Periodontal Disease 2011

- PDD Model
- Oral-Systemic connection
- Current Relevant research
- Therapy Protocols
- LANAP

“Despite the prevalence of periodontal disease among adults, it is still under diagnosed and undertreated, putting patients at risk for systemic disease.”

American Dental Association 2006

Periodontal Disease and Tooth Loss

- 20 M teeth extracted annually
- 45.9 % 65+ lost ≥6 teeth
- 25% 65+ edentulous

CDC National Center For Chronic Disease Prevention and Health Promotion; Oral Health Resources; October 27, 2006

The Silent Epidemic

74% - Periodontal Infection AGD 2002
80% - Mild to Moderate ADA 2006
90% - Age 65-64: Moderate - Severe AAP J Perio. Aug. 2005, 1408

The Silent Epidemic
David Satcher MD, Surgeon General

NZ Our Oral Health 2009

- Adults aged ≥18
- 10% are edentulous
- 33.5% have pockets >4mm, 10.5% > 5mm and 5.1% >6mm
- 49.9% have attachment loss >4mm, 27.5% > 5mm, and 13.4% >6mm


Significantly underestimated

“Despite the prevalence of periodontal disease among adults, it is still under diagnosed and undertreated, putting patients at risk for systemic disease.”

American Dental Association 2006

CHICAGO, Sept. 21, 2010/PRNewswire USNewswire. CDC

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Current Model of Periodontitis

Fibroblast growth factor 2 (FGF-2)

“Proposed mechanisms: 1. stimulated cell death of bone lining cells reducing the number of precursor cells available to form new bone; 2. cytokines inhibit osteoblast differentiation from precursors; 3. inflammatory mediators reduce production of bone matrix. This leads to an uncoupling of osteoblasts from the repair process resulting in net bone loss.”

Periodontal Pathogenesis

- Local factors
- Host susceptibility, genetics
- Environmental factors
- Nutritional deficiencies
- Tobacco/smoking
- Medications
- Age

Pathogenicity is a direct result of local factors:

1. Proteolytic enzymes
2. Toxins such as lipopolysaccharide
3. Cytokines from lymphocyte, macrophages

PDD is a biofilm disease mediated by gram negative bacteria and coupled with a host hyper-inflammatory response.

1. Porphyromonas gingivalis
2. Bacteroides forsythus
3. Treponema denticola
"The bacteria associated with periodontal diseases are predominantly gram-negative bacteria and may include A. actinomycetemcomitans, P. intermedia, B. forsythus, C. rectus, E. nodatum, P. micros, S. intermedius, and Treponema."


Host Susceptibility

- Everybody has the pathogens
- 10% are high risk
- Lifelong chronic

"These findings confirm the hypothesis that complex interactions between the microbiota and host genome are at the basis of susceptibility to periodontal disease. These findings represent a useful model to study the pathways and mechanisms of host-pathogen interactions in inflammatory diseases."


Genetics contribute to oral health differences

"We used quantitative genetic modeling, based on the genetic similarity of identical and non-identical twins, to calculate the most probable model for both filled teeth and gingival bleeding. The models revealed a strong genetic component behind the number of teeth in males (49%) and females (68%) and a weaker genetic component affecting gingival bleeding being similar for males and females (32%)."


Relationship to PVD

- PDD 2.7 times greater risk for CVD
- Evidence supports relationship between PDD and PVD, Cause/Effect?
- Oral/Systemic Connection!

"Three hundred and eighty-nine subjects from 55 pedigrees were studied. Saliva samples were collected from all subjects, and DNA was extracted. Family similarity group 5 gene C (FAM5C) mRNA expression was significantly higher in diseased versus healthy sites, and was found to be correlated to the IL-1β, IL-17A, IL-4 and RANKL mRNA levels. This study provides evidence that FAM5C contributes to aggressive periodontitis."

"Periodontitis has been found to contribute to systemic inflammatory burden including the elevation of C-reactive protein (CRP) in the general population. Atherosclerotic complications including MI and stroke are the primary causes of mortality in the ESRD (end stage renal disease) population and, the best predictor of all cause and cardiac death in this population is CRP. Consequently periodontitis may be a covert but treatable source of systemic inflammation in the ESRD population."


"The prevalence of periodontitis and cardiovascular disease is high. Associations between periodontitis and CVD have been reported in many studies when controlling for confounders. Thus physicians caring for subjects at CVD risk should consult with dentists/periodontists."


"There is strong evidence from cross-sectional studies that plasma CRP in periodontitis is elevated compared to controls. There is modest evidence on the effect of periodontal therapy in lowering levels of CRP."


"Patients on statin medication exhibit fewer signs of periodontal inflammatory injury than subjects without the statin regimen. Periodontal Inflammatory Burden Index PIBI provides a tool for monitoring inflammatory load of chronic periodontitis."


"In the present study, PD lesions predicted presence of CAD stenosis in patients with cardiovascular risk factors. PD severity was correlated to angiographic extent of coronary lesion independent of inflammatory status. Those results suggest that these patients might benefit from an intensive periodontal therapy to prevent CAD progression."


"We examined the levels of antibodies against P gingivalis, A actinomycetemcomitans, T forsythia, T denticola and CRP in 518 men with history of MI. No single bacterium but rather combinations were related to increasing relative risk for MI independent of known cardiovascular risk factors."

"It is concluded that *P. gingivalis* increased the production of IL-18 and CRP in human umbilical vein endothelial cells in a dose and time dependent manner. IL-18 and CRP may be involved in the regulation of inflammation both in periodontal disease and formation of atherosclerotic plaque in cardiovascular disease."


"There was an association between advanced periodontitis and elevated CRP levels as measured at two time points at a 10 year interval in the 60-70 year-old European males investigated. This association was adjusted for various cardiovascular risk factors. There was also an association between high levels of tooth loss and High CRP in the men studied."


"Although the strength of the reported associations is modest, the consistency of data across diverse populations and a variety of exposure and outcomes variables suggest that the findings are not spurious or attributable only to the effects of confounders. Analysis of limited data from interventional studies suggests that periodontal treatment generally results in favorable effects on subclinical markers of atherosclerosis, although such analysis also indicates considerable heterogeneity in responses."


"Recent evidence suggests that strain variation in the serum IgG response to *Porphyromonas gingivalis* occurs in periodontal disease and cardiovascular disease. A significant interaction effect between periodontal status and CV status for antibody levels...providing further support for the role of the immune response to *P. gingivalis* in the relationship between PDD and CV."


"Cancer

• Oral/Systemic connection?"

"Several studies have reported associations between periodontal disease or tooth loss and risk of oral, upper gastrointestinal, lung and pancreatic cancer in different populations. In a number of studies, these associations persisted after adjustment for major risk factors, including cigarette smoking and socioeconomic status."


"Thus, smoking is associated with specific structural alterations to the lipid-A-derived 3-OH fatty acid profile in saliva that are consistent with an oral microflore of reduced inflammatory potential. These findings provide a much needed mechanistic insight into the established clinical conundrum…"


"Our findings do not indicate an apparent association between periodontitis and incident diabetes; however, additional studies are needed for increased risk."


"There may be an interaction between periodontal disease and some systemic diseases such as diabetes mellitus. It was concluded that periodontal disease may influence the metabolic control of DM-2. Additional studies with larger sample sizes and longer follow up are necessary for a better clarification of this issue."

“After adjusting for age, smoking status, and clinical history of diabetes mellitus, these findings demonstrate a dose-response relationship between BMI and the development of periodontal disease in a population of Japanese individuals.”


“There is emerging evidence of a possible relationship between maternal periodontal diseases as a potential risk factor of adverse pregnancy outcomes, like preterm low birth weight even though not all of the actual data support such hypotheses. Further studies are clearly required to clarify the causes and/or relationships linking pathologic oral conditions and adverse pregnancy outcomes.”


“The results of our meta-analysis indicate periodontal treatment during pregnancy does not reduce the risks of pregnant women experiencing PTB and LBW. Pooled results from high quality RCTs with low bias do not support the continued treatment of periodontal disease in pregnancy to prevent PTB, LBW or both.”


“Maternal periodontal disease with systemic inflammation as measured by C-reactive protein is associated with an increased risk for preeclampsia.”


“Evidence for the link between periodontal disease and rheumatoid arthritis comes from the commonality of their pathogenesis and clinical presentation. Both diseases involve inflammatory mediators, including IL-1, IL-6, and TNF-alpha, among others. These patients will likely be unaware that perio treatment may result in improvement in their RA symptoms.”

AAP Risk Assessment
Approved by the Board of Trustees of the AAP October 2007

Risk Factors
- Certain etiologic microorganisms
- Proportion of pocket probing depths >6mm
- Extent and severity of alveolar bone loss
- Hygiene, Age, gender, gingival bleeding
- Positive family history
- Poorly controlled diabetes
- Smoking

PDD Diagnosis
2 AAP Type 0-4

AAP Statement on Diagnosis
Approved by the Board of Trustees of the AAP 2010

Establishing a Diagnosis:
- Medical & Dental history / consultation
- Consideration of risk factors: diabetes, smoking, etc.
- Consideration of diagnostic testing that may include microbiological, genetic, or biochemical assessment

PDD Tests OralDNA Labs
- DNA(bacterial) Testing with MyPerioPath® establishes bacterial risk and can help guide therapy based on causation
- DNA (genetic) Testing with MyPerioID® PST® establishes genetic risk and can help guide therapy based on genetics
- DNA (viral) Testing with OraRisk® HPV identifies HPV status (separate risk factor for oral cancers)

MyPerioID® PST®
- Identifies specific variations in the interleukin-1A and interleukin-1B genes
- Patients are either PST-positive or PST-negative
- The result is for life (test once only)

PST Positive Results
- Increased inflammatory response
- Increased bacterial counts
- 3-7 times more likely to develop severe periodontal disease
- 3-8 times more likely to lose teeth
“Knowledge of the patient’s IL-1 genotype and smoking status will improve the clinician’s ability to accurately assign prognosis and predict tooth loss. Since periodontal diseases are multifactorial, knowledge of the patient’s genotype is more important in predicting future risk than explaining past disease.”


“Genetically determined increased BOP prevalence and incidence observed in IL-1 genotype-positive subjects indicates that some individuals have a genetically determined hyper-inflammatory response that is expressed in the clinical response of the periodontal tissues.”


“Genetically determined significant association

This study demonstrates that the composite IL-1 genotype is significantly associated with the severity of adult periodontitis. It also confirmed that both IL-1 genotyping and smoking history provide objective risk factors for periodontal disease in a private practice environment.”


PST Positive Patients

- Inform patient PST status
- Re-evaluate therapy:
  - Increase recall frequency
  - Consider doxycyline
  - Move to implants
  - Slower orthodontic
- Optimal hygiene
- Chantix/smoking

MyPerioPath®

- Determines the cause of periodontal infections
- Tests for 11 pathogenic species (13 bacteria) that are known to cause periodontal disease
- Results issued securely within 5 business days

MyPerioPath® and MyPerioID® PST® Specimen Collection Process

1. LABEL: Write Patient Name and and DOB on Barcode Label then place Barcode Label lengthwise on Collection Tube
2. SWISH: Ask patient to swish vigorously for 30 seconds
3. EXPECTORATE: Ask Patient to expel into Collection Tube and seal it

Note: Specimen should be collected prior to cleaning (e.g. debridement or rinsing with antimicrobials). Probing and other evaluations OK.
MyPerioPath® Result Report

OraRisk® HPV

• A non-invasive, easy-to-use screening tool to identify the type(s) of oral HPV, a mucosal viral infection that could potentially lead to oral cancer

• Enables clinician to establish increased risk for oral cancer and determine appropriate referral and monitoring conditions

“Oral HPV16 infection was rare in healthy men, especially at younger ages, and was positively associated with current tobacco use. Oral HPV appears to be 10-fold less prevalent than infection at genital sites in men (4% vs. ~40%, respectively). It remains unclear whether this reflects reduced exposure or if the oral region is more resistant to HPV infection compared with anogenital sites.”


OraRisk® HPV test result

AAP Statement on Diagnosis

Approved by the Board of Trustees of the AAP 2010

Establishing a Prognosis and Treatment Plan:

Surgical and non-surgical therapies

Adjunctive restorative treatment

Ongoing reevaluation & maintenance

Periodontal maintenance program
# AAP Classification

- **AAP Case Type 0**
  - Gingival tissue pink, firm, no inflammation, bleeding or exudate
  - Problings 1-3 mm without MG defects
  - Alveolar Bone Loss (ABL) none
  - Furcation Involvement none
  - Tooth Mobility none

- **AAP Case Type 1**
  - Gingival tissue red, inflammation, bleeding on probing, no exudate
  - Problings <4 mm without MG defects
  - Alveolar Bone Loss (ABL) none
  - Furcation Involvement none
  - Tooth Mobility none

- **AAP Case Type 2**
  - Gingival tissue red with inflammation to alveolar crest, moderate bleeding, slight exudate
  - Problings 4-6 mm
  - Alveolar Bone Loss (ABL) slight up to 2mm
  - Furcation Involvement Grade I or II
  - Tooth Mobility slight Class I

- **AAP Case Type 3**
  - Gingival tissue inflamed, generalized bleeding and exudate
  - Problings 5-8 mm with moderate to severe bone loss
  - Alveolar Bone Loss (ABL) moderate 2-4 mm
  - Furcation Grade II-IV
  - Tooth Mobility Class II

- **AAP Case Type 4**
  - Severe destruction of periodontal tissues, gingival inflammation, moderate bleeding and exudate, immediate possible tooth loss
  - Problings >7mm
  - Alveolar Bone Loss (ABL) advanced >4mm
  - Furcation Involvement Grades II-III
  - Tooth Mobility Moderate Class II-III
Therapeutic Options

- Mechanical debridement, SRP
- Antimicrobial Therapy
- Recare Frequency
- OralDNA
- Perioscopy Therapy
- Surgery: pocket reduction, CT grafts, LANAP

Guidelines for Recare

- 6 month recare: Type 0 healthy pedo patients, early Type 1, 2 teens and adults
- 4 month recare: Type 3 adults, ortho patients, pregnant women
- 3 month recare: Type 3 & 4, smokers, diabetes, positive PST

Effectiveness of Scaling Root Planing (SRP) at reducing periodontal disease non-surgically:

Rarely effective at complete removal of plaque and periodontal pathogens. Around 50-70%, at best about 84%. This is a surgical approach to a biofilm disease!

Antimicrobial Therapy

- Carifree Treatment Rinse, CHX?
- Arrestin, Betadine PI 10% in local application
- Amoxicillin 250-500mgx40xqid acute abscess
- Periostat 20mgx180xbid modulate disease between dental visits
- Amoxicillin 250-500mg/Metronidazole 500mgx16xbid each for eight days (no alcohol)

"With Azithromycin treatment, GCF decreased significantly on day 2-7, but increased towards baseline on day 14. The was accompanied by a transient decrease in the content of IL-1B, IL-8, TNF-a and VEGF. These findings suggest azithromycin produces anti-inflammatory effects in gingiva."

Lactobacillus reuteri

The study shows that L. reuteri Prodentis acts synergistically with standard treatment (scaling and root planing, SRP) to significantly reduce probing depth and clinical attachment level which are the two most important parameters to assess the severity of periodontitis.

# Treatment Recommendations

## AAP Case Type 0-4
- **Prophy**
- *Maintain primary oral health*
- *Recare 6-12 months*
- *Monitor for changes in risk factors*
- *Always*

## AAP Case Type 0
- **Prophy**
- *Maintain primary oral health*
- *Recare 6-12 months*
- *Monitor for changes in risk factors*

## AAP Case Type 1
- **Prophy, oral hygiene instructions**
- *Maintain primary oral health*
- *Recare 6-12 months*
- *Monitor for changes in risk factors*

## AAP Case Type 2
- **Prophy, oral hygiene instructions**
- *Gross/fine scale or SRP full mouth*
- *Antimicrobial therapy*
- *Recommend OralDNA test*
- Depending on IL results, modulation
- Perioscopy site specific
- *Recare 3-6 months*
- *Monitor for changes in risk factors*

## AAP Case Type 3
- **Prophy, oral hygiene instructions**
- *SRP full mouth*
- *LANAP laser site specific*
- *Antimicrobial therapy*
- *Recommend OralDNA test*
- Depending on IL results, modulation
- *Recare 3-4 months*
- *Monitor for changes in risk factors*

## AAP Case Type 4
- **Prophy, oral hygiene instructions**
- *SRP full mouth*
- *LANAP laser site specific*
- *Antimicrobial therapy*
- *Recommend OralDNA test*
- Depending on IL results, modulation
- *Recare 3 months*
- *Monitor for changes in risk factors*
Disclosure

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LANAP
Laser Assisted New Attachment Procedure
Millennium MVP-7

Nd:YAG Characteristics

- Transparent to water/cell walls
- Highly absorbed in dark pigment – e.g. P. Gingivalis
- Moderate absorption in hemoglobin
- Not absorbed in connective tissue

Wavelength & Operating Parameters

- FR Pulsed Nd:YAG laser 1,064nm
- Free Running Pulsed microseconds (µsec)
  - @20Hz = 50,000(µsec)/sec
  - 100(µsec) on = 49,900 (µsec) off
- 0.20% duty cycle
Laser Wound Healing

- Remove diseased tissue
- Kills bacteria, remove infection
- Sufficient/stable clot
- Retard epithelium growth
- Less inflammation
- Slower histologic healing

PDD Pathogens

"P. gingivalis resides, replicates and persists within epithelial cells, macrophages, dentin tubules and calculus."


"Kill rate for P. gingivalis by Nd:YAG laser is 16 times greater than continuous wave, gated, pulsed 810 diode laser. The Nd:YAG killed P. gingivalis to a depth of 2 mm into the tissue while the CW/gated/pulsed 810 diode produced a surface effect only."


LANAP

"All LANAP-treated specimens showed new cementum and new connective tissue attachment in and occasionally coronal to the notch, whereas five of the six control teeth had a long junctional epithelium with no evidence of new attachment or regeneration. There was no evidence of any adverse histologic changes around the LANAP specimens."

"In vitro calculus removal was faster with DIs, followed by HIs and PIs. More residual calculus was found with the DIs; however, the 1% to 3% difference (93.7% clean versus 94.6% clean versus 96.9% clean with DIs, PIs, and HIs, respectively) does not seem to be clinically significant."


"An 8-year retrospective study of the LANAP demonstrated consistent mean pocket depth reduction (40%) and improved bone density (38%)."


LANAP Cases

Before/After

Courtesy Dr. L. Fred Church

Patient E

Courtesy Dr. K. Rathburn

Pocket reduction/bone density

Courtesy Dr. J. Humphrey
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