Duly 2011 **Nectors and Contract of Contra**

Treating Periodontal Disease Cuts Risk of Systemic Complications

Sponsored as a service to the medical profession by Colgate. Reprinted from Medical Tribune, July 2011. © 2011 UBM Medica.

Hong Kong International Dental Expo And Symposium (HKIDEAS), 3–5 June 2011

Treating periodontal disease cuts risk of systemic complications

Naomi Rodrig

Recent evidence suggests that periodontal interventions aimed at controlling local inflammation may reduce the risk of systemic diseases such as diabetes mellitus (DM) and cardiovascular disease (CVD), which have been associated with periodontal diseases.

According to Professor David Paquette, Dean for Education at Stony Brook University School of Dental Medicine, USA, there is ample study and case-based evidence linking both CVD and DM with periodontal inflammation.

"Periodontal disease, including gingivitis and periodontitis, is characterized by inflammatory and destructive changes such as local erythema, edema and bleeding tendency. Importantly, such chronic infection is a recognized risk factor for inflammatory changes, leading to atherosclerosis," he said.

Indeed, meta-analyses relating periodontal disease and CVD showed a clear positive association, with an odds ratio (OR) of 1.2- 2.85 for coronary heart disease (CHD) and/or stroke in individuals suffering from periodontal disease vs controls.

"For example, in the ARIC [Atherosclerosis Risk In Communities] study, 7.3 percent of periodontal disease patients had CHD, compared with only 4.0 percent of people with no periodontal disease," he said. [*Arterioscler Thromb Vasc Biol* 2001;21:1816-1822]

Subjects with periodontal disease also had a higher prevalence of intima media thickness (IMT) at the 90th percentile than other study participants (15.8 vs 7.7 percent;p=0.0001).IMT is an established surrogate marker for atherosclerotic disease. "Higher levels of CRP [C-reactive protein], a marker of inflammation associated with increased risk of heart disease, are also raised in people with periodontitis," added Paquette.

He hypothesized that potential pathways for periodontal infection-induced atherosclerosis might involve systemic exposure to periodontal pathogens and their products or direct invasion of vascular endothelium, effects on platelets, or autoimmune responses.

"In fact, several studies demonstrated the presence of periodontal bacteria in human atheroma tissue," he said. "For instance, *Porphyromonas gingivalis*

> was detected in 42 percent of atheromas using immunologic staining. Another study that analyzed carotid atheromas with PCR found that 30 percent were positive for *Tanneralla forsythensis* and 26 for *P. gingivalis,* as well as other periodontal pathogens." [J Periodontol 2000;71:1554-1560]

> Intensive treatment of periodontal disease – involv-

ing scaling, root planning, subgingival minocycline microspheres and extraction of hopeless teeth – was shown to improve endothelial function and lower CRP levels, suggesting it may cut CVD risk. [*N Engl J Med* 2007;356:911-920]

Studies that evaluated the link between DM and periodontal disease found that adult DM patients are three times more likely to present with periodontal disease than nondiabetic individuals. [*J Diab Complications* 2006;20:59-68]

"Another study showed that untreated periodontitis increases the risk of poor glycemic control $[HbA_{1c} > 9$ percent], and a meta-analysis demonstrated that diabetics had the same extent but higher severity of periodontal disease compared with nondiabetics, " said

66 Chronic infection is a recognized risk factor for inflammatory changes



66 Dental professionals

need to be engaged in programs

for overall patient wellness.

Paquette. [J Periodontol 1996;67:1085-1093]

Similar trends were observed in children and adolescents with DM, who had

increased gingival inflammation and greater periodontal destruction than their nondiabetic counterparts. [J Clin Periodontol 2007;34:294-298]

A range of periodontal pathogens was also detected in serum samples from prediabetic children in a case-control study.

"The pathogenesis is likely to be related to impaired blood flow, depressed neutrophil function and chronic exposure to advanced glycation end-products," he suggested.

As in the case of CVD, studies and meta-analyses have shown that periodontal treatment with antibiotics can improve glycemic control in type 2 DM patients with periodontitis.

"Early intervention studies suggest that short-term

improvements in surrogate outcomes for systemic conditions – such as CRP for CVD or HbA_{1c} for DM – can

> occur with periodontal or preventive therapies," stressed Paquette. "Dental professionals need to be engaged in programs for overall patient wellness."

> A survey conducted by his team revealed that the majority of dentists are convinced there is strong evidence for the link between periodon-

tal disease and CVD (71 percent) and diabetes (67 percent).

He argued that doctors and dentists need to communicate this to their patients, so that they are aware of the association. "Communication with patients on the relationship between oral and CV health and collaboration between medical professions is crucial. Dentists should promote and encourage continuity of oral care to minimize oral inflammation as part of overall healthcare," he suggested.

Think all toothpastes work the same?

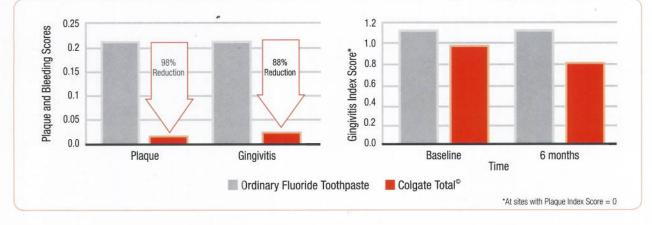
Colgate Total[®] is proven to help prevent gingival inflammation.¹

Colgate Total[®] contains a Triclosan + Copolymer formula that helps fight gingival inflammation in two ways:^{1,2,4}

Kills plaque bacteria for a full 12 hours² to help reduce plaque by up to 98% and gingivitis by up to 88%.³ Shown to directly reduce gingival inflammation⁴

Reduction compared with control

Reduction of gingival inflammation at sites without visible plaque





Refer to Colgate Total® package for approved uses

12-Hour Protection that Helps Prevent Gingival Inflammation. Better Oral Health as Part of Better Overall Health.

1. Panagakos F, et al. J Clin Dent. 2005; 16 (Suppl): S1-S20. 2. Amornchat C, et al. Mahidol Dent J. 2004; 24: 103-111. 3. Garcia-Godoy F, et al. Am J Dent. 1990; 3 (Special Issue): S15-S26. 4. Lindhe et al. J Clin Periodontol. 1993; 20: 323-334, supplemental report on file.