Surveillance Spotlight...

Current Concepts in Oral–Systemic Health

By Anthony M. Iacopino, DMD, PhD

The International Centre for Oral–Systemic Health is based at the University of Manitoba's faculty of dentistry. Its core mission was developed around interprofessional education, research and practice models where oral health is a critical component of comprehensive patient care.

As part of the educational component of its mission, the centre provides a valuable service to stakeholders in the dental community by scanning the latest research and writings as well as best practices in oral–systemic medicine. The centre is proud to partner with the *JCDA* to provide summaries of contemporary literature and news in oral–systemic health that may affect modern dental practice.

Oral Biofilms: The Origin of Cross-Reactive Antibodies Involved in Systemic Disease?

Oral inflammation in the form of gingivitis and periodontitis is the most prevalent microbial-mediated disease worldwide.[1] The etiology of these chronic inflammatory conditions is the microbial flora residing in dental plaque, an amazingly complex oral biofilm.[2]

During the last 10 years, interest in the connections between oral and systemic inflammation and overall systemic health has continued to grow.[3] In fact, many are now convinced that oral microbial burden and subsequent systemic inflammatory burden are responsible for the connection between periodontitis and chronic inflammatory conditions such as cardiovascular disease, arthritis and diabetes.[4] Despite ongoing research, the mechanism responsible for the linkage between the oral biofilms and systemic inflammatory conditions remains unknown. One of the popular hypotheses regarding this mechanism involves cross-reactivity or molecular mimicry between bacterial antigens in the oral biofilms and self antigens in host tissues.[1]

The most likely candidates in this regard are *Porphyromonas gingivalis* GroEL antigen and heat-shock proteins present in all host cells. It is thought that host antibodies formed against *P. gingivalis* GroEL are reactive against host heat-shock proteins because of their structural similarities.[1] It appears that endothelial cells in the cardiovascular tree and epithelial cells lining joint synovium are particularly vulnerable to damage caused by this antibody cross-reactivity. The cellular damage leads to dysfunction and eventual development of atherosclerotic plaques or rheumatoid complexes. In fact, a correlation between high antibody titres to heat-shock proteins that are cross-reactive with *P. gingivalis* GroEL and morbidity and mortality from atherosclerosis has already been demonstrated.[5] Most individuals with elevated cardiovascular risk have high levels of these antibodies.[6] Interestingly, individuals with no history of periodontitis do not express these antibodies and demonstrate a significantly reduced incidence of atherosclerosis.[7]

A growing body of evidence supports the notion that oral infection is associated with atherosclerosis via molecular mimicry. It is clear that oral infection makes a significant contribution to the total body burden of infection and inflammation. All health professionals are responsible for ensuring that oral infection is kept to a minimum. Effective health policy and interprofessional care must include approaches to reduce oral biofilms.

Biofilms can never be completely eliminated. The pathogenic nature of the dental plaque biofilm can be diminished by reducing the bioburden and effectively maintaining a normal oral flora with oral hygiene procedures that include daily toothbrushing, flossing and rinsing with an antimicrobial mouthrinse. ◆

References

1. Seymour GJ, Ford PJ, Cullinan MP, Leishman S, Yamazaki K. Relationship between periodontal infections and systemic disease. *Clin Microbiol Infect* 2007; 13(Suppl 4):3–10.

2. Thomas JG, Nakaishi LA. Managing the complexity of a dynamic biofilm. *J Am Dent Assoc* 2006; 137(Suppl):10S–15S.

3. Iacopino AM. Maintaining oral health in the aging population: the importance of the periodontal–systemic connection in the elderly. *Grand Rounds Oral-Systemic Med* 2006; 3:25–37.

4. Van Dyke TE. Inflammation and periodontal disease: a reappraisal. *J Periodontol* 2008; 79(Suppl 8):1501–2.

5. Ford PJ, Gemmell E, Timms P, Chan A, Preston FM, Seymour GJ. Anti-P. gingivalis response correlates with atherosclerosis. *J Dent Res* 2007; 86(1):35–40.

6. Ford PJ, Gemmell E, Hamlet SM, Hasan A, Walker PJ, West MJ, and others. Cross-reactivity of GroEL antibodies with human heat shock protein and quantification of oral pathogens in atherosclerosis. *Oral Microbiol Immunol* 2005; 20(5):296–302.

7. Wick G, Perschinka H, Millonig G. Atherosclerosis as an autoimmune disease: an update. *Trends Immunol* 2001; 22(12):665–9.

Dr. Iacopino *is dean and professor of restorative dentistry, and director of the International Centre for Oral–Systemic Health, faculty of dentistry, University of Manitoba, Winnipeg, Manitoba. Email: iacopino@cc.umanitoba.ca*