Surveillance Spotlight...

Current Concepts in Oral–Systemic Health

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The International Centre for Oral–Systemic Health, based at the University of Manitoba's faculty of dentistry, was launched in January 2008. Its core mission was developed around interprofessional education, research and practice models where oral health is a critical component of comprehensive patient care.

The centre will provide interprofessional curriculum and advanced degree programs emphasizing oral–systemic health, interprofessional clinical training experiences with multidirectional screening and referral, implementation of new interprofessional practice models, cutting-edge biomedical and clinical research, analysis of the impact of new practice models on population health and new continuing education approaches for all health professionals and the public.

As part of the educational component of its mission, the centre will provide a valuable service to stakeholders in the dental community by scanning the latest research, best practices and intellectual thought in oral–systemic medicine.

The centre is proud to partner with *JCDA* to provide summaries of contemporary literature and news in oral–systemic health that may affect modern dental practice. The topic of this installment is periodontal disease.

Marijuana and Periodontal Disease

For many years, those of us in the dental community involved with oral–systemic health have recognized that widespread acceptance within the medical community represents the ultimate step in applying advances in oral–systemic science to patient care. The February 2008 issue of the *Journal of the American Medical Association (JAMA)* may be considered a watershed event in the evolution of oral–systemic medicine. It features 2 articles on the relationship between marijuana smoking and periodontal disease and a 1-page summary of periodontal disease that includes a basic diagram of a healthy and diseased periodontium, along with tables listing major etiologic factors, signs/symptoms and prevention/treatment.[1–3]

This is not the first time *JAMA* has published information on the relationship between oral and systemic health. However, it is the first time that readers have been encouraged to become more aware of the causes, presentation and management of periodontal disease. This is significant because it signals a shift within the medical profession toward screening and referral for patients with active disease or at high risk for the development of periodontal disease. It may not be long before we see comprehensive care plans that include thorough integration of the dental team.

The 2 research articles indicate that marijuana smoking increases the risk of developing periodontal disease, even in young adults. In a study of over 900 young adults, heavy marijuana smokers were 1.6 times more likely to have at least mild periodontal disease and 3 times more likely to have severe periodontal disease at a single site compared to those who never smoked marijuana. Heavy smokers were defined as those who used marijuana an average of 40 times per year.

In addition, a significant number of regular marijuana users developed periodontal disease at a younger age than would normally be expected (30% of individuals by age 32). The effects of marijuana use were independent of other risk factors, such as tobacco use and frequency of dental visits. It has been recognized for many years that tobacco use is a major risk factor for periodontal disease, because chemicals in tobacco smoke reduce blood flow in tissues surrounding the teeth and decrease the immune response to oral bacteria. There are similarities between many of the chemical compounds in marijuana and tobacco smoke. However, there is obviously something different in marijuana smoke that will require special attention to the oral health of marijuana users. ◆

References

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Helicobacter Pylori *and Periodontal Disease*

H. pylori is back in the news. This gram negative bacterium infects nearly one-third of the total population in North America and has been implicated as the cause of duodenal and gastric ulcers. Historically, some studies have demonstrated that poor periodontal health, characterized by advanced periodontal pockets (≥ 5 mm) containing an established subgingival biofilm in the form of plaque and calculus, has been associated with *H. pylori* infection in adults (more than 50% of adults with pocket depth of ≥ 5 mm were *H. pylori* seropositive).[1] The periodontal pocket may provide an ideal microenvironment for *H. pylori* because of its unique microaerophilic architecture and urea produced by the subgingival biofilm (*H. pylori* is a urease-producing bacteria). Additionally, gastric reflux provides a constant source of *H. pylori* impregnation of the biofilm. The relationship between *H. pylori* and periodontal status is important because the oral cavity is the primary extragastric reservoir for *H. pylori* and infection by this pathogen appears to involve an oral route, thus oral health status may influence *H. pylori* infection or reinfection. The success rate for gastric eradication of *H. pylori* has been shown to be dependent on the prevalence of *H. pylori* in the oral cavity.

Recent studies now indicate that there may be significant relationships between *H. pylori* and recurrent aphthous stomatitis as well as herpes simplex virus type 1 (HSV-1).[2,3] It was demonstrated that patients with *H. pylori* infection suffered significantly more aphthous outbreaks (more than twice as many) and that these recurrences had significantly more severe symptoms than for non-infected individuals. Over a period of 12 months after *H. pylori* eradication therapy, 63% of these patients demonstrated a significant reduction in both of these parameters. In addition, for those patients who did have recurrences, the time period between these episodes was significantly longer than before treatment.

It is possible that the relationship is based on a *H. pylori*-mediated autoimmune reaction involving T-cell hypersensitivity. HSV-1 seropositivity was also associated with *H. pylori* infection (more than twice the risk for HSV-1 in individuals infected with *H. pylori*). HSV-1 and *H. pylori* share the same route of transmission, both have been isolated from ulcerations of oral and gastric mucosa, and there appears to be a linear association between the seroprevalence of antibodies against the 2 organisms. More studies will be needed to determine how *H. pylori* infection may predispose individuals to HSV-1 infection and if, similar to recurrent aphthous stomatitis, *H. pylori* eradication therapy leads to a reduction of HSV-1 infection. ◆

References

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