PERIODONTAL MEDICINE

DR.KANCHAN JAGTAP(MDS)

STEVEN OFFENBACHER- FATHER OF PERIODONTAL MEDICINE

Periodontal
(pertaining to the periodontium)

+

Medicine
(treatment of periodontal disease by non-surgical means)

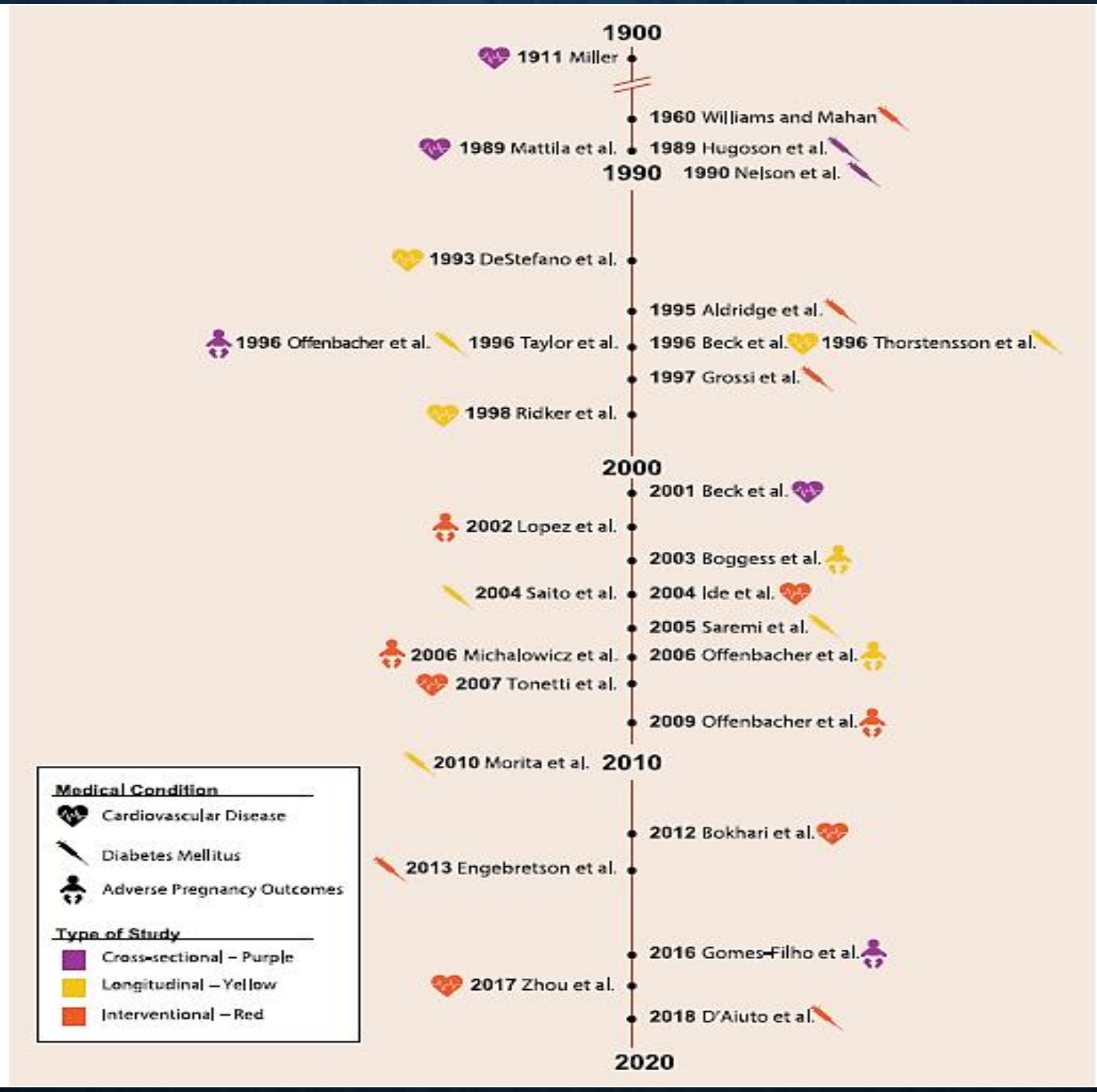
→

PERIODONTAL MEDICINE

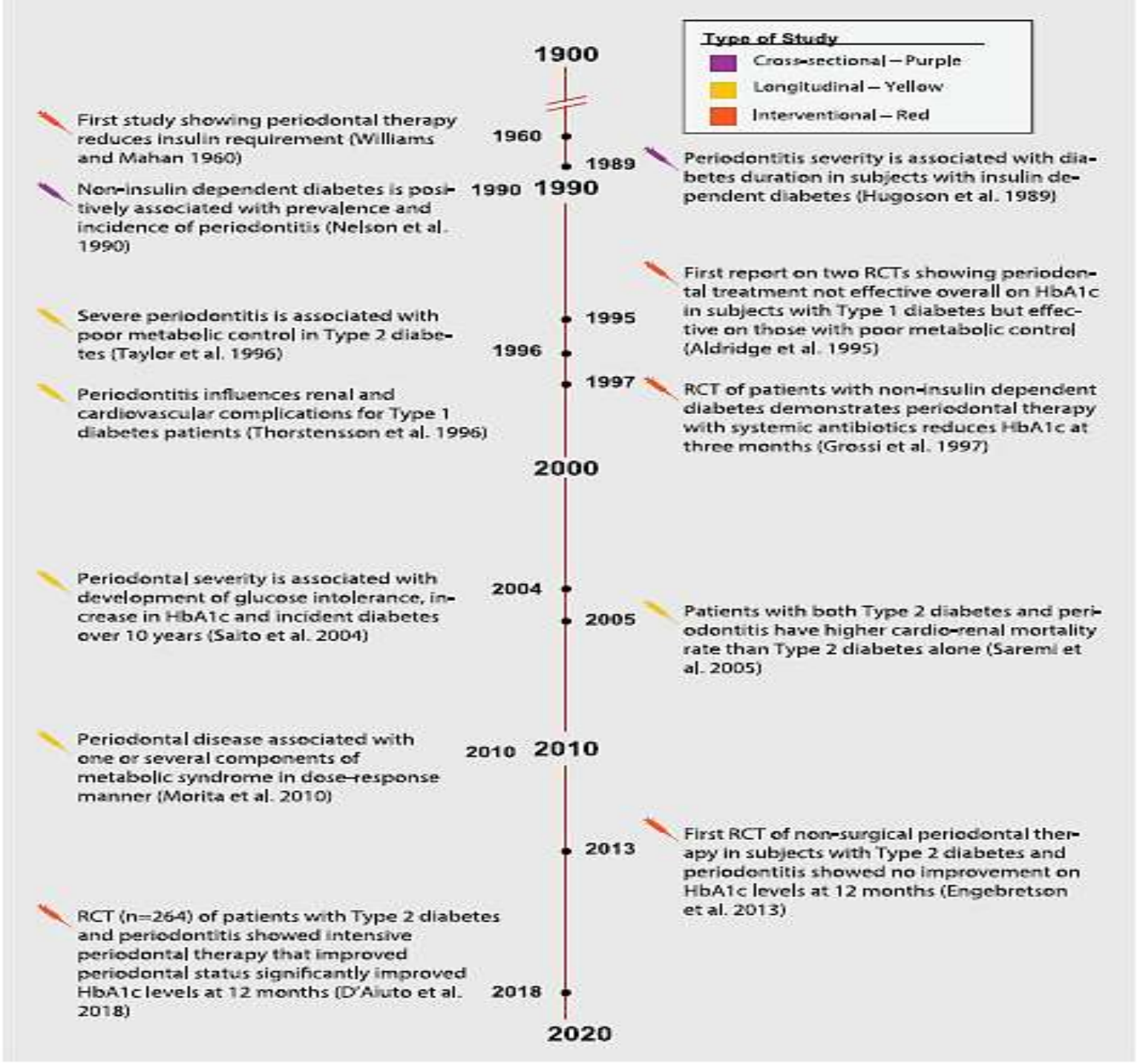
The term "Periodontal medicine" was first suggested by Steven Offenbacher (1996)

Definition-
"Rapidly emerging branch of periodontology focussing on the wealth of new data establishing a strong relationship between periodontal health or disease and systemic health or disease".

HISTORY


1900
1911 Miller
1960 Williams and Mahan
1989 Mattila et al.
1989 Hugoson et al.
1990
1990 Nelson et al.
1993 DeStefano et al.
1995 Aldridge et al.
1996 Offenbacher et al.
1996 Taylor et al.
1996 Beck et al.
1996 Thorstensson et al.
1997 Grossi et al.
1998 Ridker et al.
2000
2001 Beck et al.
2002 Lopez et al.
2003 Boggess et al.
2004 Saito et al.
2004 Ide et al.
2005 Saremi et al.
2006 Michalowicz et al.
2006 Offenbacher et al.
2007 Tonetti et al.
2009 Offenbacher et al.
2010 Morita et al.
2010
2012 Bokhari et al.
2013 Engebretson et al.
2016 Gomes-Filho et al.
2017 Zhou et al.
2018 D'Aiuto et al.
2020
Medical Condition
Cardiovascular Disease
Diabetes Mellitus
Adverse Pregnancy Outcomes
Type of Study
Cross-sectional – Purple
Longitudinal – Yellow
Interventional – Red

Different studies



CONCEPT OF "FOCAL INFECTION"

- "Oral sepsis as a cause of disease" (William Hunter, 1900)
- Superseded by "focal infection"- introduced by Frank Billings (1911)
- " Circumscribed area infected with micro-organisms which may or may not give rise to clinical manifestations" (JADA, 1951)

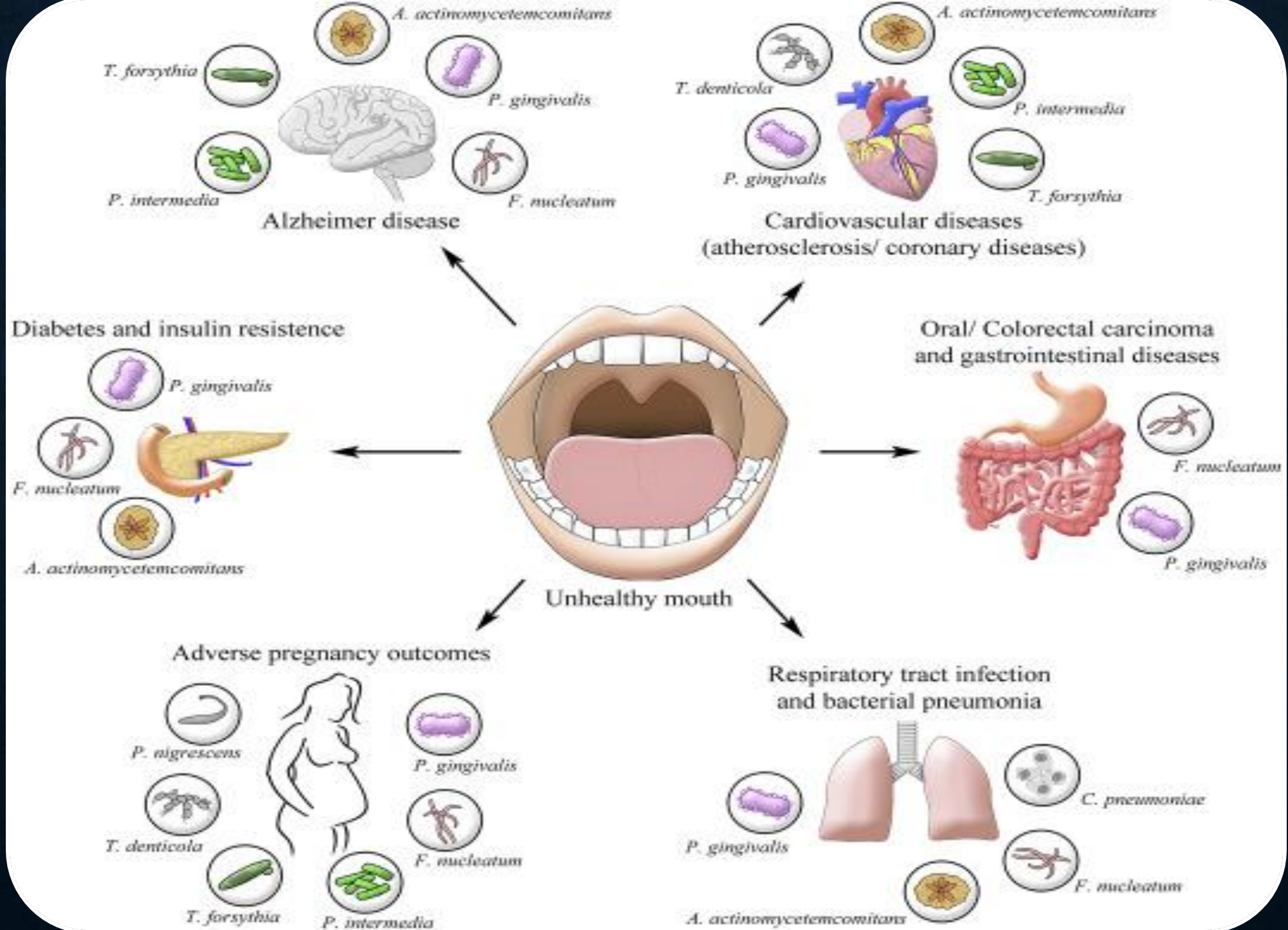
Dabelian et al (1994) identified 3 pathways-

- Metastatic infection from the oral cavity due to transient bacteremia
- Metastatic injury due to oral microbial toxins
- Metastatic inflammation due to immunologic injury caused by oral micro-organisms

2 Major mechanisms of focal infection-
1]An actual metastasis of organisms from a focus
2]The spread of toxins or their products from a remote focus to other tissues by the blood stream.

William Hunter's concept of oral cavity as focus of infection was discarded since it was not based on sound scientific evidence

Sources of Focal Infection- By Frank Billings (1912)
Facial tonsils,peritonsillar tissues and supratonsillar fossae, abcesses of gums and alveolar sockets, pyorrhoea alveolaris and septic types of gingivitis (actual periodontal disease), sinuses related to head, frontal, maxillary, ethmoidal and spheroidal region, bronchiectatic and pulmonic cavities, chronic ulcers pf the gastrointestinal tract, chronic appendicitis, cholecystitis and cholangeitis.


A. actinomycetemcomitans
T. forsythia
P. gingivalis
P. intermedia
F. nucleatum
Alzheimer disease
A. actinomycetemcomitans
T. denticola
P. intermedia
P. gingivalis
T. forsythia
Cardiovascular diseases
(atherosclerosis/ coronary diseases)
Diabetes and insulin resistence
P. gingivalis
F. nucleatum
A. actinomycetemcomitans
Oral/ Colorectal carcinoma
and gastrointestinal diseases
F. nucleatum
P. gingivalis
Unhealthy mouth
Adverse pregnancy outcomes
P. nigrescens
P. gingivalis
T. denticola
F. nucleatum
T. forsythia
P. intermedia
Respiratory tract infection
and bacterial pneumonia
C. pneumoniae
P. gingivalis
F. nucleatum
A. actinomycetemcomitans

ROLE OF INFLAMMATION IN PERIODONTAL AND SYSTEMIC DISEASES: IMPLICATIONS FOR CLINICAL PRACTICE

- Should the movement from infection model to inflammation model of disease change the way we treat our patients?

NO

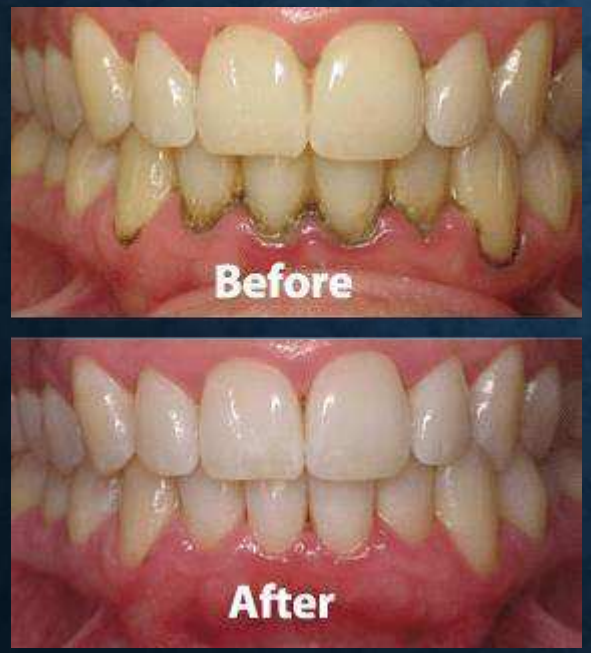
SHOULD IT?

OUT OF CONTROL INFLAMMATION

PATHWAYS OF SPREAD OF PERIODONTAL DISEASE

- Metastasis
- Inflammation
- Immunological Injury

- **How does inflammation fit into risk assessment?**
- INFECTION
- INFLAMMATION
- CLINICAL PRESENTATION



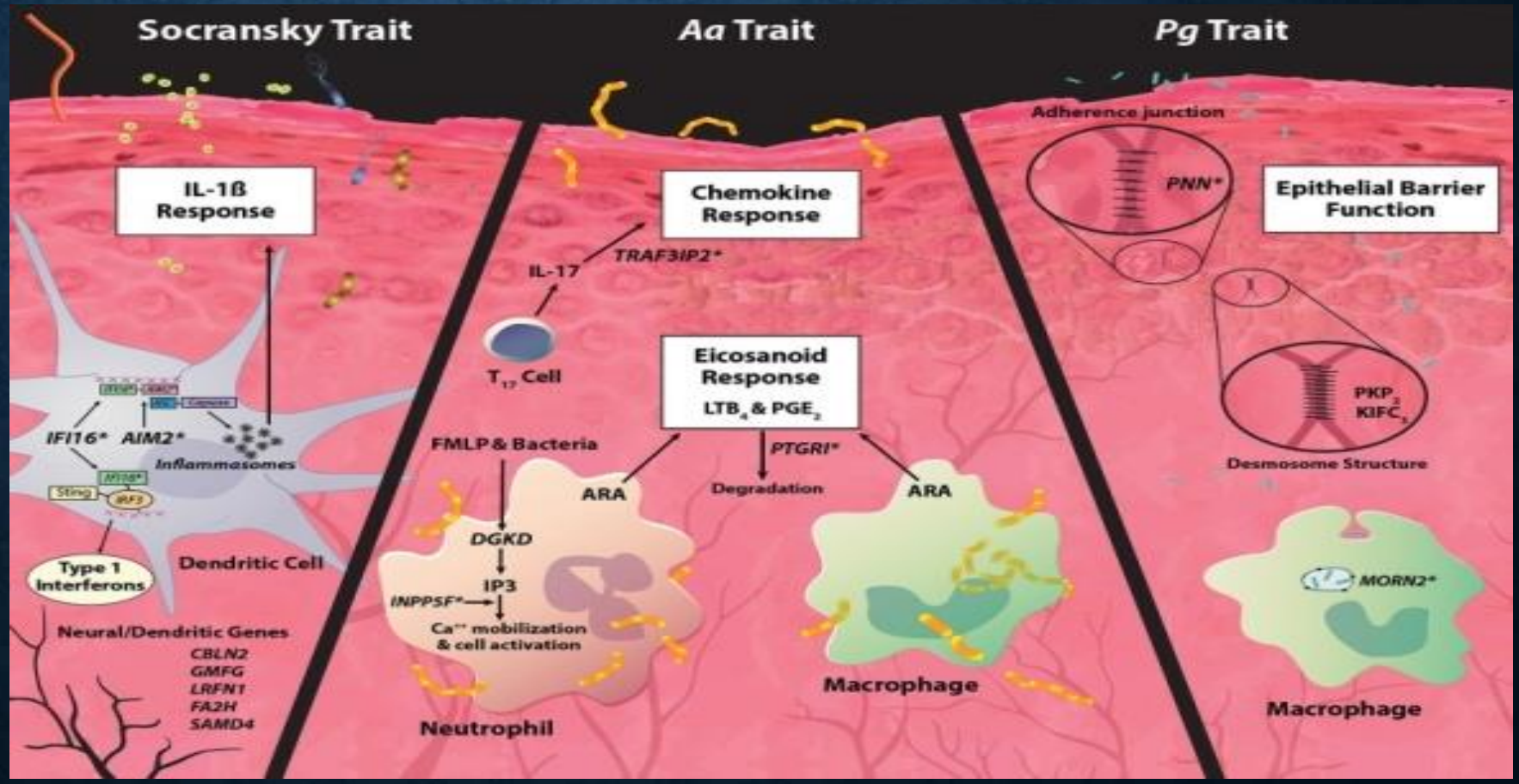
ASSOCIATION AND CAUSALITY

- Association is defined as the concurrence of two variables more often than would be expected by chance.
- Causality is defined as the relationship between an event(cause) and a second event (effect) where the second event is understood as a consequence of the first.
- Types of Causal relationships-

  Sufficient Cause, Necessary Cause, Risk Factor.
- Criteria for Causality-

  Epidemiological Association, Biological Plausibility, Impact of Intervention.

- Is there way to identify hyper responders?
- Prognosis Vs Actual Outcome
- The Effectiveness of clinical parameters & IL-1 Genotype in accurately predicting prognosis & tooth survival
- (McGuire & Nunn JP 99)



- Traditional periodontal therapy is effective for mild to moderate Periodontitis cases & localized severe cases with anatomical & other risk factors.
- Most severe cases occurs in a very small subset of the population (8-13%) & we have not identified or treated their disease effectively.

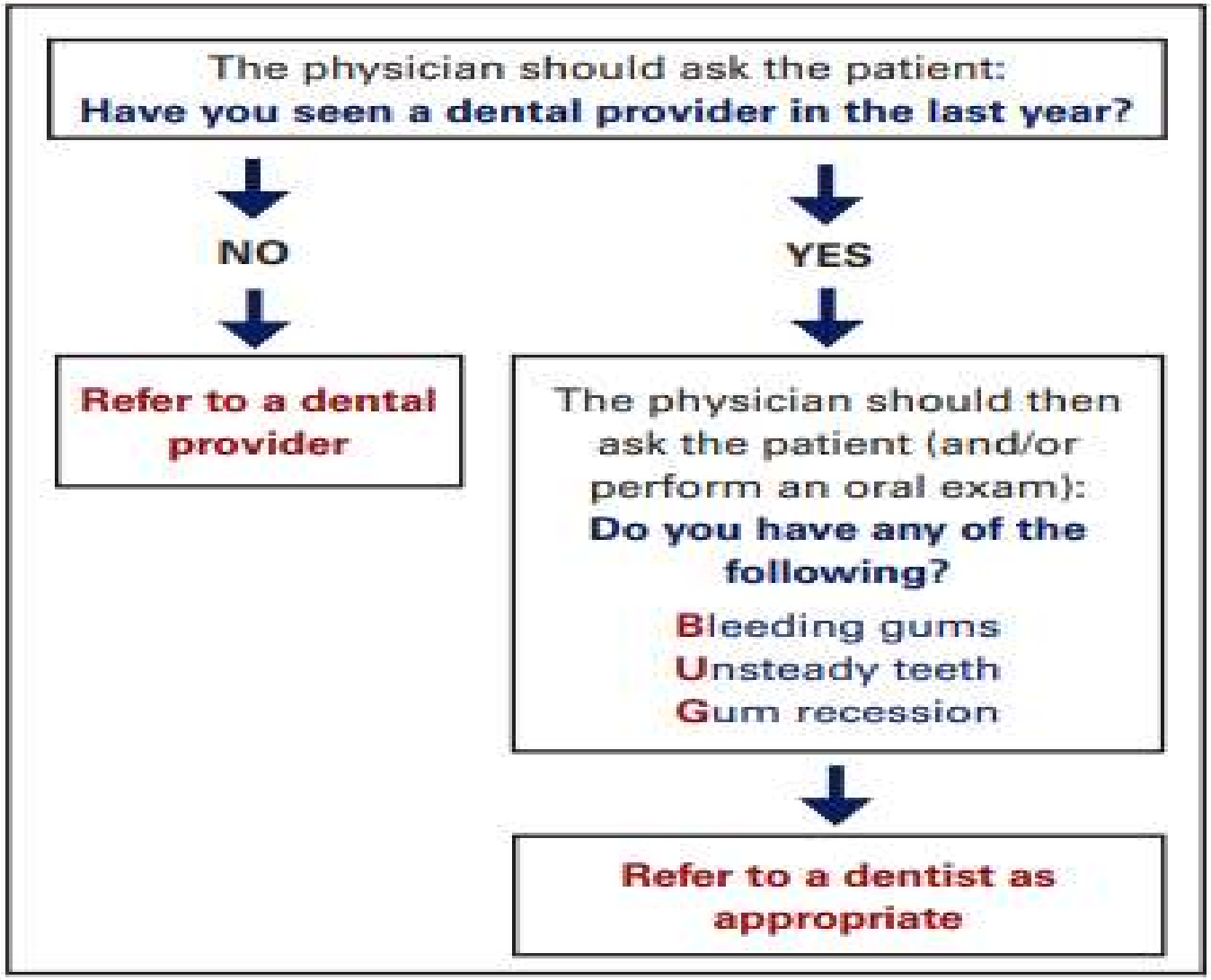
SHOULD WE BE TAKING A MORE DETAILED SYSTEMS REVIEW DURING OUR NEW PATIENT EXAM & AT RECALL?

THE SCOTTSDALE PROJECT:

- Referral to MD
- Perio ptswho have not had med, eval within 2 years or more of the following:
- 50 yrs old
- At risk for diabetes
- Hypertension
- Dyslipidemia w/frm history of CVD or stroke
- Tobacco use
- History consistent w/CVD

THE SCOTTSDALE PROJECT

Figure 4. Expert panel recommendations for physicians to screen and refer patients with signs of periodontal disease.

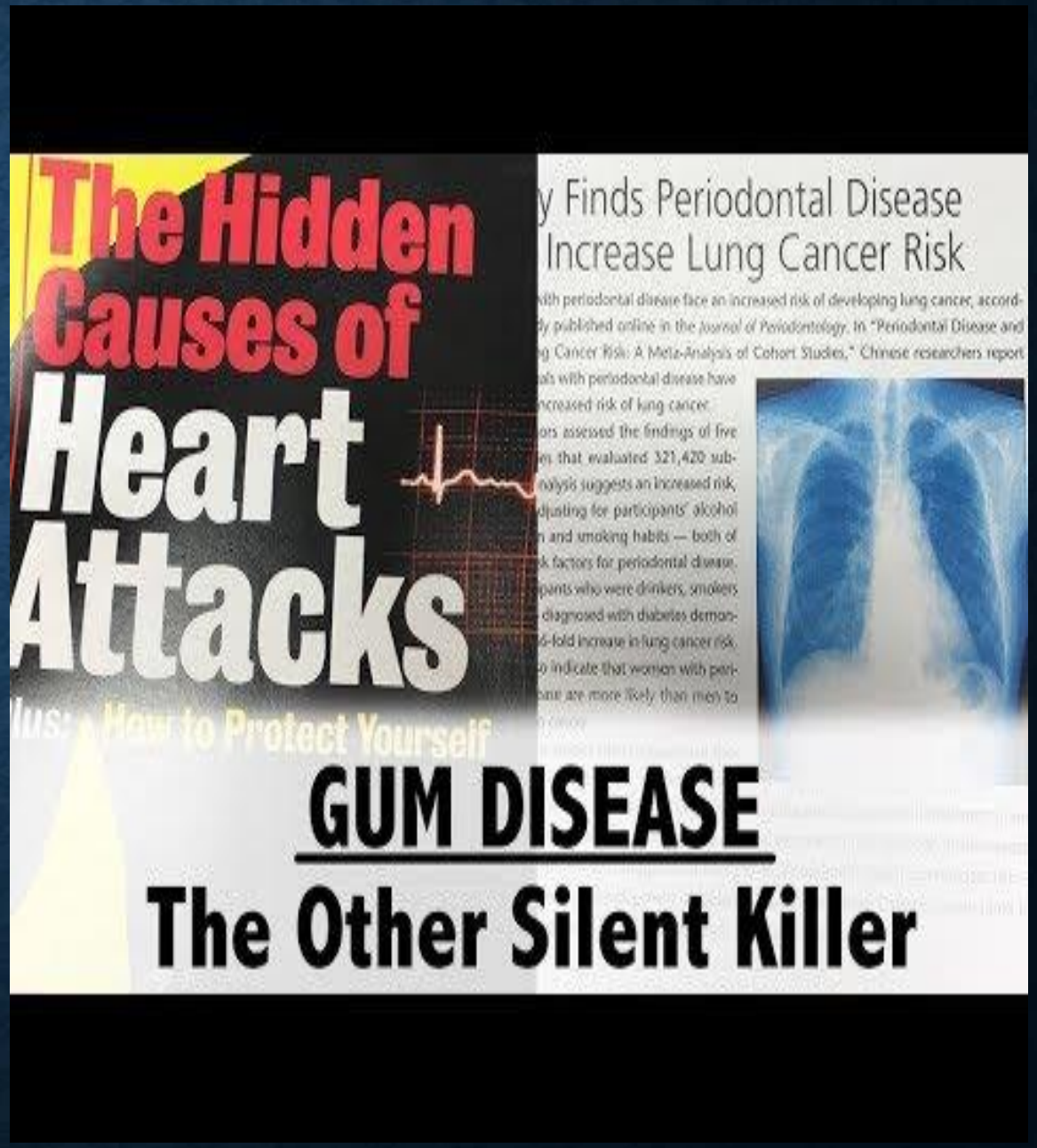


INCIDENCE OF BACTEREMIA DURING DIFFERENT DENTAL PROCEDURES

(HEIMDAHL ET AL, 1990)

Scaling and Root Planing	• % of patients with bacteremia- 70 • % of patients with Viridans group of streptococci- 55 • % of Anaerobes - 65
Surgical procedures 1) Dental Extraction 2)Third Molar surgery	• % of patients with bacteremia- 100 , 55 • % of patients with Viridans group of streptococci- 85,40 • % of Anaerobes – 75,45
Endodontic Treatment	• % of patients with bacteremia- 20 • % of patients with Viridans group of streptococci- 15 • % of Anaerobes- 5
Bilateral Tonsillectomy	• % of patients with bacteremia- 55 • % of patients with Viridans group of streptococci- 40 • % Anaerobes - 40

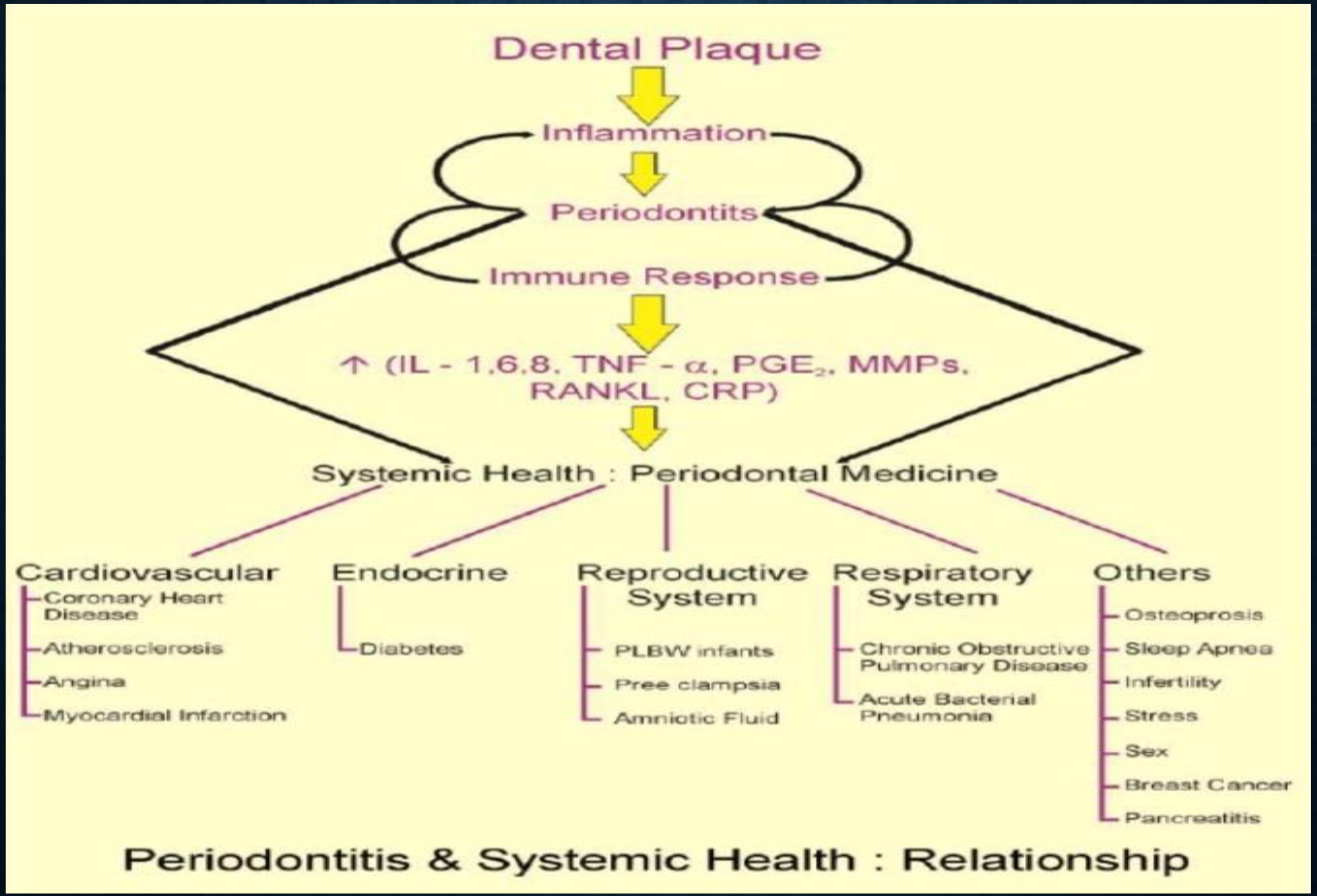
Subgingival environment as a reservoir of bacteria- total surface area of pocket epithelium in contact with subgingival bacteria and their products in a patient with generalized moderate periodontitis- estimated to be approximately the size of the palm of an adult hand with larger areas of exposure in cases of more advanced periodontal destruction (Roy C Page, 1998)



IS PERIODONTITIS A RISK FACTOR FOR SYSTEMIC DISEASES…..???

- RISK FACTOR- distinctive characteristics, exposures that increase the probability of disease outcome (Albander, 2002)
- Bradford's Criteria (1971)-

i. Temporal consistency
ii. Dose-response effect
iii. Strength of association
iv. Biologic plausibility
v. Specificity of association
vi. Consistency of the findings



Periodontitis & Systemic Health : Relationship

REPORTED ASSOCIATIONS BETWEEN PERIODONTAL DISEASE AND SYSTEMIC DISEASE

- Alzheimer's disease
- Anaemia
- Atherosclerosis
- Cancer
- COPD
- Colon cancer
- Crohn's disease
- Death
- Dementia
- Endometrosis
- Fever
- Fibromyalgia
- Gastro-oesophageal reflux disease
- Hypertension
- Infertility
- Inflammatory bowel disease
- Intellectual function
- Leukemia
- Low birth weight
- Lung cancer
- Lupus
- Metabolic syndrome

- Multiple sclerosis
- Myocardial infarction
- Obesity
- Obstructive sleep apnoea
- Osteoporosis
- Pneumonia
- Polycystic ovaries
- Miscarriage
- Mouth cancer
- Erectile dysfunction
- Fatigue
- Pre-eclampsia
- Premature birth
- Psoriasis
- Renal disease
- Rhematoid arthritis
- Stroke
- Stomach ulcers

Mark Bartold, 2012

SYSTEMS AFFECTED BY PERIODONTAL DISEASE :

- Cardiovascular System
- Reproductive System
- Endocrine System
- Respiratory System
- Musculoskeletal System
- Renal System

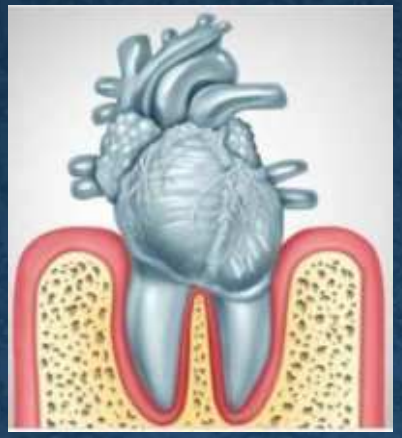
PERIODONTAL DISEASE AND CARDIOVASCULAR DISEASE

EPIDEMIOLOGICAL EVIDENCE

- Individuals with Periodontitis:
- Are 25% more likely to develop coronary heart disease.
- As periodontitis severity increases, so does the probability of CVD.
- Exhibit increased serum biomarkers and clinical markers (mediators of inflammation) of endothelial dysfunction and atherosclerosis.

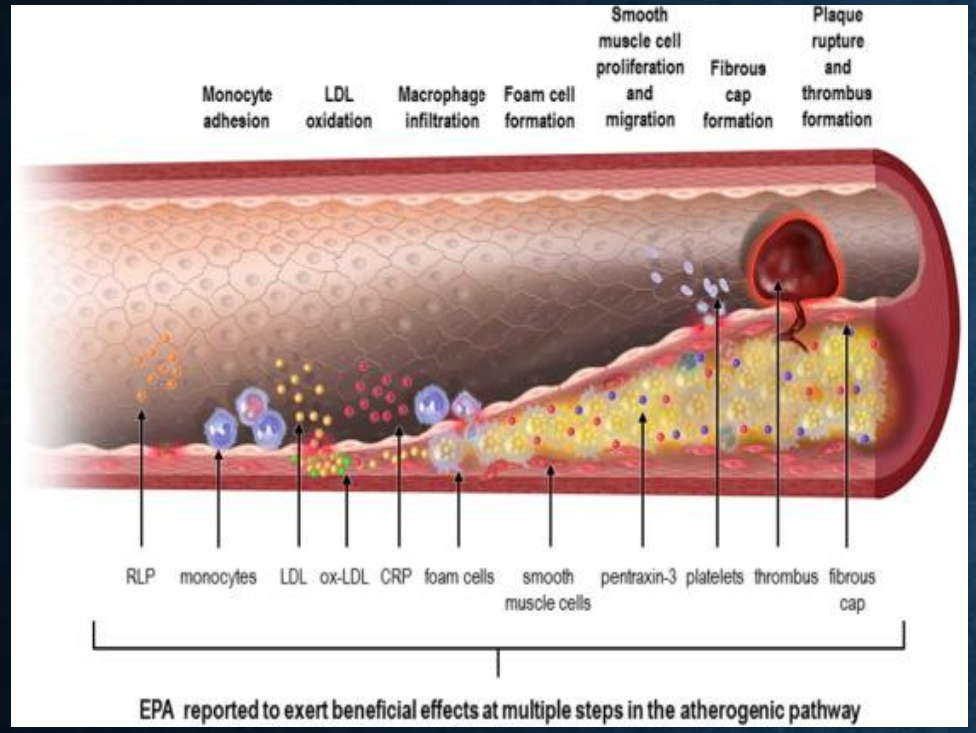
I. 30% increase in C-reactive protein (CRP)levels.

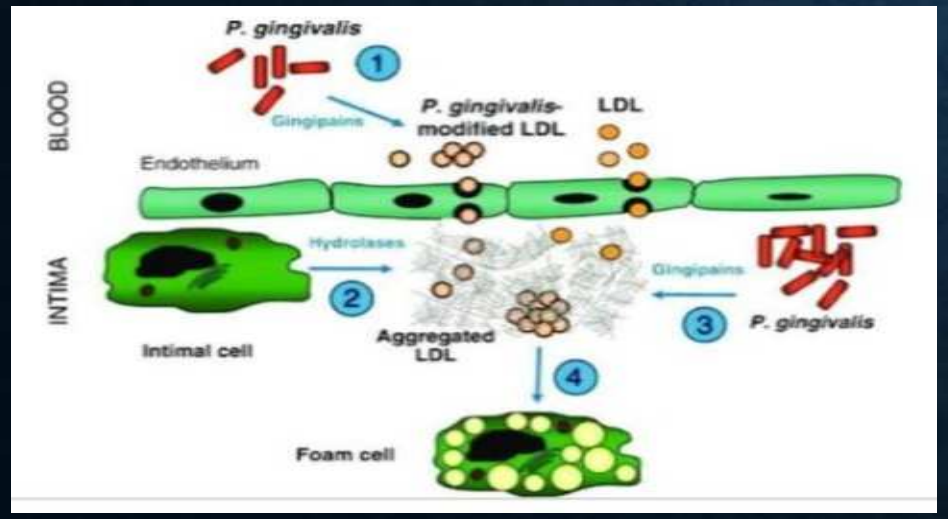
II. 11% increase in cholesterol (LDL).



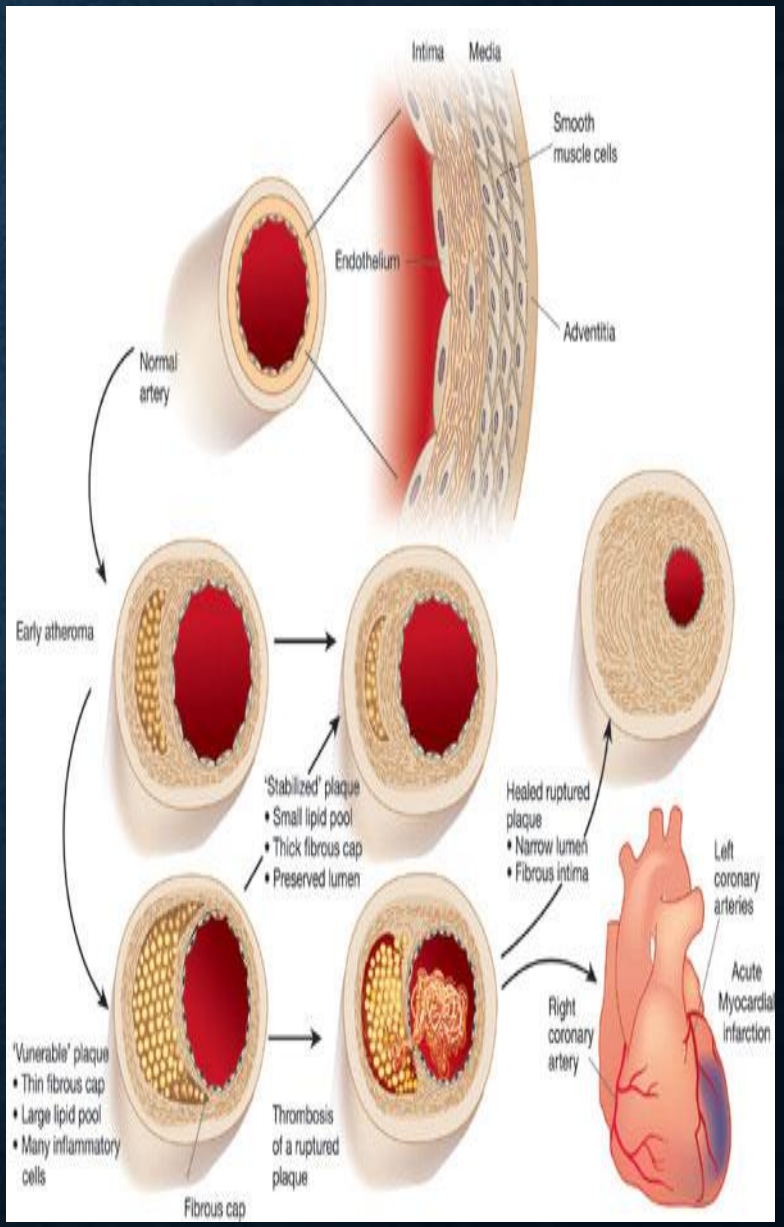
- Individuals with Periodontitis are at increased risk:-
- 2.1 times for Acute Myocardial Infarction
- 4.3 times for Stroke
- 2.3 times for Peripheral vascular diseases.

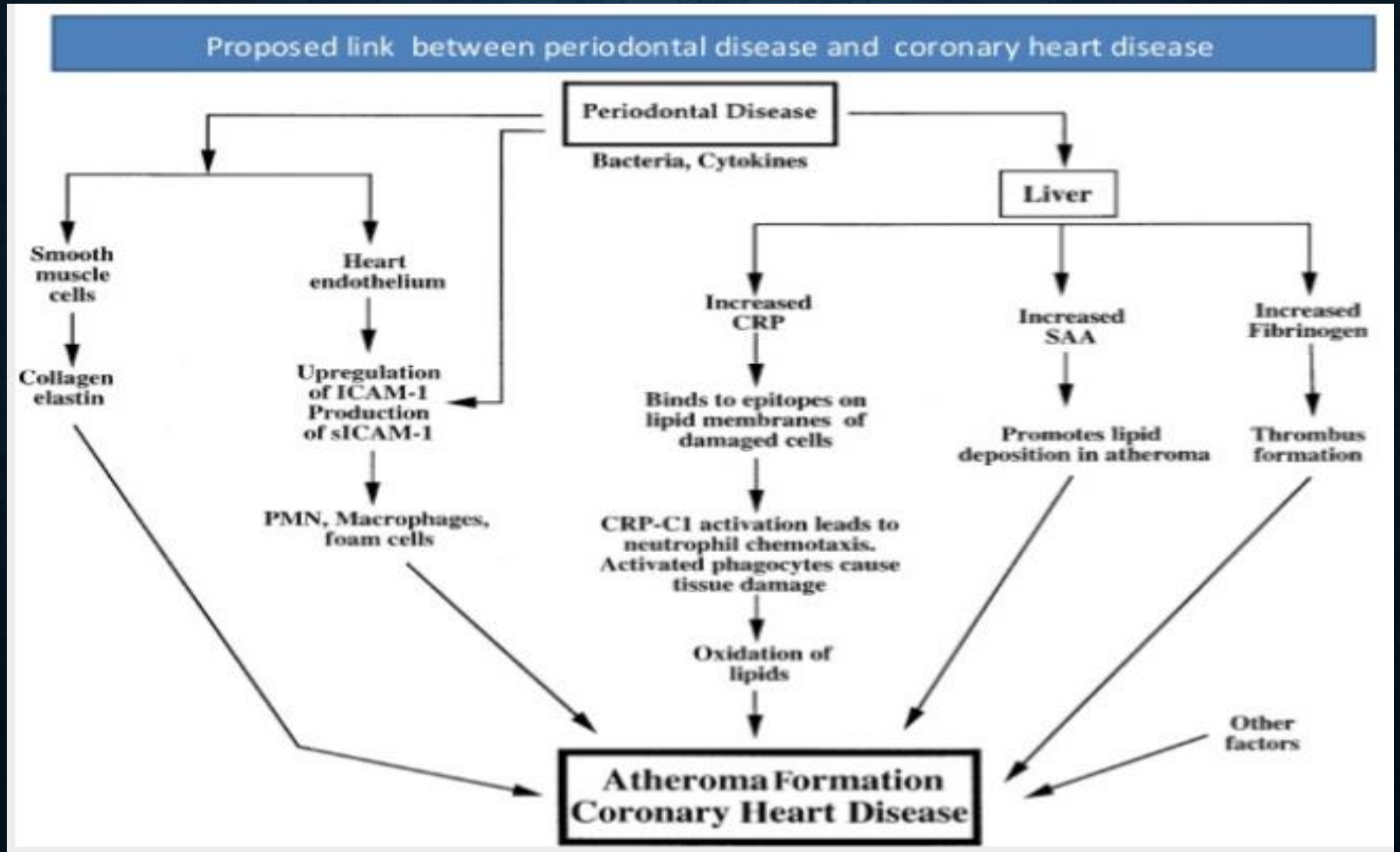
STEPS IN ATHEROSCLEROSIS

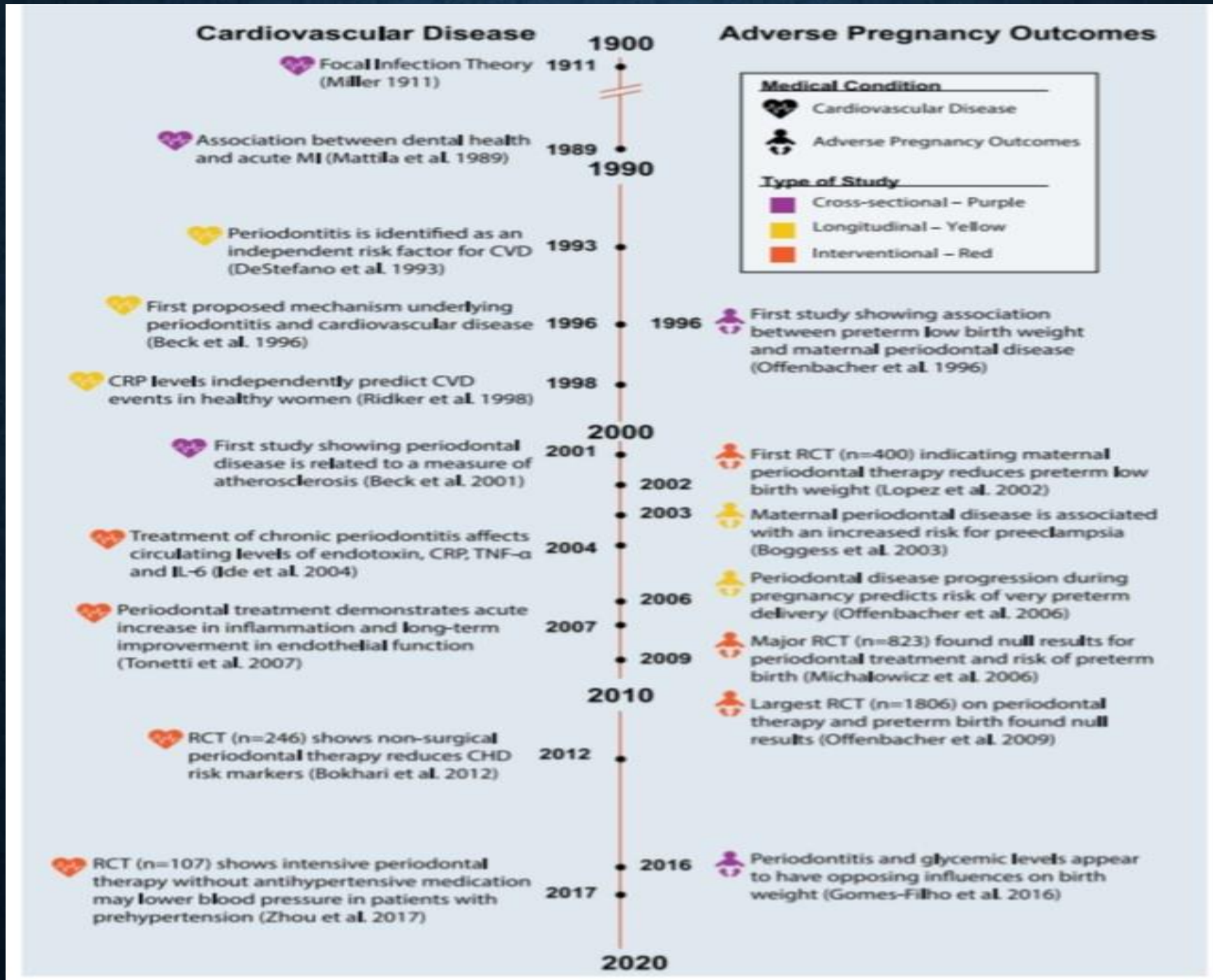




- Endothelial dysfunction
- Adhesion of platelets and leukocytes
- Infiltration of monocytes
- Formation of foam cells
- Smooth muscle cells proliferation
- Atheroma formation
- Thromboembolic event




Proposed link between periodontal disease and coronary heart disease
Periodontal Disease
Bacteria, Cytokines
Liver
Smooth muscle cells
Collagen elastin
Heart endothelium
Upregulation of ICAM-1 Production of sICAM-1
PMN, Macrophages, foam cells
Increased CRP
Binds to epitopes on lipid membranes of damaged cells
CRP-C1 activation leads to neutrophil chemotaxis. Activated phagocytes cause tissue damage
Oxidation of lipids
Increased SAA
Promotes lipid deposition in atheroma
Increased Fibrinogen
Thrombus formation
Other factors
Atheroma Formation
Coronary Heart Disease


Cardiovascular Disease
Adverse Pregnancy Outcomes
1900
1911
Focal Infection Theory (Miller 1911)
1989
Association between dental health and acute MI (Mattila et al. 1989)
1990
1993
Periodontitis is identified as an independent risk factor for CVD (DeStefano et al. 1993)
1996
First proposed mechanism underlying periodontitis and cardiovascular disease (Beck et al. 1996)
1996
First study showing association between preterm low birth weight and maternal periodontal disease (Offenbacher et al. 1996)
1998
CRP levels independently predict CVD events in healthy women (Ridker et al. 1998)
2000
2001
First study showing periodontal disease is related to a measure of atherosclerosis (Beck et al. 2001)
2002
First RCT (n=400) indicating maternal periodontal therapy reduces preterm low birth weight (Lopez et al. 2002)
2003
Maternal periodontal disease is associated with an increased risk for preeclampsia (Boggess et al. 2003)
2004
Treatment of chronic periodontitis affects circulating levels of endotoxin, CRP, TNF-α and IL-6 (Ide et al. 2004)
2006
Periodontal disease progression during pregnancy predicts risk of very preterm delivery (Offenbacher et al. 2006)
2007
Periodontal treatment demonstrates acute increase in inflammation and long-term improvement in endothelial function (Tonetti et al. 2007)
2009
Major RCT (n=823) found null results for periodontal treatment and risk of preterm birth (Michalowicz et al. 2006)
2010
Largest RCT (n=1806) on periodontal therapy and preterm birth found null results (Offenbacher et al. 2009)
2012
RCT (n=246) shows non-surgical periodontal therapy reduces CHD risk markers (Bokhari et al. 2012)
2016
Periodontitis and glycemic levels appear to have opposing influences on birth weight (Gomes-Filho et al. 2016)
2017
RCT (n=107) shows intensive periodontal therapy without antihypertensive medication may lower blood pressure in patients with prehypertension (Zhou et al. 2017)
2020
Medical Condition
Cardiovascular Disease
Adverse Pregnancy Outcomes
Type of Study
Cross-sectional – Purple
Longitudinal – Yellow
Interventional – Red

MICROBIOLOGICAL EVIDENCE

- Direct association between an increased risk of atherosclerosis and the presence of periodontal pathogens such as-

i. C. rectus

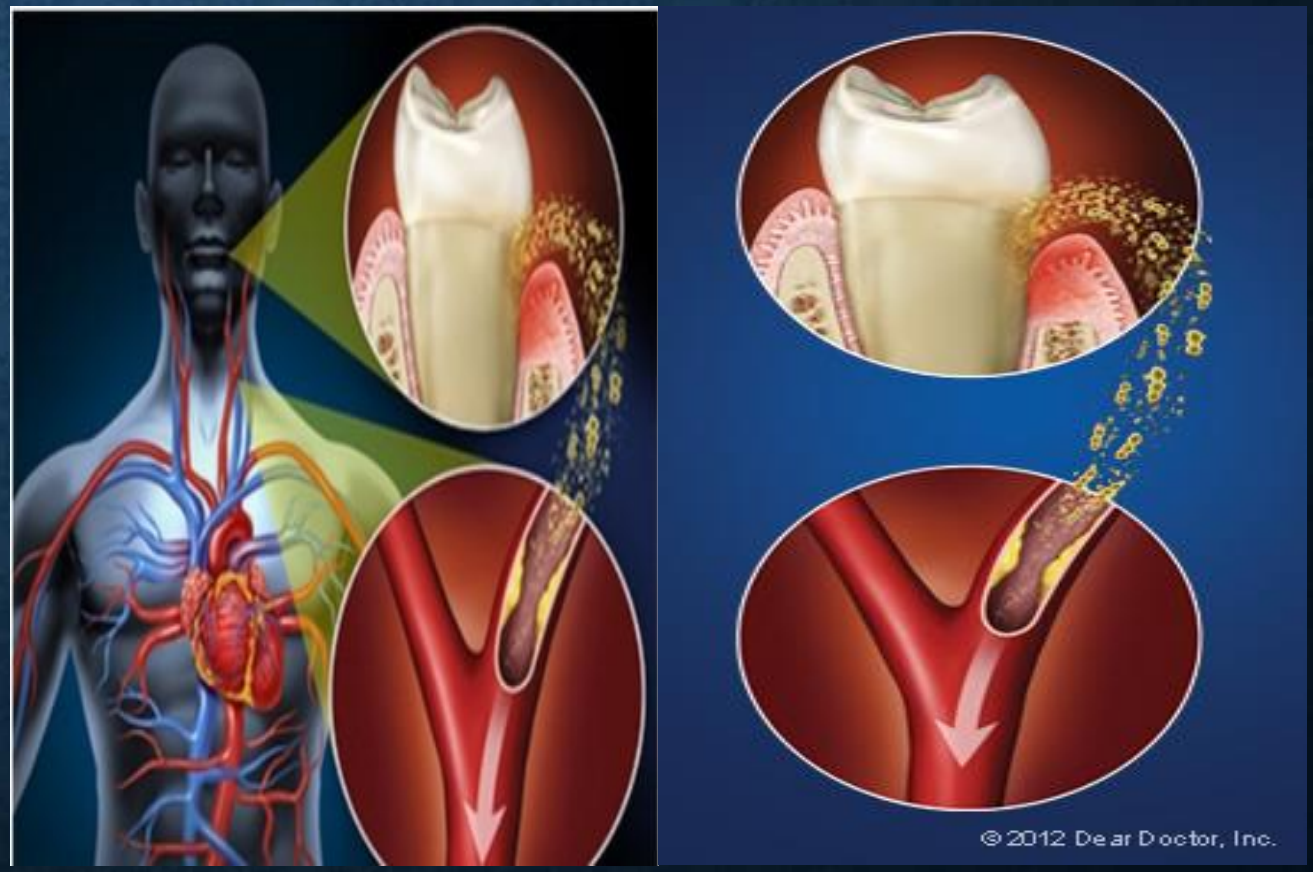
ii. P.micros

iii. A.actinomycetemcomitans

iv. P.gingivalis

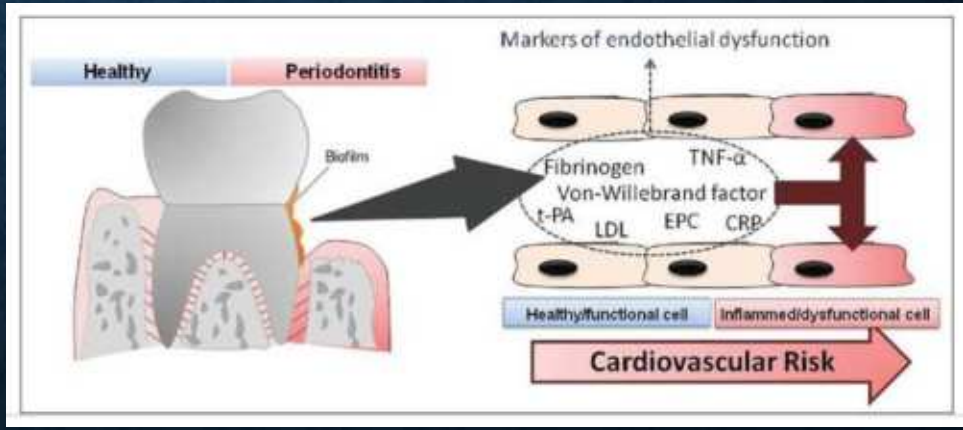
v. T.denticola

vi. T.forsythensis



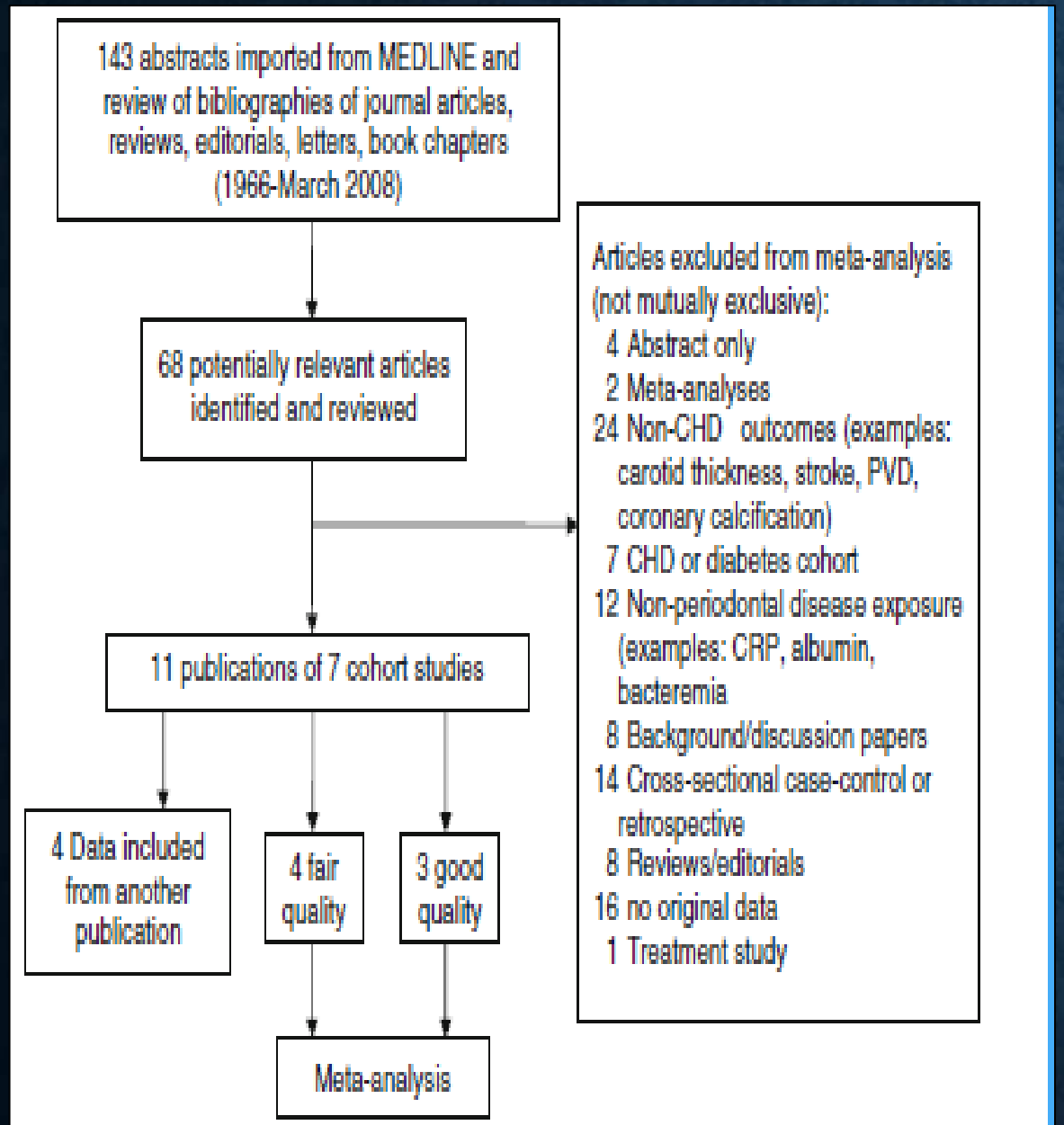
EVIDENCE OF ASSOCIATION BETWEEN PERIODONTAL DISEASE & CARDIOVASCULAR DISEASE

- Research documenting association between Periodontal Disease and Cardiovascular Disease is recent with earliest study published in 1989 by Mattila and associates. This cross-sectional study is the first study to report association between CVD and PD. After this landmark study, a number of observational studies implicating periodontitis for etiopathogenesis were published, however, few of the studies did not find any association.



- Periodontal Disease and Coronary Heart Disease Incidence: A Systematic Review and Meta-analysis- prospective cohort studies

J Gen Intern Med. 2008 ; 23(12): 2079–2086.



European Heart Journal Supplements (2010) 12 (Supplement B), B3–B12
doi:10.1093/eurheartj/suq003

European workshop in periodontal health and cardiovascular disease—scientific evidence on the association between periodontal and cardiovascular diseases: a review of the literature

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KEYWORDS
Periodontal diseases;
Cardiovascular disease;
Oral health;
Systemic inflammation;
Cardiovascular events;
Periodontal pathogens

In the last 10 years, a rising number of epidemiological investigations have studied the possible association between chronic oral infections and cardiovascular diseases (CVD). These studies were based on the hypothesis that periodontal diseases (PD), may confer an independent risk for CVD. There is, however, still controversy whether these associations are causal or whether there are common aetiological factors common to both diseases (residual confounding). The objective of this paper was to review the possible association between PD and CVD on both the epidemiological association and the possible preventive and treatment implications.

Although the reported epidemiological studies have shown a significant, albeit week associations, we still lack properly designed clinical trials demonstrating that these chronic infections are independent factors of cardiovascular risk. The use of surrogate variables assessing the infective load and measures of subclinical atherosclerosis have clearly shown, not only a significant pathogenic relationship, but also a significant impact after periodontal therapy.

From a public health perspective, if further studies consistently identify PD as a risk factor for CHD and treatment studies show benefit, the implications are significant, since PD is mostly avoidable and treatable when not prevented. In addition, good preventive dental care has multiple other benefits, particularly on quality of life. Furthermore, identifying individuals at higher risk for CHD than predicted by traditional risk factors could facilitate treatment of risk factors known to decrease CHD events in high-risk individuals and this might be significant given the high prevalence of PD in the population and the common problem of CHD.

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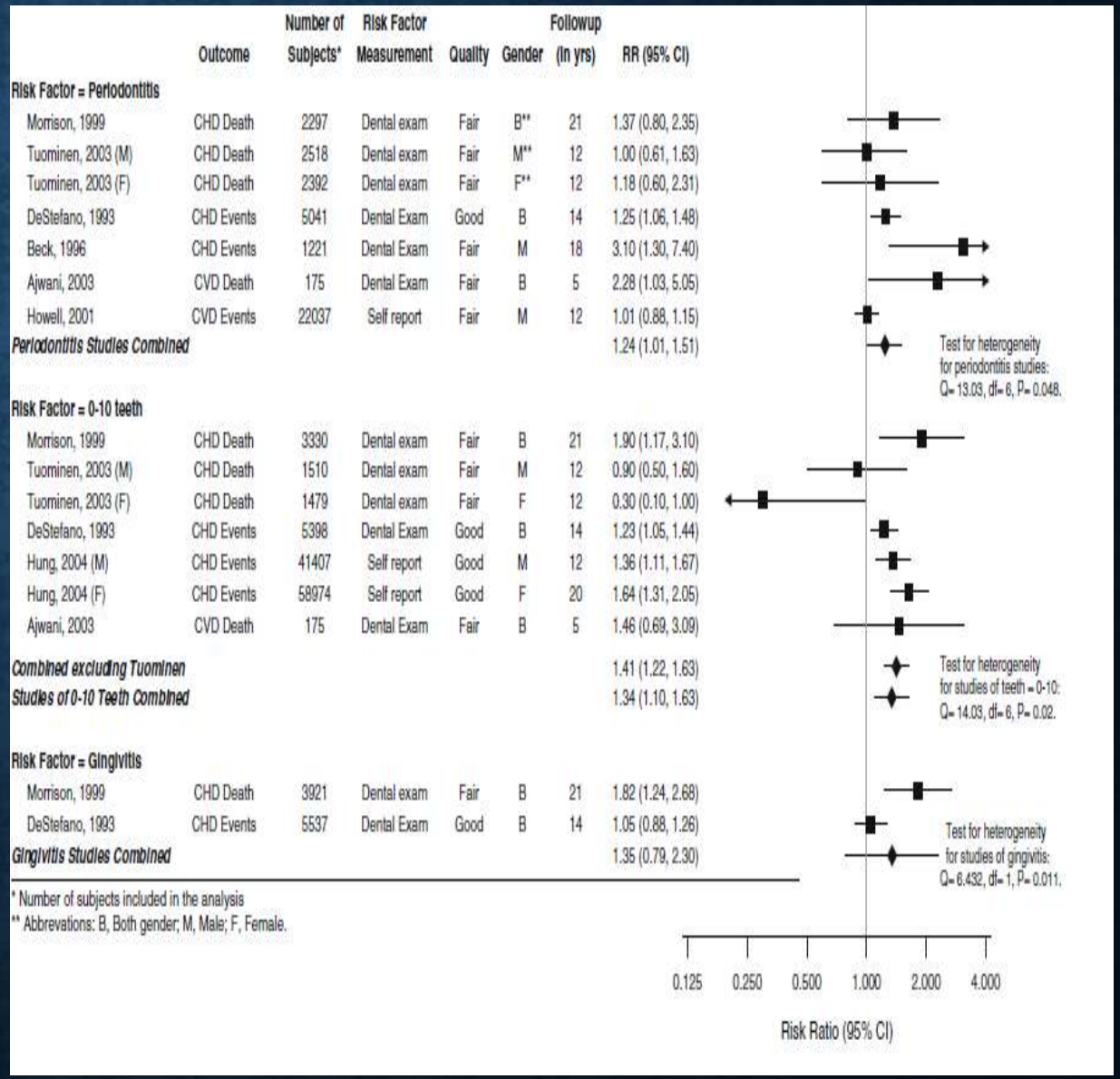
Systematic reviews and Meta-analysis of Periodontal Disease and Coronary Heart Disease

Study (quality rating)	Demographics	Dental assessment method; exposures measured	Follow-up (years); loss to follow-up (%); outcomes studied; outcome assessment	Variables adjusted for	Baseline prevalence of periodontal disease (%)
Helsinki Aging Study[18] (fair*)	175 M and F ages 78-85	Dental exam; PD, tooth loss	5; <2%; mortality, CVD mortality; Finnish Death Registry	Age, sex, TC, HDL, BP, smoking, BMI, prevalent CHD, social class	Periodontitis: 46
Dental Longitu-dinal Study VA[19] (fair*†)	1,094 M veterans ages 21-81 (mean 42.7), free of known chronic illnesses	Radiographic and dental exams: PD, bone loss, clinical probe depth	18; NR; Total CHD (MI, CHD death, angina), stroke	Age, BMI, smoking, sBP, dBP, TC, family history of CHD, alcohol	Mean bone loss score >1: 21 periodontitis: NR
NHANES[20] (good)	9,760 M and F ages 25-74 without known CHD	Dental exam; no. decayed teeth, periodontal clas-sification and index, oral hygiene index, no. teeth	14; <10%; CHD and total mortality, CHD admission; death certs, medical records, interviews	Age, sex, race, education, marital status, sBP, TC, BMI, DM, activity, alcohol, poverty index, smoking	Gingivitis: 23 Periodontitis: 18 Edentulous: 22
PHS[21] (good)	22,037 M physicians ages 40-84 without stroke, MI, TIA, or cancer	Self report; tooth lossPD	12; <1%; stroke, all CVD, CV death, non-fatal MI, annual surveys, medical record review	Age, sex, smoking, DM, BP, BMI, exercise, alcohol	Periodontitis: 12



PERIODONTITIS AND ATHEROSCLEROTIC CARDIOVASCULAR DISEASE

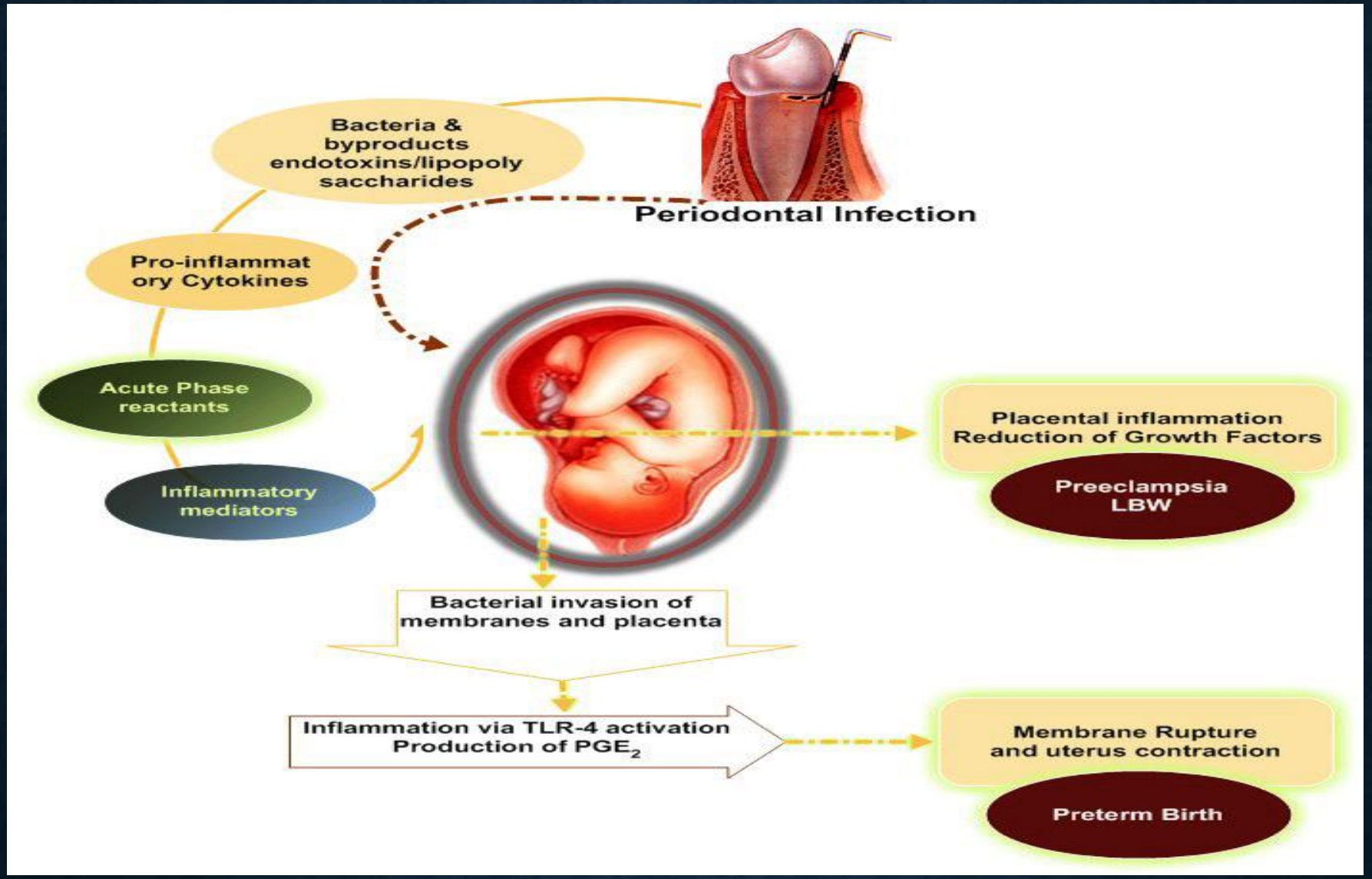
- Aim of this consensus report is to provide health professionals especially cardiologist, periodontist & general dental practitioners a better understanding of link of atherosclerotic cardiovascular disease and periodontitis on the basis of current information and approach to reducing the risk for primary and secondary atherosclerotic CVD events in patients with periodontitis.

American journal of cardiology and journal of periodontology editor's consensus report: Vincent, Friedwald, kenneth S, Kornman et al. J periodontol 2009:80(7);1021-1032

CONCLUSION

- Periodontal Disease is a risk factor or marker for coronary heart disease that is independent of traditional CHD risk factors including socioeconomic status. Further research in this important area of public health is warranted.

PERIODONTAL DISEASE AND PREGNANCY OUTCOMES



Offenbacher (1996), Jeffcoat (2001), Jarjoura(2005): Proposed that periodontal disease is a risk factor for LBW.

Scaling and Root planning in pregnant females reduced the incidence of preterm birth(Jeffcoat 2001)

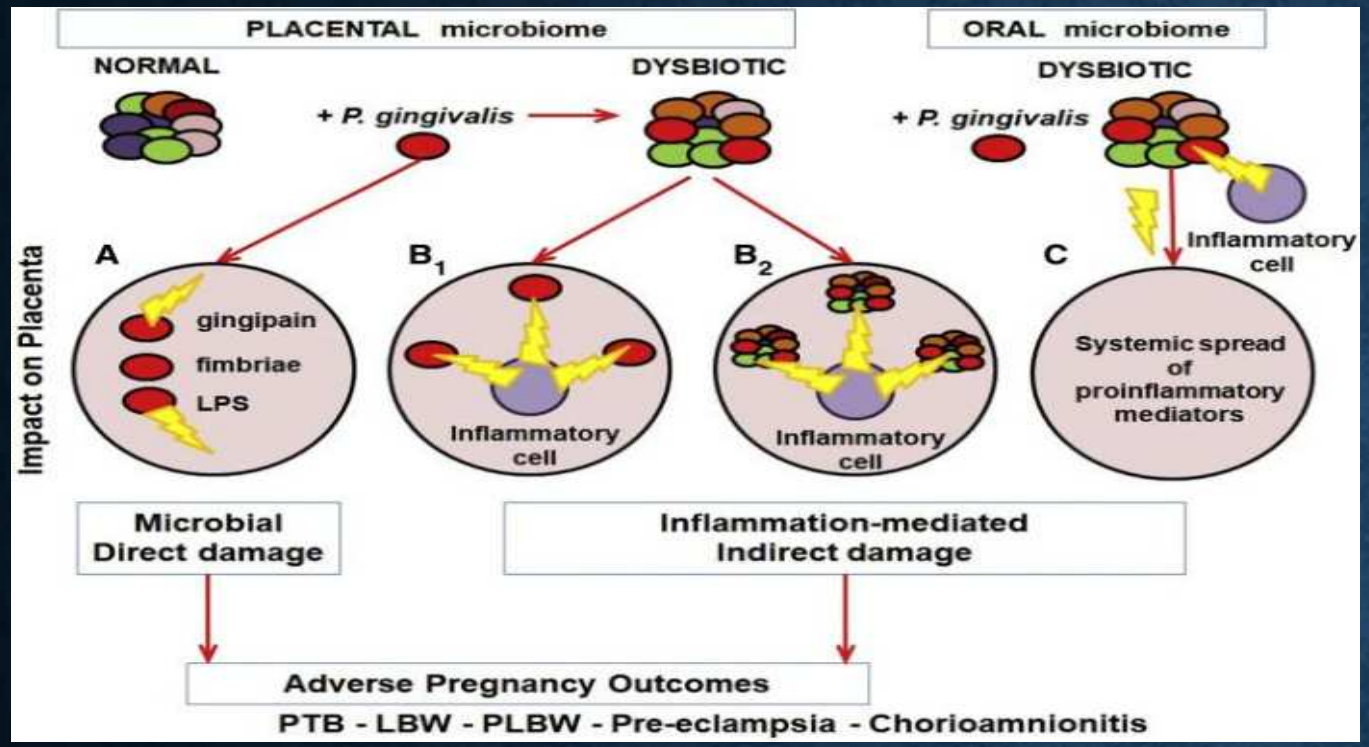
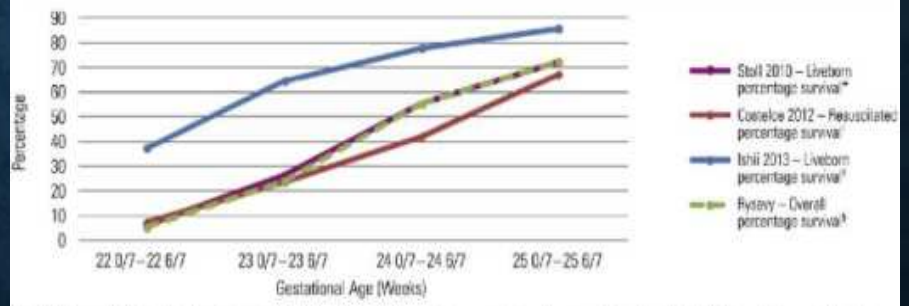


TABLE 1 Newborn mortality before discharge[1] and up to 120 days[2] in the Neonatal Research Network from 2008 to 2012 and survival without major morbidity[1]

Gestational week	Newborns surviving to discharge per 100 births[1]	Newborns surviving to approximately 120 days of life per 100 births[2]	Newborns surviving to discharge without major morbidity per 100 births[1]
22	7	6.2	0
23	32	30.9	5
24	62	61.0	11
25	77	75.7	22
26	85	83.2	32
27	90	89.0	48
28	94	93.5	60

COMPLETED WEEKS OF GESTATION AT BIRTH (using last menstrual period)	CHANCE OF SURVIVAL
21 weeks and less	0%
22 weeks	0-10%*
23 weeks	10-35%
24 weeks	40-70%
25 weeks	50-80%
26 weeks	80-90%
27 weeks	>90%
30 weeks	>95%
34 weeks	>98%

FIGURE 1
Percentage of survival by gestational age



*Stoll BJ, Hansen NI, Bell EF, Shankaran S, Laptook AR, Walsh MC, et al. Neonatal outcomes of extremely preterm infants from the NICHD Neonatal Research Network. Eunice Kennedy Shriver National Institute of Child Health and Human Development Neonatal Research Network. Pediatrics 2010;126:443–56.

†Costeloe KL, Hennessy EM, Haider S, Stacey F, Marlow N, Draper ES. Short term outcomes after extreme preterm birth in England: comparison of two birth cohorts in 1995 and 2006 (the EPICure studies). BMJ 2012;345:e7976.

‡Ishii N, Kono Y, Yonemoto N, Kusuda S, Fujimura M. Outcomes of infants born at 22 and 23 weeks' gestation. Neonatal Research Network, Japan. Pediatrics 2013;132:62–71.

§Rysavy MA, Li L, Bell EF, Das A, Hintz SR, Stoll BJ, et al. Between-hospital variation in treatment and outcomes in extremely preterm infants. Eunice Kennedy Shriver National Institute of Child Health and Human Development Neonatal Research Network. N Engl J Med 2015;372:1801–11.

ACOG. Periviable birth. Am J Obstet Gynecol 2015.

Types of adverse pregnancy outcomes- Still birth, Fetal death, Premature rupture of membranes, Preterm birth, Low birth weight, Pre-eclampsia.

Evidence grade associating periodontitis with preterm birth and/or low birth weight: II. A systematic review of randomized trials evaluating the effects of periodontal treatment

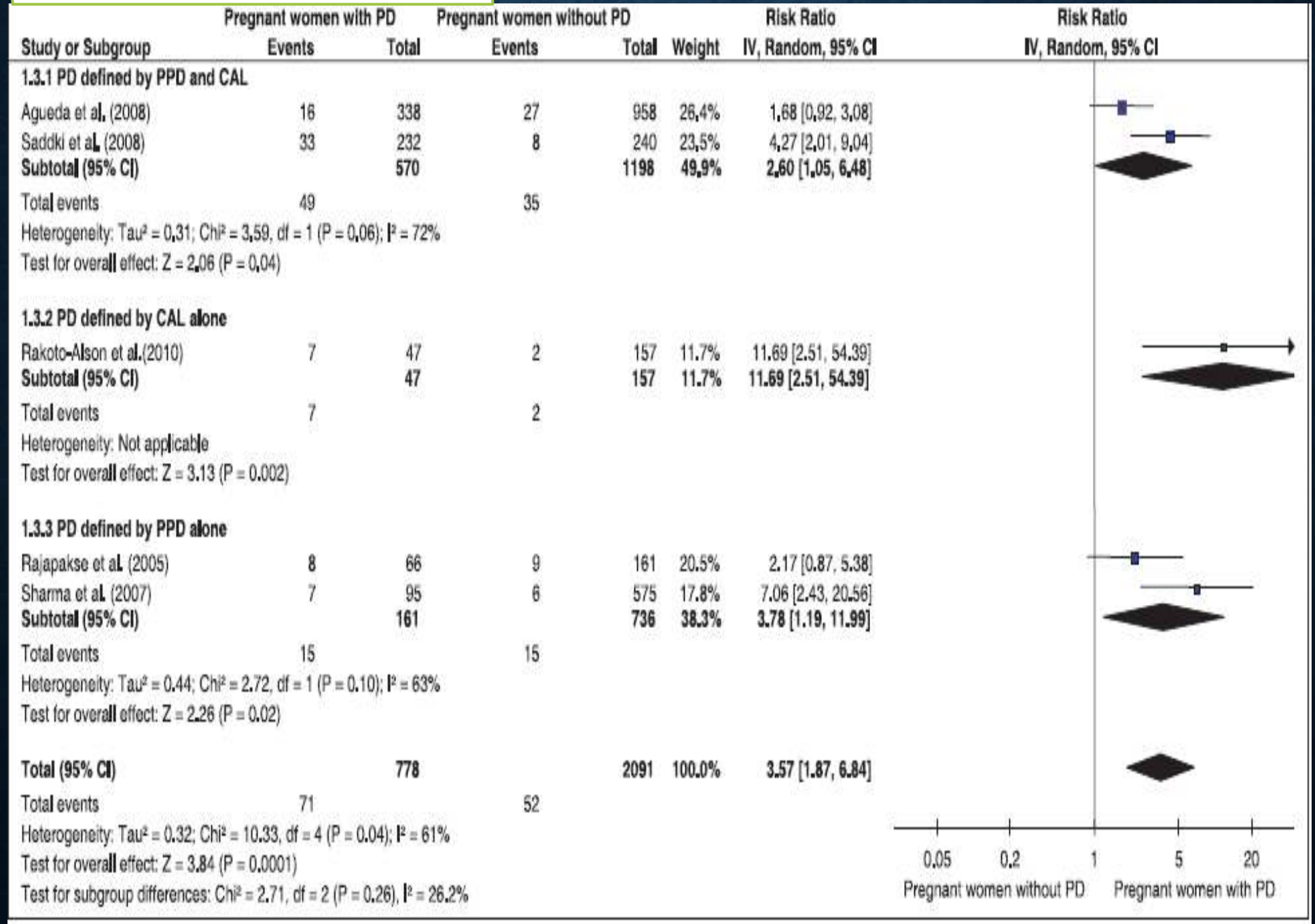
Chambrone L, Guglielmetti MR, Pannuti CM, Chambrone LA.
J Clin Periodontol 2011; 38: 902-914.

EPIGENETIC LINK FOR PERIODONTITIS AS A RISK FOR PRE-TERM DELIVERY

- Recent studies have reported that epigenetic alterations could be induced in the placenta by oral bacteria which could lead to alteration of the placental phenotype and influence development of the fetus.

Bobetsis YA et al in 2007

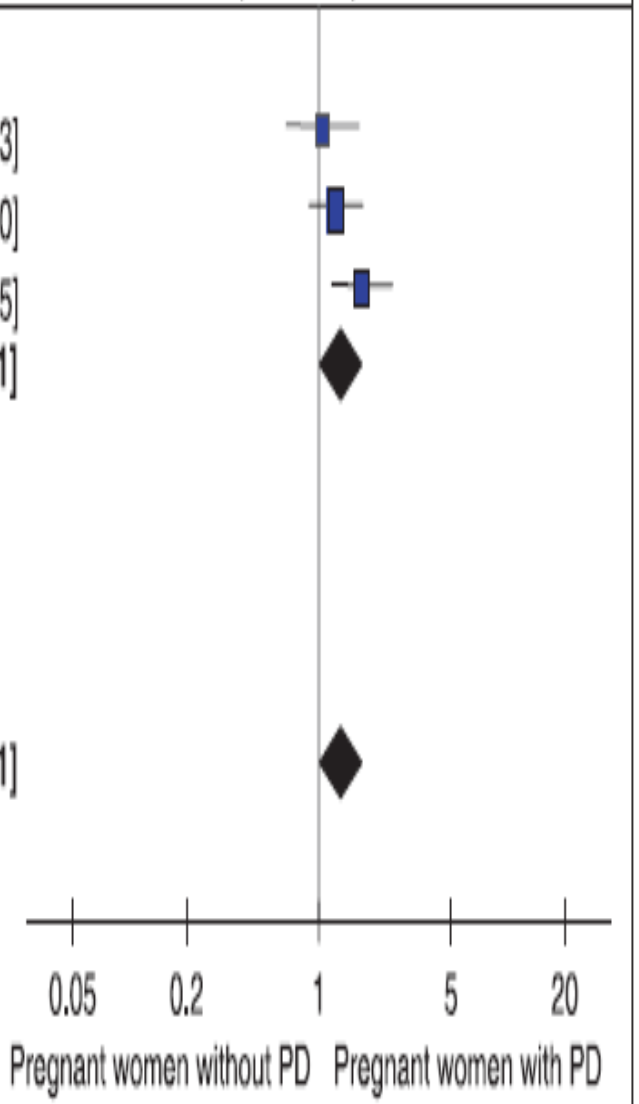
Pre –term and Low birth weight



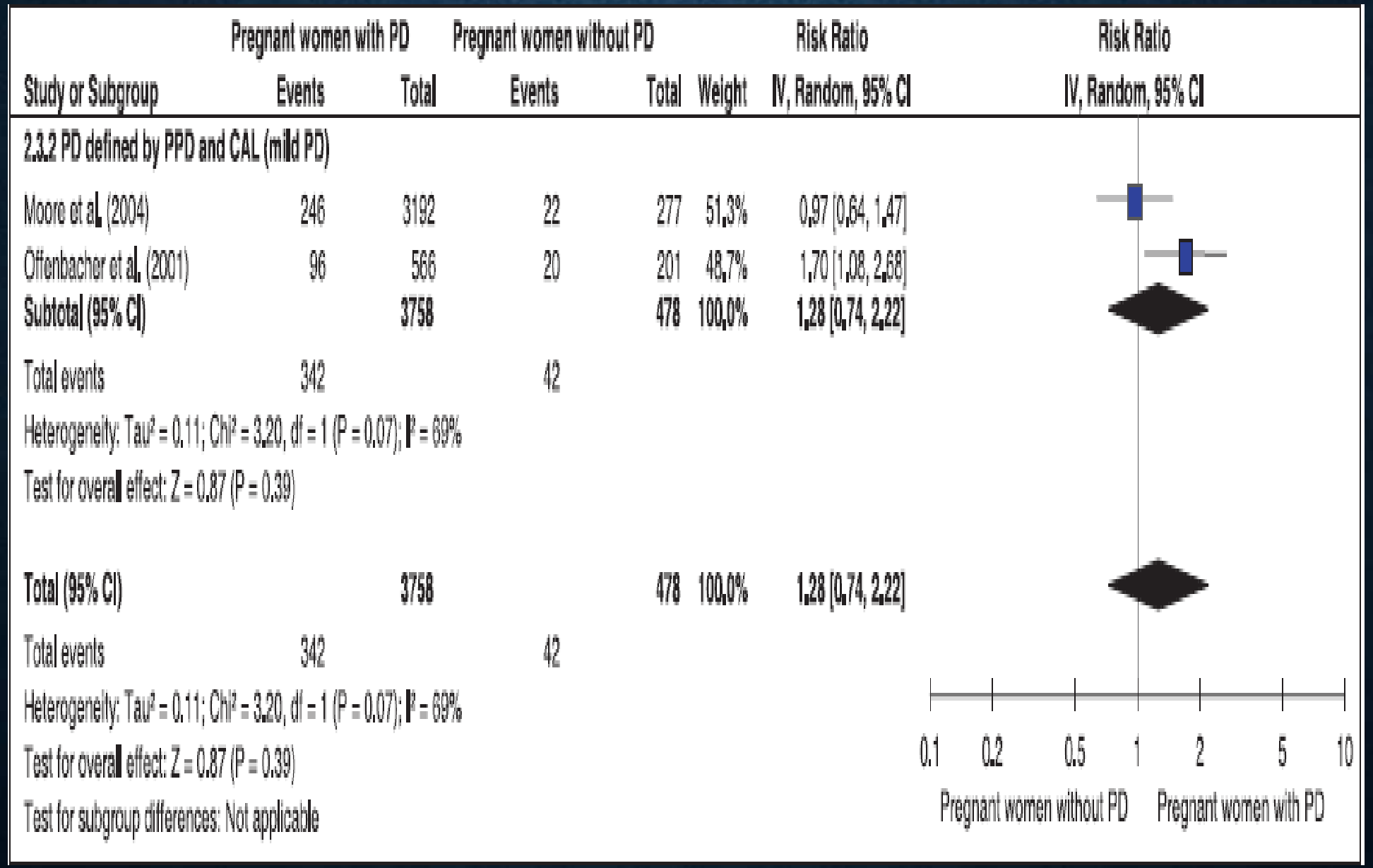
Study or Subgroup	Pregnant women with PD Events	Total	Pregnant women without PD Events	Total	Weight	Risk Ratio IV, Random, 95% CI
1.3.1 PD defined by PPD and CAL						
Agueda et al. (2008)	16	338	27	958	26.4%	1.68 [0.92, 3.08]
Saddki et al. (2008)	33	232	8	240	23.5%	4.27 [2.01, 9.04]
Subtotal (95% CI)		**570**		**1198**	**49.9%**	**2.60 [1.05, 6.48]**
Total events	49		35			
Heterogeneity: Tau² = 0.31; Chi² = 3.59, df = 1 (P = 0.06); I² = 72%						
Test for overall effect: Z = 2.06 (P = 0.04)						
1.3.2 PD defined by CAL alone						
Rakoto-Alson et al.(2010)	7	47	2	157	11.7%	11.69 [2.51, 54.39]
Subtotal (95% CI)		**47**		**157**	**11.7%**	**11.69 [2.51, 54.39]**
Total events	7		2			
Heterogeneity: Not applicable						
Test for overall effect: Z = 3.13 (P = 0.002)						
1.3.3 PD defined by PPD alone						
Rajapakse et al. (2005)	8	66	9	161	20.5%	2.17 [0.87, 5.38]
Sharma et al. (2007)	7	95	6	575	17.8%	7.06 [2.43, 20.56]
Subtotal (95% CI)		**161**		**736**	**38.3%**	**3.78 [1.19, 11.99]**
Total events	15		15			
Heterogeneity: Tau² = 0.44; Chi² = 2.72, df = 1 (P = 0.10); I² = 63%						
Test for overall effect: Z = 2.26 (P = 0.02)						
Total (95% CI)		**778**		**2091**	**100.0%**	**3.57 [1.87, 6.84]**
Total events	71		52			
Heterogeneity: Tau² = 0.32; Chi² = 10.33, df = 4 (P = 0.04); I² = 61%						
Test for overall effect: Z = 3.84 (P = 0.0001)						
Test for subgroup differences: Chi² = 2.71, df = 2 (P = 0.26), I² = 26.2%						

Pre-term birth with mild periodontitis

Study or Subgroup	Pregnant women with PD Events	Total	Pregnant women without PD Events	Total	Weight	Risk Ratio IV, Random, 95% CI
2.1.1 PD defined by PPD and CAL (mild PD)						
Moore et al. (2004)	242	3192	20	277	26.4%	1.05 [0.68, 1.63]
Offenbacher et al. (2001)	132	566	38	201	39.7%	1.23 [0.89, 1.70]
Offenbacher et al. (2006)	112	588	32	285	33.9%	1.70 [1.18, 2.45]
Subtotal (95% CI)		**4346**		**763**	**100.0%**	**1.32 [1.01, 1.71]**
Total events	486		90			
Heterogeneity: $Tau^2 = 0.02$; $Chi^2 = 3.01$, df = 2 (P = 0.22); $I^2 = 34\%$						
Test for overall effect: Z = 2.05 (P = 0.04)						
Total (95% CI)		**4346**		**763**	**100.0%**	**1.32 [1.01, 1.71]**
Total events	486		90			
Heterogeneity: $Tau^2 = 0.02$; $Chi^2 = 3.01$, df = 2 (P = 0.22); $I^2 = 34\%$						
Test for overall effect: Z = 2.05 (P = 0.04)						
Test for subgroup differences: Not applicable						



Low birth weight with mild periodontitis



Periodontal Therapy May Reduce the Risk of Preterm Low Birth Weight in Women With Periodontal Disease: A Randomized Controlled Trial

Néstor J. López,* Patricio C. Smith,* and Jorge Gutierrez†

Background: Recent studies have suggested that periodontal disease is a risk factor for preterm low birth weight (PLBW). A randomized controlled trial was undertaken to help further evaluate the proposed association between periodontal disease and PLBW.

Methods: Four hundred pregnant women with periodontal disease, aged 18 to 35, were enrolled while receiving prenatal care in Santiago, Chile. Women were randomly assigned to either an experimental group (n = 200), which received periodontal treatment before 28 weeks of gestation or to a control group (n = 200) which received periodontal treatment after delivery. Previous and current pregnancies and known risk factors were obtained from patient medical records and interviews. The primary outcome assessed was the delivery at less than 37 weeks of gestation or an infant weighing less than 2,500 g.

Results: Of the 400 women enrolled, 49 were excluded from the analyses for different reasons. The incidence of PLBW in the treatment group was 1.84% (3/163) and in the control group was 10.11% (19/188), (odds ratio [OR] 5.49, 95% confidence interval [CI] 1.65 to 18.22, $P = 0.001$). Multivariate logistic regression analysis showed that periodontal disease was the strongest factor related to PLBW (OR 4.70, 95% CI 1.29 to 17.13). Other factors significantly associated with such deliveries were: previous PLBW (OR 3.98, 95% CI 1.11 to 14.21), less than 6 prenatal visits (OR 3.70, 95% CI 1.46 to 9.38), and maternal low weight gain (OR 3.42, 95% CI 1.16 to 10.03).

Conclusions: Periodontal disease appears to be an independent risk factor for PLBW. Periodontal therapy significantly reduces the rates of PLBW in this population of women with periodontal disease. *J Periodontol 2002;73:911-924.*

KEY WORDS

Clinical trials, controlled; clinical trials, randomized; infant, low birth weight; periodontal diseases/adverse effects; infant, premature; risk factors.

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† Hospital San José, Servicio de Salud Metropolitano Norte.

Low birth weight and its 2 components, preterm birth and intrauterine growth restriction, are major predictors of perinatal mortality and morbidity.[1,2] Multiple factors have been associated with preterm delivery and low birth weight[3,4] and some authors have emphasized the heterogeneity of the causes of preterm birth.[5,6]

Convincing evidence has associated preterm birth with infection, especially genito-urinary infections, which appear to be an important factor in the premature rupture of membranes.[4,7] Several studies have linked bacterial vaginosis with preterm birth.[8-10] However, treatment of vaginosis has not led to definite conclusions on its efficacy in reducing preterm delivery[11-14] and the impact of such intervention on preterm birth rate remains unclear.[15] Results of 2 case-control studies[16,17] and a concurrent cohort study[18] showed that periodontal diseases may be a potential independent risk factor for preterm birth and low birth weight after adjusting for several known risk factors.

Periodontal diseases are a group of infectious diseases resulting in inflammation of gingival and periodontal tissues and progressive loss of alveolar bone. The periodontal infection is initiated and sustained by several bacteria, predominantly Gram-negative, anaerobic and microaerophilic bacteria that colonize the subgingival area. Host defense mechanisms play an integral role in the

PERIODONTAL DISEASE AND PRE-ECLAMPSIA : A SYSTEMATIC REVIEW

Aim: The aim of this systematic review was to evaluate the published scientific evidence for this possible relationship between periodontal disease and pre-eclampsia.

Kunen A et al
J Clin Periodontal 2010

Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease: A randomized controlled study. – II.

(López et al. *J Periodontol* 2002;73:911-924.)

Incidence of Preterm Births (PTB), Low Birth Weight (LBW), Both (PLBW)

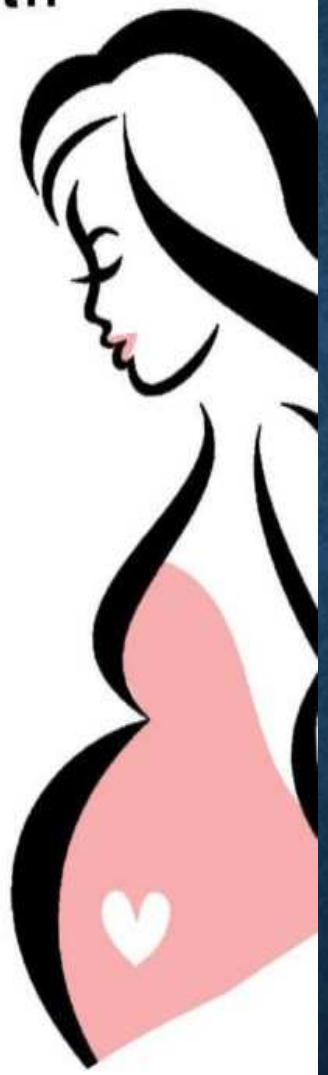
	Treatment Group (n = 163)		Control Group (n = 188)	
	N%	N	%	P Value
Intention-to-treat analysis				
PTB	21.10	12	6.38	0.017
LBW	10.55	7	3.72	0.083
PLBW	31.63	19	10.11	0.001
Protocol analysis				
PTB	21.22	12	6.38	0.001
LBW	10.61	7	3.72	0.11
PLBW	31.84	19	10.11	0.003

Odds Ratio = 5.49 (C.I. 1.65 to 18.22); P = 0.001

PLBW = Preterm/Low Birth Weight

Pregnancy & Oral Health

THE HEALTHIER MOUTH THE HEALTHIER YOUR BABIES MOUTH WILL BE TOO



KEEP YOUR MOUTH HEALTHY

Hormonal Changes while pregnant can cause yout gums to be sore, swollen and/or

Pregnancy can make some dental problems worse

MORNING SICKNESS
If you experience morning sickness, rinse with a teaspoon of baking soda mixed with water after you get sick. You want to avoid stomach acid repeatedly coming into contact with your teeth.

PREGNANCY TUMORS & YOUR GUMS
During the trimester some woman may experience overgrowths of tissue called 'pregnancy tumors' on gums. This swelling is typically found between the teeth and is believed to be caused by excess plaque.

KNOW THE FACTS

14-20 WEEKS Dental Work is safe during pregnancy and is recommended during weeks 14-20 of pregnancy.

40% Approximately 40% of pregnant women have some form of periodontal desease. Having your oral health checked while pregnant is important.

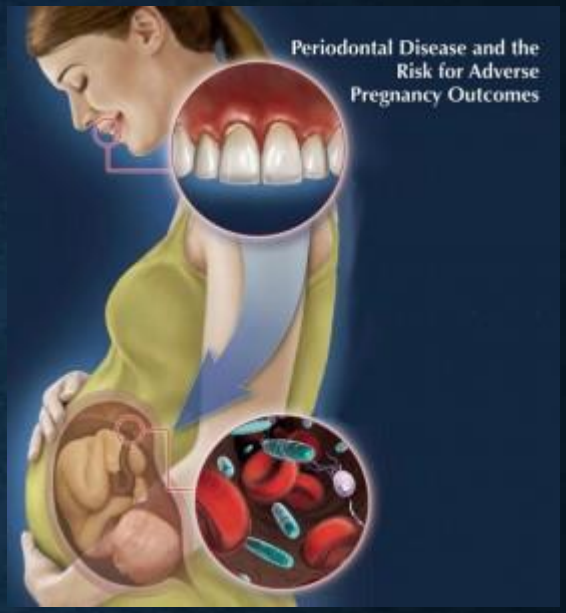
7x Studies have shown that pregnant woman with periodontal desease are 7x more at risk of delivering a preterm low birth weight baby.

Table 1. Risk Factors for Preterm Delivery

Risk factors	*Modifiable?*	*Intervention to modify risk factor improves neonatal outcomes?*
Bacterial vaginosis	Yes	No
Black race	No	—
Cocaine or heroin use	Yes	Yes
History of abdominal surgery	No	—
History of cervical conization or a loop electrosurgical excision procedure of the cervical transformation zone	No	—
History of preterm delivery	No	—
Infections of the urinary and genital tracts	Yes	Yes
Intrauterine infection	Yes	Yes
Low prepregnancy body mass index (< 19.8 kg per m^2)	Yes	Yes
Medical disorders such as thyroid disease, diabetes mellitus, or hypertension	Yes	Yes
Mother's work is physically strenuous	Yes	Yes
Multiple gestation pregnancy	No	—
Periodontal disease	Yes	No
Polyhydramnios or oligohydramnios	No	—
Sexually transmitted infections (i.e., chlamydia, gonorrhea, and trichomoniasis)	Yes	Yes
Short pregnancy interval (< 18 months between pregnancies)	Yes	Yes
Shortened cervix (< 25 mm before 28 weeks' gestation)	No	—
Tobacco use	Yes	Yes
Uterine anomalies	No	—
Vaginal bleeding caused by placental abruption or placenta previa	No	—

Information from reference 7.

(Lopez et al., 2002) investigated whether the women who had gingivitis and received treatment before birth (n=406) reduced the risk of preterm lowweight children comparing to women who had periodontal (n=233) disease and were treated after delivery. The study concluded that periodontal disease is an independent risk factor for preterm birth and low birth weight.



Lopez, NJ et al

Periodontal therapy may reduce the risk of preterm low birth weight in women with periodontal disease : a randomized controlled trial

J. Periodontology. 2002 Aug;73(8):911-924

CONCLUSION

- Thus, promotion of the early detection and treatments of periodontal disease in young women before and during pregnancy is recommended to prevent periodontal disease and their consequences on pregnancy outcomes.

- There is no significant evidence to implicate periodontal disease for occurrence of pre-ecclampsia.

- The other adverse effect on reproductive system like delayed conception, decreased sperm count, erectile dysfunction need further exploration.

Maternal Oral Therapy to Reduce Obstetric Risk (MOTOR)

- NIH funded, 1806 women, 3 sites, opened 2004
- Scaling and root planing in 2nd trimester vs within 4 weeks postpartum.
- Offenbacher, UNC
- Effect of Periodontal Therapy on Rate of Premature Delivery: A Randomized Controlled Study, (OBGYN Sept 2009)
- Conclusion: Perio therapy did not reduce incidence of prematurity

PERIODONTAL DISEASE AND FERTILITY

A study conducted at Western Australian University has indicated that women suffering from Periodontal Disease take longer time to get conceived than women with healthy periodontium suggesting that periodontal disease has adverse effect on conception and fertility.

Professor Roger Hart
European Society of Human
Reproduction and Embryology 2011

EFFECT OF PERIODONTAL DISEASE ON SPERM COUNT

- A study of 56 men aged between 23-52 by the Bikur Holim Hospital, Jerusalem, and the Hebrew University Hadassah School of Dental Medicine found that more than half the men with low sperm counts or no sperm at all displayed developed gum disease.

- The subjects also received periodontal examinations. Overall, they had poor gum health. Only 13% had healthy gums. Fully 50% suffered from gingivitis, with 32% having chronic periodontal disease and 5% aggressive periodontitis.

British Dental Health Foundation (BDHF), 2009

ASSOCIATION BETWEEN CHRONIC PERIODONTITIS AND ERECTILE DYSFUNCTION

- Periodontal disease and erectile dysfunction both share common risk factors and are associated with cardiovascular diseases.

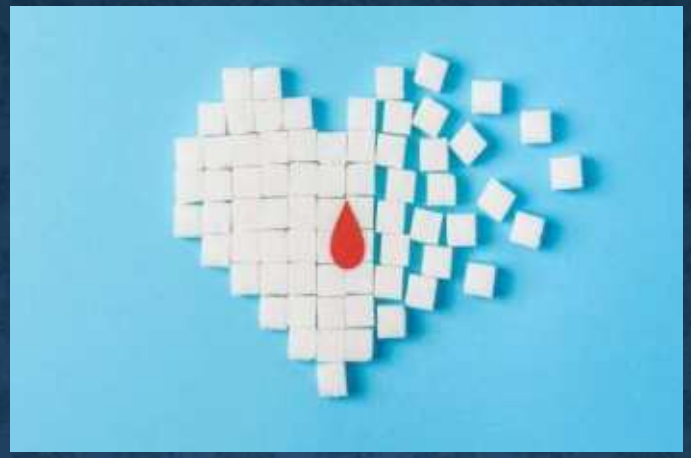
- Based on this attempts were made to asses the relationship between periodontal disease and erectile dysfunction.

CONCLUSION FOR ASSOCIATION BETWEEN PERIODONTAL DISEASE AND REPRODUCTIVE SYSTEM

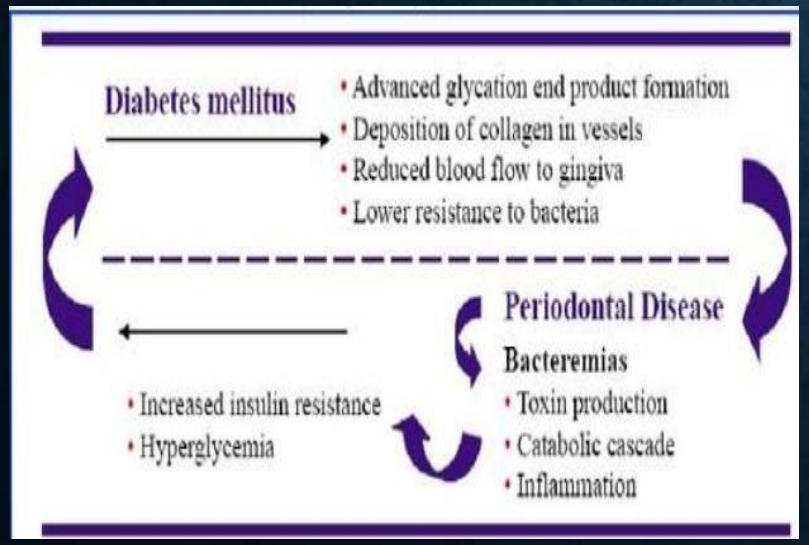
- Numerous epidemiological studies have been conducted across the world and have found an association between periodontitis and PT/LBW delivery.

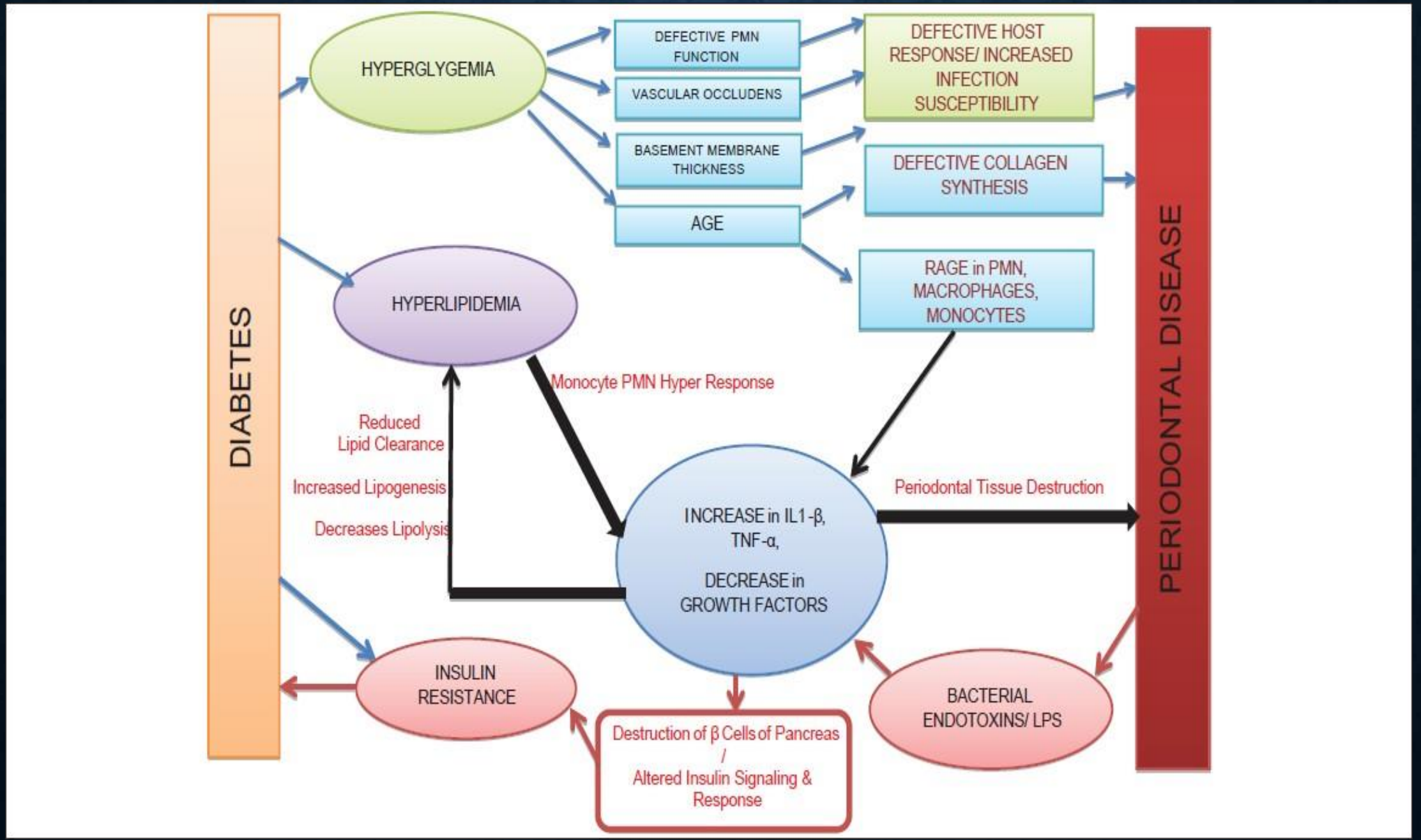
- Considering periodontal diseases as a risk factor for preterm birth, interventional studies have been performed to evaluate impact of periodontal treatment on pregnancy outcomes and conflicting results were found.

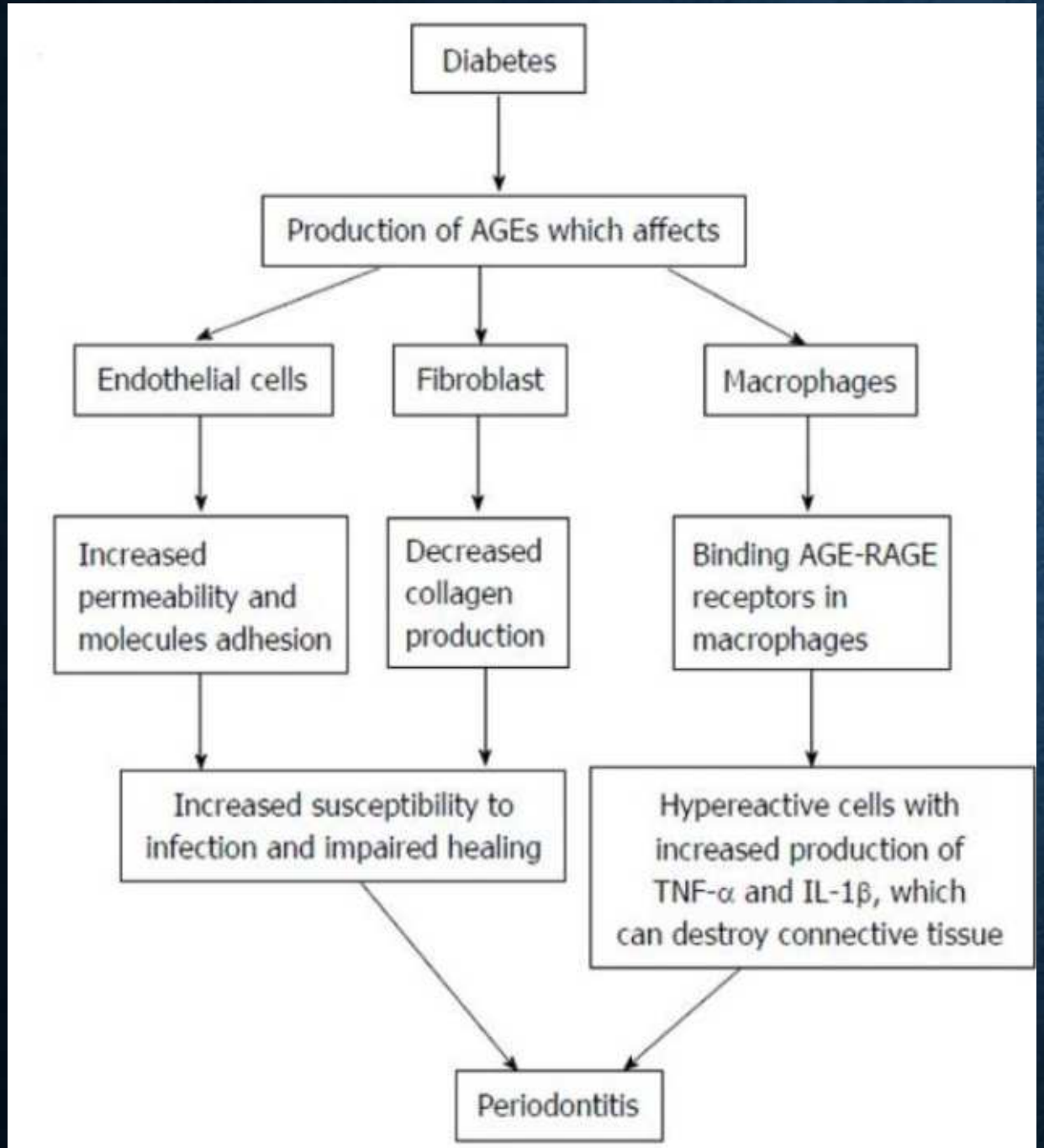
- Despite apparent conflicting data, the majority of studies report that periodontal treatment is safe for pregnant women and improve periodontal status.

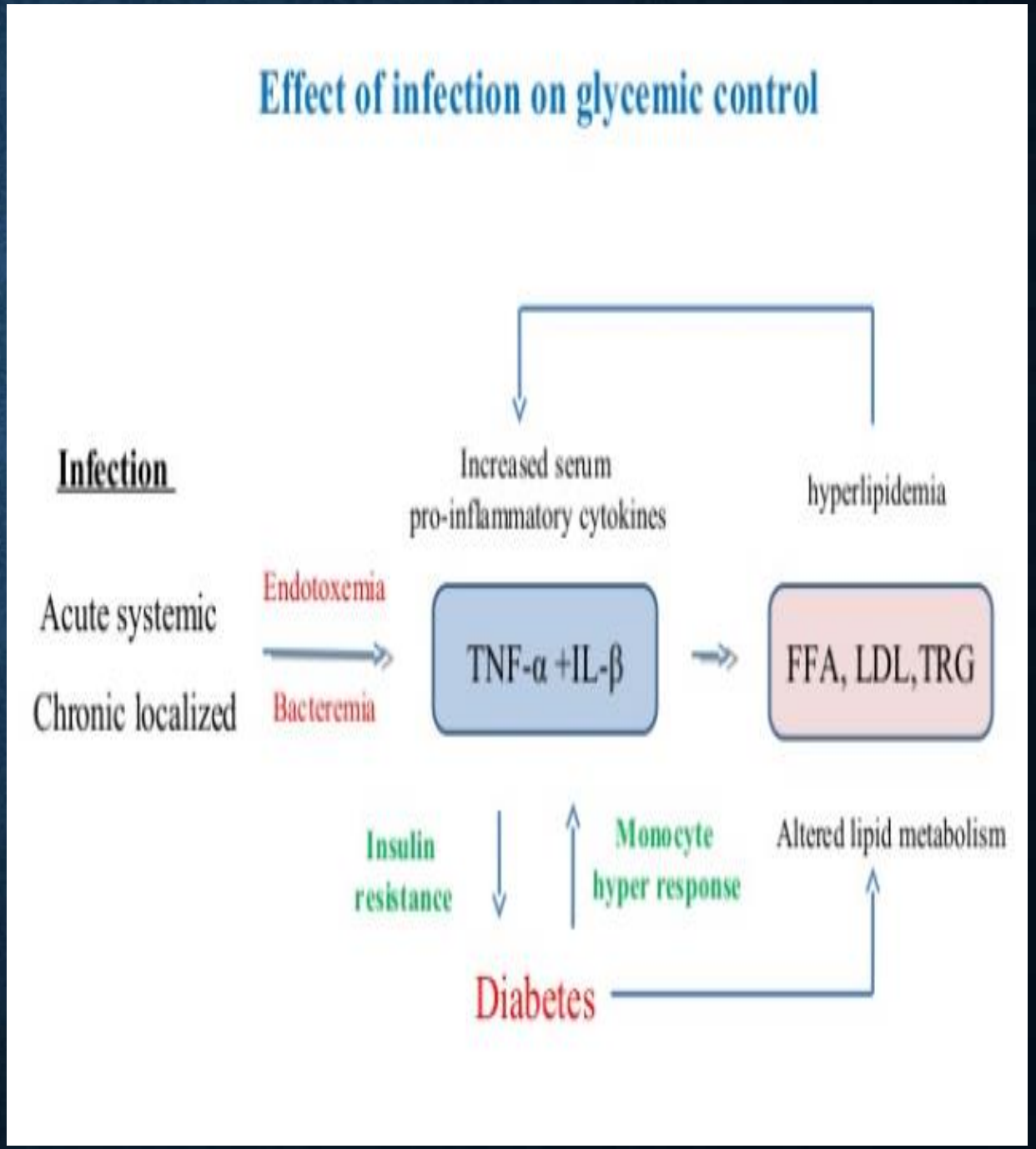


DIABETES & PERIODONTAL DISEASE




DIABETES
HYPERGLYGEMIA
DEFECTIVE PMN FUNCTION
VASCULAR OCCLUDENS
BASEMENT MEMBRANE THICKNESS
AGE
DEFECTIVE HOST RESPONSE/ INCREASED INFECTION SUSCEPTIBILITY
DEFECTIVE COLLAGEN SYNTHESIS
RAGE in PMN, MACROPHAGES, MONOCYTES
HYPERLIPIDEMIA
Monocyte PMN Hyper Response
Reduced Lipid Clearance
Increased Lipogenesis
Decreases Lipolysis
INCREASE in IL1-β, TNF-α,
DECREASE in GROWTH FACTORS
Periodontal Tissue Destruction
PERIODONTAL DISEASE
INSULIN RESISTANCE
Destruction of β Cells of Pancreas / Altered Insulin Signaling & Response
BACTERIAL ENDOTOXINS/ LPS


Diabetes
Production of AGEs which affects
Endothelial cells
Fibroblast
Macrophages
Increased permeability and molecules adhesion
Decreased collagen production
Binding AGE-RAGE receptors in macrophages
Increased susceptibility to infection and impaired healing
Hypereactive cells with increased production of TNF-α and IL-1β, which can destroy connective tissue
Periodontitis


Effect of infection on glycemic control
Infection
Increased serum pro-inflammatory cytokines
hyperlipidemia
Acute systemic
Chronic localized
Endotoxemia
Bacteremia
TNF-α +IL-β
FFA, LDL,TRG
Insulin resistance
Monocyte hyper response
Altered lipid metabolism
Diabetes

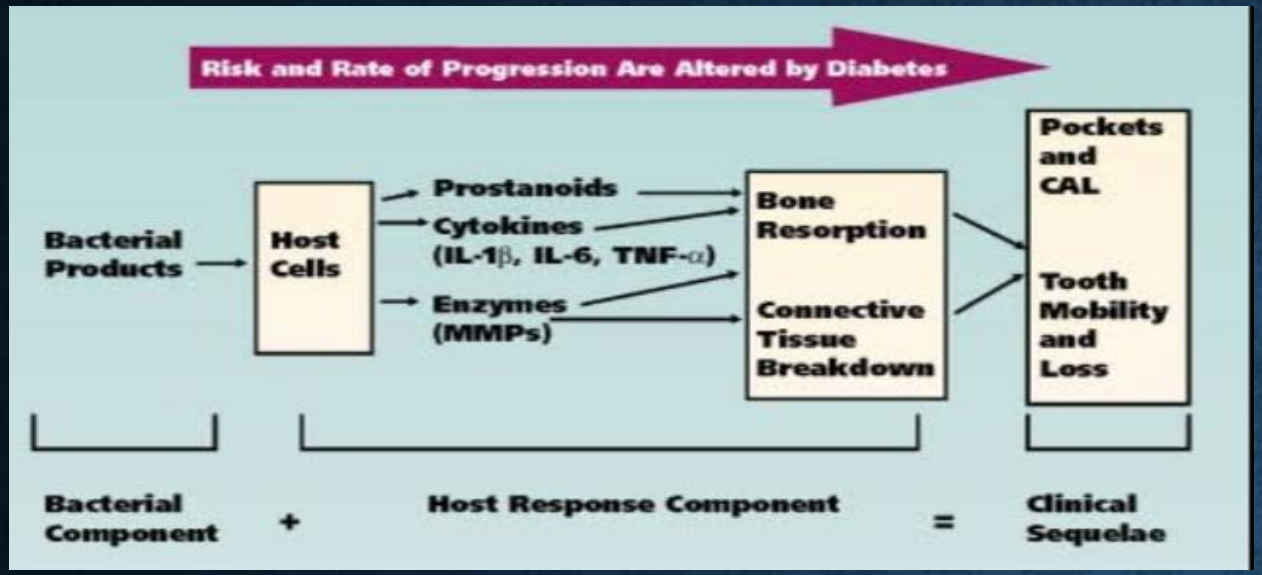
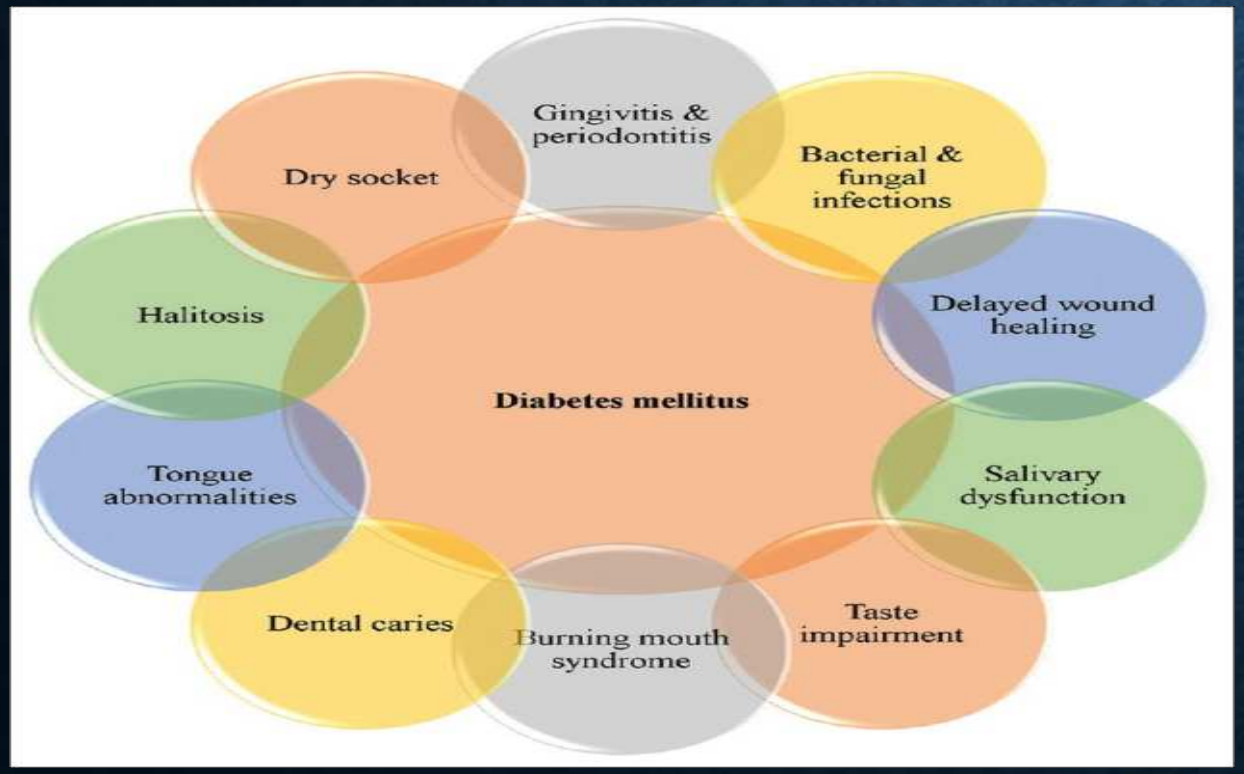
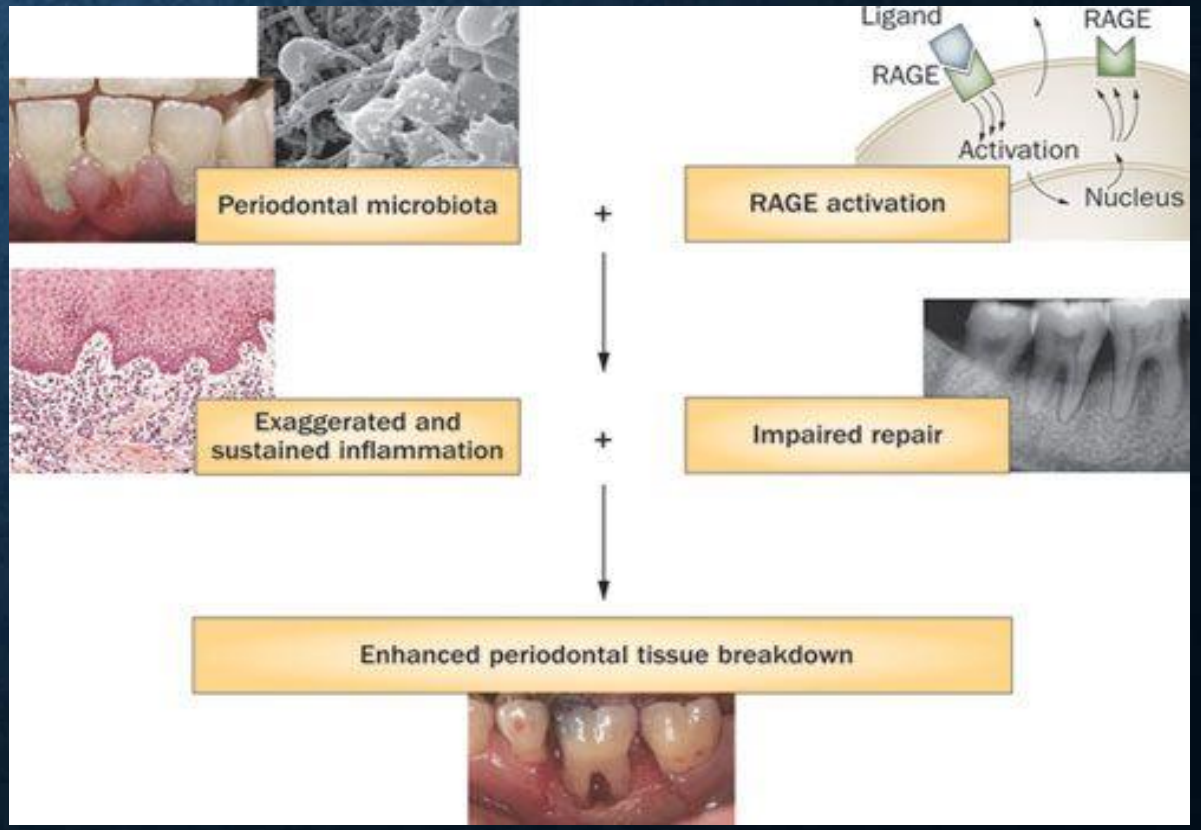


Table 1. Periodontal Findings Suggesting Undiagnosed or Poorly Controlled Diabetes.

- Enlarged, hemorrhagic tissues
- Numerous mobile teeth
- Multiple periodontal abscesses occurring at the same time
- More rapid bone loss and deepening of pockets than expected, given the patient's level of plaque and calculus
- Poor healing after periodontal treatment





J Clin Periodontol 2013; 40 (Suppl. 14): S106–S112 doi: 10.1111/jcpe.12077

Journal of Clinical Periodontology

Diabetes and periodontal diseases: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases

Iain L. C. Chapple[1], Robert Genco[2] and on behalf of working group 2 of the joint EFP/AAP workshop*

[1]Periodontal Research Group & MRC Centre for Immune Regulation, University of Birmingham School of Dentistry, Birmingham, UK; [2]University at Buffalo, Oral Biology and Microbiology and Immunology, Buffalo, NY, USA

Chapple ILC, Genco R, and on behalf of working group 2 of the joint EFP/AAP workshop. Diabetes and periodontal diseases: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases. J Clin Periodontol 2013; 40 (Suppl. 14): S106–S112. doi: 10.1111/jcpe.12077.

Abstract

Background: Diabetes and periodontitis are complex chronic diseases with an established bidirectional relationship. There is long-established evidence that hyperglycaemia in diabetes is associated with adverse periodontal outcomes. However, given the ubiquity of periodontal diseases and the emerging global diabetes epidemic, the complications of which contribute to significant morbidity and premature mortality, it is timely to review the role of periodontitis in diabetes.

Aims: To report the epidemiological evidence from cross-sectional, prospective and intervention studies for the impact of periodontal disease on diabetes incidence, control and complications and to identify potential underpinning mechanisms.

Epidemiology: Over the last 20 years, consistent and robust evidence has emerged that severe periodontitis adversely affects glycaemic control in diabetes and glycaemia in non-diabetes subjects. In diabetes patients, there is a direct and dose-dependent relationship between periodontitis severity and diabetes complications. Emerging evidence supports an increased risk for diabetes onset in patients with severe periodontitis.

Biological mechanisms: Type 2 diabetes is preceded by systemic inflammation, leading to reduced pancreatic β-cell function, apoptosis and insulin resistance. Increasing evidence supports elevated systemic inflammation (acute-phase and oxidative stress biomarkers) resulting from the entry of periodontal organisms and their virulence factors into the circulation, providing biological plausibility for the effects of periodontitis on diabetes. AGE (Advanced Glycation Endproducts)–RAGE (Receptor for AGEs) interactions and oxidative-stress-mediated pathways provide plausible mechanistic links in the diabetes to periodontitis direction.

Interventions: Randomized controlled trials (RCTs) consistently demonstrate that mechanical periodontal therapy associates with approximately a 0.4% reduction in HbA1C at 3 months, a clinical impact equivalent to adding a second drug to a pharmacological regime for diabetes. RCTs are needed with larger numbers of subjects and longer term follow-up, and if results are substantiated, adjunctive periodontal therapies subsequently need to be evaluated. There is no current evidence to support adjunctive use of antimicrobials for periodontal management of diabetes patients.

Guidelines: Given the current evidence, it is timely to provide guidelines for periodontal care in diabetes patients for medical and dental professionals and recommendations for patients/the public.

Key words: association; complications; diabetes mellitus; gestational diabetes; HbA1C; incident; intervention; mechanisms; periodontal disease; periodontitis; type 2 diabetes

Accepted for publication 14 November 2012

The proceedings of the workshop were jointly and simultaneously published in the Journal of Clinical Periodontology and Journal of Periodontology.

The Relationship Between Diabetes Mellitus and Destructive Periodontal Disease: A Meta-Analysis

Nilo Guliberto Martins Chávarry[a,b]/Mario Vianna Vettore[c]/ Carmelo Sansone[a]/Aubrey Sheiham[d]

Purpose: The aim of this study was to systematically review the studies on the association between diabetes mellitus (DM) and destructive periodontal disease.

Methods: The methods applied include a literature search strategy, inclusion and exclusion criteria for selecting the studies, characteristics of the studies, quality assessment and meta-analysis. Data sources included PubMed, EMBASE, SciELO and LILACS. Selected papers were articles relating to human studies investigating whether or not diabetes is a risk factor for periodontitis and if it influences the response to periodontal therapy. Those papers that were published between January 1980 and June 2007 were retrieved.

Results: Of the 2440 identified studies, 49 cross-sectional and eight longitudinal studies met the inclusion criteria. Twenty-seven of the 49 cross-sectional studies that are included in this review detected more periodontal disease in diabetic subjects compared with non-diabetic subjects. The greater risk of periodontal disease progression was associated with type 2 DM, and one study associated DM with response to periodontal therapy. Methodological flaws of most of the studies included inadequate control for confounders, insufficient statistical analysis and lack of information about sampling design. Random effect model showed a significant association with clinical attachment level (mean difference = 1.00 [CI 95% = 0.15 to 1.84]) and periodontal pocket depth (mean difference = 0.46 [CI 95% = 0.01 to 0.91]) between type 2 diabetics and non-diabetics.

Conclusions: Type 2 DM can be considered a risk factor for periodontitis. More studies are needed to confirm the harmful effects of type 1 DM on periodontal disease.

Key words: diabetes mellitus, meta-analysis, periodontal disease, periodontitis, systematic review

Oral Health Prev Dent 2009; 7: 107–127. *Submitted for publication: 24.12.07; accepted for publication: 03.03.08.*

Moderate chronic periodontal disease is common in most countries (Albandar, 2002a, b; Sheiham and Netuveli, 2002). Poor oral hygiene and bacterial biofilms composed of anaerobic Gram negative microorganisms are the main causative factors for periodontal breakdown (Christersson, 1993). In addition, the habit of smoking and diabetes mellitus (DM) have been described as the risk factors for periodontal disease initiation and progression (Obeid and Bercy, 2000; Albandar, 2002a, b). Other putative risk factors for periodontal disease include psychosocial factors, socioeconomic conditions and age (Albandar, 2002a, b; Vettore et al, 2003).

Most narrative review papers claim an association between DM and periodontal disease (Belting et al, 1964; Campbell, 1967; Hugoson and Jordan, 1982; Mealey and Oates, 2006). However, these review papers did not follow adequate protocols that are applied in systematic reviews (Alderson et al,

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Accepted: 4 July 2017
DOI: 10.1111/jcpe.12808

ORIGINAL ARTICLE

WILEY Journal of Clinical Periodontology

Scientific evidence on the links between periodontal diseases and diabetes: Consensus report and guidelines of the joint workshop on periodontal diseases and diabetes by the International Diabetes Federation and the European Federation of Periodontology

Mariano Sanz[1] | Antonio Ceriello[2,3] | Martin Buysschaert[4] | Iain Chapple[5] | Ryan T. Demmer[6] | Filippo Graziani[7] | David Herrera[1] | Søren Jepsen[8] | Luca Lione[9] | Phoebus Madianos[10] | Manu Mathur[11] | Eduard Montanya[12] | Lior Shapira[13] | Maurizio Tonetti[14] | Daniel Vegh[15]

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Sanz, Mariano; Ceriello, Antonio; Buysschaert, Martin; Chapple, Iain; Demmer, Ryan T.; Graziani, Filippo; Herrera, David; Jepsen, Søren; Lione, Luca; Madianos, Phoebus; Mathur, Manu; Montanya, Eduard; Shapira,

Abstract

Background: Diabetes and periodontitis are chronic non-communicable diseases independently associated with mortality and have a bidirectional relationship.

Aims: To update the evidence for their epidemiological and mechanistic associations and re-examine the impact of effective periodontal therapy upon metabolic control (glycated haemoglobin, HbA1C).

Epidemiology: There is strong evidence that people with periodontitis have elevated risk for dysglycaemia and insulin resistance. Cohort studies among people with diabetes demonstrate significantly higher HbA1C levels in patients with periodontitis (*versus* periodontally healthy patients), but there are insufficient data among people with type 1 diabetes. Periodontitis is also associated with an increased risk of incident type 2 diabetes.

DOES PERIODONTAL TREATMENT IMPROVE GLYCEMIC CONTROL IN DIABETIC PATIENTS? A META-ANALYSIS OF INTERVENTION STUDIES

- This meta-analysis was initiated on 10 intervention studies to quantify the effects of periodontal treatment on HbA1c level among diabetic patients, to explore possible causes for the discrepant reports, and to make recommendations for future studies.

S.-J. Janket, A. Wightman, A.E. Baird, T.E. Van Dyke and J.A. Jones

J Dent Res 2005 84: 1154

Does Periodontal Treatment Improve Glycemic Control in Diabetic Patients? A Meta-analysis of Intervention Studies

S.-J. Janket, A. Wightman, A.E. Baird, T.E. Van Dyke and J.A. Jones

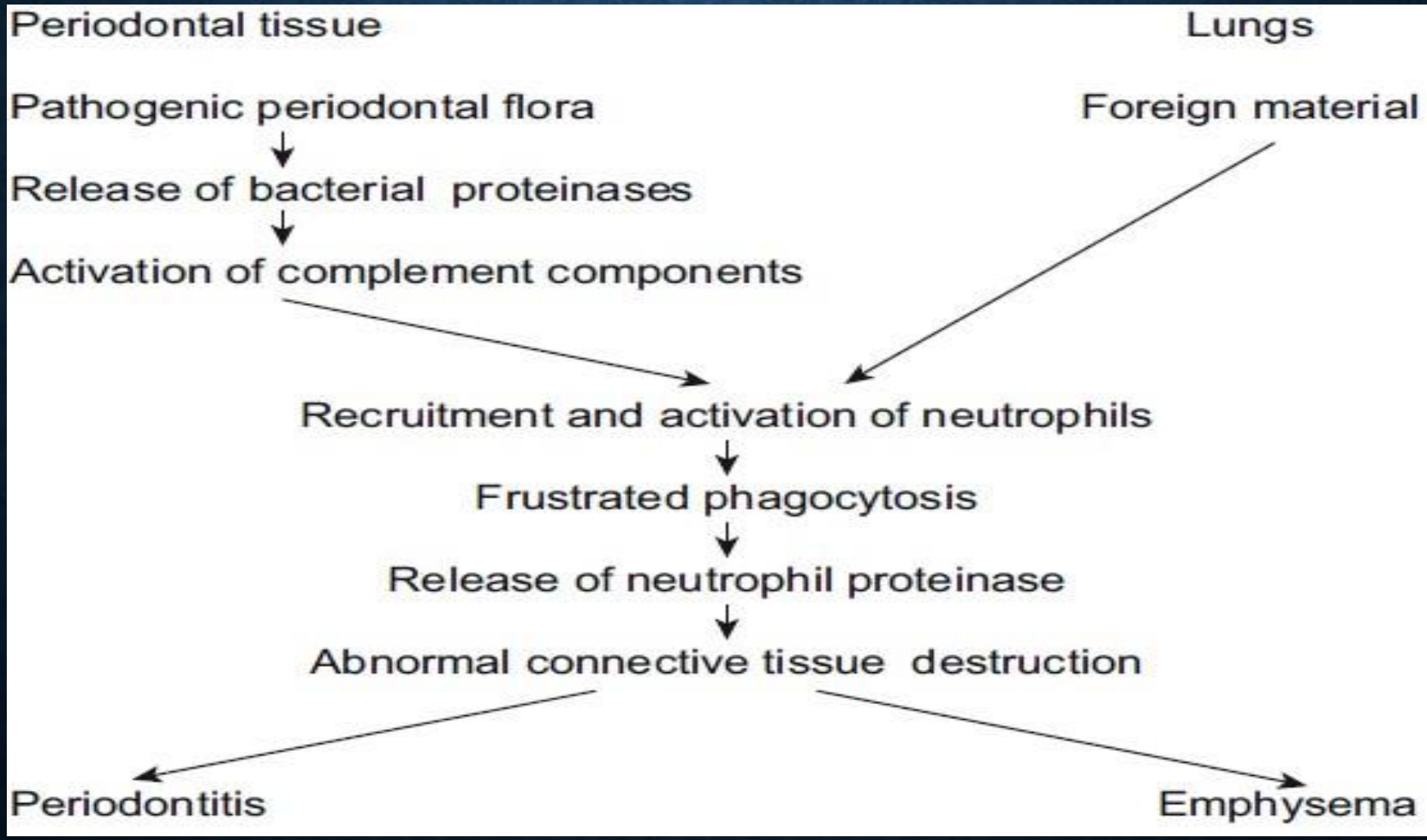
J DENT RES 2005 84: 1154

Conclusion

- Periodontal therapy with antibiotics appeared to decrease HbA1c levels by statistically non-significant 0.71% among patients with type 2 diabetes.
- Although this percent improvement in glycemic control may be of value to some patients, the evidence currently available was not strong enough for us to reject the null hypothesis, "periodontal treatment does not affect glycemic control in patients with diabetes".
- The present meta-analysis suggests that periodontal treatment leads to an improvement of glycemic control in type 2 diabetic patients for atleast 3 months.

RESPIRATORY SYSTEM AND PERIODONTAL DISEASE

RESPIRATORY SYSTEM AND PERIODONTAL DISEASE



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Risk of Periodontal Diseases in Patients With Chronic Obstructive Pulmonary Disease

A Nationwide Population-based Cohort Study

Te-Chun Shen, MD, Pei-Ying Chang, MD, Cheng-Li Lin, MS, Chia-Hung Chen, MD, Chih-Yen Tu, MD, Te-Chun Hsia, MD, Chuen-Ming Shih, MD, PhD, Wu-Huei Hsu, MD, Fung-Chang Sung, MPH, PhD, and Chia-Hung Kao, MD

Abstract: Several studies have reported an association between chronic obstructive pulmonary disease (COPD) and periodontal diseases. However, a large-scale population-based cohort study was previously absent from the literature. Therefore, we evaluated the risk of periodontal diseases in patients with COPD in a nationwide population.

From the National Health Insurance claims data of Taiwan, we identified 22,332 patients with COPD who were newly diagnosed during 2000 to 2010. For each case, two individuals without COPD were randomly selected and frequency matched by age, sex, and diagnosis year. Both groups were followed up till the end of 2011.

The overall incidence of periodontal diseases was 1.19-fold greater in the COPD group than in the comparison group (32.2 vs 26.4 per 1000 person-years; 95% confidence interval [CI] 1.15−1.24). Compared with non-COPD patients, the adjusted hazard ratios of patients with COPD increased with the number of emergency room visits (from 1.14 [95% CI 1.10−1.19] to 5.09 [95% CI 4.53−5.72]) and admissions (from 1.15 [95% CI 1.10−1.20] to 3.17 [95% CI 2.81−3.57]). In addition, the adjusted hazard ratios of patients with COPD treated with inhaled corticosteroids (1.22, 95% CI 1.11−1.34) and systemic corticosteroids (1.15, 95% CI 1.07−1.23) were significantly higher than those of patients not treated with corticosteroids.

Patient with COPD are at a higher risk of developing periodontal diseases than the general population. Our results also support that the risk of periodontal diseases is proportional to COPD control. In addition, patients who receive corticosteroid treatment are at a higher risk of developing periodontal diseases.

(*Medicine* 94(46):e2047)

Abbreviations: CAD = coronary artery disease, CD8 = cluster of differentiation 8, CI = confidence interval, CKD = chronic kidney disease, COPD = chronic obstructive pulmonary disease, ER = emergency room, HR = hazard ratio, ICD-9-CM = International Classification of Disease 9th Revision Clinical Modification, LHID2000 = Longitudinal Health Insurance Database 2000, NHI = National Health Insurance, NHRI = National Health Research Institutes.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is characterized by persistent and usually progressive airflow limitation and is associated with an enhanced chronic inflammatory response in the airways and the lungs. Comorbidities that occur frequently in patients with COPD include certain infections, cardiovascular diseases, diabetes, metabolic syndrome, skeletal muscle dysfunction, osteoporosis, anxiety, depression, impaired cognitive function, and lung cancer.[1] These comorbid conditions can have a significant impact on the prognosis of the disease.

Periodontal diseases are defined as any disorder of the tissues surrounding and supporting the teeth; the term usually refers to the inflammatory disorders of gingivitis and periodontitis.[2] These diseases are highly prevalent and may affect up to 90% of the worldwide population.[3] They are mainly caused by the bacterial biofilm that accumulates on the teeth, but other genetic and environmental factors also contribute to the conditions.[4,5] Compared with COPD, similar comorbid diseases such as cardiovascular diseases, diabetes, and osteoporosis have been reported in association with periodontal diseases.[6–8]

Patients who have an underlying respiratory disorder may face some special challenges in establishing and maintaining oral health. These factors include the illness itself and the associated medical therapies.[9] Several studies have reported the association between COPD and periodontal diseases.[10–14] For example, Zeng et al[12] recently performed a meta-analysis using 14 observational studies and identified a significant association between periodontal diseases and COPD (odds ratio (OR) 2.08, 95% confidence interval [CI] 1.48−2.91, $P < 0.001$).

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F-CS and C-HK contributed equally.
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Conception and design: TCS, PYC, CHC, and CYT; administrative support: TCH, CMS, WHH, FCS, and CHK; collection and assembly of data: all authors; data analysis and interpretation: TCS, CLL, FCS, and CHK; manuscript writing: TCS, PYC, CLL, FCS, and CHK; final approval of manuscript: all authors.
There are no conflicts of interests.

ISSN: 0025-7974
DOI: 10.1097/MD.0000000000002047

BIOLOGIC PLAUSIBILITY

- Dental Plaque may serve as a reservoir for pulmonary pathogens responsible for aspiration pneumonia in high risk patients, for eg, intensive care, dentate elderly with poor oral health, residents of long term care facilities.

Azarapazhooh et al.

A Systematic Review of the Preventive Effect of Oral Hygiene on Pneumonia and Respiratory Tract Infection in Elderly People in Hospitals and Nursing Homes: Effect Estimates and Methodological Quality of Randomized Controlled Trials

Sjögren P, Nilsson E, Forsell M, Johansson O, Hoogstraate J.

Journal of the American Geriatrics Society
Volume 56, Issue 11 pages 2124–2130, November 2008

Periodontal Disease and Risk of Chronic Obstructive Pulmonary Disease: A Meta-Analysis of Observational Studies

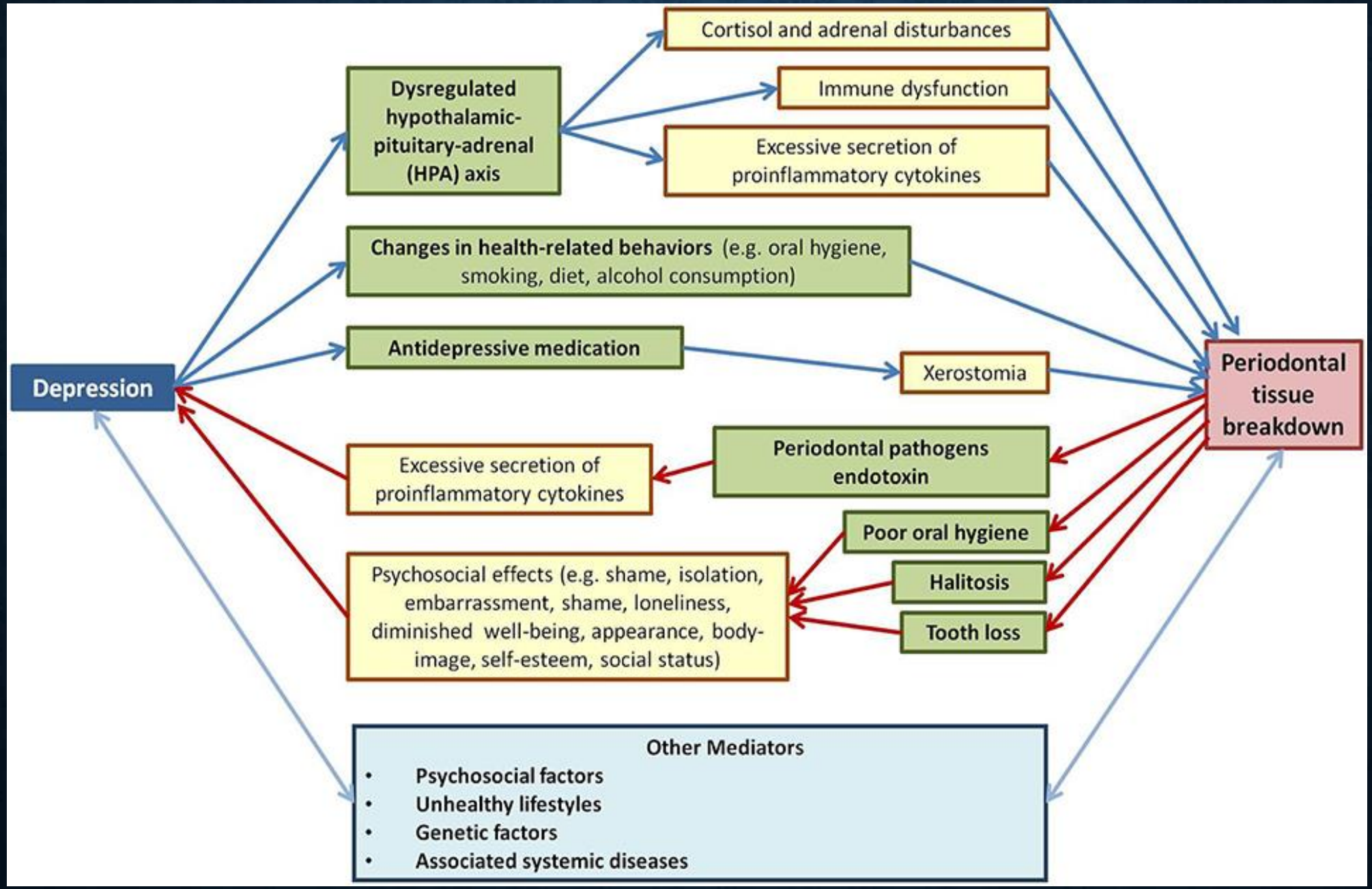
PLUSONE october 2012

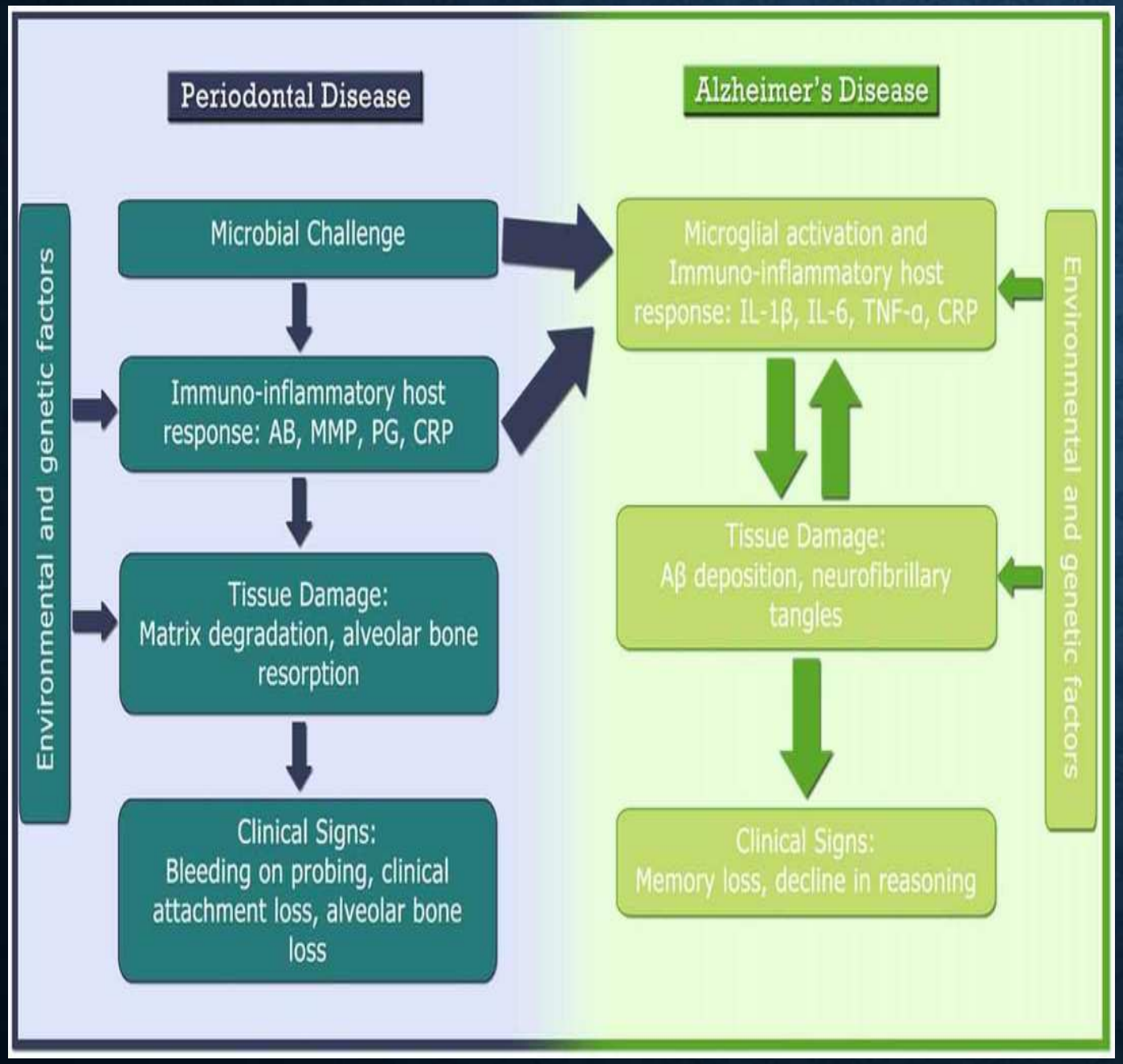
Xian-Tao Zeng,Ming-Li Tu,Dong-Yan Liu, Dong , Zheng, Jing Zhang, Wei Dong Leng

CONCLUSION

- Based on current evidence, Periodontal disease is a significant and independent risk factor of COPD.
- However, whether a causal relationship exists, remains unclear. Moreover, we suggest performing randomized controlled trails to explore whether periodontal interventions are beneficial in regulating COPD pathogenesis and progression.

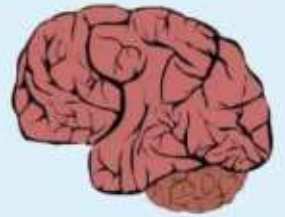
NERVOUS SYSTEM AND PERIODONTAL DISEASE


Depression
Dysregulated hypothalamic-pituitary-adrenal (HPA) axis
Cortisol and adrenal disturbances
Immune dysfunction
Excessive secretion of proinflammatory cytokines
Changes in health-related behaviors (e.g. oral hygiene, smoking, diet, alcohol consumption)
Antidepressive medication
Xerostomia
Periodontal tissue breakdown
Excessive secretion of proinflammatory cytokines
Periodontal pathogens endotoxin
Poor oral hygiene
Halitosis
Tooth loss
Psychosocial effects (e.g. shame, isolation, embarrassment, shame, loneliness, diminished well-being, appearance, body-image, self-esteem, social status)
Other Mediators
Psychosocial factors
Unhealthy lifestyles
Genetic factors
Associated systemic diseases



1. Alzheimer's Disease

Alzheimer's is a progressive, degenerative brain disease that gradually destroys a person's ability to think, reason, and recall memories.



Alzheimer's disease develops as a result of loss of neurons and synapses in brain.

- Alzheimer's disease accounts for 60-80% of all the cases. Currently, more than five million Americans have Alzheimer's.
- Alzheimer's disease is diagnosed with the help of a complete medical history, medical imaging and blood tests to rule out other probable causes.

PERIODONTAL DISEASE & CEREBROVASCULAR ACCIDENT

- A **stroke**, or **cerebrovascular accident** (**CVA**), is the rapid loss of brain function due to disturbance in the blood supply to the brain. This can be due to ischemia (lack of blood flow) caused by blockage (thrombosis, arterial embolism), or a haemorrhage
- Biological plausibility of this is same as that of cardiovascular disease

Association between periodontal disease and stroke- a meta-analysis

Sfyroeras GS, Roussas N, Saleptsis VG, Argyriou C, Giannoukas AD.
Journal of Vascular Surgery
Volume 55, Issue 4, Pages 1178–1184

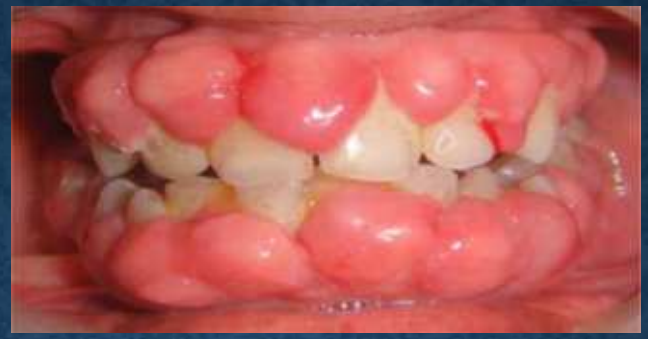
RETROSPECTIVE STUDY INCLUDED IN META ANALYSIS

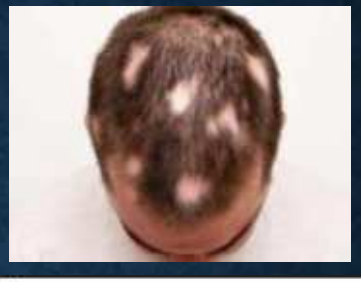
Authors	*Study population*	*Periodontal diagnosis*
Grau et al[15]	Case-control study: 303 patients with cerebrovascular ischemia, 300 population and 168 hospital controls aged 18-75 years	CAL ≥6 mm
Elter et al[16]	9415 dentate and 1491 edentulous adults	Periodontal AL, measured at 6 sites per tooth. Number of sites with AL 3 + mm/measured sites, X 100
Sim et al[17]	Case-control study: 265 patients with nonfatal chronic stroke and 214 controls	CAL ≥6 mm
Lee et al[18]	5123 subjects aged >60 years	Number of teeth CAL ≥2 mm and CAL ≥3 mm
Dorfer et al[19]	Case-control study: 303 consecutive patients with ischemic stroke or TIA, and 300 controls aged 18-75 years	Number of teeth, CAL ≥6 mm, radiographic bone loss, gingival index
Pradeep et al[20]	Case-control study: 100 patients and 100 controls aged 33-68 years	Plaque index, gingival index, probing pocket depth and CAL
Kim et al[21]	Case-control study: 165 patients and 214 nonstroke control subjects	CAL

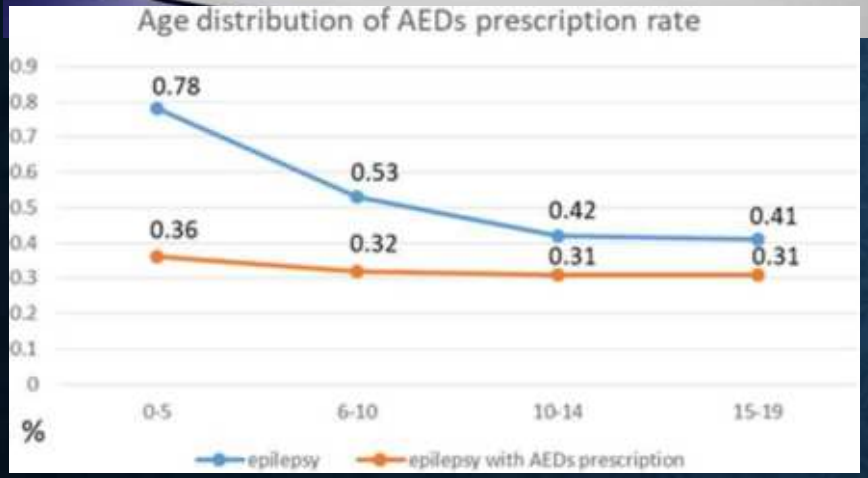
BMI, Body mass index; *CAL*, clinical attachment level; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein; *TIA*, transient ischemic attack.

Once the seizure is over :

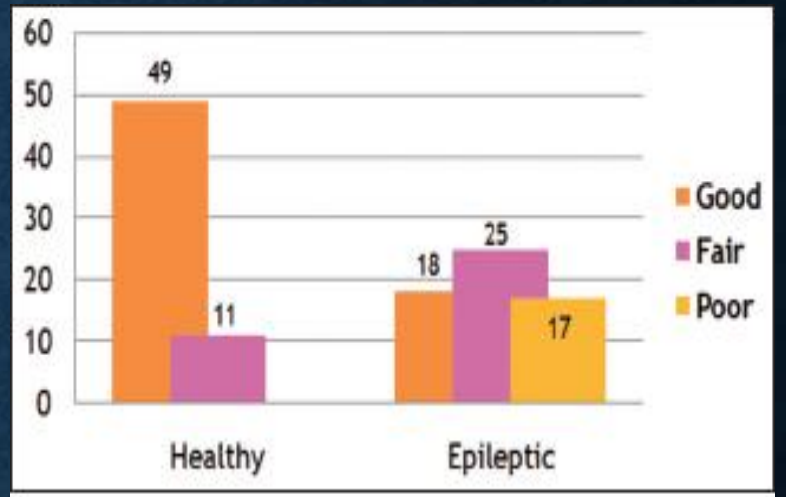
1. Do not undertake further dental treatment that day.
2. Try to talk to the patient to evaluate the level of consciousness .
3. Do not allow the patient to leave the office if his or her level of awareness is not fully restored.
4. Contact the patient's family, if he or she is alone .
5. Do a brief oral examination for sustained injuries.







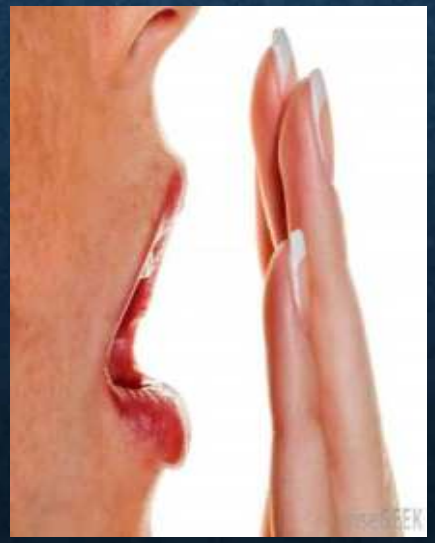
ALOPECIA-EPILEPSY-PYORRHEA-INTELLECTUAL DISABILITY SYNDROME

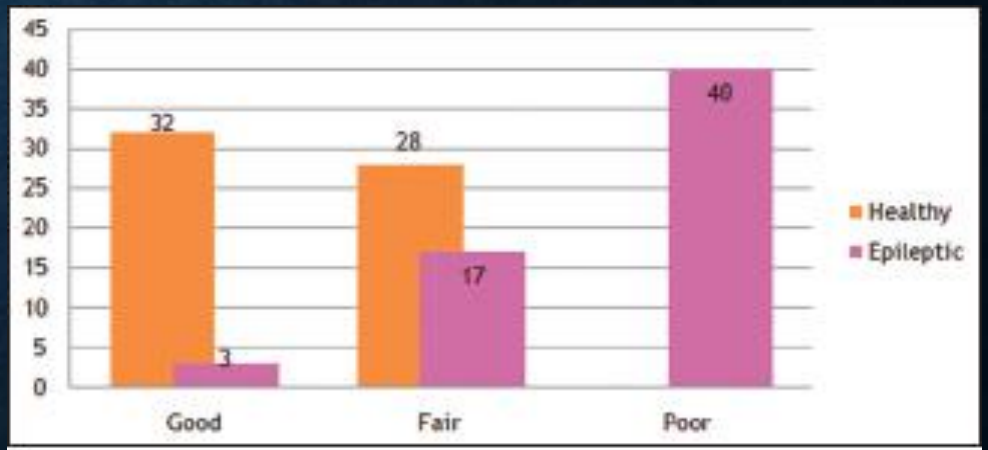


Graph 1: Comparison of oral hygiene status between healthy and epileptic group

Problems that a Dentist May Encounter

- ***Trauma***
- Generalized tonic–clonic seizures often cause minor oral injuries, such as tongue biting,16 but also frequently lead to tooth injuries17 and in some cases to maxillofacial trauma.18
- Patients with epilepsy can be at increased risk of fracture because enzyme-inducing antiepileptic drugs (e.g., phenytoin, phenobarbital, carbamazepine) alter the metabolism and clearance of vitamin D and have been associated with osteopenia and osteomalacia.





Graph 2: Comparison of the plaque index score between healthy and epileptic group

Surgical Science, 2012, 3, 47-52
http://dx.doi.org/10.4236/ss.2012.31008 Published Online January 2012 (http://www.SciRP.org/journal/ss)

Management of Epileptic Patients in Dentistry

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ABSTRACT

Epilepsy has direct negative effects on sufferers' general dental condition and oral health, both of which are further affected by inadequate oral hygiene; poor oral hygiene itself is often also caused by epilepsy-related poor health. Consequently, tooth loss, caries and periodontal disease occur increasingly often in epilepsy sufferers and they need more dental treatment. However, in fact the epileptic patients can receive fewer and simpler treatment modalities. The aim of this study was to review and synthesize recent studies on dental treatment in epilepsy patients and to mention potential triggers for seizures in dental practice.

Keywords: Epilepsy; Dental; Seizure; Prosthodontic Treatment

1. Introduction

The human brain consists of millions of neurons, their extensions, and the supportive tissues found between those neurons. All brain cells have the ability to produce electrical currents and conduct them to other cells. It is by transmitting such electrical signals that the brain functions. In other words, it is the conduction of these electrical currents that enables us to act, to speak and to feel [1,2].

Seizures can be defined as the discontinuity of normal brain functions due to sudden electrical discharges which may be either excessive or inadequate; these result in episodic convulsions (such as involuntary motion), disturbances in perception, or alterations in consciousness. The outcome of such excessive discharge during electrical conduction is called seizure [2-4].

Epilepsy is a disease that involves seizures which are characterized by an alteration of perception, behavior and mental activities, as well as by involuntary muscle contractions, temporary loss of consciousness and chronic changes in neurological functions that result from abnormal electrical activity in the brain [3,5,6]. Epileptic seizures are reversible and recur frequently [2].

For centuries epilepsy was thought to be a disease related to the supernatural. Although Hippocrates claimed that epilepsy is a naturally occurring disease, the misbelieve that the cause of epilepsy was supernatural was common until the neuropathologic origin of epilepsy was reported in the 19th century [7]. In the early part of that century, John Hughlings Jackson defined Jocksonian seizures; in the middle of the 19th century, Robert Bently Todd defined the paralyses that may develop after long-term seizures [2]. Today, the diagnosis of an epileptic patient required at least three seizure episodes [1].

The aim of this study was to review and synthesize recent studies on dental treatment in epilepsy patients and outline the special concerns that dentists should take into account when providing care to these patients.

We searched the dental literature with Medline/Pubmed with an emphasis on peer-reviewed journals and Science Citation Index Expanded. Key words used were epilepsy, dental, seizure and prosthodontic treatment. We also scrutinized common textbooks on removable and fixed proshodontics. For additional information a hand search from relevant data were searched, too.

2. Epidemiology and Prevalence

Epilepsy is a disease that is frequently encountered by oral and maxillofacial surgery practices [2]. It is thought to affect millions of people worldwide, and has a prevalence of 0.5% - 0.9% in the general population [1,2,8]. Chapman *et al.* have reported that, epileptic seizures are the second most common medical incident in dental surgeries. They have stated that statistically every dentist notice in his/her professional life 1.5 times generalized tonic-clonic seizures by the patients [9].

It has been reported that the disease occurs independent of race, age and gender [2,10]. However, epilepsy has been occur more frequently in men than in women [2,5, 10,11].

Epilepsy has been observed most frequently in children under 1 year of age and in people over the age of 75

OBESITY AND PERIODONTAL DISEASE

Obesity and periodontitis

- Obesity leads to hyperglycemia what will lead to ROS production.
- There is an increased metabolic rate that produces high ROS levels.
- LDL and fatty acids are incremented both in obesity and periodontitis.
- There is a higher NADPH oxidase activity in obesity.
- Obesity and periodontitis ROS production together can cause an oxidative stress situation

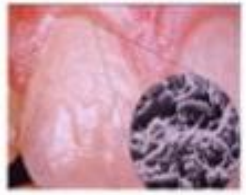
Alterations in enteral microflora

Adipocyte inflammation production

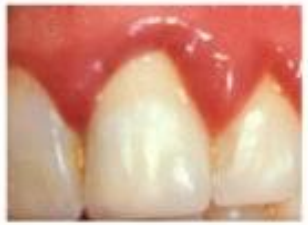
Dietary Factors Affecting Oral Bacterial Food Source Substrate

Other Genetic and Environmental Factors

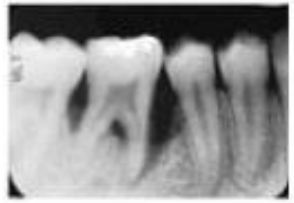
Pathologic oral flora



Local Gingival Inflammation



Immune System Host Response



Periodontal Disease Progression

Etiologic Factors and Pathophysiology of Obesity

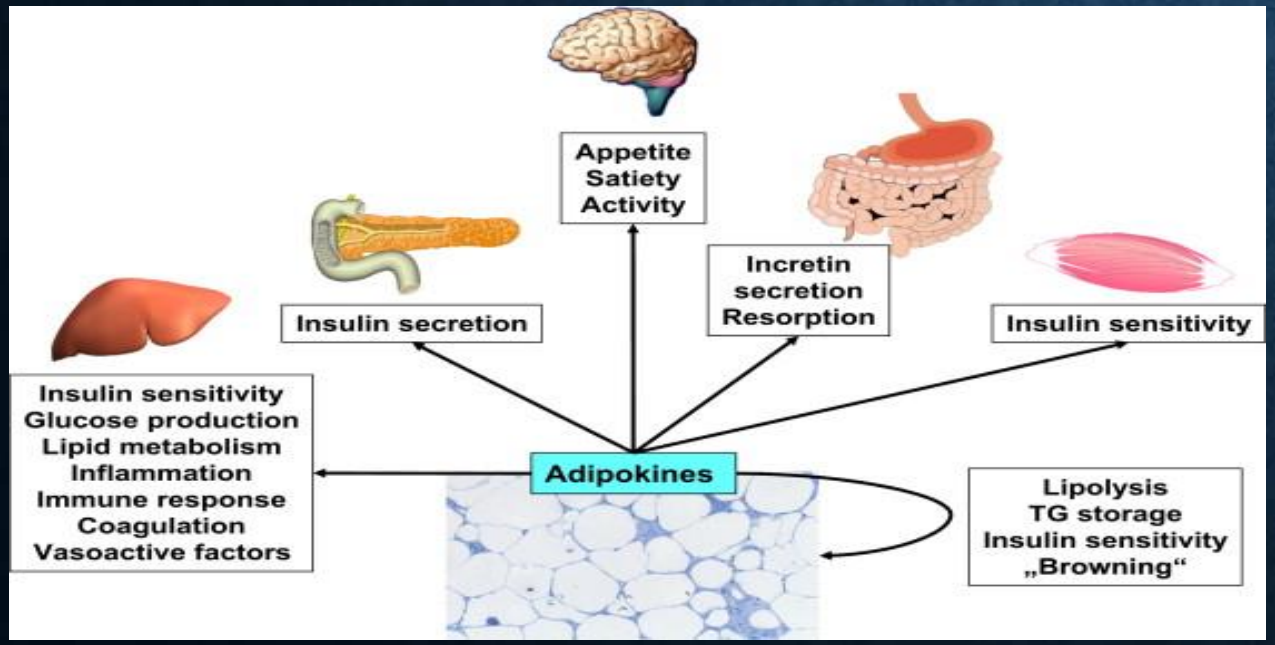
Etiologic Factors and Pathophysiology of Chronic Periodontitis

Clinical Disease

OBESITY

- Reactive oxygen species are products of normal cellular metabolism but over-production of reactive oxygen species induces damage by oxidizing DNA, lipids and proteins.
- Obesity increases the circulation of reactive oxygen species which in turn causes gingival oxidative damage and progression of periodontitis

Parveen Dahiya, Reet Kamal, and Rajan GuptaObesity, periodontal and general health: Relationship and management.



Diet → Free fatty acids → Obesity

1. Adipocytes secrete proinflammatory cytokines into the plasma

TNFα

2. TNFα inhibits insulin signaling leads to IR.[45] Also, free fatty acids cause apoptosis of B cells via ceramide and nitrous oxide, leading to IR

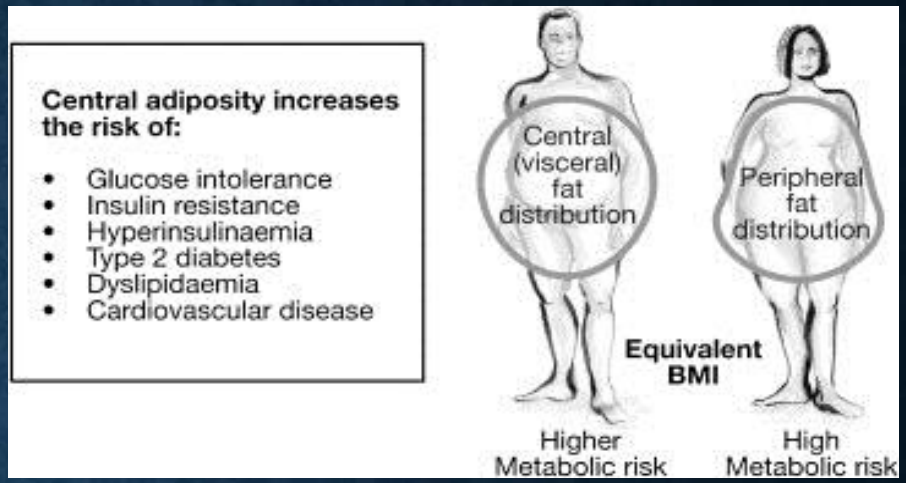
Insulin resistance (IR)

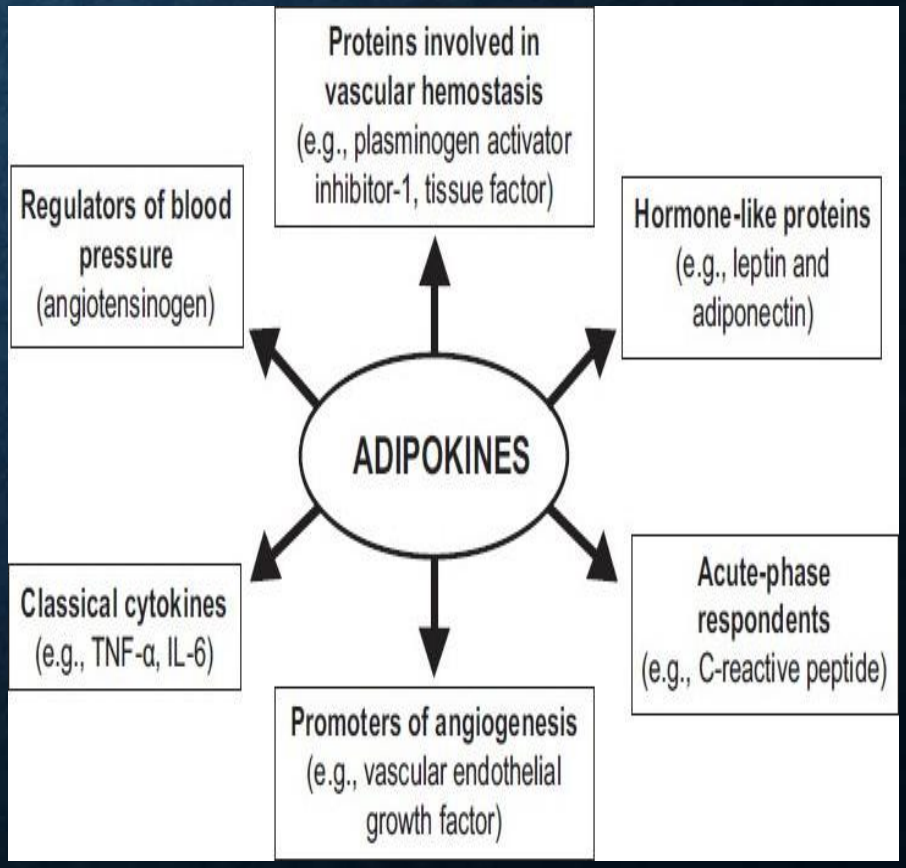
Diabetes mellitus

3. Diabets associated with a hyperinflammatory state

Periodontal infections

4. Periodontal tissues primed by hyperinflammatory state and exhibit exaggerated response to infecting organisms





PERIODONTOLOGY 2000

Obesity and periodontal disease

Christine Seel Ritchie

Definition and current prevalence

Classification

Obesity is an excess amount of body fat in proportion to lean body mass, to the extent that health is impaired (31). The most commonly used measure of body fat is the body mass index, which is defined as a person's weight, in kilograms, divided by the square of his/her height in meters. The World Health Organization and the National Heart, Lung and Blood Institute (NHLBI) define overweight as a body mass index of 25–29.9 and obesity as a body mass index of ≥30 (58, 88). Childhood obesity is defined as a body mass index for age and gender that is greater than the 95th percentile (6). The full classification for overweight and obesity, developed by the National Institutes of Health through an expert panel that reviewed data from approximately 300 studies, is shown in Table 1.

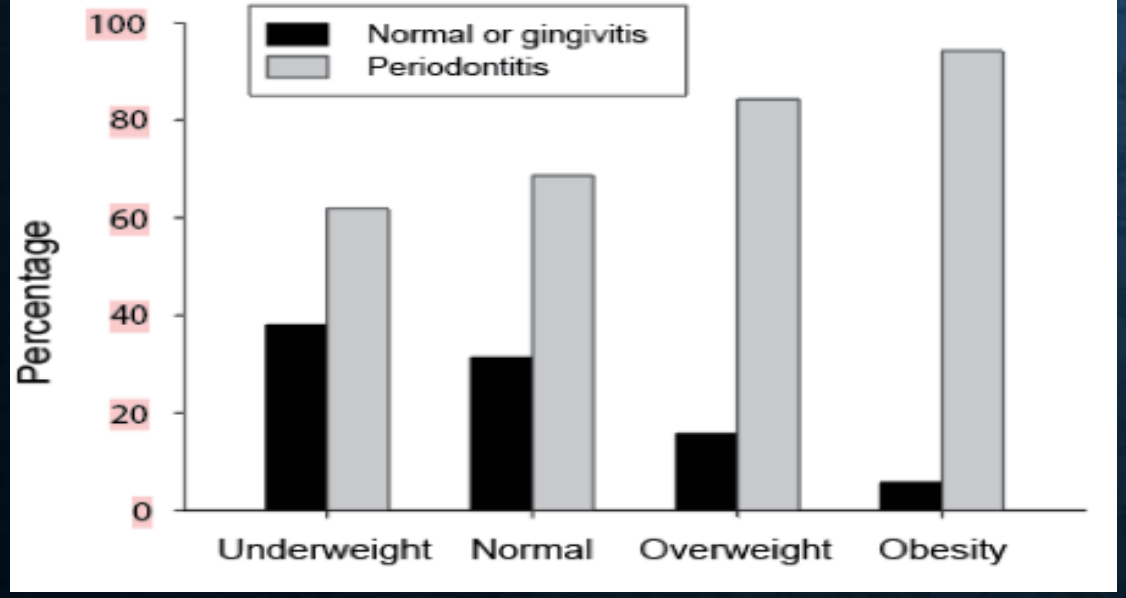
Waist circumference is also an important indicator of visceral abdominal fat. Evidence suggests that abdominal fat carries a higher health risk than peripheral fat, and that the visceral fat component has the strongest correlation with increased risk. A high-risk waist circumference is considered to be ≥88 cm for women and ≥102 cm for men (50).

Prevalence and trends

Over the period 1960–1980, the prevalence of overweight and obesity among adults, and of overweight among children, was relatively constant. About 13% of adults were obese and 5% of children were overweight. However, data from the National Health and Nutrition Examination Survey III (1988–1991) showed that obesity in adults and overweight in children had markedly increased from the previous survey (38, 74). These trends continued such that approximately 34% (59 million) of American adults now meet the criterion for obesity. More than 65% of the United States adult population have a body mass index of ≥25 kg/m²; and 35.8% of children aged 6–11 years, and 16.1% of adolescents aged 12–19 years, are overweight (48). Thus, in a relatively short time period, the prevalence of obesity among adults has doubled, and the prevalence of overweight among children and adolescents has tripled.

With the exception of sub-Saharan Africa, trends internationally have mirrored those seen in the USA. The International Obesity Task Force estimates that over 1 billion adults are overweight, including 312 million who are obese. Because Asians experience obesity related disease complications at lower body mass indices, new criteria for Asians define overweight as a body mass index of ≥ 23. Using this criteria, the number of adults globally who are overweight is closer to 1.7 billion (34).

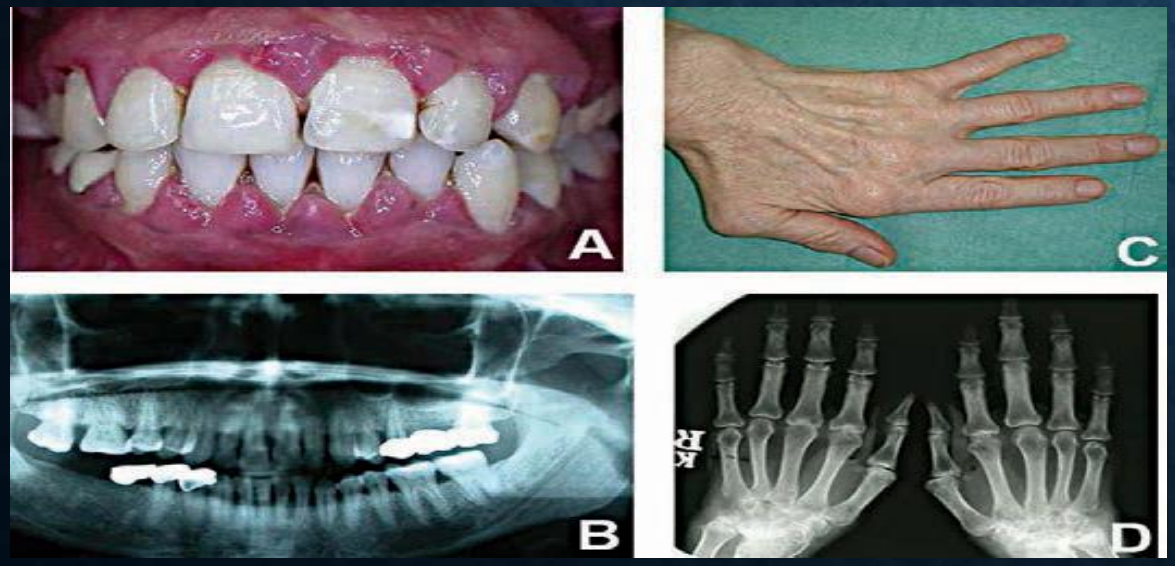
Twin studies (71), and other longitudinal data (6, 59), clearly demonstrate a genetic component in human obesity. However, recent increases in obesity prevalence cannot be solely explained by changes in the gene pool. Predisposition to obesity is probably influenced by numerous susceptibility genes, accounting for variations in energy needs, fuel utilization, metabolic characteristics, and taste preferences. Although influenced by genetic variability, the three factors believed to constitute most of the etiology of obesity are metabolic factors, diet, and physical inactivity. Metabolic factors, such as resting energy expenditure (the number of calories burned at rest) and the thermic effect of food (energy expended during digestion, transport, metabolism and storage of food), vary among individuals but do not appear to be a major component in explaining risk for developing obesity (88). Large portion sizes, high fat intakes, and easy access to calorically sweetened beverages, all play a role in the development of obesity (23, 30). Longitudinal data suggests a particularly important role of reduced physical activity. In a 5-year prospective study of over 12,000 Finnish adults, sedentary individuals were almost twice as likely to experience substantial weight gain as physically active men and women (88). For children, decreased participation in organized sports, changes in

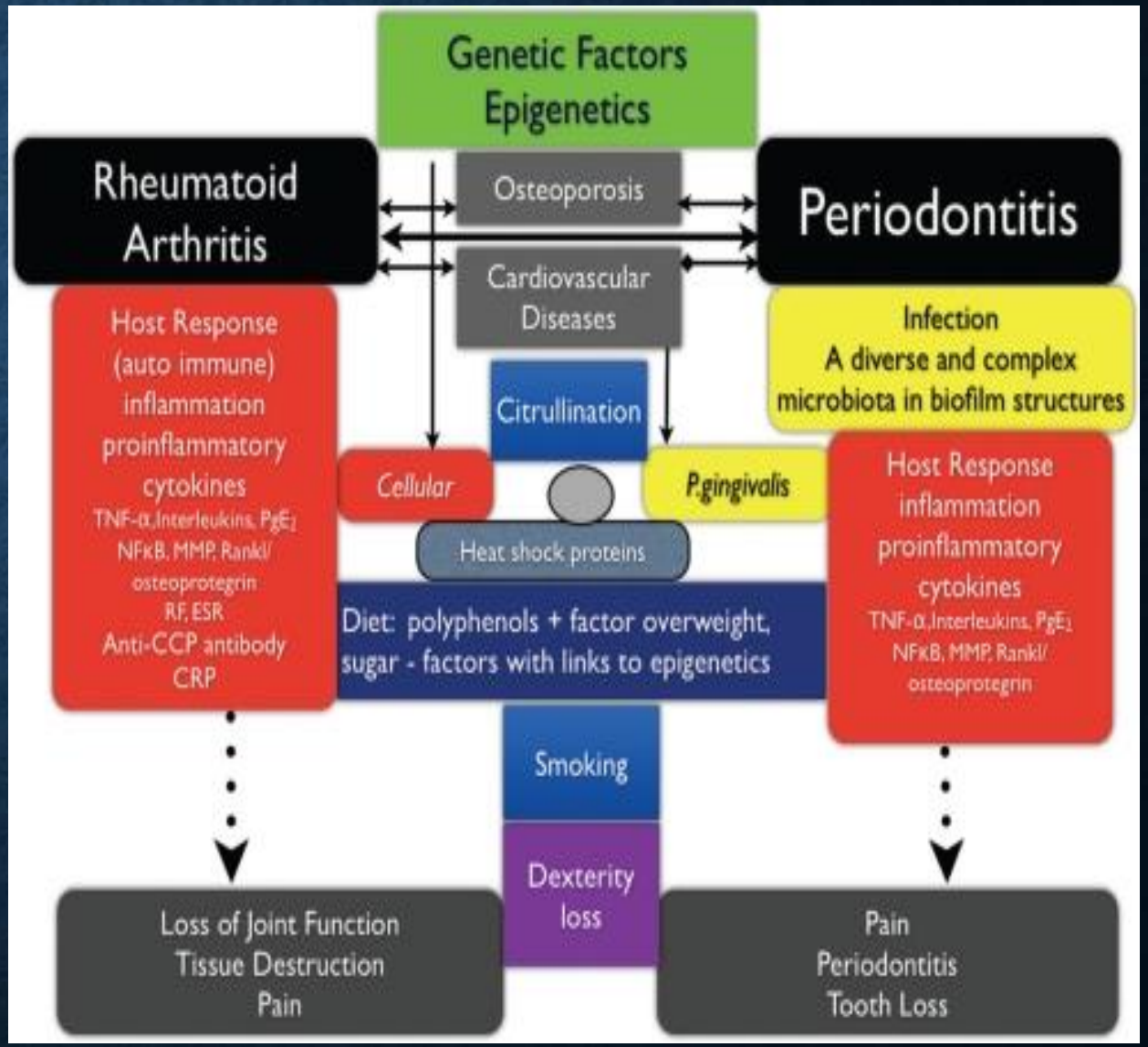


	Obesity Treatment Considerations	
☐	High inflammatory burden associated with obesity	☐
☐	Metabolic dysfunction including insulin resistance and endocrine dysfunction	☐
☐	Gut microbiome associated with obesity (high levels of *Firmicutes and Enterobacteriaceae spp.*)	☐
☐	Comorbidities with obesity-associated systemic disease that may affect the periodontium including Diabetes Mellitus, Cardiovascular disease, Cerebrovascular disease, and dislipidemia	☐
☐	High ghrelin and low leptin levels	☐
	Periodontal Treatment Considerations	
☐	High levels of BOP	☐
☐	High periodontal inflammatory burden (PISA score, salivary/GCF proinflammatory cytokines)	☐
☐	Large quantities of plaque and high levels of anaerobic bacteria within the plaque biofilm	☐
☐	Chronic periodontitis	☐

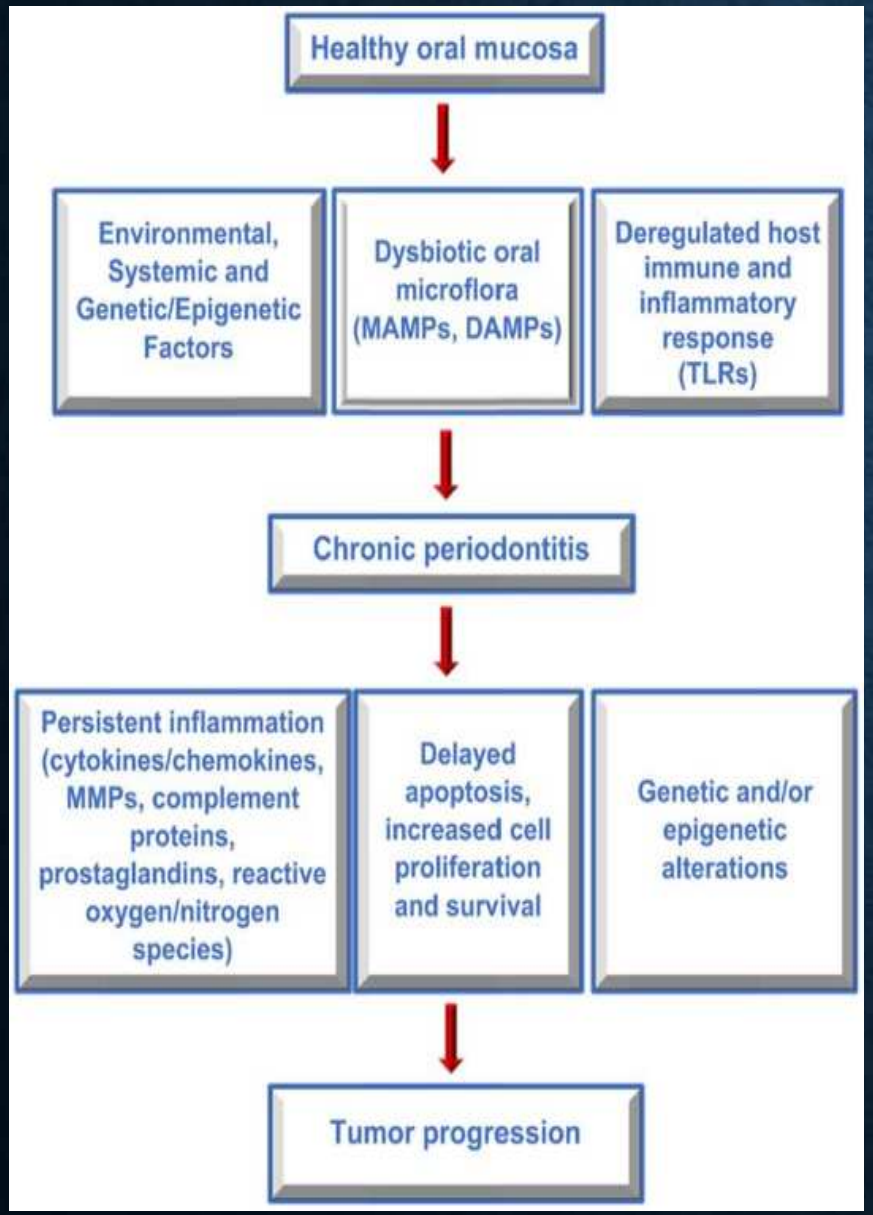
PERIODONTITIS & RHEUMATOID ARTHRITIS

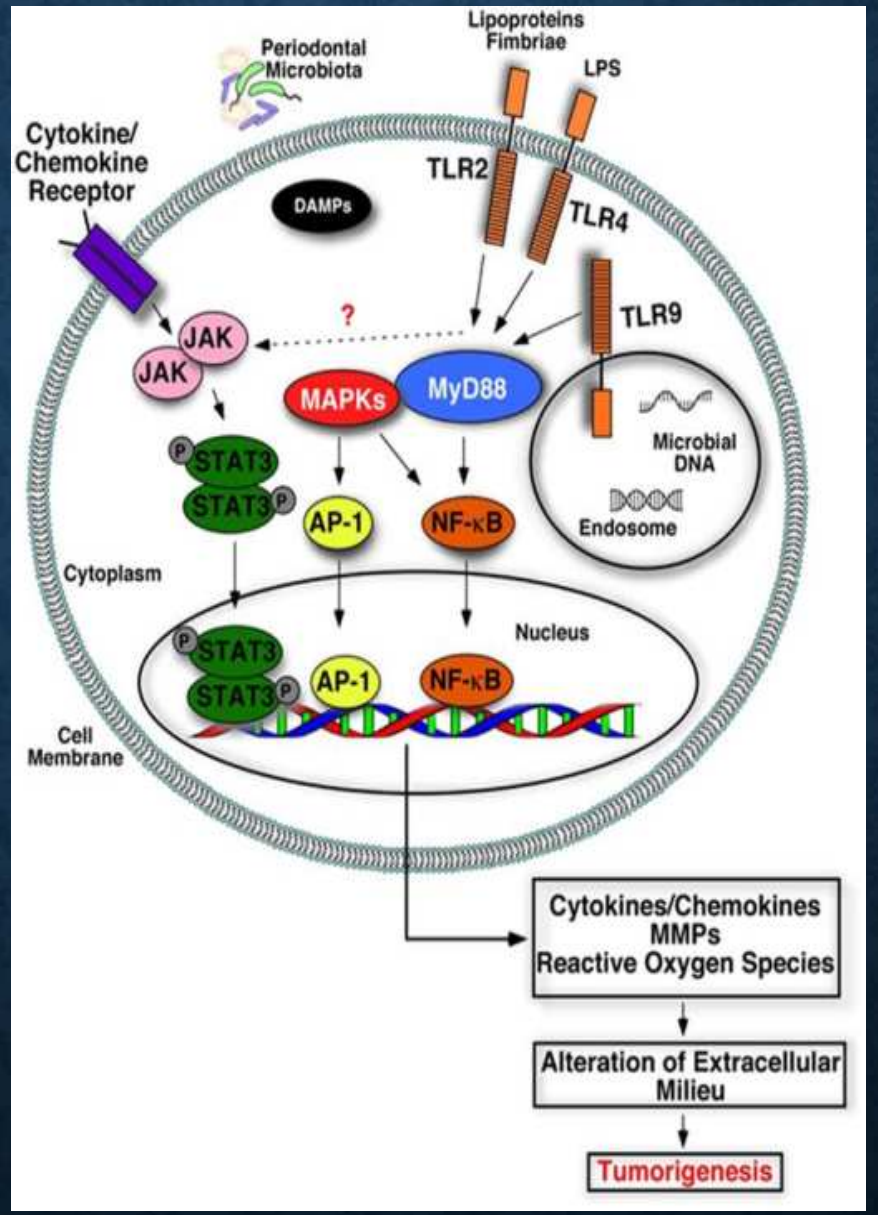
Periodontitis	Rheumatoid arthritis
Chronic immune-inflammatory disease	Chronic auto-inflammatory disease
P.gingivalis the main etiological agent w some autoimmunity	Bacteria/peptide is an adjunct antigen in autoantibody production
Genetic (HLA-DR) and environmental influences	Genetic (HLA-DR) and environmental influences
Increased IL-1, TNF, PGE2, NFkB, MMPs, RANL/RANKL/OPG, osteoclast activation	Increased IL-1, TNF, PGE2, NFkB, MMPs, RANL/RANKL/OPG, osteoclast activation
Th1, Th2 and Th17	Th1=Th2 and Th17
Bacterial DNA of anaerobes and high antibody titers against HSP of *P.gingivalis, P. Melanogenicus and P. Intermedia*	Bacterial DNA of anaerobes and high antibody titers against HSP of *P.gingivalis, P. Melanogenicus and P. Intermedia*

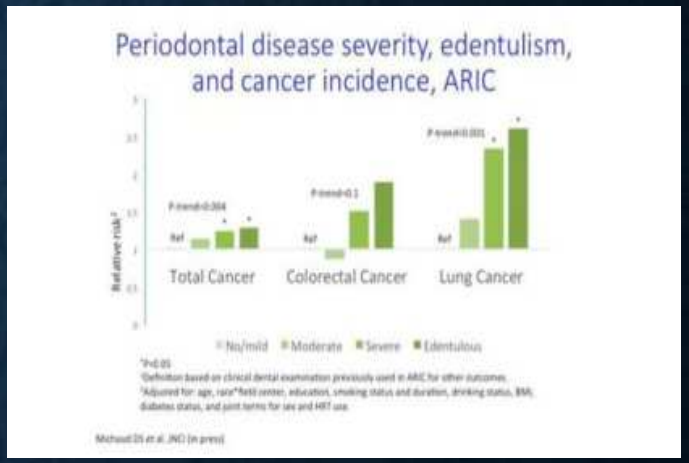




NEOPLASIA & PERIODONTAL DISEASE







IJHS

Review Article

Prevalence of periodontal disease, its association with systemic diseases and prevention

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ABSTRACT

Periodontal diseases are prevalent both in developed and developing countries and affect about 20-50% of global population. High prevalence of periodontal disease in adolescents, adults, and older individuals makes it a public health concern. Several risk factors such as smoking, poor oral hygiene, diabetes, medication, age, hereditary, and stress are related to periodontal diseases. Robust evidence shows the association of periodontal diseases with systemic diseases such as cardiovascular disease, diabetes, and adverse pregnancy outcomes. Periodontal disease is likely to cause 19% increase in the risk of cardiovascular disease, and this increase in relative risk reaches to 44% among individuals aged 65 years and over. Type 2 diabetic individuals with severe form of periodontal disease have 3.2 times greater mortality risk compared with individuals with no or mild periodontitis. Periodontal therapy has been shown to improve glycemic control in type 2 diabetic subjects. Periodontitis is related to maternal infection, preterm birth, low birth weight, and preeclampsia. Oral disease prevention strategies should be incorporated in chronic systemic disease preventive initiatives to curtail the burden of disease in populations. The reduction in the incidence and prevalence of periodontal disease can reduce its associated systemic diseases and can also minimize their financial impact on the health-care systems. It is hoped that medical, dental practitioners, and other health-care professionals will get familiar with perio-systemic link and risk factors, and need to refer to the specialized dental or periodontal care.

Keywords: Periodontal disease, epidemiology, risk factors, systemic disease, preventive strategy

Introduction

Periodontal disease is a chronic inflammatory disease of periodontium and its advanced form is characterized by periodontal ligament loss and destruction of surrounding alveolar bone.[1] It is the main cause of tooth loss and is considered one of the two biggest threats to the oral health.[1,2] There are approximately 800 species of bacteria identified in the oral cavity[3] and it is hypothesized that complex interaction of bacterial infection and host response, modified by behavioral factors such as smoking, can result in periodontal disease.[4]

The aim of the review is two-fold: (1) To evaluate the prevalence of periodontal disease in different populations, risk factors, and its association with systemic diseases and (2) to discuss the strategies and measures to prevent and control periodontal disease.

Prevalence of Periodontal Disease

Periodontal disease is the most common oral condition of human population.[5] The prevalence and incidence statistics of periodontal diseases vary because of bias, case misclassification, and the number of teeth and the sites examined.[6] According to the Canadian Health Measures Survey 2007-2009, the measurement of loss of periodontal ligament attachment is considered the gold standard in reporting the prevalence of periodontal disease.[7] National Health and Nutrition Examination Survey (NHANES) determined the attachment loss (AL) and probing depth (PD) at six sites of all teeth (excluding third molars) for the estimation of periodontal disease in the U.S.[8]

The world Health Organization (WHO) has maintained global oral health data bank using community periodontal index (CPI).[9] This global oral health data from large epidemiological studies from different countries were gathered to show the distribution of periodontal disease in adolescents, adults and elderly populations (Figures 1-3).[9] CPI index score ranges from 0 to 4 and describes the periodontal condition of individuals at population level. CPI score 0 represents no periodontal disease; score 1 means gingival bleeding on probing; score 2 shows the presence of calculus and bleeding; score 3 indicates shallow periodontal pockets of

Journal of Clinical Periodontology

J Clin Periodontol 2008; 35 (Suppl. 8): 333–337 doi: 10.1111/j.1600-051X.2008.01278.x

Journal of Clinical Periodontology

Periodontal diseases and health: Consensus Report of the Sixth European Workshop on Periodontology

Denis Kinane[1] and Phillippe Bouchard[2] on behalf of group E of the European Workshop on Periodontology*

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Kinane D, Bouchard P. Periodontal diseases and health: Consensus Report of the Sixth European Workshop on Periodontology. J Clin Periodontol 2008; 35 (Suppl. 8): 333–337. doi: 10.1111/j.1600-051X.2008.01278.x.

Abstract

Introduction: The remit of this group was to update the knowledge base on periodontal diseases and health.

Material and Methods: The literature was systematically searched and critically reviewed in five specific topics.

Results: *Prevalence of periodontitis:* The data suggest a trend towards a lower prevalence of periodontitis in recent years.

Adverse pregnancy outcome: The findings indicate a likely association between periodontal disease and an increased risk of adverse pregnancy outcomes. There is no evidence that treating periodontal disease decreases the rate of adverse pregnancy outcomes.

Prevalence and distribution of periodontal pathogens: Genetic analysis of bacteria has demonstrated an unanticipated diversity within species. Carriage rates and particular subsets of these species vary between ethnic groups. Few of these differences can be related to differences in disease prevalence.

Diabetes mellitus: Evidence on the association supports the concept of increased severity but not extent of periodontitis in subjects with poorly controlled diabetes. It is inconclusive that periodontal treatment results in improved metabolic control.

Cardiovascular diseases: Evidence suggests that having periodontitis contributes to the total infectious and inflammation burden and may contribute to cardiovascular events and stroke in susceptible subjects. The impact of periodontal therapy must be further investigated.

Key words: cardiovascular; diabetes mellitus; periodontitis; preterm birth; prevalence

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Conflict of interest and source of funding statement

Group E participants declare that they had no conflict of interests.
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*D'Aiuto Francesco, UK; Hugoson Anders, Sweden; Kilian Mogens, Denmark; Kocher Thomas, Germany; Loos Bruno, The Netherlands; Madianos Phaebus, Greece; Norderyd Ola, Sweden; Papapanou Panos, USA; Persson Rutger, Switzerland; Pihlström Bruce, USA; Rylev Mette, Denmark; Salvi Giovanni, Switzerland; Shapira Lior, Israel; Wimmer Gernot, Germany.

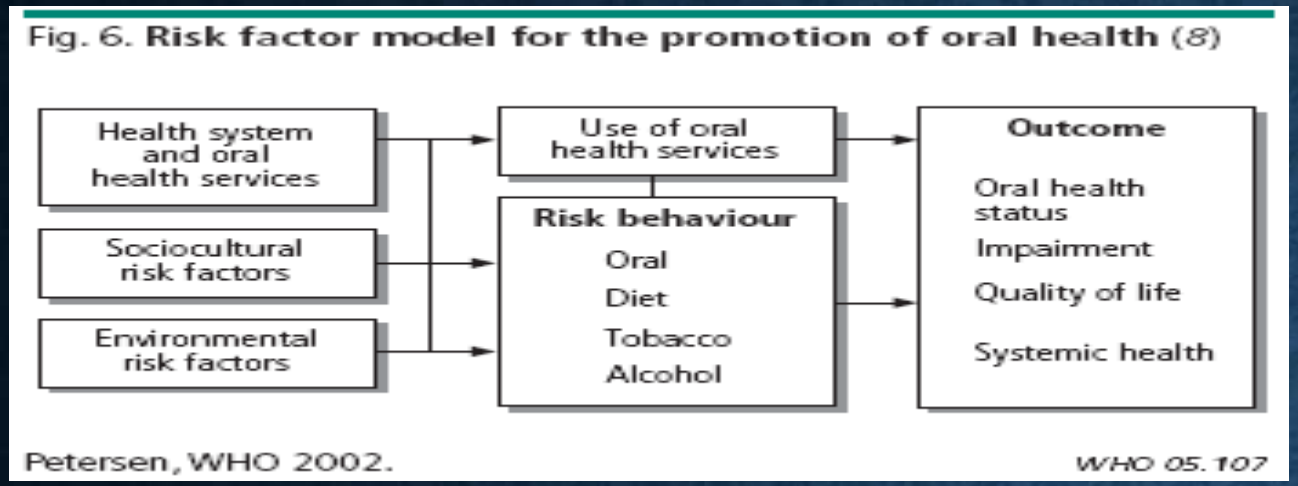


Fig. 6. Risk factor model for the promotion of oral health (8)
Health system and oral health services
Sociocultural risk factors
Environmental risk factors
Use of oral health services
Risk behaviour
Oral
Diet
Tobacco
Alcohol
Outcome
Oral health status
Impairment
Quality of life
Systemic health
Petersen, WHO 2002.
WHO 05.107

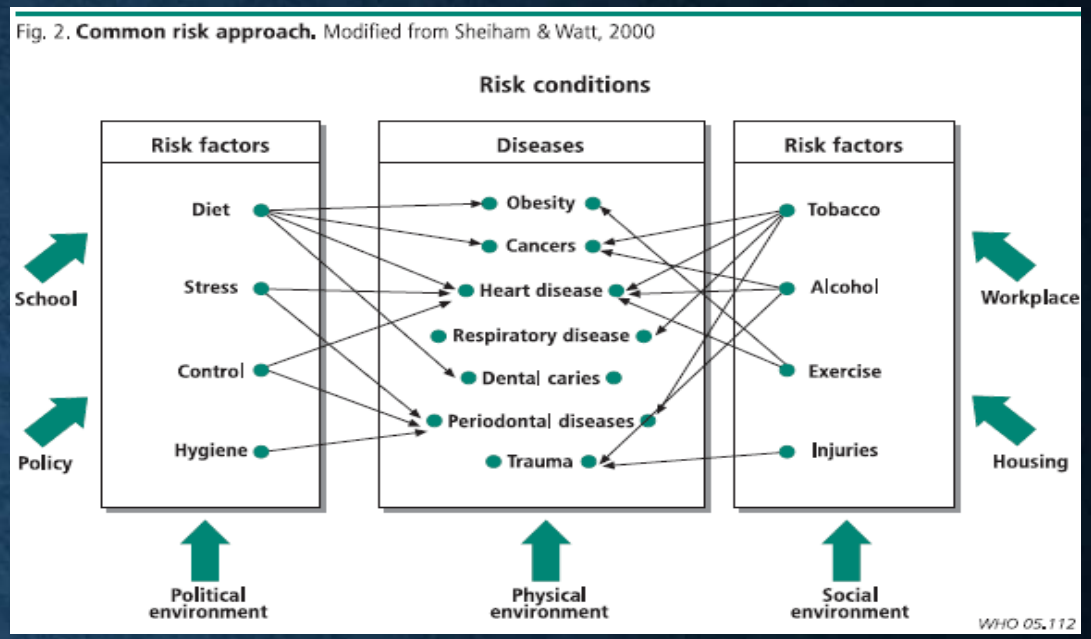
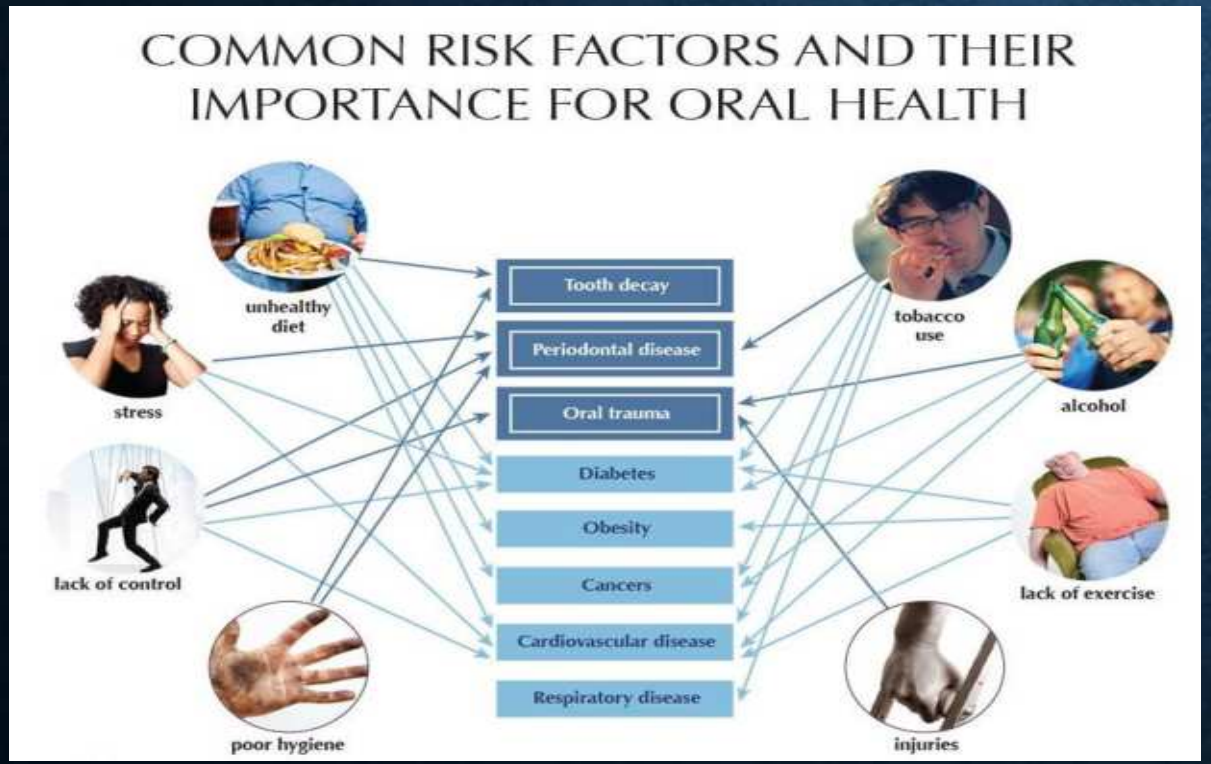
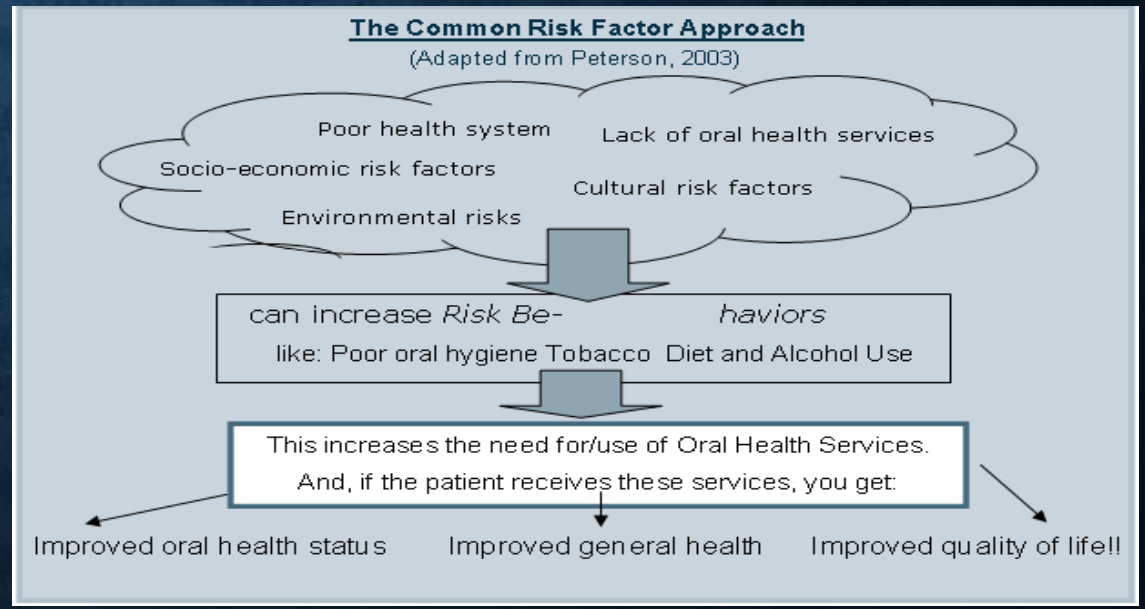
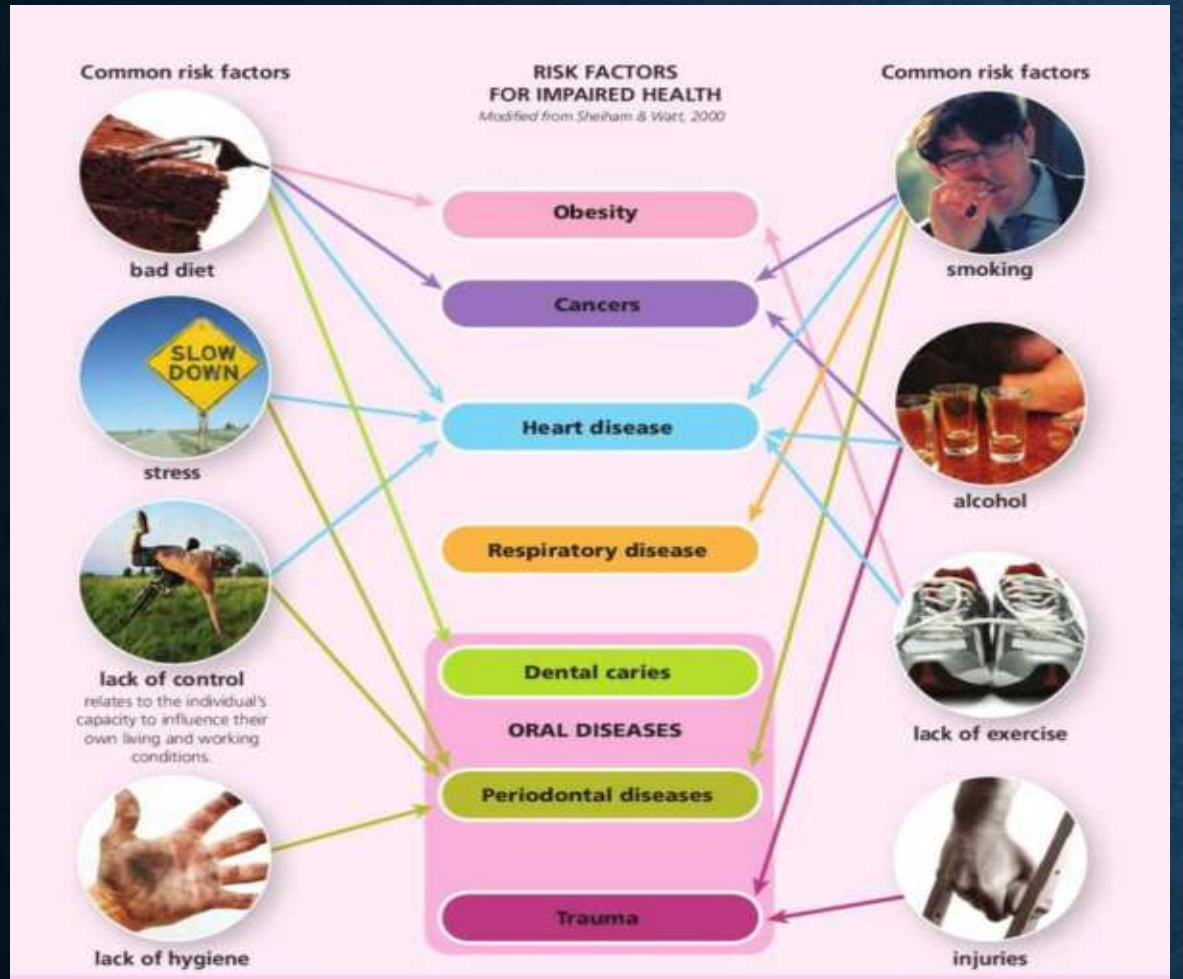


Fig. 2. Common risk approach. Modified from Sheiham & Watt, 2000
Risk conditions
Risk factors
Diseases
Risk factors
Diet
Stress
Control
Hygiene
Obesity
Cancers
Heart disease
Respiratory disease
Dental caries
Periodontal diseases
Trauma
Tobacco
Alcohol
Exercise
Injuries
School
Policy
Workplace
Housing
Political environment
Physical environment
Social environment
WHO 05.112


COMMON RISK FACTORS AND THEIR IMPORTANCE FOR ORAL HEALTH
unhealthy diet
stress
lack of control
poor hygiene
tobacco use
alcohol
lack of exercise
injuries
Tooth decay
Periodontal disease
Oral trauma
Diabetes
Obesity
Cancers
Cardiovascular disease
Respiratory disease


The Common Risk Factor Approach
(Adapted from Peterson, 2003)
Poor health system
Lack of oral health services
Socio-economic risk factors
Cultural risk factors
Environmental risks
can increase Risk Be- haviors
like: Poor oral hygiene Tobacco Diet and Alcohol Use
This increases the need for/use of Oral Health Services.
And, if the patient receives these services, you get:
Improved oral health status
Improved general health
Improved quality of life!!



"As I see it, every day you do one of two things: build health or produce disease."
Adelle Davis, pioneering US nutritionist, 1907–74

Chronic diseases, including oral diseases, are of long duration, generally progressing slowly and usually non-communicable. A small number of common causes (risk factors) are responsible for most chronic diseases. Major risk factors, such as tobacco use, physical inactivity and a diet high in fat, salt and sugar, contribute to a range of chronic diseases, such as obesity, diabetes, cardiovascular diseases and oral diseases.

The risk factors for chronic diseases compound over time, resulting in higher levels of chronic disease as age increases. Poverty and chronic disease are linked into a vicious cycle; chronic diseases can exacerbate poverty and the poor have greater exposure to risk and less access to health services.

Risk Factors	Risk determinants	Risk indicators	Risk predictors
Tobacco smoking	Genetic factors	HIV/AIDS	Previous history of periodontal disease
Diabetes	Age	Osteoporosis	Bleeding on probing
Pathogenic bacteria	Gender	Infrequent dental visits	
Microbial tooth deposit	Socioeconomic status		
	Stress		

Without Good Periodontal Health, You Can't Have Good General Health.

Periodontal disease (gum disease) can affect your general health and it can be affected by your general health. Here are a few examples:

Respiratory Infections[2-5]

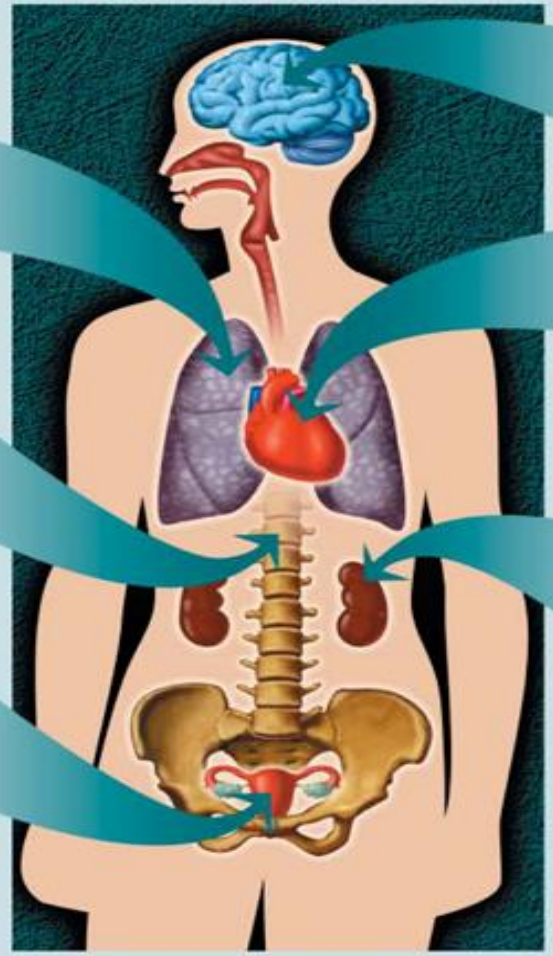
- Inhaling bacteria from the mouth and throat can lead to pneumonia
- Dental plaque buildup creates a dangerous source of bacteria that can be inhaled into the lungs

Severe Osteopenia[11]

- Reduction in bone mass (osteopenia) is associated with gum disease and related tooth loss
- Severity has been connected to tooth loss in postmenopausal women

Preterm or Low Birthweight Babies[17,18]

- Women with advanced gum disease may be more likely to give birth to an underweight or preterm baby[17]
- Oral microbes can cross the placental barrier, exposing the fetus to infection.[17]



Stroke[1]

- Those with adult periodontitis may have increased risk of stroke

Heart Disease[6-10]

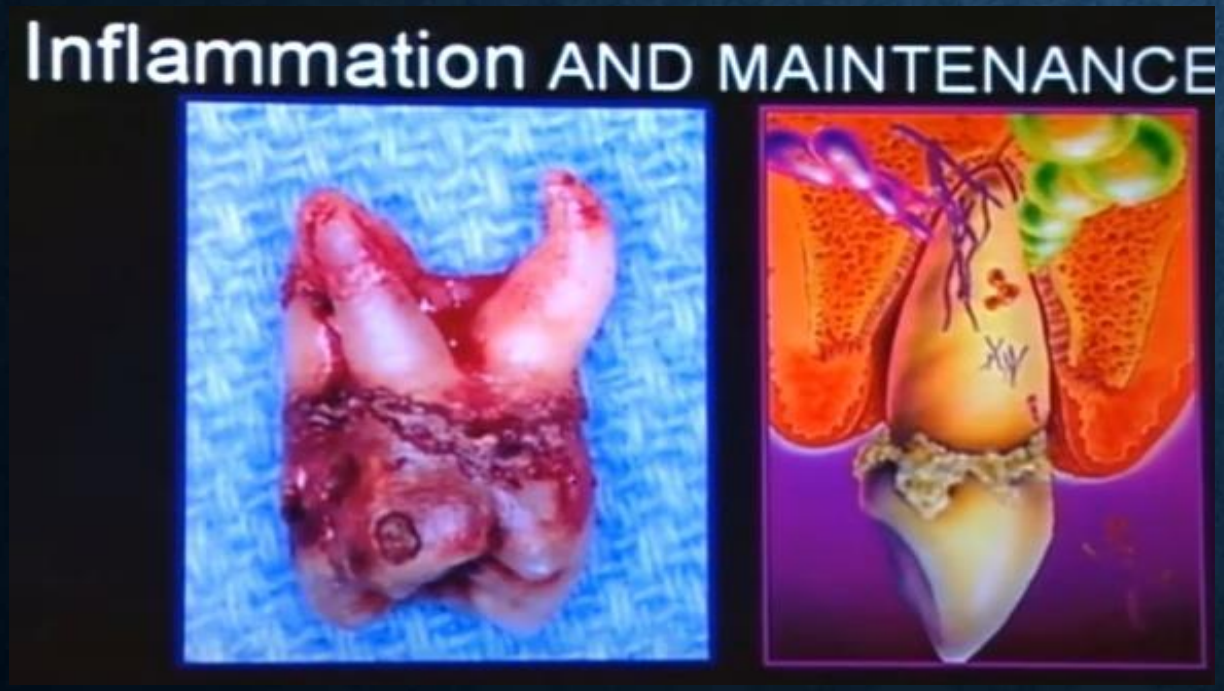
- Those with adult periodontitis may have increased risk of fatal heart attack …[1,6,7]
- And are more likely to be diagnosed with cardiovascular disease[1]
- Bacteria from the mouth may cause clotting problems in the cardiovascular system[6]

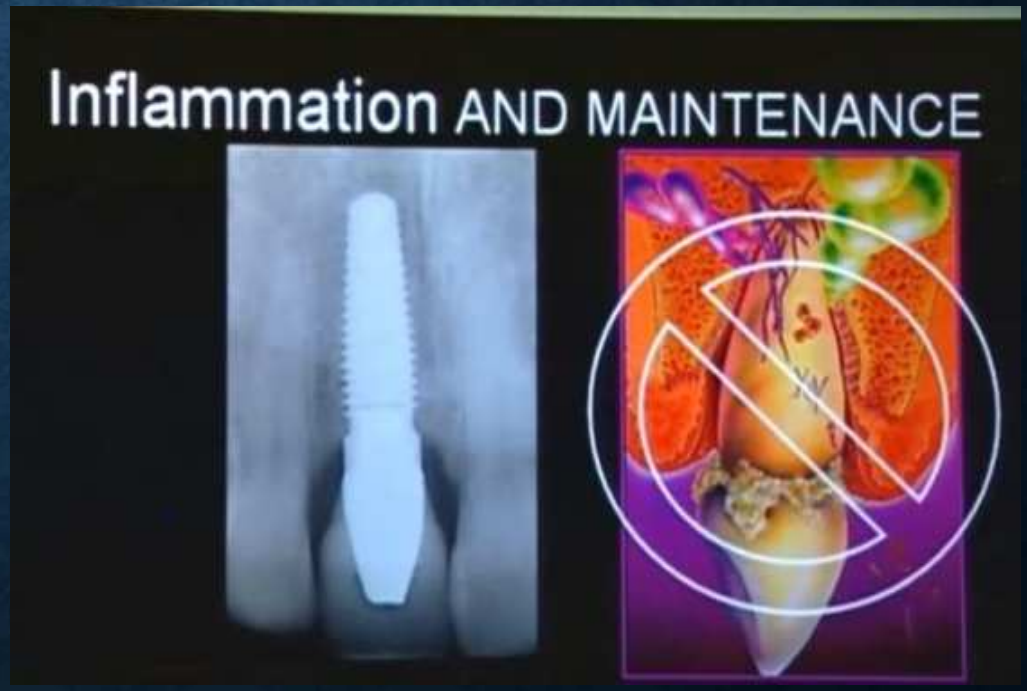
Uncontrolled Diabetes[12-16]

- Chronic periodontal disease can disrupt diabetic control[12-15]
- Diabetes can alter the pocket environment, contributing to bacterial overgrowth[14]
- Smokers with diabetes increase their risk of tooth loss by 20 times[13]
- People with type II diabetes are 3 times as likely to develop periodontal disease than are nondiabetics[13]

Do You Have Adult Periodontitus? What Are You Doing About It?

- SHOULD THE MOVEMENT FROM INFECTION MODEL TO INFLAMMATION MODEL CHANGE THE WAY IN WHICH WE PLAN THE TREATMENT FOR OUR PATIENTS…???





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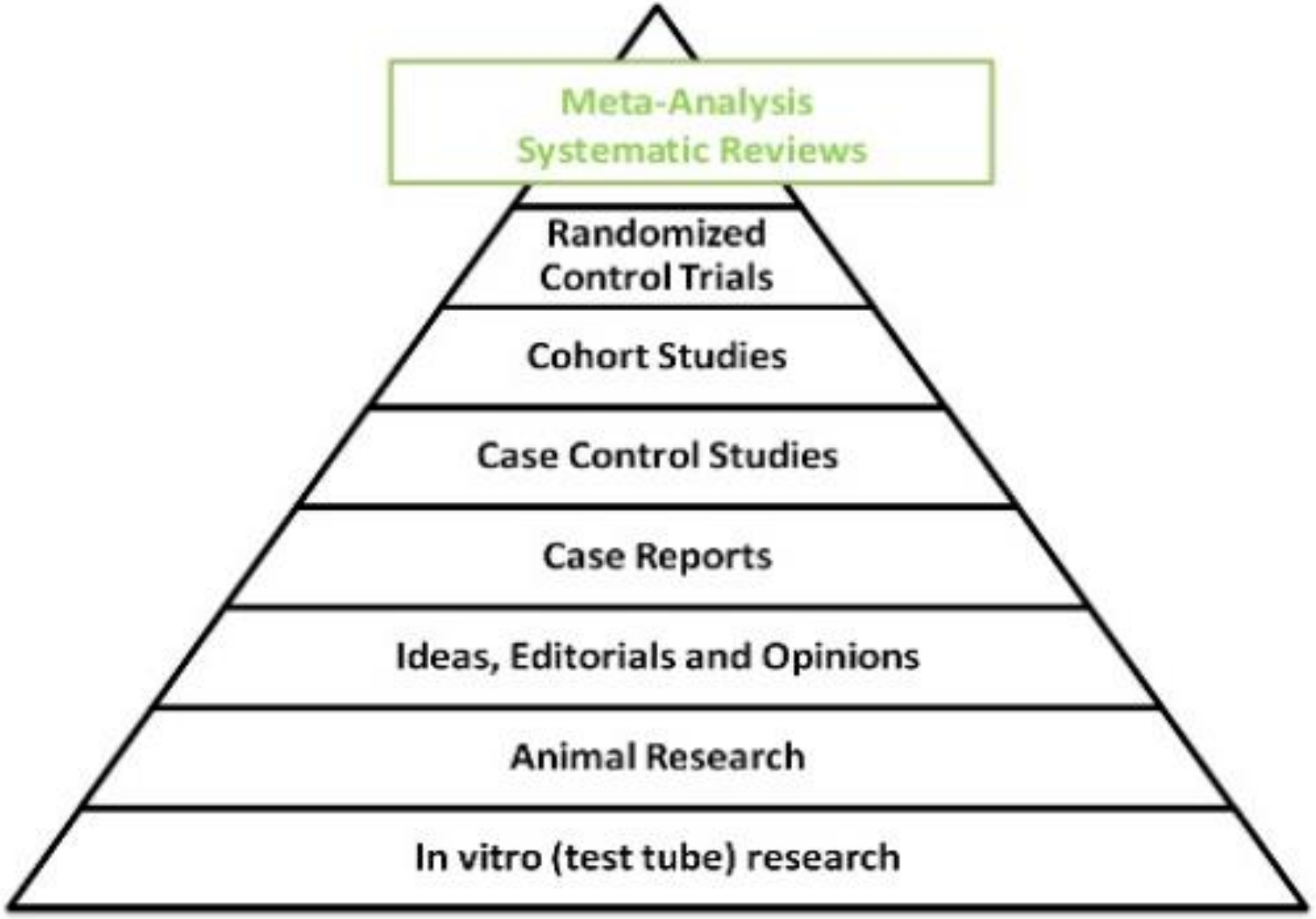

Periodontitis and Systemic Diseases Proceedings of a workshop jointly held by the European Federation of Periodontology and American Academy of Periodontology.

Co-edited by Maurizio Tonetti and Kenneth S. Kornman

Volume 40, Issue Supplement s14, April 2013

TABLE 2–2. The Strength of Association of Local and Systemic Factors with Destructive Periodontal Disease

Factor	*Case Report Studies*	*Case-Control Studies*	*Cross-Sectional Studies*	*Longitudinal Studies*	*Intervention Studies*
Specific bacteria					
P. gingivalis	Yes	Yes	Yes	Yes	Yes
B. forsythus	Yes	Yes	Yes	Yes	Yes
P. intermedia	Yes	Yes	Yes	Yes	Yes
Gender					
Male	Yes	NR	Yes	NR	NR
Age	Yes	Yes	Yes	No (to 7th decade)	NR
Diabetes mellitus					
Type 2	Yes	Yes	Yes	Yes	Yes (treatment reduces glycosylated hemoglobin)
Type 1	Yes	Yes	Yes	NR	NR
Smoking	NR	Yes	Yes	Yes	Yes (smokers heal poorly)
Osteoporosis	Yes	Yes	Yes	NR	NR
Stress, distress, coping	Yes	Yes	Yes	NR	NR
PMN disorders	Yes	Yes	NR	Yes (case series)	NR
Genetic factors (IL-1 polymorphisms)	NR	Yes	NR	NR	NR
Dietary calcium	NR	Yes	Yes	NR	NR
Preexisting periodontal disease	Yes	Yes	Yes	Yes	Yes

NR = not reported, or not relevant; PMN = polymorphonuclear.

Adapted from Genco RJ. Current view of risk factors for periodontal diseases. J Periodontal 1996;67(Suppl):1041–9.

Medical History Form

Date _____

Name _____ Home Phone ( ) _____
Last First Middle

Address _____ Business Phone ( ) _____
Number Street

City _____ State _____ Zip Code _____

Occupation _____ Social Security No. _____

Date of Birth ___/___/___ Sex M F Height _____ Weight _____ Single _____ Married _____
mo. day yr.

Name of Spouse _____ Closest Relative _____ Phone ( ) _____

If you are completing this form for another person, what is your relationship to that person? _____

Referred by _____

For the following questions, *circle yes or no*, whichever applies. Your answers are for our records only and will be considered confidential. Please note that during your initial visit you will be asked some questions about your responses to this questionnaire and there may be additional questions concerning your health.

1.	Are you in good health?	Yes	No
2.	Has there been any change in your general health within the past year?	Yes	No
3.	My last physical examination was on _____		
4.	Are you now under the care of a physician?	Yes	No
	If so, what is the condition being treated? _____		
5.	The name and address of my physician(s) is _____		
6.	Have you had any serious illness, operation, or been hospitalized in the past 5 years?	Yes	No
	If so, what was the illness or problem? _____		
7.	Are you taking any medicine(s) including non-prescription medicine?	Yes	No
	If so, what medicine(s) are you taking? _____		
8.	Do you have or have you had any of the following diseases or problems?		
a.	Damaged heart valves or artificial heart valves, including heart murmur or rheumatic heart disease	Yes	No
b.	Cardiovascular disease (heart trouble, heart attack, angina, coronary insufficiency, coronary occlusion, high blood pressure, arteriosclerosis, stroke)	Yes	No
	1. Do you have chest pain upon exertion?	Yes	No
	2. Are you ever short of breath after mild exercise or when lying down?	Yes	No
	3. Do your ankles swell?	Yes	No
	4. Do you have inborn heart defects?	Yes	No
	5. Do you have a cardiac pacemaker?	Yes	No
c.	Allergy	Yes	No
d.	Sinus trouble	Yes	No
e.	Asthma or hay fever	Yes	No
f.	Fainting spells or seizures	Yes	No
g.	Persistent diarrhea or recent weight loss	Yes	No
h.	Diabetes	Yes	No
i.	Hepatitis, jaundice or liver disease	Yes	No
j.	AIDS or HIV infection	Yes	No
k.	Thyroid problems	Yes	No
l.	Respiratory problems, emphysema, bronchitis, etc.	Yes	No
m.	Arthritis or painful swollen joints	Yes	No
n.	Stomach ulcer or hyperacidity	Yes	No
o.	Kidney trouble	Yes	No
p.	Tuberculosis	Yes	No
q.	Persistent cough or cough that produces blood	Yes	No
r.	Persistent swollen glands in neck	Yes	No
s.	Low blood pressure	Yes	No
t.	Sexually transmitted disease	Yes	No
u.	Epilepsy or other neurological disease	Yes	No
v.	Problems with mental health	Yes	No
w.	Cancer	Yes	No
x.	Problems of the immune system	Yes	No

(over)

Figure 3–1. Medical history form, side 1.

9. Have you had abnormal bleeding?	Yes	No
a. Have you ever required a blood transfusion?	Yes	No
10. Do you have any blood disorder such as anemia?	Yes	No
11. Have you ever had any treatment for a tumor or growth?	Yes	No
12. Are you allergic or have you had a reaction to:		
a. Local anesthetics	Yes	No
b. Penicillin or other antibiotics	Yes	No
c. Sulfa drugs	Yes	No
d. Barbiturates, sedatives, or sleeping pills	Yes	No
e. Aspirin	Yes	No
f. Iodine	Yes	No
g. Codeine or other narcotics	Yes	No
h. Other _____		
13. Have you had any serious trouble associated with any previous dental treatment?	Yes	No
If so, explain _____		
14. Do you have any disease, condition, or problem not listed above that you think I should know about?	Yes	No
If so, explain _____		
15. Are you wearing contact lenses?	Yes	No
16. Are you wearing removable dental appliances?	Yes	No
Women		
17. Are you pregnant?	Yes	No
18. Do you have any problems associated with your menstrual period?	Yes	No
19. Are you nursing?	Yes	No
20. Are you taking birth control pills?	Yes	No

Chief Dental Complaint _____

I certify that I have read and understand the above. I acknowledge that my questions, if any, about the inquiries set forth above have been answered to my satisfaction. I will not hold my dentist, or any other member of his/her staff, responsible for any errors or omissions that I may have made in the completion of this form.

_____
Signature of Patient

For completion by the dentist.

Comments on patient interview concerning medical history: _____

Significant findings from questionnaire or oral interview: _____

Dental management considerations: _____

_____ _____
(Date) Signature of Dentist

Medical history update:

Date	Comments	Signature

Figure 3–1. Medical history form, side 2.

NON-SMOKERS ZONE

CONCLUSION

- Greater emphasis on inflammation on patient education
- Expand medical history to include more systems review
- Currently there are no chairside diagnostics to identify Hyper Responders.
- Treat more aggresively
- Consider short term antibiotic therapy or host modulation therapy to help control inflammation in hyperinflammatory patients
- Retain fewer questionable teeth.
- Encourage risk factor modification
- Develop relationships with Physicians & create referal network

THANKYOU