this data was compiled in 2000, this percentage (2.5%) did not change.[18]

Given the strength of evidence which suggests that periodontal disease has the potential to evoke a systemic inflammatory response and

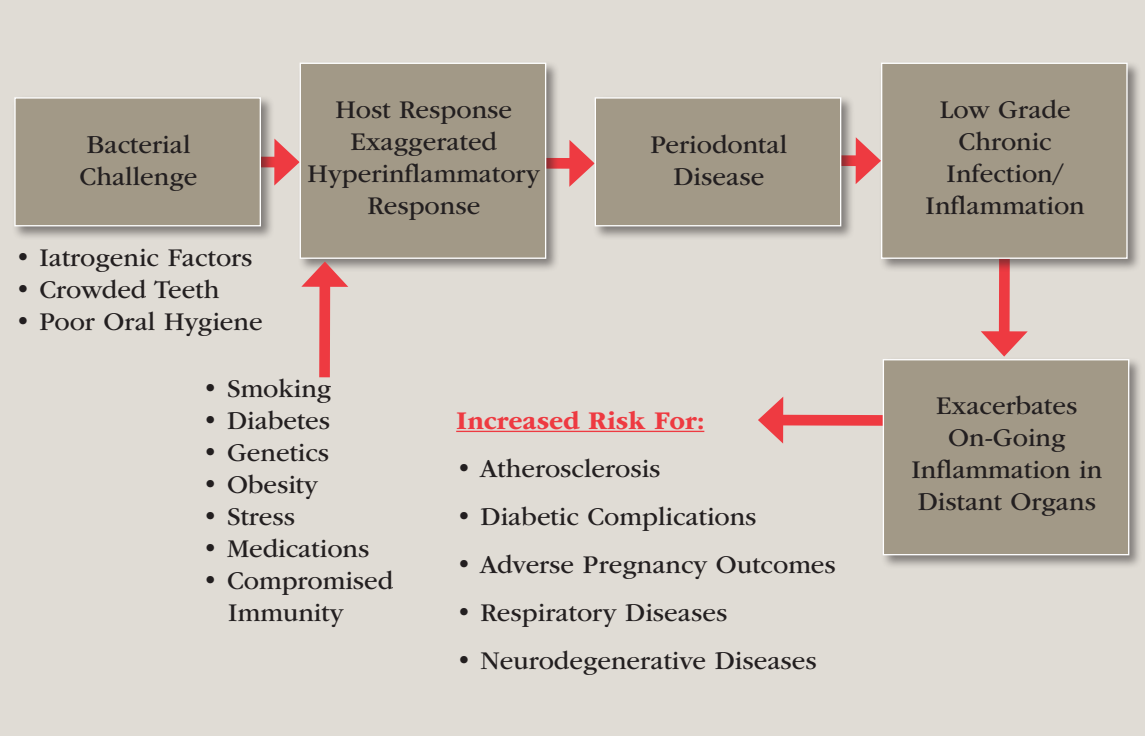

Figure 3. The risk continuum that illustrates the link between periodontal disease and systemic inflammatory diseases. (Source: Casey Hein.)

The concept of the risk continuum that links periodontal disease to inflammatory driven disease states has been adopted from the work of Beck and Offenbacher[12] who proposed that "periodontal disease must be thought of as a disease process that is an exposure for a systemic disease or condition (outcome) rather than the outcome itself." In this continuum of risk, bacterial challenge of anaerobic Gram negative microorganisms initiates the cascade of events. We now know that although bacteria are essential in initiating the etiological process associated with periodontal infection, the presence of bacteria alone are insufficient to explain the loss of supporting structures of the periodontium. How the host responds (our immune response) plays a major role in determining whether the infection is contained within the soft tissue of the periodontium, or whether various risk factors (e.g., smoking, diabetes, genetics, obesity, stress, certain medications or compromised immunity) may incite an exaggerated or hyperinflammatory response to the bacterial challenge. With an exuberant inflammatory response in overdrive trying to rid the body of periodontal infection, the sequelae are a breakdown of supporting tissues of the periodontium, the classical manifestations of periodontal disease, and the potential for vasculature access of periodontal pathogens and systemic inflammation. Instead of thinking of periodontal disease as a "dentro-centrically defined entity" it is helpful to think of periodontal disease as a chronic, low grade infection.[12] It is multiple, long term, low grade infections which cause on-going inflammation throughout the body which have been implicated in increasing risk for many systemic diseases and conditions. Periodontal infection has the potential to induce systemic inflammation in organs distant to the oral cavity, resulting in increased risk for systemic diseases and conditions many of which are life threatening.

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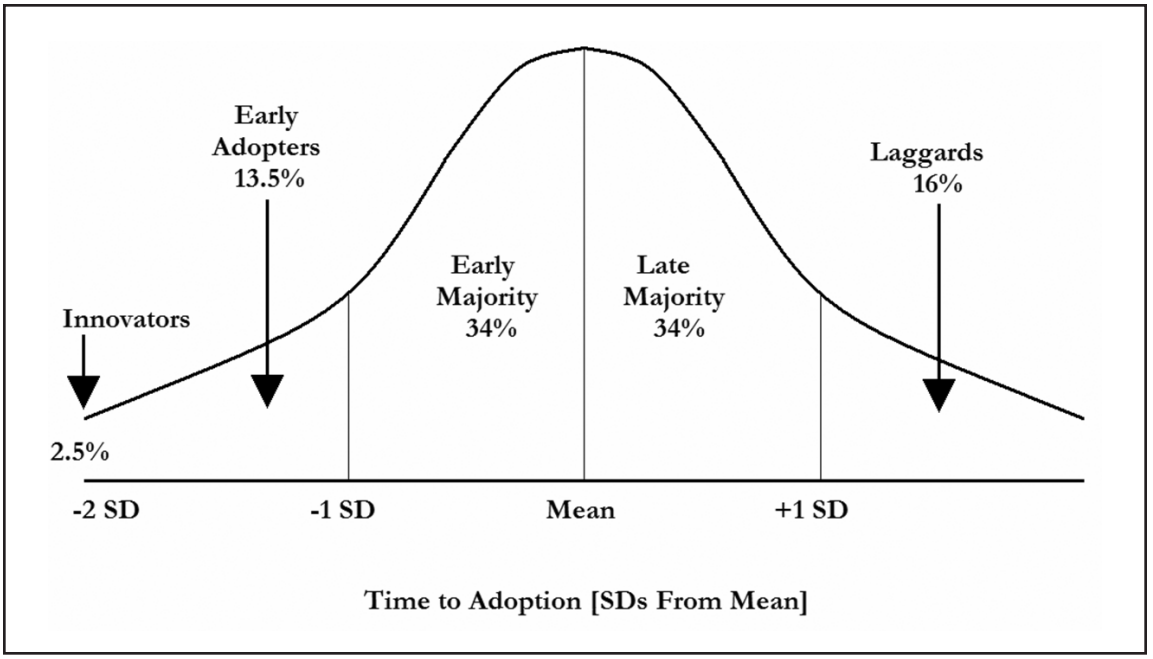

Figure 4. Rogers EM. Adopter Categorization on the Basis of Innovation; Diffusion of Innovations, 4th ed. New York, NY: Free Press.1995

increase the risk for a number of serious diseases and conditions, the real mystery is why do clinicians wait for 5 and 6 mm probing depths to intercept periodontal disease?

Early adopter or someplace in the middle of the bell curve?

Figure 4 illustrates the bell curve associated with the adoption curve of new technologies. The rate of change in healthcare often runs parallel to this curve. Many of us who follow the trends in clinical practice have seen evidence of "early adopters" of this body of science, clinicians in the top 15-16% of the adoption curve. These are individual dental and dental hygiene practitioners who have embraced the significance of periodontal-systemic interrelationships and integrated a progressive approach to early identification of periodontal disease, and appropriate treatment and referral. They have also taken the initiative to increase awareness with the non-dental provider communities. It comes as no surprise that some of these are also SSC members. My guess is that these early adopters will also be the practitioners most likely to benefit from increased referral from the medical community—just another added benefit of rethinking the significance of periodontal disease.

Endnotes

1. Berwick, DM. Disseminating innovations in health care. *JAMA*. 2003;289(15):1969-1975.
2. University of Texas Southwestern Medical Center. National Cholesterol Education Program. Available at: http://www.utsouthwestern.edu/utsw/cda/dept27717/files/97623.html. Accessed July 30, 2007.
3. Report on the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. *Arch Intern Med*. 1988;148:36-69.
4. Pasternak RC, Smith SC, Jr., Bairey-Merz CN, et al. ACC/AHA/NHLBI Advisory on the use and safety of statins. *J Am Coll Cardiol*. 2002;40:567-572.
5. Hujoel PP, White BA, Garcia RI, et al. The dentogingival epithelial surface area revisited. *J Periodontol Res*. 2001;36(1):48-55.
6. Beck JD, Eke P, Heiss G, et al. Periodontal disease and coronary heart disease: a reappraisal of the exposure. *Circulation*. 2005;112 (1):19-24.
7. Dorn BR, Dunn WA, Progulske-Fox A. Invasion of human coronary artery cells

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by periodontal pathogens. *Infect Immun*. 1999; 67 (11):5792–5798.
8. Holmstrup P, Poulsen AH, Andersen L, Skuldbol T, Fiehn NE. Oral infections and systemic diseases. *Dent Clin North Am*. 2003;47 (3):575-598. Review.
9. Li X, Kolltveit KM, Tronstad L, et al. Systemic diseases caused by oral infection. *Clin Microbiol Rev*. Oct. 2000, p 547-558.
10. Garcia RI, Henshaw MM, et al. Relationship between periodontal disease and systemic health. *Periodontol 2000*. 2001;25:21-36.
11. Geerts SO, Nys M, De MP, et al. Systemic release of endotoxins induced by gentle mastication: association with periodontitis severity. *J Periodontol*. 2002;73 (1):73-78.
12. Beck JD, Offenbacher S. Relationships among clinical measures of periodontal disease and their associations with systemic markers. *Ann Periodontol* 2002;7:79-89.
13. Cobb C. Diagnosis and treatment of periodontal disease: a crisis of direction. *Triage*. 2005;1(1):6-14.
14. Flemmig TF. Periodontitis. *Ann Periodontol* 1999;4:32-37.
15. Thomas MV, Mealey BL. Formulating a periodontal diagnosis and prognosis. In: Rose LF, Mealey BL, Genco RJ, Cohen DW, eds., *Periodontics: Medicine, Surgery, Implants*. C. V. Mosby Co., St. Louis, pp. 172-199, 2004.
16. American Academy of Periodontology. Parameters of care. *J Periodontol* 2000;71:847-883.
17. Orban B, Kohler J. Die physiologische Zahn-fleischtasche, Epithelansatz und Epitheltie fenwucherung. *Z Stomatol* 1924 22:353.
18. Brown LJ, Johns BA, Wall TP. The economics of periodontal disease. *Periodontology 2000* 2002;29:223-234.
19. Hein C. Proceedings and consensus opinion from the global oral and systemic health summit; present evidence and future directions. Published as a Special Supplement to *Grand Rounds in Oral-Sys Med*. 2007;1.

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