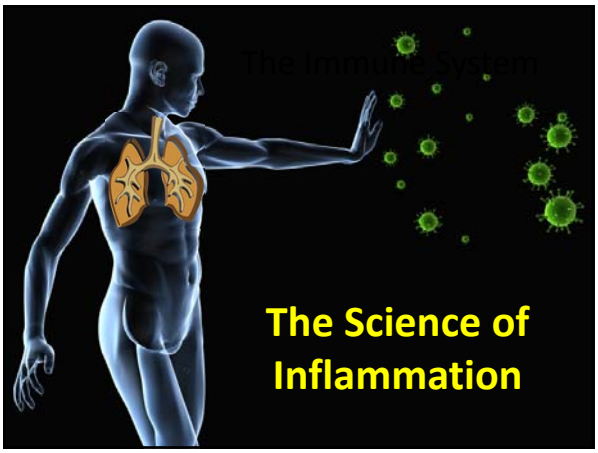
The New Era of Wellness

The Science and Application of the "Mouth-Body" Connection

Inflammation and Interdisciplinary Care

Lee Ostler DDS

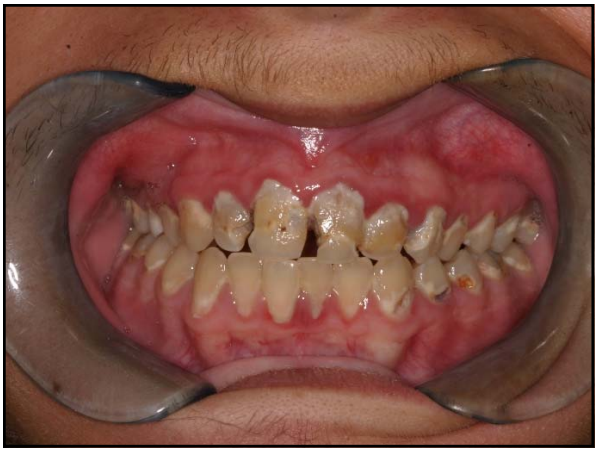


The Concept of Immunity

- Innate Immunity: Defenses against any pathogen
- Adaptive Immunity: Defenses against specific pathogens

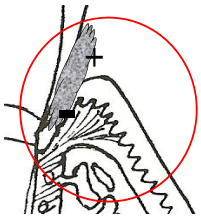
1 Innate (Nonspecific)		2 Adaptive (Acquired)
1st line of defense	2nd line of defense	3rd line of defense

• Physical barriers: intact skin & mucous membranes
• Chemical barriers
• Genetic barriers
• Ciliary hair
• pH (skin & GI)
• Fluids & secretions
• Normal microflora (commensals)
	• Antigen non-specific
	• No memory
	• PAMPs interact w/ PRR (TLRs)
	• Inflammation
	• Phagocytosis
	• Complement
	• Fever
	• Acute phase proteins & antimicrobial substances
		• Natural & artificial
		• Specialized lymphocytes
		  • T cells
		  • B cells
		• Antibodies
		• Long-term – memory
		• Vaccination
		• Immune serum
		• Improves w/ exposures



Periodontal Biofilms

Bacteria, endotoxins and cytokines enter the general circulation through diseased gum tissue and become available to the rest of the body.



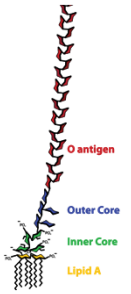
Portal of entry is diseased tissue stripped of protective epithelium barrier.

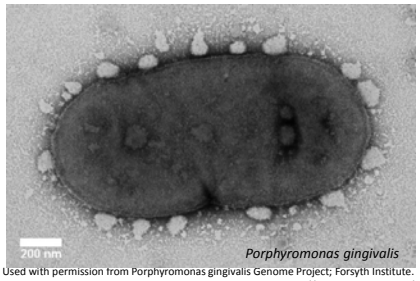
Red Complex Bacteria

1. *Porphyromonas gingivalis*
2. *Tannerella forsythia*
3. *Treponema denticola*

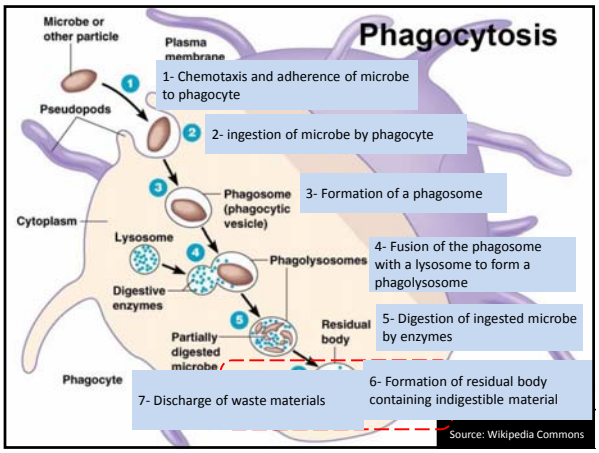
How Gram Negative Bacteria Affect Inflammation

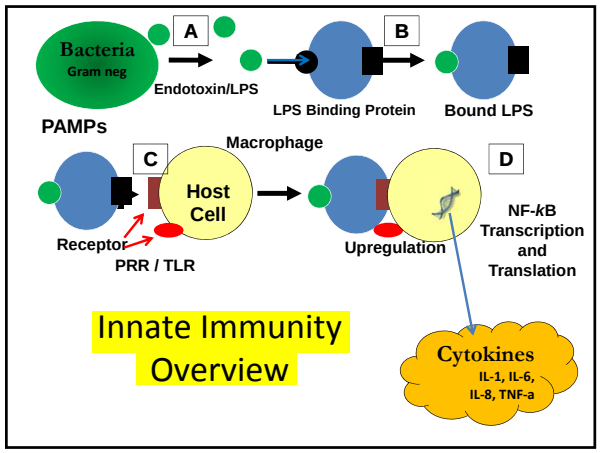
Shedding of LPS remnants in-vivo upon death and lysis/phagocytosis of Gram-Negative Bacteria, and/or release from WBC

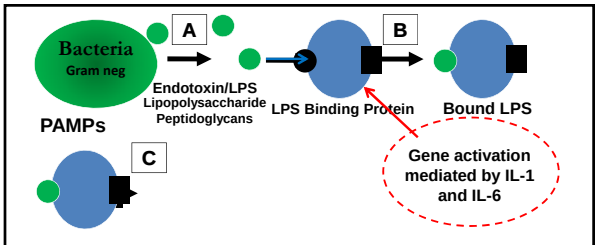




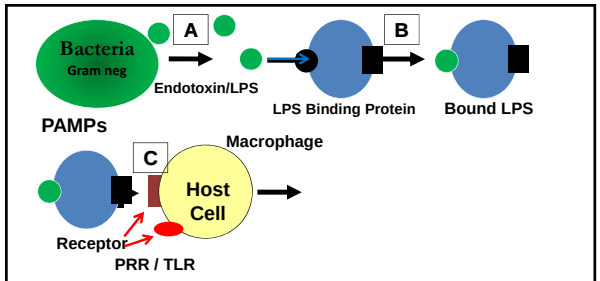
Used with permission from Porphyromonas gingivalis Genome Project; Forsyth Institute. http://www.pgingivalis.org/



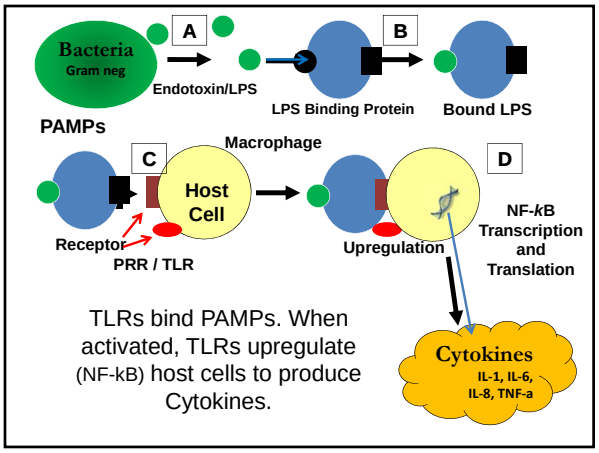




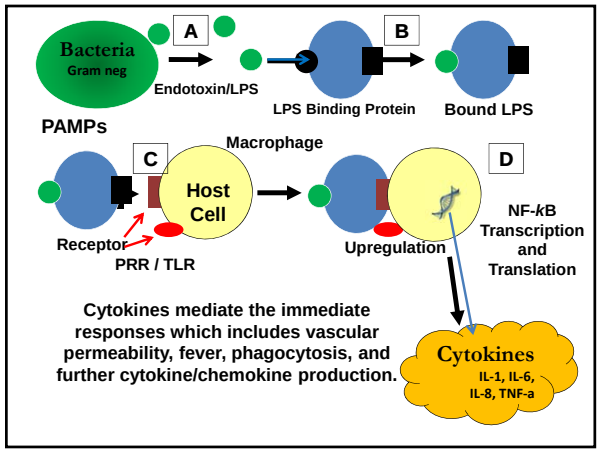
PAMPs - "Pathogen Associated Molecular Patterns"
- Provoke response from immune system
- Molecules common to all pathogens and distinguish-able from host ("self") molecules



PRRs & TLRs
"Pattern Recognition Receptors"
"Toll-Like Receptors"



TLRs bind PAMPs. When activated, TLRs upregulate (NF-kB) host cells to produce Cytokines.



Cytokines mediate the immediate responses which includes vascular permeability, fever, phagocytosis, and further cytokine/chemokine production.

1. Cytokine Feedback Loops
2. Coagulation Pathway
3. Compliment Pathway

Inflammation & Damage

F

Coagulation pathway

Compliment activation

Cytokine feedback loops

D

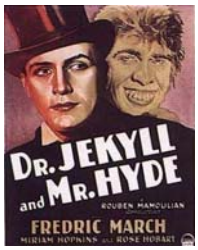
Upregulation

NF-*k*B Transcription and Translation

E

Cytokines

IL-1, IL-6, IL-8, TNF-a



What Is Inflammation?

Good

Acute

Local

Local & Systemic

Chronic

Bad

What Is Chronic Inflammation

Systemic inflammation is the presence of proinflammatory proteins circulating in the blood stream, exerting their effects on tissues of the body generally/systemically.

Good

Acute

Local

Local & Systemic

Chronic

Bad

Whole-Body Inflammatory Burden

Non-Modifiable Risks
- Age & Gender
- Genetics SNPs
- Epigenetics
- Ethnicity / Race
- Family history CVD, stroke, diabetes, cancer

Systemic Factors
- GI conditions
- Respiratory conditions
- Diabetes
- Obesity
- Osteoporosis / Osteopenia
- Rheumatoid arthritis
- Psoriasis
- Lupus
- Alzheimer's
- Cancer
- Depression
- Chronic infections

Increased Inflammatory Burden = Increased Risk for Systemic Disease

Environmental Factors
- Diet / Nutrition
- Stress
- Inactivity
- Smoking
- Excessive alcohol

Periodontal disease
- Specific Pathogens
- IL-1 Genotype

Oral Factors
- Oral Biofilm (Red/Orange)
- Xerostomia
- Altered mouth chemistry
- Faulty dentistry
- Lack of dental care

The CRP Cascade

Inflammatory Challenge

Cellular & Vascular activation

Systemic Plasma-Humoral Immune activation

Adhesion Molecules "Chemokines"

Inflammatory Proteins "Cytokines"

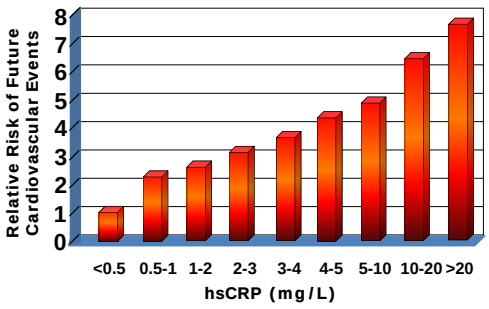
IL-1, TNFα

Interluken-6

Liver

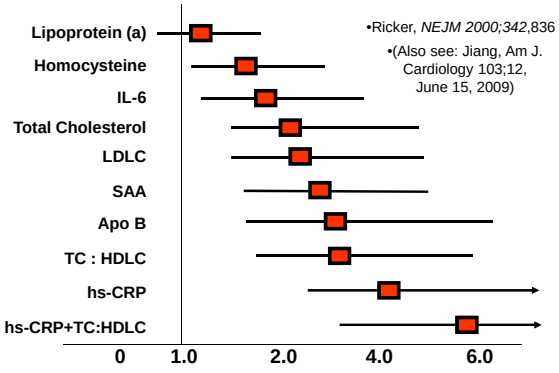
C-reactive protein

Clinical Predictive Value of Very Low as Well as Very High Levels of hsCRP



•(Ridker PM, Rifai N, et.al., *NEJM*, Nov 14, 2002 Vol. 347:1557-1565 No 20.
•Yeh ETH, Willerson JT. *Circulation* 2003;107:370.)

Risk Factors for Future Cardiovascular Event



- Ricker, *NEJM 2000;342*,836
- (Also see: Jiang, Am J. Cardiology 103;12, June 15, 2009)

Consequences of Periodontal Disease

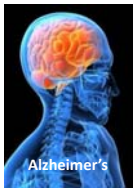
2 → Bacteria and endotoxins in blood spread through body → Systemic Disease

Chronic Bacterial Insult

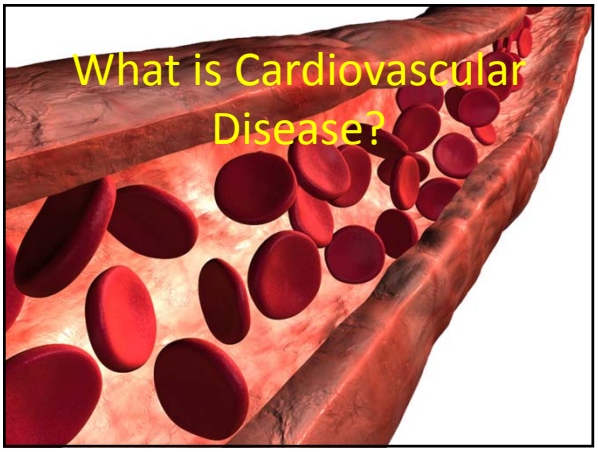
1 → Tissues Break Down → Periodontal Disease

Periodontal Disease is Linked…

- Heart disease & Stroke
- Diabetes
- Alzheimer's
- Rheumatoid arthritis
- Cancer
- Pregnancy complications
- Lung disease
- Osteoporosis
- Obesity
- High blood pressure
- Kidney disease



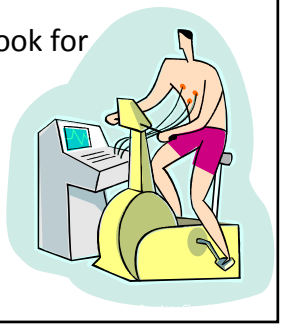
What is Cardiovascular Disease?



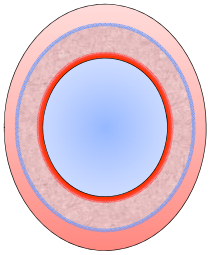
The "Old" View

Stress Test

- Not designed to look for atherosclerosis

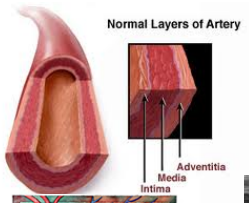


The New View

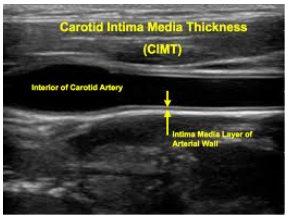


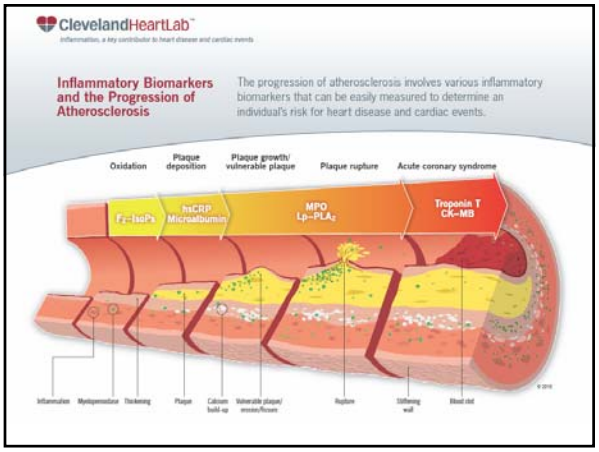
Inflammation is now recognized as the key process in atherosclerosis.

"Arterologist" vs "Lumenologist"



Intimal hyperplasia is the thickening of the intima of a blood vessel and is the universal response of a vessel to injury.





Clin J Dent Res. 2012;15(1):25-9

Lipoprotein-associated phospholipase A2 and serum lipid levels in subjects with chronic periodontitis and hyperlipidemia.

Zhou SY, Xiao WM, Ouyang XY

Department of Periodontlogy, Peking University School, Beijing, People's Republic of China.

Abstract

OBJECTIVE: To evaluate the relationships between clinical periodontal parameters and levels of lipoprotein-associated phospholipase A2 (Lp-PLA2) and lipid profile markers in subjects with or without hyperlipidaemia.

METHODS: Forty chronic periodontitis (CP) subjects with hyperlipidaemia (CP/HPL group), 40 systemically healthy CP subjects (CP group) and 20 systemically and periodontally healthy subjects (control group) were enrolled. The clinical periodontal parameters, the serum concentrations of Lp-PLA2, lipid profiles including total cholesterol (TC), triglyceride (TG), bleeding on probing and WBC counts

CONCLUSION: Elevated level of Lp-PLA2 is associated with periodontal inflammation, indicating that periodontal treatment could reduce the risk of cardiovascular disease in CP subjects with hyperlipidaemia.

was positively associated with bleeding on probing and WBC counts.

CONCLUSION: Elevated level of Lp-PLA2 is associated with periodontal inflammation, indicating that periodontal treatment could reduce the risk of cardiovascular disease in CP subjects with hyperlipidaemia.

Conclusion: Bacteria believed to be important contributors to clinical periodontal disease are positively associated with novel inflammatory markers [Lp-PLA$_2$] recently shown to have prognostic value for incident coronary artery disease.

2689 Periodontal Bacteria and Novel Systemic Inflammatory Markers in INVEST

M. DESVARIEUX, Z. MALLAT, A. TEDGUI, R.T. DEMMER, D.R. JACOBS, Jr., and P.N. PAPAPANOU, Columbia University, New York, NY, USA, Institut National de la Santé et de la Recherche Médicale, Paris, France, University of Minnesota, Minneapolis, USA

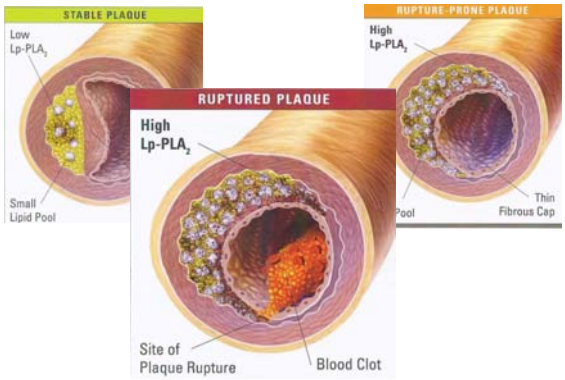
Objective: To investigate the relationship between periodontal bacterial colonization and novel inflammatory markers relevant to incident coronary artery disease.

Methods: The Oral Infections and Vascular Disease Epidemiology Study(INVEST) enrolled subjects aged ≥55 years, in northern Manhattan. Participants in the current analysis (n=598) were 60% female, tri-ethnic (58% Hispanic, 22% Black, 18% White, 2% other) with mean age (±SD) 69±9). In the two most posterior teeth/quadrant eleven periodontal microbes were quantified from dental plaques (n=4,866) in 8 sites/mouth (mesio-lingual in the maxilla and mesio-buccal in the mandible) using DNA-DNA checkerboard hybridization. Secretory-phospholipase A2 (s-PLA2) activity and lipoprotein-associated PLA2 (Lp-PLA2) was assessed systemically from stored plasma samples. We examined the cross-sectional relationship between *Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola* and *Tannerella forsythia* and both s-PLA2 activity and Lp-PLA2 activity. A standardized bacterial burden score was computed by: 1) in transforming laboratory derived bacterial values; and 2) dividing values for each individual bacteria and person, by the population standard deviation for that bacteria. Etiologic burden (EB) was defined by summing standardized values across the four bacteria and represents the absolute combined level of these species, equating standard deviation units (SDU) across bacteria. In separate analyses, we used ANOVA to assess levels of s-PLA2 or Lp-PLA2 across EB tertiles. Analyses were adjusted for age, gender, race/ethnicity, education, diabetes, smoking, systolic blood pressure, HDL-cholesterol and LDL-cholesterol.

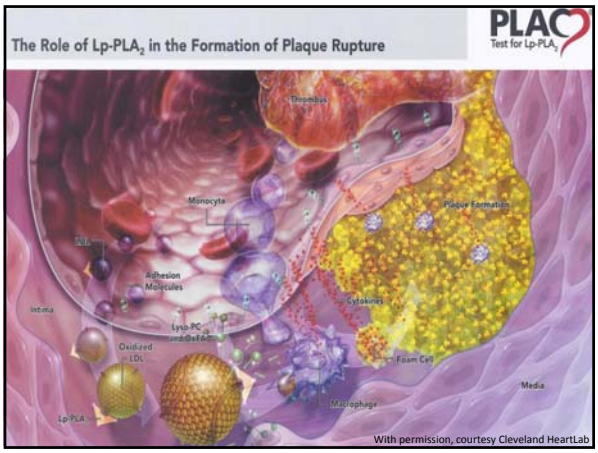
Results: Mean EB was 324±4 SDU and mean s-PLA2 activity and Lp-PLA2 activity values were 0.76±0.56 nmol/min/ml and 30.4±10.9 nmol/min/ml, respectively. s-PLA2 values increased across tertiles of EB, as follows: T1=0.71±0.05, T2=0.74±0.04, T3=0.83±0.05 nmol/min/ml (p for trend=0.12). Lp-PLA2 values were: T1=29.4±0.9, T2=29.1±0.74, T3=32.9 nmol/min/ml (p for trend=0.04).

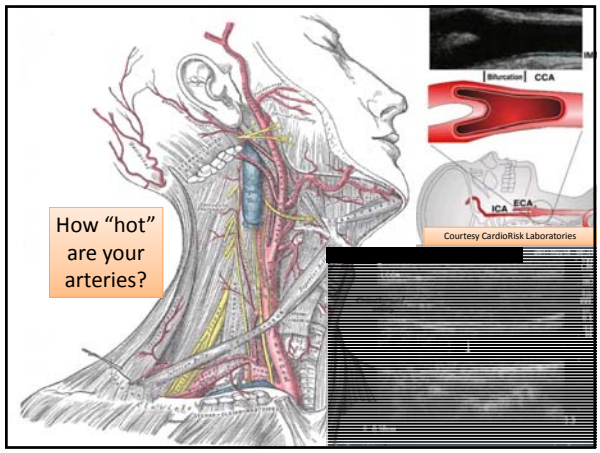
Conclusion: Bacteria believed to be important contributors to clinical periodontal disease are positively associated with novel inflammatory markers recently shown to have prognostic value for incident coronary artery disease.

Desvarieux M, Mallat Z, et al. *IADR* 86th General Session, July 2008. http://iadr.confex.com/iadr/2008Toronto/techprogram/abstract_106763.htm

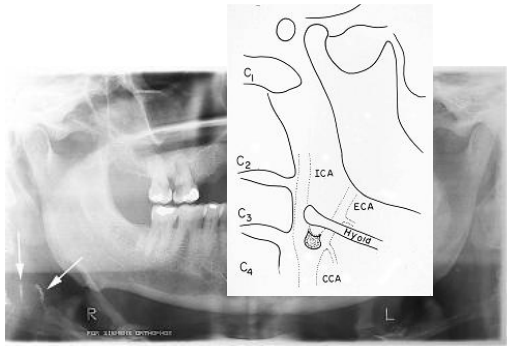


With permission, courtesy Cleveland HeartLab

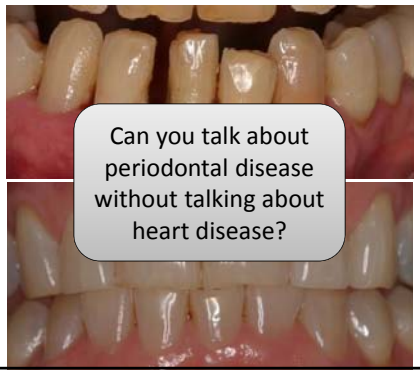




Panoramic Carotid Calcification



What is Periodontal Disease?



What is Periodontal Disease

Can a person have periodontal disease before there is bleeding or tissue breakdown – or any other clinical signs or symptoms?

What comes first?

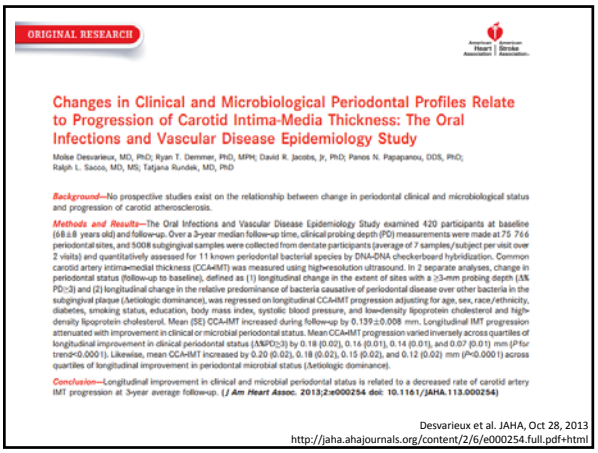
The periodontal pocket or the microbial profile?

Which is worse?

Shallow pockets or deep pockets?

Three Studies

1. Changes in Clinical and Microbiological Periodontal Profiles Relate to Progression of Carotid Intima-Media Thickness; Desvarieux
2. Evaluating Clinical Periodontal Measures as Surrogates for Bacterial Exposure; Demmer
3. Bacterial Signatures in Thrombus Aspirates of Patients with Myocardial Infarction; Pessi



ORIGINAL RESEARCH

Changes in Clinical and Microbiological Periodontal Profiles Relate to Progression of Carotid Intima-Media Thickness: The Oral Infections and Vascular Disease Epidemiology Study

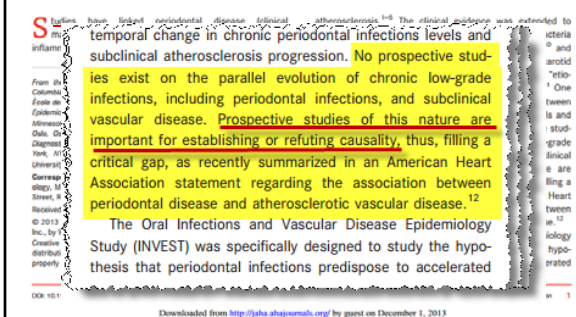
Moïse Desvarieux, MD, PhD; Ryan T. Demmer, PhD, MPH; David R. Jacobs, Jr, PhD; Panos N. Papapanou, DDS, PhD; Ralph L. Sacco, MD, MS; Tatjana Rundek, MD, PhD

Background—No prospective studies exist on the relationship between change in periodontal clinical and microbiological status and progression of carotid atherosclerosis.

Methods and Results—The Oral Infections and Vascular Disease Epidemiology Study examined 420 participants at baseline (68±8 years old) and follow-up. Over a 3-year median follow-up time, clinical probing depth (PD) measurements were made at 75 766 periodontal sites, and 5008 subgingival samples were collected from dentate participants (average of 7 samples/subject per visit over 2 visits) and quantitatively assessed for 11 known periodontal bacterial species by DNA-DNA checkerboard hybridization. Common carotid artery intima-medial thickness (CCA-IMT) was measured using high-resolution ultrasound. In 2 separate analyses, change in periodontal status (follow-up to baseline), defined as (1) longitudinal change in the extent of sites with a ≥3-mm probing depth (ΔN PD≥3) and (2) longitudinal change in the relative predominance of bacteria causative of periodontal disease over other bacteria in the subgingival plaque (Δetiologic dominance), was regressed on longitudinal CCA-IMT progression adjusting for age, sex, race/ethnicity, diabetes, smoking status, education, body mass index, systolic blood pressure, and low-density lipoprotein cholesterol and high-density lipoprotein cholesterol. Mean (SE) CCA-IMT increased during follow-up by 0.139±0.008 mm. Longitudinal IMT progression attenuated with improvement in clinical or microbial periodontal status. Mean CCA-IMT progression varied inversely across quartiles of longitudinal improvement in clinical periodontal status (ΔN/PD≥3) by 0. 18 (0.02), 0.14 (0.01), 0.14 (0.01), and 0.07 (0.01) mm (*P* for trend=0.000 1). Likewise, mean CCA-IMT increased by 0.20 (0.02), 0.18 (0.02), 0.15 (0.02), and 0.12 (0.02) mm (*P*<0.0001) across quartiles of longitudinal improvement in periodontal microbial status (Δetiologic dominance).

Conclusion—Longitudinal improvement in clinical and microbial periodontal status is related to a decreased rate of carotid artery IMT progression at 3-year average follow-up. (***J Am Heart Assoc.* 2013;2:e000254 doi: 10.1161/JAHA.113.000254**)

Desvarieux et al. JAHA, Oct 28, 2013
http://jaha.ahajournals.org/content/2/6/e000254.full.pdf+html



temporal change in chronic periodontal infections levels and subclinical atherosclerosis progression. No prospective studies exist on the parallel evolution of chronic low-grade infections, including periodontal infections, and subclinical vascular disease. Prospective studies of this nature are important for establishing or refuting causality, thus, filling a critical gap, as recently summarized in an American Heart Association statement regarding the association between periodontal disease and atherosclerotic vascular disease.[12]

The Oral Infections and Vascular Disease Epidemiology Study (INVEST) was specifically designed to study the hypothesis that periodontal infections predispose to accelerated

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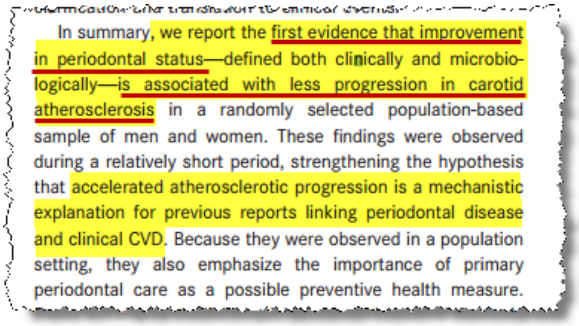

In summary, we report the first evidence that improvement in periodontal status—defined both clinically and microbiologically—is associated with less progression in carotid atherosclerosis in a randomly selected population-based sample of men and women. These findings were observed during a relatively short period, strengthening the hypothesis that accelerated atherosclerotic progression is a mechanistic explanation for previous reports linking periodontal disease and clinical CVD. Because they were observed in a population setting, they also emphasize the importance of primary periodontal care as a possible preventive health measure.

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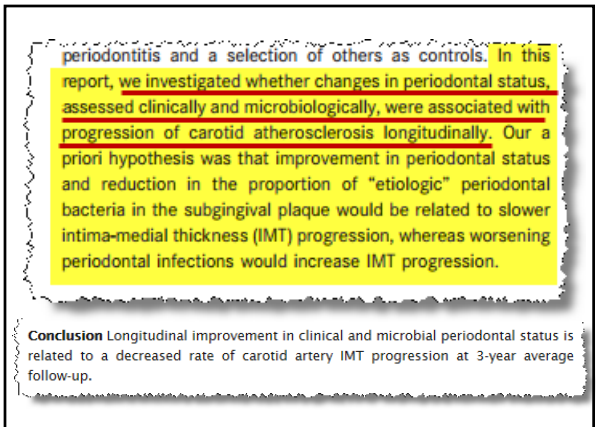


periodontitis and a selection of others as controls. In this report, we investigated whether changes in periodontal status, assessed clinically and microbiologically, were associated with progression of carotid atherosclerosis longitudinally. Our a priori hypothesis was that improvement in periodontal status and reduction in the proportion of "etiologic" periodontal bacteria in the subgingival plaque would be related to slower intima-medial thickness (IMT) progression, whereas worsening periodontal infections would increase IMT progression.

Conclusion Longitudinal improvement in clinical and microbial periodontal status is related to a decreased rate of carotid artery IMT progression at 3-year average follow-up.

http://jaha.ahajournals.org/content/2/6/e000254.short

Discussion / Conclusions

- Etiologic Bacteria = "Red Complex" bacteria
- First evidence – improvement in periodontal status (clinical & microbiological) associated with less progression in atherosclerosis.
- Longitudinal (temporal) change in perio status is concurrent with longitudinal carotid artery IMT progression.
- Dose-response relationship between IMT progression and perio pocket changes and presence of etiologic bacteria.
  - Improvement in perio status (clinical and biological) realized slower IMT progression.
  - Etiologic bacteria considered "causal" (perio), most closely linked to atherosclerotic progression

http://jaha.ahajournals.org/content/2/6/e000254.short

Discussion / Conclusions

- "Pre-clinical" – low threshold perio measures (≥3mm) strongly correlate with etiologic perio bacteria, inflammation and atherosclerotic progression.
- Systemic translation of local infection is more related to bacterial levels than overt clinical disease.
- 3 mm pocket depth should not be assumed to be healthy. "Pre-clinical" periodontal disease cannot be ignored.
- Avg 0.03mm/yr difference in IMT score with deteriorating vs improving perio health status
  - Thresholds of clinical significance - reference:
    - 0.03mm/yr increase in IMT associated with 230% increase risk for coronary events (Hodis et al; progression study)
    - 0.0082mm/yr (Crouse et al; statin study)

http://jaha.ahajournals.org/content/2/6/e000254.short

Discussion / Conclusions

- Study supports the role of high risk bacteria and atherosclerotic vascular disease (ASVD)
- ASVD is improved with effective perio therapy
- BOP is harmful to health – regardless of pocket depth, if there are high risk bacteria present
- Judging perio health based on probing depths is now obsolete from a scientific point of view.

http://jaha.ahajournals.org/content/2/6/e000254.short

Research article

Open Access

Evaluating clinical periodontal measures as surrogates for bacterial exposure: The Oral Infections and Vascular Disease Epidemiology Study (INVEST)

Ryan T Demmer[1], Panos N Papapanou[2], David R Jacobs[3,4] and Moïse Desvarieux[1,3,5]*

Conclusions

Clinical exposure definitions of periodontal disease should incorporate relatively shallow pockets to best reflect whole mouth exposure to bacterial burden.

Demmer et al. Evaluating clinical periodontal measures as surrogates for bacterial exposure: INVEST Study. BMC Med Res Method, 2010,10:2
www.biomedcentral.com/1471-2288/10/2

Discussion / Conclusions

- Low-severity sites not often regarded as clinical disease.
- Periodontal bacterial burden highly correlated with low-severity (≥ 3mm).
  - Perio - etilogic bacteria burden = "Red Complex" (Aa, Pg, TD, Tf)
- B.O.P. strongly assoc with bacterial burden and is more pronounced in shallow than in deep periodontal pockets.
- Low-severity thresholds have strongest correlations with etiologic bacteria.
  - This does not imply that high-severity sites are not pathologic. (Note: high severity thresholds skewed due to lower prevalence, access to care issues with affected study population, & severe sites more predisposed to treatment/removal).

Demmer et al. Evaluating clinical periodontal measures as surrogates for bacterial exposure: INVEST Study. BMC Med Res Method, 2010,10:2
www.biomedcentral.com/1471-2288/10/2

Discussion / Conclusions

- Low threshold definitions of clinical periodontal disease tend to optimize associations with cardiovascular disease biomarkers.
- Highlights importance of subclinical periodontal infection (i.e. "low-severity") in the context of periodontal infection and cardiovascular disease risk.
- Shallow pockets → gingivitis / periodontitis PLUS subclinical pathological processes with systemic effects.
- Shallow sites might be considered as "nascent" disease; (i.e beginning, starting, developing, emerging).

Demmer et al. Evaluating clinical periodontal measures as surrogates for bacterial exposure: INVEST Study. BMC Med Res Method, 2010,10:2
www.biomedcentral.com/1471-2288/10/2

Discussion / Conclusions

"The finding that pocket depth and bleeding on probing definitions performed as well as, and often better than, attachment loss definitions might have been anticipated when considering that pocket depth and bleeding tend to be better markers of current infection while attachment loss better reflects historical disease."

Demmer et al. Evaluating clinical periodontal measures as surrogates for bacterial exposure: INVEST Study. BMC Med Res Method, 2010,10:2
www.biomedcentral.com/1471-2288/10/2

Discussion / Conclusions

While the relative risk for bacterial colonization in deep vs. shallow periodontal pockets is high, the prevalence of deep pockets is often low in epidemiological settings. Therefore, in absolute terms, much of the attributable risk from exposure to pathogenic bacteria would likely occur in relatively shallow periodontal pockets. Specifically, our findings highlight the potential importance of using clinical definitions that include less severe periodontal disease when such disease is viewed as a model of infection in studies of systemic disease risk.

Demmer et al. Evaluating clinical periodontal measures as surrogates for bacterial exposure: INVEST Study. BMC Med Res Method, 2010,10:2 www.biomedcentral.com/1471-2288/10/2

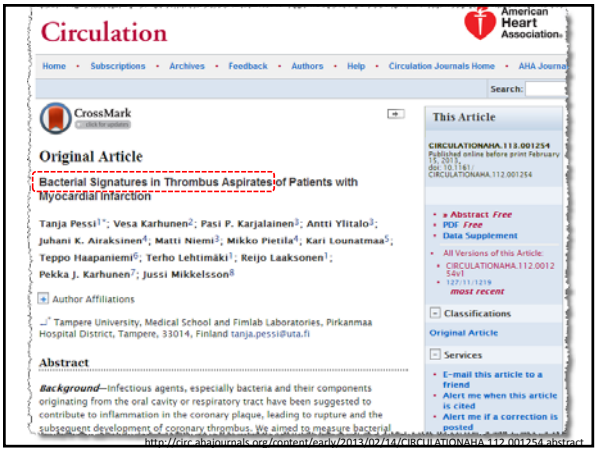
Discussion / Conclusions

"Substantial exposure to pathological periodontal microbiology likely occurs in shallow periodontal pockets that do not yet exhibit commonly accepted clinical signs of frank periodontal disease. …
Results of this nature highlight the potential importance of subclinical periodontal infection in the epidemiological context of periodontal infection and cardiovascular disease risk. Relatively shallow periodontal sites not only have the potential to develop gingivitis and subsequent periodontitis but might also be undergoing subclinical pathological processes that could have systemic effects. Therefore some shallow periodontal sites might actually be considered as nascent disease."

Demmer et al. Evaluating clinical periodontal measures as surrogates for bacterial exposure: INVEST Study. BMC Med Res Method, 2010,10:2 www.biomedcentral.com/1471-2288/10/2

What does this mean?

- Gingivitis & subclinical perio disease should not be trivialized, normalized, or "watched".
  - No more "bloody prophys".
- B.O.P. is disease; is assoc with high levels of etiologic bacterial burden
- Shallow pockets are associated with systemic pathology.
  - Low threshold perio tends to optimize associations with cardiovascular disease biomarkers.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Original Article

Bacterial Signatures in Thrombus Aspirates of Patients with Myocardial Infarction

Tanja Pessi[1*]; Vesa Karhunen[2]; Pasi P. Karjalainen[3]; Antti Ylitalo[3]; Juhani K. Airaksinen[4]; Matti Niemi[3]; Mikko Pietila[4]; Kari Lounatmaa[5]; Teppo Haapaniemi[6]; Terho Lehtimäki[1]; Reijo Laaksonen[1]; Pekka J. Karhunen[7]; Jussi Mikkelsson[8]

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Abstract

Background—Infectious agents, especially bacteria and their components originating from the oral cavity or respiratory tract have been suggested to contribute to inflammation in the coronary plaque, leading to rupture and the subsequent development of coronary thrombus. We aimed to measure bacterial

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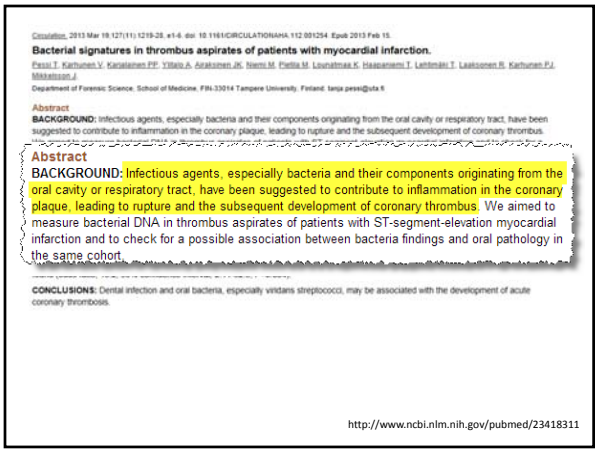
Circulation. 2013 Mar 19;127(11):1219-28. e1-6. doi: 10.1161/CIRCULATIONAHA.112.001254. Epub 2013 Feb 15.

Bacterial signatures in thrombus aspirates of patients with myocardial infarction.

Pessi T, Karhunen V, Karjalainen PP, Ylitalo A, Airaksinen JK, Niemi M, Pietila M, Lounatmaa K, Haapaniemi T, Lehtimäki T, Laaksonen R, Karhunen PJ, Mikkelsson J.

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Abstract

BACKGROUND: Infectious agents, especially bacteria and their components originating from the oral cavity or respiratory tract, have been suggested to contribute to inflammation in the coronary plaque, leading to rupture and the subsequent development of coronary thrombus.

Abstract

BACKGROUND: Infectious agents, especially bacteria and their components originating from the oral cavity or respiratory tract, have been suggested to contribute to inflammation in the coronary plaque, leading to rupture and the subsequent development of coronary thrombus. We aimed to measure bacterial DNA in thrombus aspirates of patients with ST-segment-elevation myocardial infarction and to check for a possible association between bacteria findings and oral pathology in the same cohort.

CONCLUSIONS: Dental infection and oral bacteria, especially viridans streptococci, may be associated with the development of acute coronary thrombosis.

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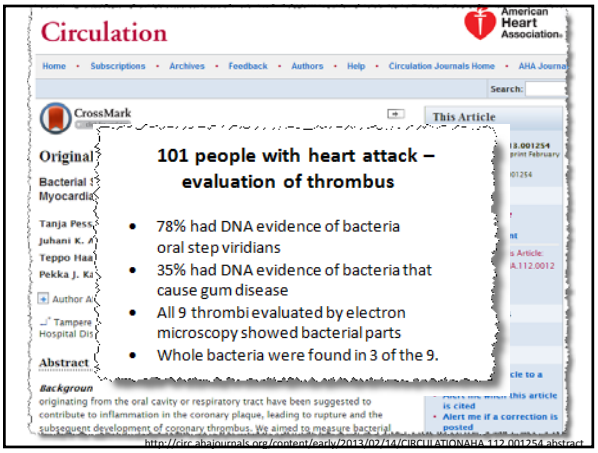
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This Article

101 people with heart attack – evaluation of thrombus

- 78% had DNA evidence of bacteria oral step viridians
- 35% had DNA evidence of bacteria that cause gum disease
- All 9 thrombi evaluated by electron microscopy showed bacterial parts
- Whole bacteria were found in 3 of the 9.

Abstract

originating from the oral cavity or respiratory tract have been suggested to contribute to inflammation in the coronary plaque, leading to rupture and the subsequent development of coronary thrombus. We aimed to measure bacterial

http://circ.ahajournals.org/content/early/2013/02/14/CIRCULATIONAHA.112.001254.abstract

Bacterial signatures in thrombus aspirates of patients with myocardial infarction.

Abstract

BACKGROUND: Infectious agents, especially bacteria and their components originating from the oral cavity or respiratory tract, have been suggested to contribute to inflammation in the coronary plaque, leading to rupture and the subsequent development of coronary thrombus. We aimed to measure bacterial DNA in thrombus aspirates of patients with ST-segment-elevation myocardial infarction and to check for a possible association between bacteria findings and oral pathology in the same cohort.

METHODS AND RESULTS: Thrombus aspirates and arterial blood from patients with ST-segment-elevation myocardial infarction undergoing primary percutaneous coronary intervention (n=101; 76% male; mean age, 63.3 years) were analyzed with real-time quantitative polymerase chain reaction with specific primers and probes to detect bacterial DNA from several oral species and Chlamydia pneumoniae. The median value for the total amount of bacterial DNA in thrombi was 16 times higher than that found in their blood samples. Bacterial DNA typical for endodontic infection, mainly oral viridans streptococci, was measured in 78.2% of thrombi, and periodontal pathogens were measured in 34.7%. Bacteria-like structures were detected by transmission electron microscopy in all 9 thrombus samples analyzed, whole

CONCLUSIONS: Dental infection and oral bacteria, especially viridans streptococci, may be associated with the development of acute coronary thrombosis.

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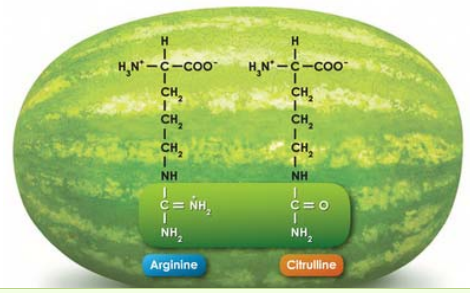
Hippocrates

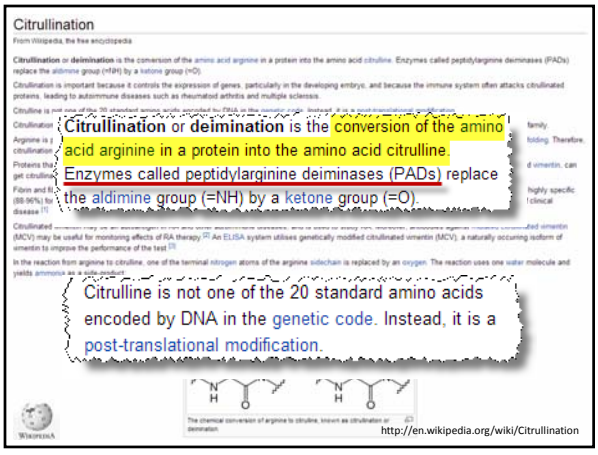
Father Of Western Medicine And Dentistry

Suggested pulling teeth could cure arthritis

Citrullination



Citrulline – an amino acid first isolated from watermelon in 1914. *Citrullus* is Latin word for watermelon.



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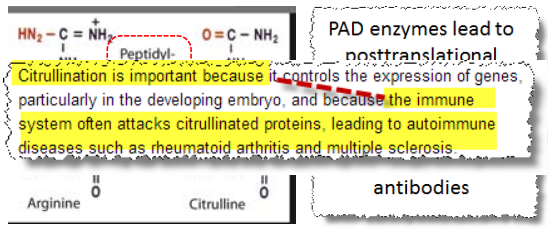
Citrullination



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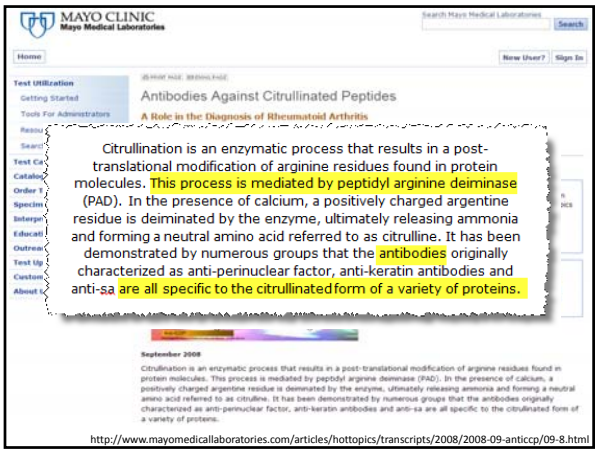

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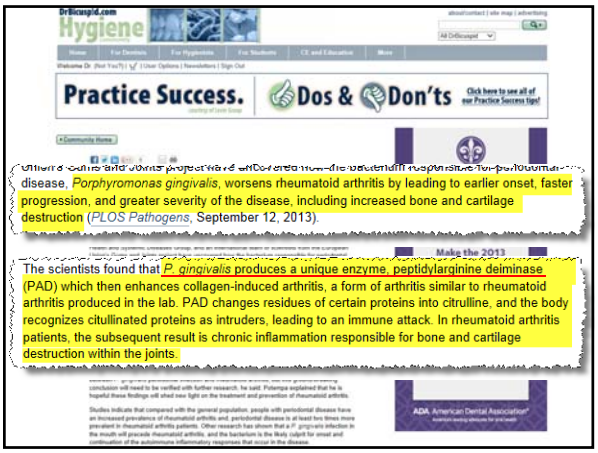

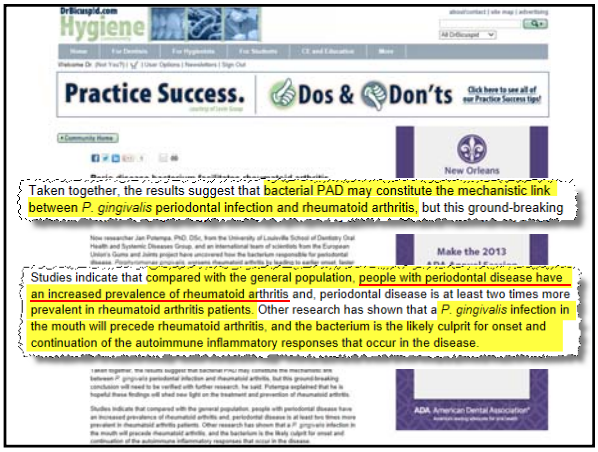

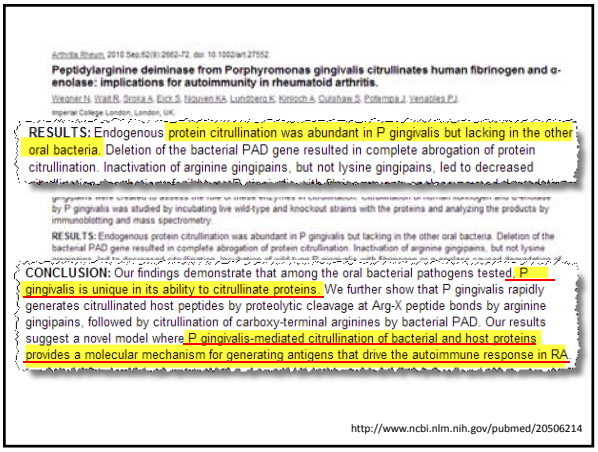

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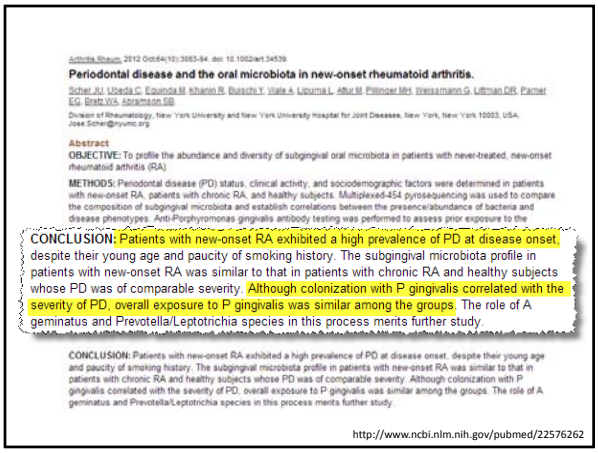

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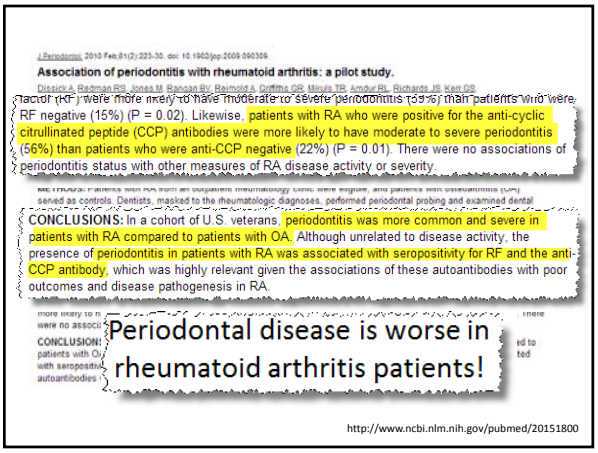

http://www.ncbi.nlm.nih.gov/pubmed/20151800

Identification of oral bacterial DNA in synovial fluid of patients with arthritis with native and failed prosthetic joints.

Abstract

OBJECTIVE: We examined the presence of bacterial DNA in synovial fluids of native or clinically aseptically failed prosthetic joints from patients having periodontal disease and arthritis to determine whether there is bacterial spread from the oral cavity to the joints.

joint. Pooled subgingival plaque samples were collected, followed by clinical periodontal examination. Bacterial DNA was extracted from the collected

RESULTS: Of the 36 patients, bacterial DNA was detected in the synovial fluid samples from 5 patients (13.9%): 2 with RA (1 native and 1 failed prosthetic joints) and 3 with OA (1 native and 2 failed prosthetic joints). Of these 5 patients, 2 were diagnosed with periodontitis and had identical bacterial clones (Fusobacterium nucleatum and Serratia proteamaculans, respectively) detected in both the synovial fluid and the dental plaque samples. Fusobacterium nucleatum was the most prevalent, detected in 4 of the 5 positive samples. No cultures were done and no patients were treated with antibiotics or developed clinical infection.

CONCLUSIONS: The present findings of bacterial DNA in the synovial fluid suggest the possibility of organisms translocating from the periodontal tissue to the synovium. We suggest that patients with arthritis or failed prosthetic joints be examined for the presence of periodontal diseases and be treated accordingly.

http://www.ncbi.nlm.nih.gov/pubmed/22426587

Medical costs fall with gum treatment, study finds

By Alex Nixon

Published: Wednesday, March 27, 2013, 12:01 a.m.

Overall medical costs are lower for patients with rheumatoid arthritis and for pregnant women if they are treated for gum disease, according to a study of claims data released by United Concordia, the dental insurance subsidiary of Highmark Inc.

The findings follow similar results released by the company that appear to show a link between lower overall health costs for people with diabetes, heart disease and stroke and good oral health. The latest study found that people with rheumatoid arthritis who received treatment for gum disease had lower annual medical costs fall $3,964; pregnant women had $2,430 in lower costs.

United Concordia hired Dr. Margorie Jeffcoat, a dentist and dean emeritus of the University of Pennsylvania's School of Dental Medicine, to perform the study of its claims data, the results of which are expected to be published later this year.

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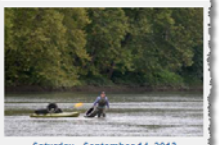

Saturday - September 14, 2013

http://triblive.com/business/businessbriefs/3732619-74/costs-lower-disease#axzz2ezD0TV31

The Science of Inflammation

Medical Problem – Dental Solution

The Oral-Systemic Connection

This is what makes periodontal disease a medical problem, & why physicians and dentists MUST work together to co-manage their patients.