



## **The Oral-Systemic Connection**

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How Oral (Dental) Health Affects Systemic (Medical) Health

**A White Paper for Physicians, Dentists, and Patients**

By

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Restorative and Cosmetic Dentistry

for a Healthy Natural Smile

Temporomandibular Disorders and Dental Sleep Medicine

*\*Suggested Additional Readings following*

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## **A. Introduction**

Only recently have the medical and dental professions realized the intimate connection between oral (dental) health and systemic (medical) health. Miller's "Focal Theory of Infection" (pub. 1891), popular in the early 1900's, held that cavities, periodontal (gum) disease, and dental infections could have long-ranging effects and could cause systemic disease in other organ systems of the body. This concept lost credence and was thoroughly denounced even into the 1980's.

Since the 1990's, evidence has been heavily mounting that there is, indeed, an oral-systemic connection, and that compromised oral health is associated with compromised systemic health. The mature biofilm of periodontal infection stimulates a strong local inflammatory response and allows bacteria and bacterial by-products to enter the bloodstream. It turns out that Miller's 1891 Focal Theory of Infection may be real, causing foci of inflammation and/or infection in distant body systems.

Oral disease has been associated with cardio-vascular disease, heart attack, stroke, Metabolic Disorder, diabetes, complications of pregnancy, kidney disease, cancer, and respiratory illness. Conversely, oral health has been associated with lessened risk of these conditions. Following is more detailed information about these associations, and how treatment of oral disease improves systemic health outcomes; how working in partnership with the physician, dentists can contribute to the health, longevity, and well-being of our patients.

## **B. Connection Between Periodontal Disease and C-Reactive Protein (CRP)**

Medical evidence has now established connections between oral infection, the resultant inflammation, and distant inflammatory disease. Death and phagocytosis of oral bacteria (especially gram-negative bacteria) cause formation and release of endotoxins, cytokines, and various groups of immune and inflammatory up-regulators. The various cytokines involved in feedback loops, the coagulation pathway, and the compliment pathway contribute to acute and chronic inflammation, and cause the liver to produce C-Reactive Protein (CRP). hs-CRP levels are a strong indicator of a future cardiovascular event, and current evidence indicates that CRP levels are an indicator of ongoing inflammation, a predictor of future inflammatory disease occurrence, and likely a contributing factor to those ongoing disease processes. Periodontal therapy can decrease CRP levels. Pre-treatment and post-treatment CRP levels should be measured and the level is often seen to drop after our periodontal therapy.

## **C. Periodontitis and Cardiac Disease**

Inflammation, up-regulated by periodontal disease, is the major oral-systemic connection, and systemic inflammation is the silent killer. Periodontal disease is a medical problem with a dental solution.

The "old" view of cardiovascular disease is that of athero-sclerotic plaques "clogging the pipes" of the arteries; narrowing the arteries and obstructing arterial blood flow. Inflammation is now seen as a major factor in atherosclerosis. Inflammation increases oxidation of LDL, increasing fatty plaque in the vessel wall, and thinning of the epithelial lining. These fatty plaques can calcify and further narrow the arterial lumen, or they

can rupture and initiate a blood clot, occluding the lumen, and initiating an infarct of the downstream tissues. Dental disease increases the circulating levels of fibrinogen and white blood cells. Oral bacteria are also found in the atherosclerotic lesions and thrombi, indicating that infection in these arteries is a component of the disease, either directly or as a result, again, of the inflammatory process.

Studies show a strong statistical link between poor oral hygiene, poor dental (periodontal) health, and cardiovascular disease. Patients with heart attack, stroke, and EKG changes have, by study and statistics, higher levels of periodontal disease and the resultant inflammatory burden.

#### **D. Periodontitis and Stroke**

As noted above, studies indicate a strong correlation between the infection and inflammation of periodontal disease. Poor oral hygiene and periodontal disease are risk factors for a CVA stroke involving ischemia or blockage of cerebral blood vessels.

The infectious/inflammatory process outlined above involving the cytokine pathways, coagulation pathways, and complement pathways can cause equivalent damage in both the cardiac and cranial vessels. Treatment of periodontal disease and improved periodontal health are correlated with lessened risk of ischemic stroke.

#### **E. Periodontitis and Pregnancy**

Periodontitis and its related inflammation have been shown to be associated with pre-term delivery and low birth-weight infants. Studies indicate that periodontal infection can lead to placental-fetal inflammatory exposure and fetal inflammation, resulting in pre-term delivery. Pre-term infants face a higher risk of neurological, respiratory, and behavioral problems, learning disabilities, and metabolic abnormalities. Studies also indicate that periodontal infection may have deleterious effects on the growth and development of the fetus and infant, in addition to those caused by pre-term delivery.

Pregnancy complications are associated with the inflammatory factors of systemic, urinary tract, and periodontal infections. Pregnancy complications are shown to be highly associated with higher CRP and cytokine levels associated with periodontal pathogens. The inflammatory mediators may cross the placental barrier, causing fetal development deformities and loss of viability.

While we do not know if treating periodontal disease in pregnant women will improve pregnancy outcomes, we do know that periodontal health is associated with improved pregnancy outcomes and improved fetal health.

#### **F. Obesity and Diabetes**

Studies have shown a strong correlation between periodontal disease and obesity, and that the incidence of periodontitis is 76% higher in obese young adults. Excess adipose tissue actually secretes a variety of cytokines and hormones involved in the inflammatory process, and periodontal disease shares many of the same inflammatory pathways. The adipokines (leptin, resistin, and adiponectin) are active players in the body's

inflammatory response and may aggravate the immunological side-effects of periodontal disease. Similarly, the inflammatory mediators of periodontal disease may aggravate the factors leading to obesity.

Periodontal disease is also highly associated with Diabetes and Metabolic Disorder, and studies indicate that patients with periodontal disease have greater difficulty in controlling their blood sugar levels.

The inflammation of periodontal disease appears to be a major contributor to the pathogenesis, complications, and poor metabolic control of diabetes. The metabolic and inflammatory pathways initiated by the periodontal infection appear to adversely affect pancreatic beta-cell health, insulin-resistance, blood-sugar control, and HgA1c levels. Periodontal infection also increases the incidence and severity of Metabolic Syndrome (obesity, lipid abnormalities, hypertension, hyperglycemia, diabetes/insulin disorder). Metabolic Syndrome appears to be worsened by the inflammation of periodontal disease and the formation of Advanced Glycation End-products (AGEs) during periods of hyperglycemia and oxidative stress. These and other inflammatory mediators are shown to cause tissue destruction associated with Diabetes and Metabolic Syndrome. Treatment of periodontal disease is improved with proper treatment of diabetes, and diabetes treatment improves periodontal outcomes.

## **G. Kidney Disease**

Chronic Kidney Disease (CKD) is more common in adults who are partially or fully edentulous. Research suggests that the same inflammatory response which causes the bone loss and tooth loss of periodontal disease also causes the damage of CKD.

Adults with tooth loss and periodontal disease have twice the incidence of kidney disease; overlapping inflammatory pathways are the common factor.

## **H. Respiratory Problems and Lung Disease**

Recent research implicates the mouth and throat as sources of respiratory infections. Normal oral bacteria can be aspirated and cause lung infections; this appears to be increased in the presence of periodontal infection and resultant increase in the number and types of pathogenic bacteria present. Chronic Obstructive Pulmonary Disease (COPD) also decreases the clearance of bacteria from the lungs and increases the severity of the problem. Lung disease is correlated with the clinical, bacterial, and inflammatory markers of periodontal disease. Periodontal disease inflammatory pathways, outlined previously, may also initiate and aggravate inflammation and swelling of the lung tissues.

## **I. Cancer**

Statistically, patients with periodontal disease have a 63% increase in pancreatic cancer, 33-36% increase in lung cancer, 50% increase in kidney cancer, and a 30+% increase in blood cell cancers. Head and neck cancers are much more common in patients with periodontal disease, and men with periodontal disease showed a 14% overall increase in cancer rates. University studies also show a correlation between H. pylori (possibly of oral

origin) and stomach cancer. Incidentally, Human Papilloma Virus (HPV), when found orally and genitally is a strong predictor/cause of oral and cervical cancers.

## **J. Osteoporosis**

University studies show a link between periodontal disease and osteoporosis. The low bone density of osteoporosis is definitely associated with periodontal bone loss and tooth loss; all increase with advancing age, though the correlation is independent of age. Tooth loss from periodontal disease correlates with menopause in women, osteopenia (decreased bone density and possible precursor to osteoporosis), use of oral contraceptives, and increased periodontal infection.

Treatments for osteoporosis include decreased use of birth control pills, post-menopausal hormone replacement therapy, calcium/vitamin D therapy, and dental periodontal therapy. Oral therapy includes dental scaling to remove bacterial deposits, hygiene-access surgery, localized antibiotic therapy, and systemic drug therapy. Research is ongoing to determine the cause and effect relationship between osteoporosis and periodontal bone loss, focusing on common inflammatory pathways and related changes in bone and calcium metabolism.

## **K. Gastric Ulcers**

Periodontal disease, periodontal bone loss, and tooth loss are associated with gastric ulcers. Investigators have found that the ulcer-causing bacterium, *Helicobacter pylori* (*H. pylori*), is much more common in the dental plaque of patients with gastric (peptic) ulcers than in those without ulcers. *H. pylori*, associated with both stomach ulcers and stomach cancer, “was significantly more common in the stomachs of patients with gum disease than in those without the condition.” (Cowen). *H. pylori* is found in the deeper pockets associated with periodontal disease and in the stomachs of patients with gastric ulcers. Infection from the oral cavity appears to be a causative factor, and gastric ulcer treatment should include treatment of any periodontal disease present.

## **L. Arthritis**

Patients with rheumatoid arthritis (RA) have eight times the incidence of periodontitis compared to non-RA patients. Periodontitis and RA are both inflammatory diseases, sharing common inflammatory pathways. Treatment of the infection and inflammation of periodontal disease improved the signs and symptoms of RA, as reported in a recent study by Case Western University and University Hospitals of Cleveland. Older studies had shown that antibiotics given to treat the RA improved the arthritis condition, but actually treated the periodontitis.

Both RA and periodontitis inflammatory pathways involve Tumor Necrosis Factor-alpha (TNF-alpha) and when RA/periodontitis patients were treated with periodontal tooth-scaling therapy and/or anti-TNF-alpha drug therapy, improvement in RA symptoms was seen in those patients receiving periodontal therapy and in those receiving the drug; greatest improvement occurred in those patients receiving both. Control of periodontitis-associated inflammatory mediators, by either means, improved the RA.

Non-smokers with moderate-to-severe gum disease have nine times the incidence of RA compared to non-periodontitis controls and those with periodontitis have higher blood levels of an antibody associated with more severe RA. Antibodies to *P. gingivalis*, a periodontitis-involved bacteria, cross-react to chemical mediators (citrullinated proteins) of rheumatoid arthritis.

Inflammation from periodontitis and from RA share common pathways and common factors. Increase in one aggravates the other; treatment of one improves outcomes in the other.

### **M. Obstructive Sleep Apnea (OSA) and Sleep Disordered Breathing (SDB)**

Obstructive Sleep Apnea, which is treatable with C-PAP and/or dental appliances, can cause or worsen excessive daytime sleepiness, obesity, diabetes, hypertension, stroke, kidney disease, misdiagnosed “ADHD,” bruxism (excessive tooth grinding), headaches and marital disharmony. OSA and SDB are increased with use of alcohol, benzodiazepines, opioids, and Viagra.

Though C-PAP is highly effective, dental sleep appliances have greater compliance and are indicated in those patients intolerant of C-PAP. OSA and SDB cause changes in chemical, oxidative, and hormonal pathways, initiating and worsening many cardiovascular diseases, hypertension, cardiac arrhythmias (especially atrial fibrillation), MI, stroke, heart failure and atherosclerosis. OSA causes repetitive hypoxias with sympathetic stimulation of the carotid bodies and resultant hypertension. Dementia is often a function of OSA.

OSA and SDB are also causative factors in obesity, diabetes type II, increased risk of gestational hypertension, and gestational diabetes. Increased gestational blood pressure, often with no snoring, can lead to fetal agitation. Pregnancy with snoring is often predictive of gestational hypertension. Untreated severe obstructive sleep apnea doubles the 12-year mortality rate in the general population. Newly developed atrial fibrillation is often a function of obstructive sleep apnea.

Anti-depressants cause sedation in one third of patients, and many of these patients are OSA patients. These OSA patients, successfully treated with CPAP or Oral Sleep Appliances, may still be sleepy as a result of taking their anti-depressants.

### **N. Snoring and Sleep Disordered Breathing (SDB)**

Snoring (even “benign” snoring) vibrations have been shown to cause neural damage in the upper airway neurons and muscles, causing decreased airway neuro-muscular reflex, decreased dilator muscle function (not totally reversible, even with treatment), impaired dilation reflex and upper airway collapse during sleep.

Snoring can, therefore, cause progressive obstructive sleep apnea with the damage not reversible, even with treatment. Early snoring and apnea treatment can lessen future apnea and sleep-disordered breathing problems.

Down’s Syndrome patients experience a high (50-80%) rate of sleep-disordered breathing problems.

Pediatric tonsil/adenoid problems and snoring are diagnostic of SDB and predictive of future Obstructive Sleep Apnea.

### **O. Gastro-Esophageal Reflux Disease (GERD)**

Gastro-Esophageal Reflux Disease can cause rapid destruction of the teeth as well as severe metabolic and physiologic problems. Solving this problem from the medical, dental and emotional standpoints is critical for the long-term health and well-being of our patients. (See Section R on Dental Erosion, p.229)

### **P. Headaches And Temporomandibular Disorders (“TMD” or “TMJ”)**

Headaches and facial pain are often the result of clenching, grinding, and bite imbalances. Treatment of “TMD” or “TMJ” can help resolve migraines, tension headaches, neurological problems, equilibrium issues, facial pain and associated psychological/emotional problems.

Nocturnal bruxism (often with headaches, facial pain and jaw pain) is associated with Obstructive Sleep Apnea and evidence indicates it may be the result of CNS-activation of jaw and airway dilator muscles to open the airway.

### **Q. Oral Ulcers: Recurrent Aphthous Ulcers (RAU) and Burning Mouth Syndrome (BMS)**

Recurrent Aphthous Ulcers (RAU) and Burning Mouth Syndrome (BMS) may be the result of many factors, including food sensitivities, chemical (toothpaste, mouthrinse) sensitivities, vitamin/mineral deficiencies, drug reactions, and metabolic changes. RAU share many common causes with burning mouth syndrome and burning tongue syndrome; proper diagnosis can result in appropriate, not merely palliative, treatment.

### **R. Dental Erosion and Systemic Factors**

Dental erosion is loss of tooth structure caused by chemical (usually acid) solutions dissolving tooth structure. The erosion can occur on any or all of the teeth and be the result of intrinsic (body-produced) or extrinsic (food or drink) acid attack. Severe dental changes can result in bite collapse, dental disfiguration, and oral destruction. Intrinsic acid attack may be the result of G.E.R.D., nausea, vomiting from gastro-intestinal and metabolic disturbances; also diseases of the stomach, intestines, pancreas and liver, as well as pregnancy and dysmenorrhea. Gastritis and sinusitis can trigger gagging, vomiting, and G.E.R.D. Many gastro-intestinal, neurological, metabolic, endocrine, and Ob/Gyn disorders can trigger gastric acid reflux. Psychological disorders such as anorexia and bulimia can trigger voluntary or involuntary release of stomach acids into the mouth, as can side-effects of many medications. Many medications such as anti-psychotics, tranquilizers, muscle relaxants, anti-anxiety medications, anti-depressants, and acid-reducers can cause severely decreased salivation (dry-mouth) so that there is not enough saliva to wash away and buffer oral acids; decay and erosion then become rampant.

Sodas, energy drinks, sports drinks, flavored waters, and drinks for metabolite replacement/rehydration (recommended for sports activities, Crohn's Disease, and other metabolic disorders) are usually highly acidic and tooth-destructive. These drinks must be used sparingly, carefully, and with specific protective protocols, which we can provide. Toothbrushing should always be avoided immediately after oral acid exposure to protect temporarily weakened enamel.

It is important to note that careful dental sleuthing can often define the oral pattern (position, surfaces, speed, path and type) of chemical erosion, and determine the origin and cause. Often, diagnosis requires accurate dental models to differentiate erosion vs. bruxism/wear vs. erosion with wear vs. toothbrush abrasion vs. abfractions ("stress corrosion") vs. combination etiologies. We can then refer for proper medical management and provide protective protocols. Definitive dental treatment is usually postponed until the problems are under medical and psychological control.

### **S. Xerostomia (Dry Mouth) with Dental Decay and Erosion**

Xerostomia (dry mouth, decreased salivation) can be the result of age, primary or secondary Sjogren's Disease, rheumatoid problems, and many metabolic/systemic disorders. Many of the current medications for anxiety, sleep, G.E.R.D., muscle problems, hypertension, stress, depression and ADHD can cause decreased salivation and dry-mouth.

The use of multiple medications almost guarantees that many of our patients on medications will experience xerostomia. (Medication change can often reduce the problem severity.) Dry mouth indicates lack of saliva, so there is not enough saliva to wash away or buffer the intrinsic, extrinsic or bacterial acids attacking the teeth. Severe tooth erosion and decay can result, and be aggravated by the use of hard, acidic candies for the dry mouth. This destruction can occur with a 40% loss of salivation; the patient may not notice or report a dry mouth until there is a 70% or greater loss of salivation. We can diagnose xerostomia, recommend oral-protective protocols, and refer back for medical and pharmacological consult.

### **T. Denture Problems**

Ill-fitting and worn dentures can cause bite collapse. Malnutrition can result from decreased protein consumption with increased carbohydrate intake. The loss of jaw and bite support can result in temporomandibular joint ("TMJ") problems and muscle dysfunction with subsequent oral pain, facial pain and headache. Worn dentures can also result in decreased esthetics, patient embarrassment, unwillingness to socialize, isolation, and decreased quality of life. Dementia appears to be aggravated by a bad bite (especially with bad dentures) and the resultant improper brain function. Properly made dentures, often combined with implants, can greatly improve one's outlook, quality of life, and function.

## Suggested Additional Readings

My thanks to Dr. Lee Ostler, Jr. All links were active at time of printing.

### **CONNECTION BETWEEN PERIODONTITIS & C-REACTIVE PROTEIN (CRP):**

**Inflammation, C-Reactive Protein, and Atherothrombosis.** [... There is abundant clinical evidence demonstrating that many biomarkers of inflammation are elevated years in advance of first ever myocardial infarction (MI) or thrombotic stroke and that these same biomarkers are highly predictive of recurrent MI, recurrent stroke, diabetes, and cardiovascular death. In daily practice, the inflammatory biomarker in widest use is high-sensitivity C-reactive protein (hsCRP).... This article ... offers the possibility that other disorders characterized by inflammation, such as periodontal disease, may have an indirect role by influencing the risk, manifestation, and progression of vascular events.] Ridker, P.M., Silvertown, J.D. *Journal of Periodontology* (2008, August), 79(8s), 1544-1551. <http://www.joponline.org/doi/full/10.1902/jop.2008.080249>

### **PERIODONTITIS & CARDIAC DISEASE:**

**Angiographically confirmed coronary heart disease and periodontal disease in middle-aged males.** [There was an association between coronary heart disease and poor periodontal status in the middle-aged males investigated. This association was independent of diabetes and all other cardiovascular risk factors investigated.] Briggs, J.E., McKeown, P.P., et al. *Journal of Periodontology*, (2006, January), 77(1), 95-102. doi:10.1902/jop.2006.77.1.95

[http://www.joponline.org/doi/abs/10.1902/jop.2006.77.1.95?prevSearch=keywordsfield%3AC-reactive\\_protein](http://www.joponline.org/doi/abs/10.1902/jop.2006.77.1.95?prevSearch=keywordsfield%3AC-reactive_protein)

**Cardiovascular disease and the role of oral bacteria.** [...Over the decades our understanding of the pathogenesis of CVD has increased, and infections, including those caused by oral bacteria, are more likely involved in CVD progression than previously thought. While many studies have now shown an association between periodontal disease and CVD, the mechanisms underpinning this relationship remain unclear. This review gives a brief overview of the host-bacterial interactions in periodontal disease and virulence factors of oral bacteria before discussing the proposed mechanisms by which oral bacterial may facilitate the progression of CVD.] Leishman, S.J., Do, H.L., et al. *Journal of Oral Microbiology* (2010, December), 2, 5781. doi:10.3402/jom.v2i0.5781

<http://www.journaloforalmicrobiology.net/index.php/jom/article/view/5781/6549>

**Markers of systemic bacterial exposure in periodontal disease and cardiovascular disease risk: A systematic review and meta-analysis.** [...Periodontal disease with elevated bacterial exposure is associated with CHD events and early atherogenesis (CIMT), suggesting that the level of systemic bacterial exposure from periodontitis is the biologically pertinent exposure with regard to atherosclerotic risk.] Mustapha, I.Z., Debrey, S., et al. *Journal of Periodontology* (2007 December), 78(12), 2289-2302.

<http://www.joponline.org/doi/abs/10.1902/jop.2007.070140>

### **PERIODONTITIS & STROKE:**

**The association between cumulative periodontal disease and stroke history in older adults.** [Based on the results of this study, there is evidence of an association between cumulative periodontal disease, based on PHS, and a history of stroke. However, it is unclear whether cumulative periodontal disease is an independent risk factor for stroke or a risk marker for the disease.] Lee, H.J., Garcia, R.I., *Journal of Periodontology* (2006, October), 77(10), 1744-1754. doi:10.1902/jop.2006.050339

<http://www.joponline.org/doi/abs/10.1902/jop.2006.050339?journalCode=jop>

### **PERIODONTITIS & PREGNANCY:**

**Periodontal diseases and health: Consensus report of the sixth European workshop on periodontology.** [... Adverse pregnancy outcome: The findings indicate a likely association between periodontal disease and an

increased risk of adverse pregnancy outcomes. ...The impact of periodontal therapy must be further investigated.] Kinane, D., Bouchard, P., et al. *Journal of Clinical Periodontology* (2008, September), 35(s8), 333-337.

<http://onlinelibrary.wiley.com/doi/10.1111/j.1600-051X.2008.01278.x/abstract>

***Fusobacterium nucleatum* induces premature and term stillbirths in pregnant mice: Implication of oral bacteria in preterm birth.** [*Fusobacterium nucleatum* is a gram-negative anaerobe ubiquitous to the oral cavity. It is associated with periodontal disease. It is also associated with preterm birth and has been isolated from the amniotic fluid, placenta, and chorioamniotic membranes of women delivering prematurely. Periodontal disease is a newly recognized risk factor for preterm birth. This study examined the possible mechanism underlying the link between these two diseases. ...This study represents the first evidence that *F. nucleatum* may be transmitted hematogenously to the placenta and cause adverse pregnancy outcomes. The results strengthen the link between periodontal disease and preterm birth. Our study also indicates that invasion may be an important virulence mechanism for *F. nucleatum* to infect the placenta.] Han, Y.W., Redline, R.W., et al. *Infection and Immunity* (2004, April), 72(4), 2272– 2279. doi:10.1128/IAI.72.4.2272-2279.2004

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC375172/>

**Intrauterine growth restriction, low birth weight, and preterm birth: Adverse pregnancy outcomes and their association with maternal periodontitis.** [It has been suggested that periodontitis is associated with systemic alterations such as adverse pregnancy outcomes. However, some conflicting results have been reported. This case-control study was conducted to determine the association between maternal periodontitis and preterm birth (PTB), low birth weight (LBW), and intrauterine growth restriction (IUGR)...Results emphasize the importance of periodontal care in prenatal health programs.] Siqueira, F.M., Cota, L.O.M., Costa, J.E. *Journal of Periodontology* (2007, December), 78(12), 2266-2276.

<http://www.joponline.org/doi/abs/10.1902/jop.2007.070196>

**Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease: A randomized controlled trial.** [Pregnant women who receive treatment for their periodontal disease can reduce their risk of giving birth to a low birth-weight or pre-term baby. ...] Lopez, N.J., et al. *Journal of Periodontology* (2002, August), 73(8) 911-924.

<http://www.joponline.org/doi/abs/10.1902/jop.2002.73.8.911>

## **OBESITY & DIABETES:**

**Association of periodontal parameters with metabolic level, systemic inflammatory markers in type 2 diabetes patients.** [Backgrounds: While world-wide evidence tends to prove that diabetes adversely affects periodontal health, there is insufficient clue that periodontitis may aggravate metabolic controlling and systemic inflammation. This study... aims to clarify the relationship of periodontal parameters with metabolic level as well as systemic inflammatory markers in diabetes patients. ...] Chen, L., Wei, B., et al. *Journal of Periodontology* (2010, March), 81(3), 364-371. doi:10.1902/jop.2009.090544

<http://www.joponline.org/doi/abs/10.1902/jop.2009.090544?journalCode=jop>

**Clinical and metabolic changes after conventional treatment of type 2 diabetic patients with chronic periodontitis.** [The aim of this study was to compare the response to conventional periodontal treatment between patients with or without type 2 diabetes mellitus from a clinical and metabolic standpoint. ...] Faria-Almeida, R., Navarro, A., et al. *Journal of Periodontology* (2006, April), 77(4), 591-598. doi: 10.1902/jop.2006.050084

<http://www.joponline.org/doi/abs/10.1902/jop.2006.050084>

**Obesity and periodontal disease in young, middle-aged, and older adults.** [Background: The growing prevalence of increased body weight and obesity in the United States has raised significant public health concerns. ... Conclusions: In a younger population, overall and abdominal obesity are associated with increased prevalence of periodontal disease, while underweight (BMI <18.5) is associated with decreased prevalence...] Al-Zahrani, M.S., Bissada, N.F., et al. *Journal of Periodontology* (2003, May), 74, 610-615.

<http://www.joponline.org/doi/abs/10.1902/jop.2003.74.5.610>

### **KIDNEY DISEASE:**

**Clinical and serologic markers of periodontal infection and chronic kidney disease.** [Background: Chronic kidney disease and its concomitant sequelae represent a major public health problem. Recent data suggest periodontal infection contributes to chronic kidney disease. Methods: This United States population-based study of 4,053 adults  $\geq 40$  years of age investigated the association between chronic kidney disease and clinical measures and serologic markers of periodontal infection. ...] Fisher, M.A., Taylor, G.W., et al. *Journal of Periodontology* (2008, September), 79(9), 1670-1678.

<http://www.joponline.org/doi/abs/10.1902/jop.2008.070569>

### **RESPIRATORY PROBLEMS & LUNG DISEASE**

**Involvement of periodontopathic anaerobes in aspiration pneumonia.** [Increasing evidence has linked the anaerobic bacteria forming periodontopathic biofilms with aspiration pneumonia in elderly persons.] Okuda, K. et al. *Journal of Periodontology* (2005, November), 76(11-s), 2154-2160.

<http://www.joponline.org/doi/abs/10.1902/jop.2005.76.11-S.2154>

### **OSTEOPOROSIS:**

**Periodontal diseases and osteoporosis: Association and mechanisms.** [There is increasing evidence that osteoporosis, and the underlying loss of bone mass characteristic of this disease, is associated with periodontal disease and tooth loss. ... Current evidence including several prospective studies supports an association of osteoporosis with the onset and progression of periodontal disease in humans. ... Both periodontal disease and osteoporosis are serious public-health concerns in the United States. ... This paper reviews the current evidence on the association between periodontal disease and osteoporosis.] Wactawski-Wende, J. *Annals of Periodontology* (2001, December), 6(1), 197-208.

<http://www.joponline.org/doi/abs/10.1902/annals.2001.6.1.197>

**The relationship between bone mineral density and periodontitis in postmenopausal women.** [Skeletal BMD is related to interproximal alveolar bone loss and, to a lesser extent, to clinical attachment loss, implicating postmenopausal osteopenia as a risk indicator for periodontal disease.] Tezal, M., Grossi, S.G., et al. *Journal of Periodontology* (2000, September), 71(9), 1492-1498.

<http://www.joponline.org/doi/abs/10.1902/jop.2000.71.9.1492>

### **GASTRIC ULCERS:**

**Are dental plaque, poor oral hygiene, and periodontal disease associated with *Helicobacter pylori* infection?** [The microorganism *Helicobacter pylori* has been closely linked to chronic gastritis, peptic ulcer, gastric cancer, and mucosa-associated lymphoid tissue (MALT) lymphoma. Despite the current treatment regimens that lead to successful management of *H. pylori*-positive chronic gastritis, the reinfection rate is high. ...] Anand, P.S., Nandakumar, K. *Journal of Periodontology* (2006, April), 77(4), 692-698.

<http://www.joponline.org/doi/abs/10.1902/jop.2006.050163>

### **ARTHRITIS:**

**Is there a relationship between rheumatoid arthritis and periodontal disease?** [Because of several similar features in the pathobiology of periodontitis and rheumatoid arthritis, in a previous study we proposed a possible **relationship** between the two diseases. ... **Conclusions:** The results of this study provide further evidence of a significant association between periodontitis and rheumatoid arthritis. ...] Mercado, F., Marshall, R.I., et al. *Journal of Clinical Periodontology* (2011, June), 72(6) 779-787.

<http://www.blackwell-synergy.com/doi/abs/10.1034/j.1600-051x.2000.027004267.x?journalCode=cpe>

### **TEMPOROMANDIBULAR PROBLEMS:**

**Inflammatory cytokines activity in temporomandibular joint disorders: a review of literature.** [Cytokines are important polypeptides mediators of acute and chronic inflammation. These molecules act as a complex immunological network, ... In spite of some controversial findings, in general high levels of pro-inflammatory

cytokines have been correlated with signs and symptoms of temporomandibular disorders (TMD) such as internal derangement and osteoarthritis. These mediators promote degradation of cartilage and bone joint by inducing release of proteinases and other inflammatory molecules...]. Campos, M.I.G., Campos, P.S.F., et al. *Brazilian Journal of Oral Sciences* (2006, July-September), 5(18).

[http://www.fop.unicamp.br/brjorals/temp2/c18\\_Art1\\_inflammatory.pdf](http://www.fop.unicamp.br/brjorals/temp2/c18_Art1_inflammatory.pdf)

### **DENTAL EROSION & SYSTEMIC FACTORS**

**HIV Infection and Bone Loss Due to Periodontal Disease** [Purpose: The goal of this study was to determine whether HIV infection and/or high-risk behaviors associated with HIV infection are related to alveolar bone loss in a sample of subjects screened at a dental school clinic. ... Conclusion: These results suggest that HIV infection is not related to alveolar bone loss in individuals with high risk behaviors for HIV infection. These results also suggest that previously reported relationships between HIV infection and increased alveolar bone loss may be explained by other factors, such as smoking. Individuals in this study population with risk behaviors associated with HIV infection, smoked at a high rate, and due to the smoking behavior have a high rate of periodontal disease.] Aichelmann-Reidy, M.E., Wrigley, D., et al. *Journal of Periodontology* (2010, June), 81(6), 877-884. <http://www.joponline.org/doi/abs/10.1902/jop.2010.070675>

### **XEROSTOMIA WITH DENTAL DECAY & EROSION**

**Using Probiotics to help patients be proactive: Novel approach can enable the prevention of root caries in a periodontal geriatric population.** [As the US population ages, ...a greater percentage of patients are keeping more teeth. ... [T]eeth retention for a large portion of adults may require some periodontal therapy.... Age- or medication-induced xerostomia diminishes the innate ability of saliva's protective response. Root surfaces are uniquely more susceptible to caries as they are more porous and likely to develop biofilms and, ultimately, dental caries. ... Recently, a new oral probiotic entered the marketplace that can uniquely help prevent root surface decay. By continually inoculating the oral cavity with probiotic bacteria that out-compete naturally occurring *S. mutans*, an environment is created that combats the development of dental caries. ] Oxford, G.E. *Inside Dentistry* (2011, March), 7(3), 96-100. <http://editiondigital.net/publication/?i=61413>