Parodontale Medizin-
Parodontitis und Allgemeinerkrankungen

G. Wimmer

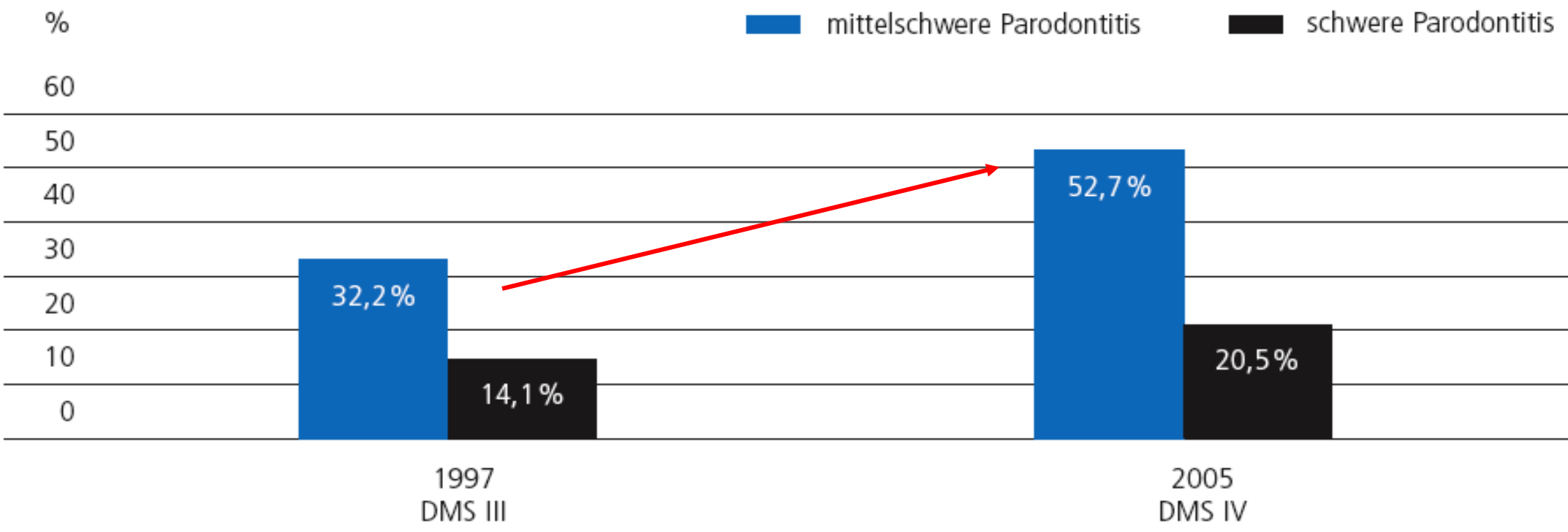
Vierte Deutsche Mundgesundheitsstudie (DMS IV)

Kurzfassung

Institut der Deutschen Zahnärzte (IDZ)
im Auftrag von Bundeszahnärztekammer und
Kassenzahnärztlicher Bundesvereinigung

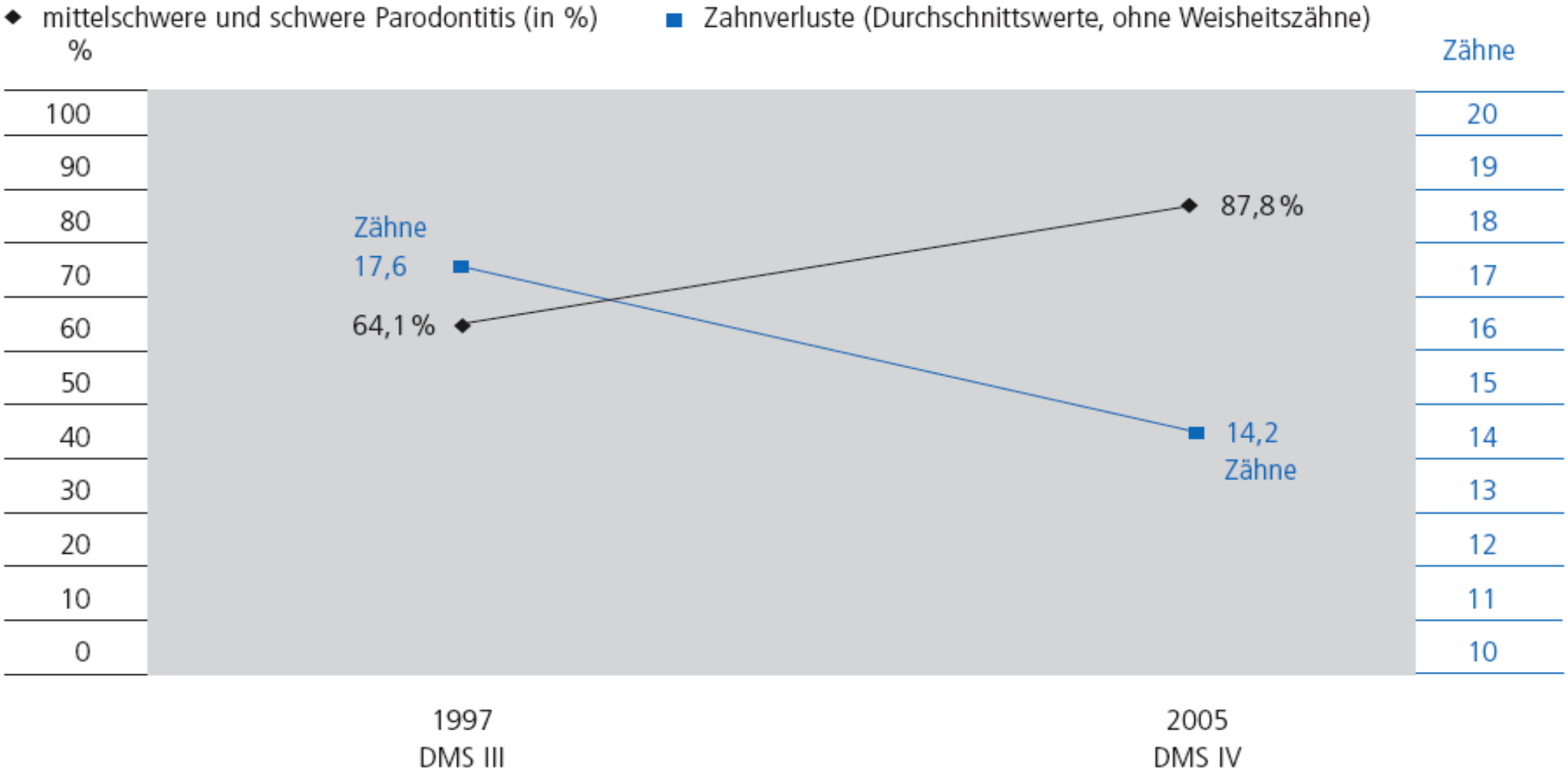
Deutliche Zunahme von Parodontalerkrankungen

Entwicklung der mittelschweren und schweren Parondontalerkrankungen bei 35- bis 44-jährigen Erwachsenen von 1997 bis 2005



Institut der Deutschen Zahnärzte (IDZ), November 2006

Entwicklung von mittelschweren und schweren Parodontalerkrankungen sowie Zahnverlusten bei 65- bis 74-jährigen Senioren von 1997 bis 2005



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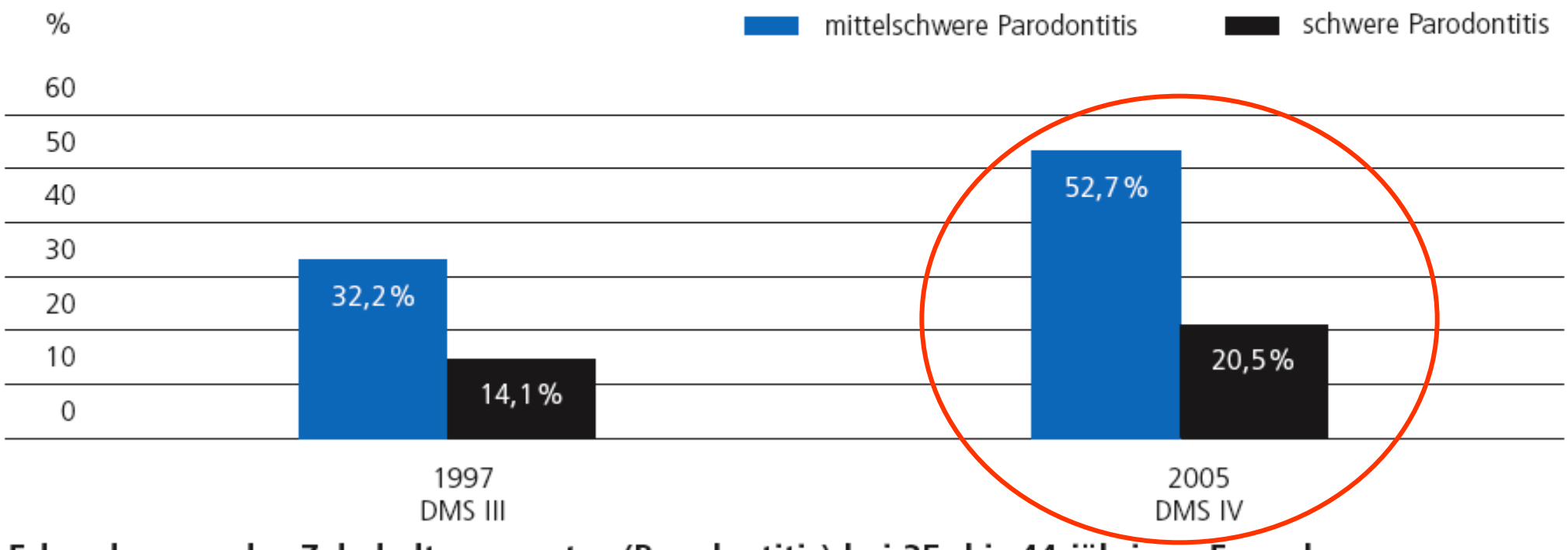
Entwicklung der mittelschweren und schweren Parondontalerkrankungen bei 35- bis 44-jährigen Erwachsenen von 1997 bis 2005



Erkrankungen des Zahnhalteapparates (Parodontitis) bei 35- bis 44-jährigen Erwachsenen

	Gesamt	Männer	Frauen	Raucher	Nichtraucher	Übergewichtige (Body-Mass-Index >25)
Mittelschwere Parodontitis	52,7 %	57,2 %	48,2 %	53,2 %	52,1 %	55,4 %
Schwere Parodontitis	20,5 %	21,8 %	19,1 %	27,1 %	17,1 %	24,6 %

Periodontitis is a major public health issue because : it is common, it is a source of social inequality, it reduces quality of life, it reduces chewing function and impairs aesthetics, it causes tooth loss and disability, it is responsible for a substantial proportion of edentulism and masticatory dysfunction, it has an impact on escalating dental costs and it is a chronic disease with possible impact on general health. Periodontitis disproportionately affects certain groups: it is more prevalent and s evere in i) socially disadvantaged and specific ethnic groups; and ii) smokers, people with diabetes and obese.

Baehni P, & Tonetti MS. Group 1 of the European Workshop on Periodontology. (2010)
Conclusions and consensus statements on periodontal health, policy and education in Europe.

"**The global burden of oral diseases is among the most common,** non-communicable diseases. Their impact on individuals and communities is considerable in terms of pain and suffering, impairment of function and reduced quality of life and cost of treatment".
Article 19 of the recent United Nations General Assembly declaration of 2011 further states "… renal, oral and eye diseases pose a major health burden for many countries and (that) these diseases share common risk factors and can benefit from common responses to non-communicable diseases."

(FDI, World Dental Parliament, 2012)

Übersicht

- Was sind parodontale Erkrankungen?

  (Einteilung, Pathogenesemodel)

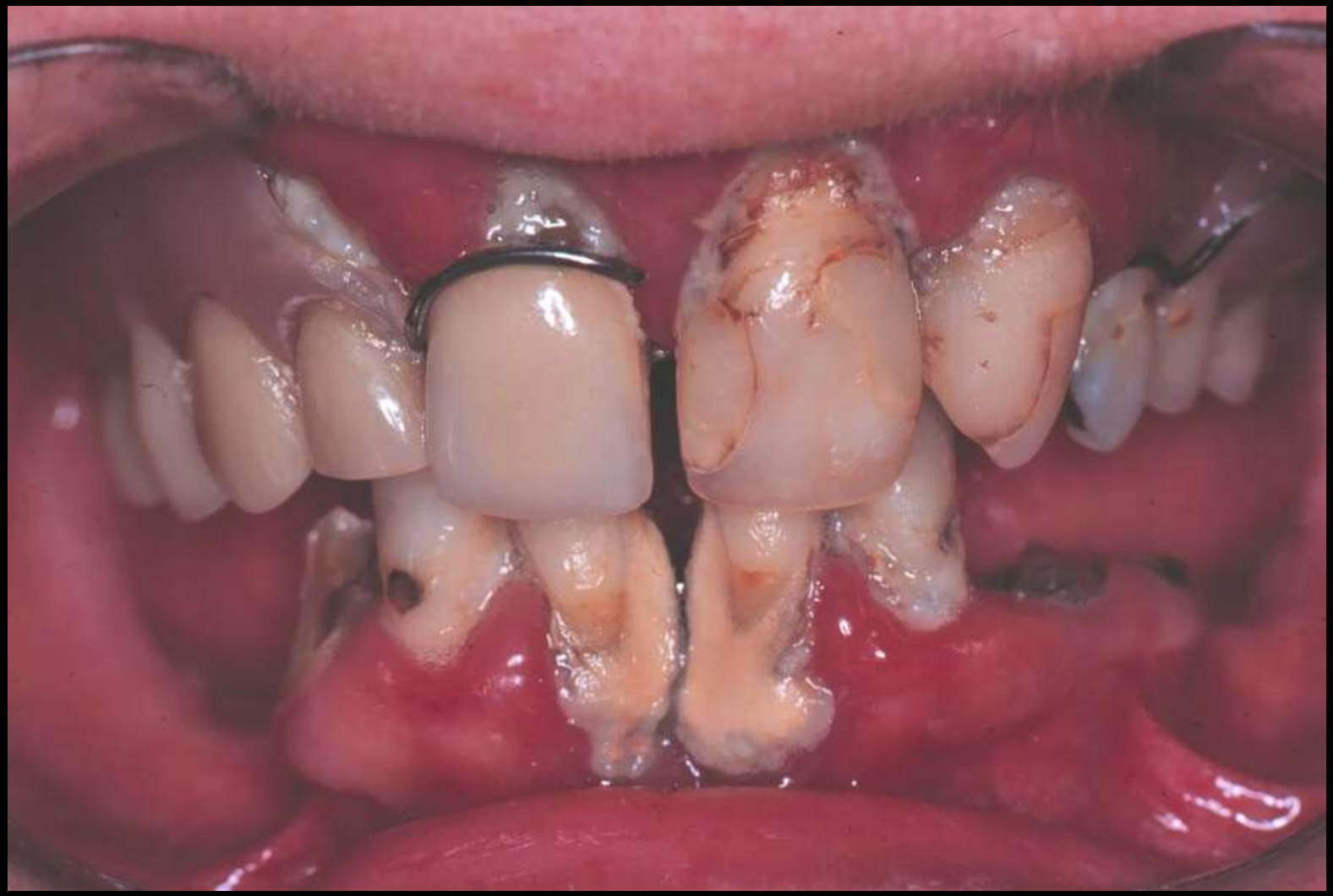
- Parodontale Erkrankungen und ihre

  Wechselwirkung mit anderen Erkrankungen

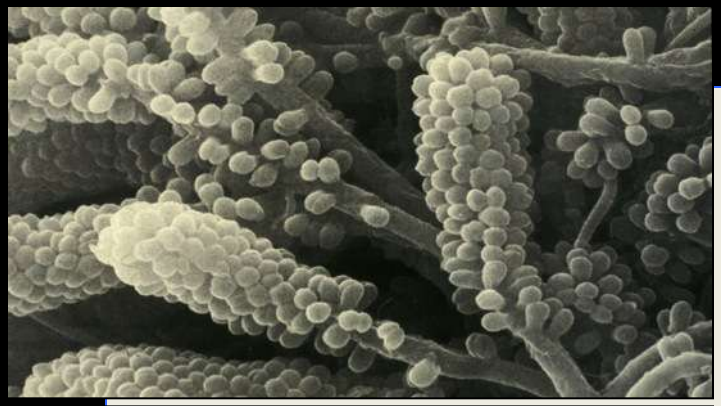
Medizinische Universität Graz

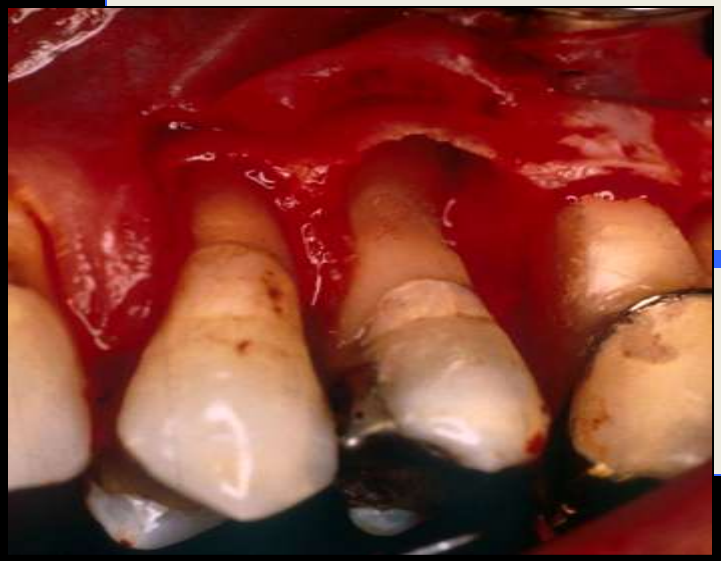
Früher: Unspezifische Plaquehypothese

Zunahme der allgemeinen Plaquemenge



führt per se zur Parodontitis





Spezifische Bakterien

↓

Wirt

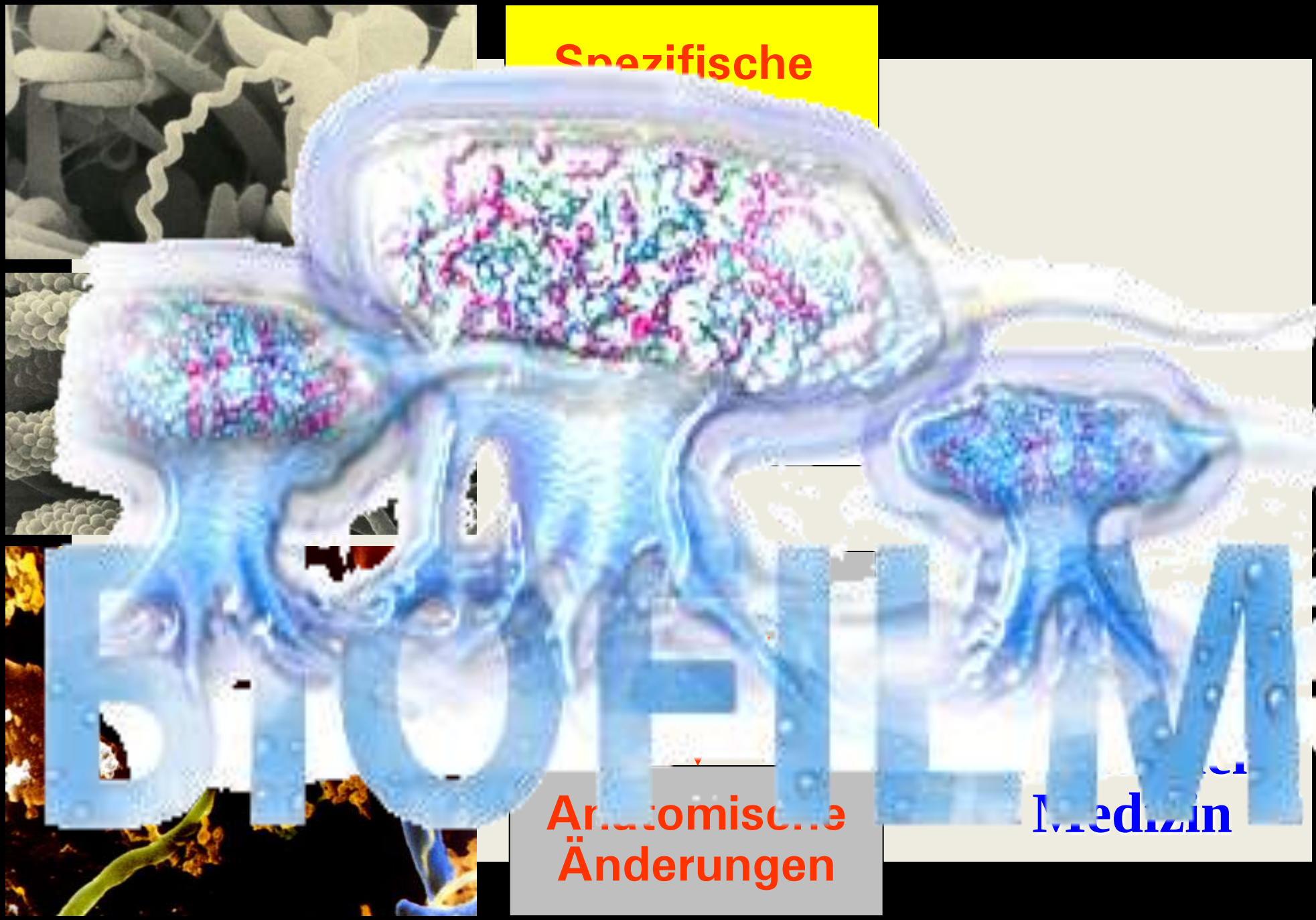
↓

Metabolische Änderungen

↓

Anatomische Änderungen

Traditionelle Medizin


Spezifische
BIOFILM
Medizin
Änderungen

The ISME Journal (2012) 6, 1176–1185

www.nature.com/ismej

ORIGINAL ARTICLE

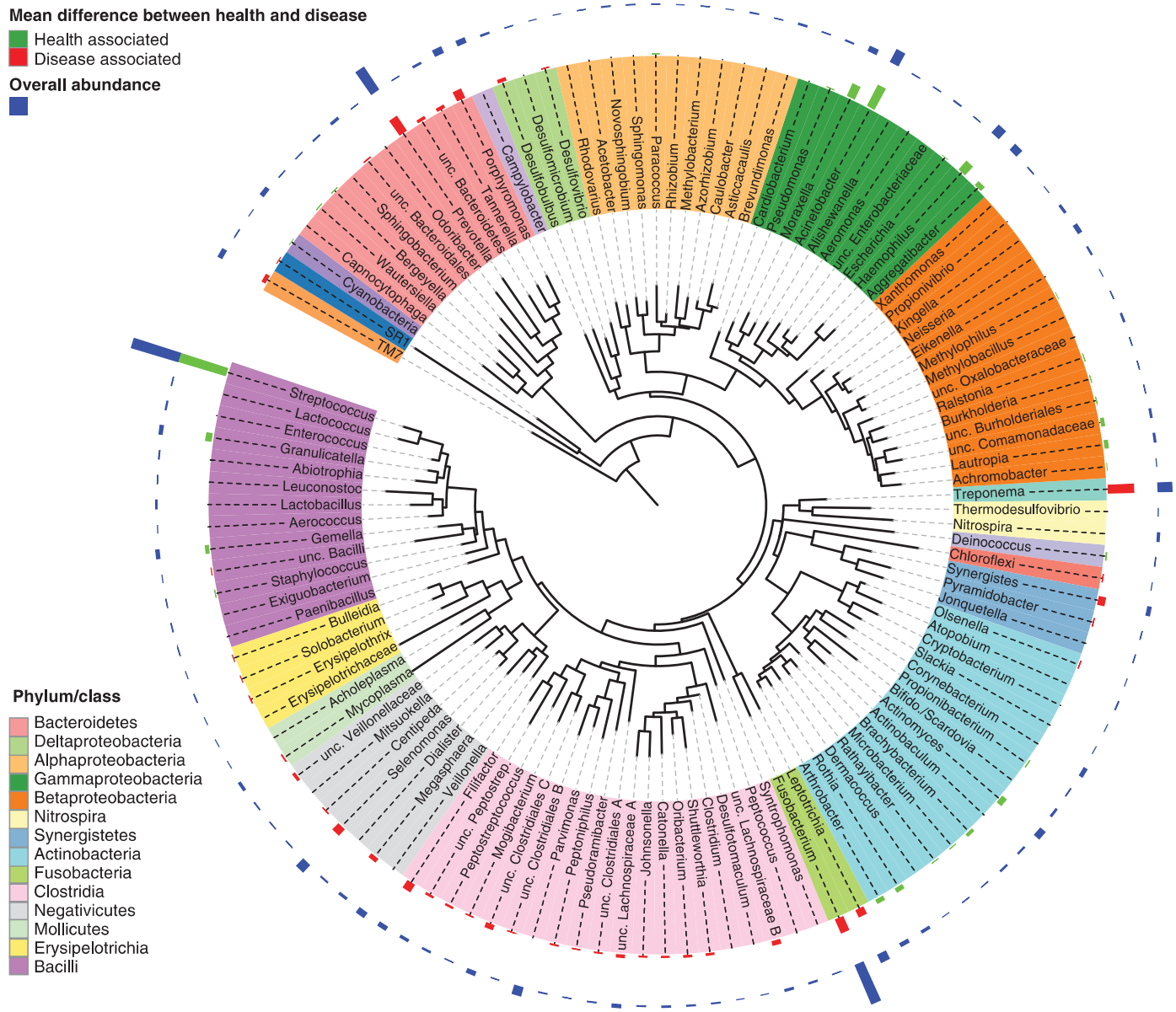
Distinct and complex bacterial profiles in human periodontitis and health revealed by 16S pyrosequencing

Ann L Griffen[1,6], Clifford J Beall[2,6], James H Campbell[3,6], Noah D Firestone[2], Purnima S Kumar[4], Zamin K Yang[3], Mircea Podar[3,5] and Eugene J Leys[2]

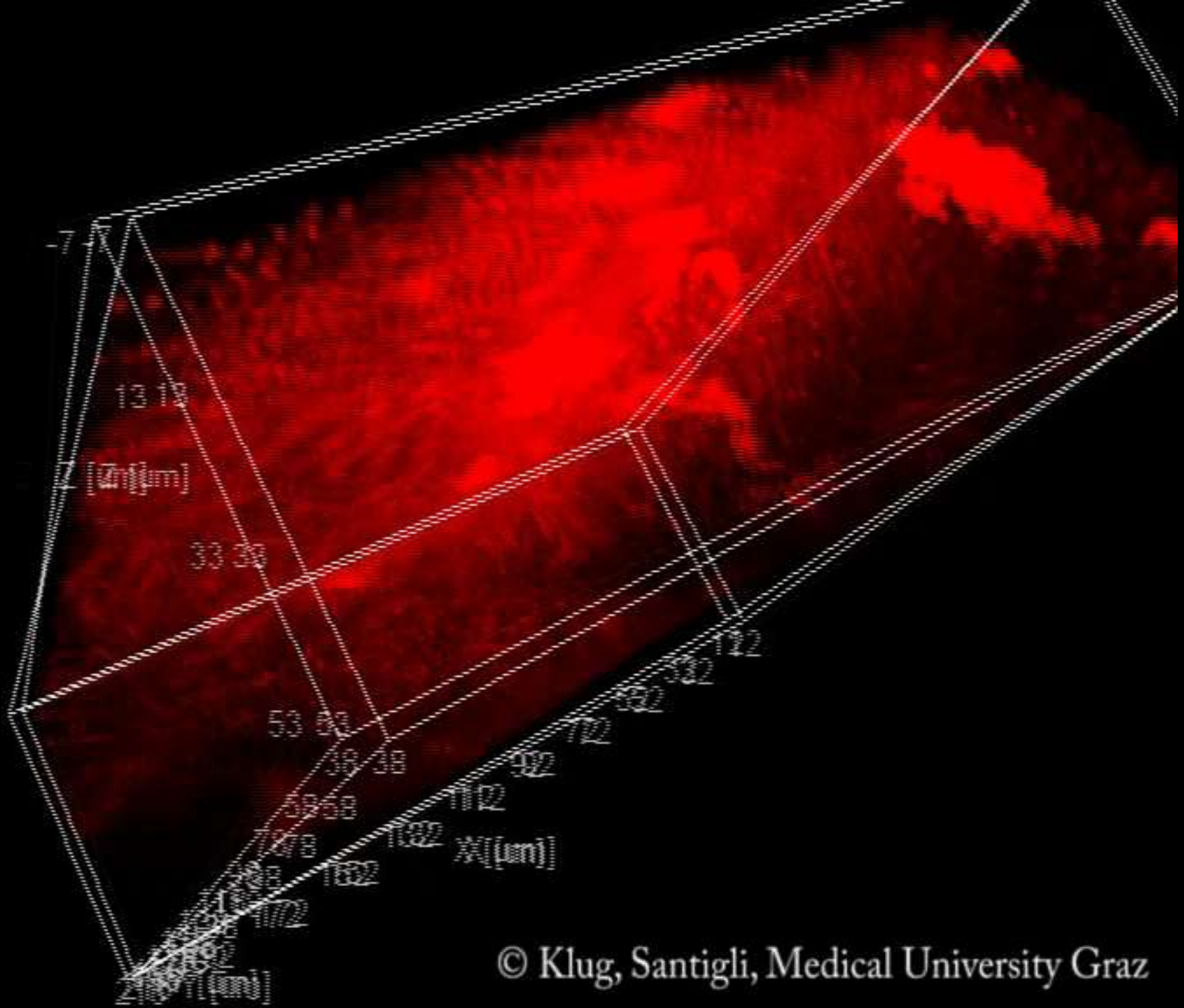
[1]*Division of Pediatric Dentistry and Community Oral Health, The Ohio State University College of Dentistry, Columbus, OH, USA;* [2]*Division of Oral Biology, The Ohio State University College of Dentistry, Columbus, OH, USA;* [3]*Biosciences Division, Oak Ridge National Laboratory, Oak Ridge, TN, USA;* [4]*Division of Periodontology, The Ohio State University College of Dentistry, Columbus, OH, USA and* [5]*Genome Science and Technology Program, University of Tennessee, Knoxville, TN, USA*

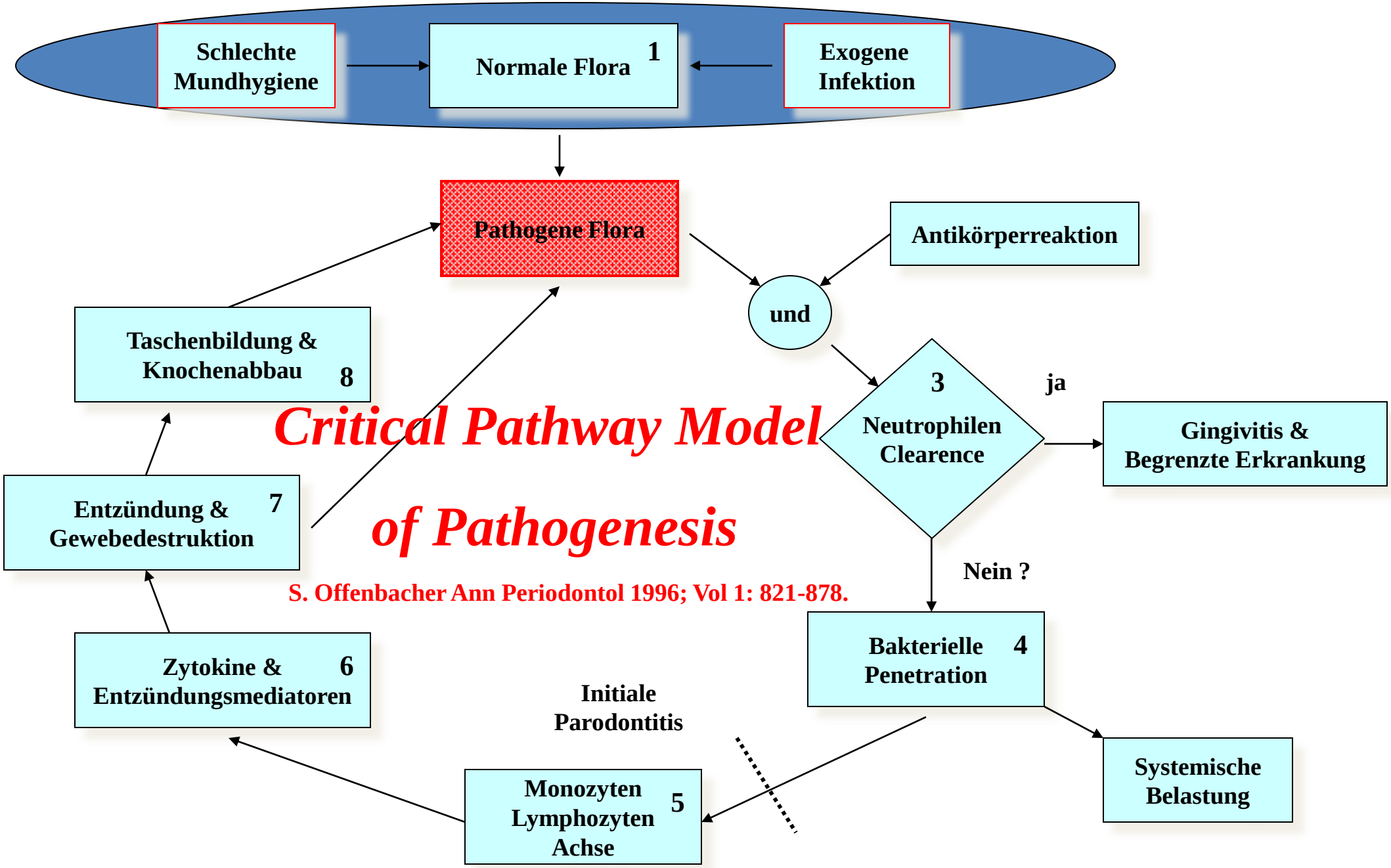
- Periodontitis has a ***polymicrobial etiology*** within the framework of a complex microbial ecosystem. With advances in sequencing technologies, comprehensive studies to elucidate bacterial community differences have recently become possible (454 sequencing of 16S rRNA genes).
- ***Differences between health- and periodontitis-associated bacterial communities*** were observed at all phylo-genetic levels, and analysis showed distinct community profiles in health and disease.
- Community ***diversity was higher in disease***, and 123 species were identified that were significantly more abundant in disease, and 53 in health.
- Elucidation of these differences in community composition provides a basis for further understanding the pathogenesis of periodontitis.

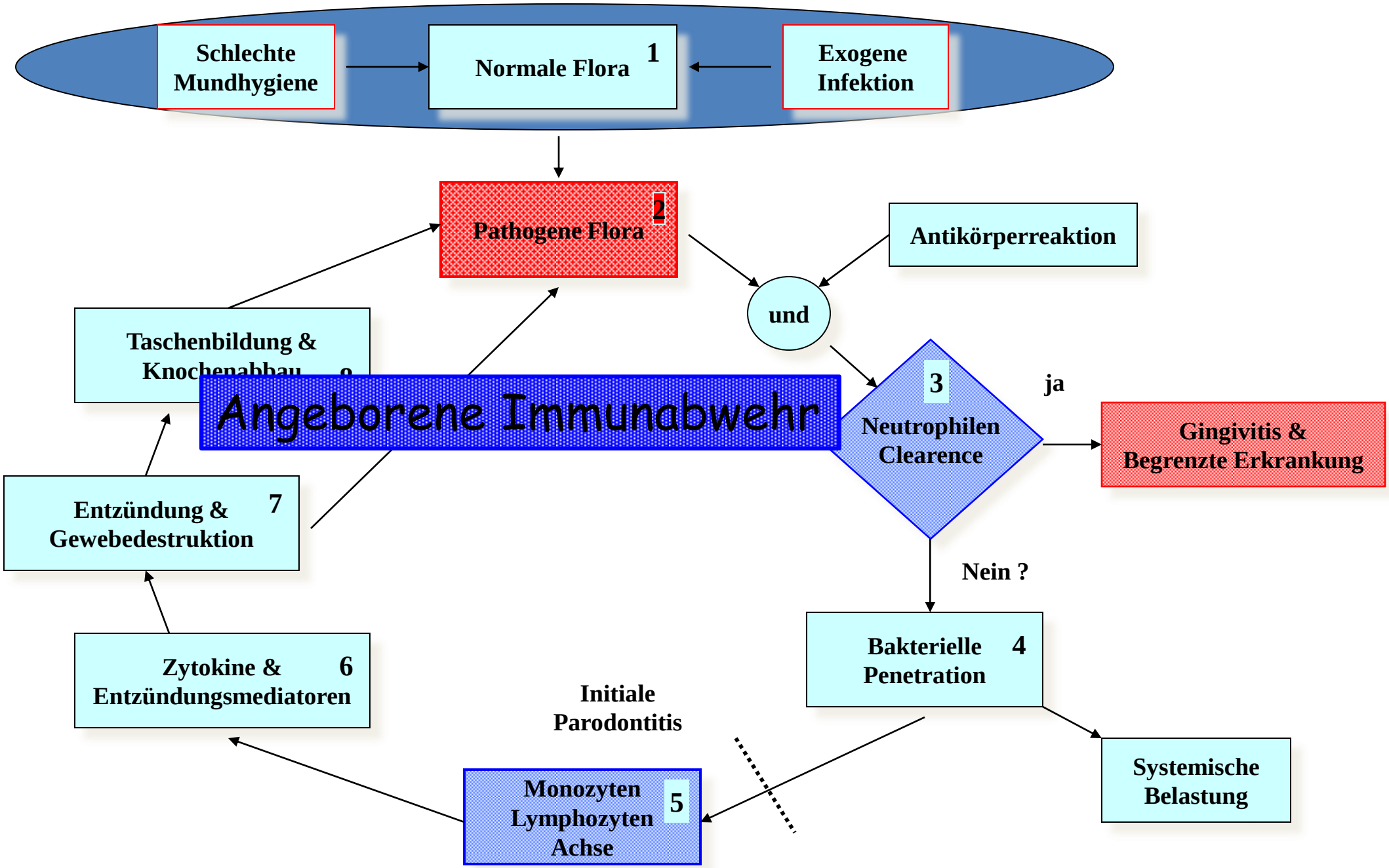
Griffen AL. et al. Distinct and complex bacterial profiles in human periodontitis and health revealed by 16S pyrosequencing. ISME J 2012, 6(6):1176.



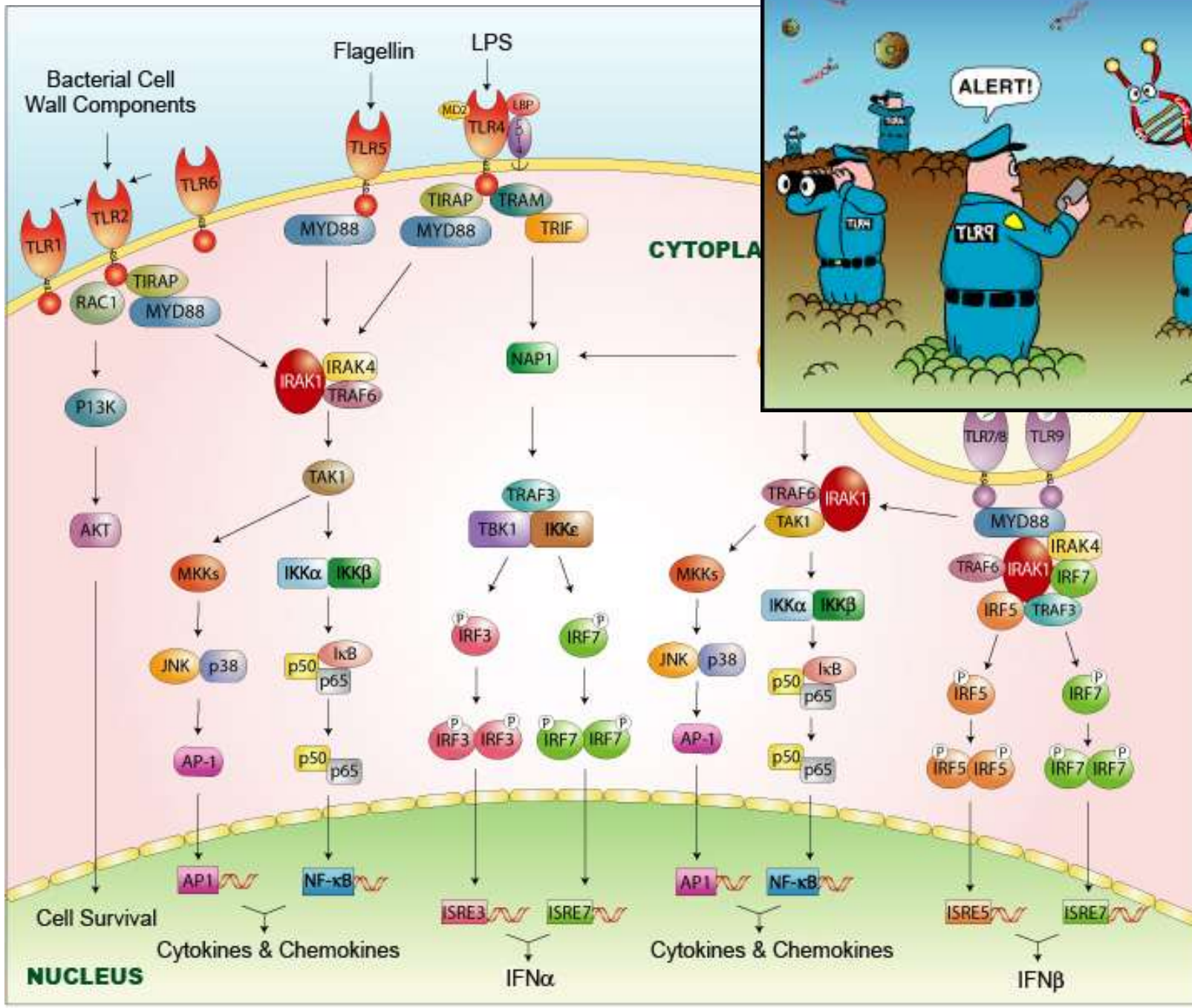
Circular maximum likelihood phylogenetic tree at level of genus

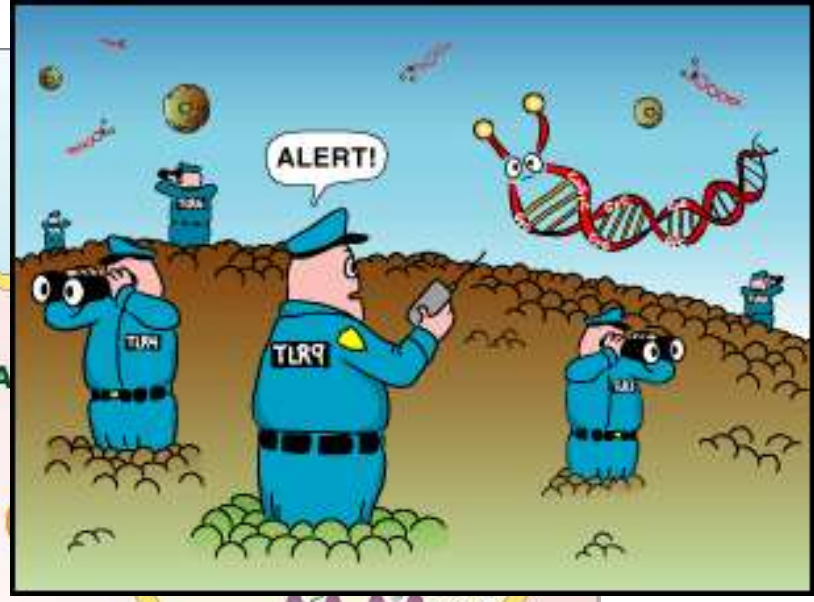


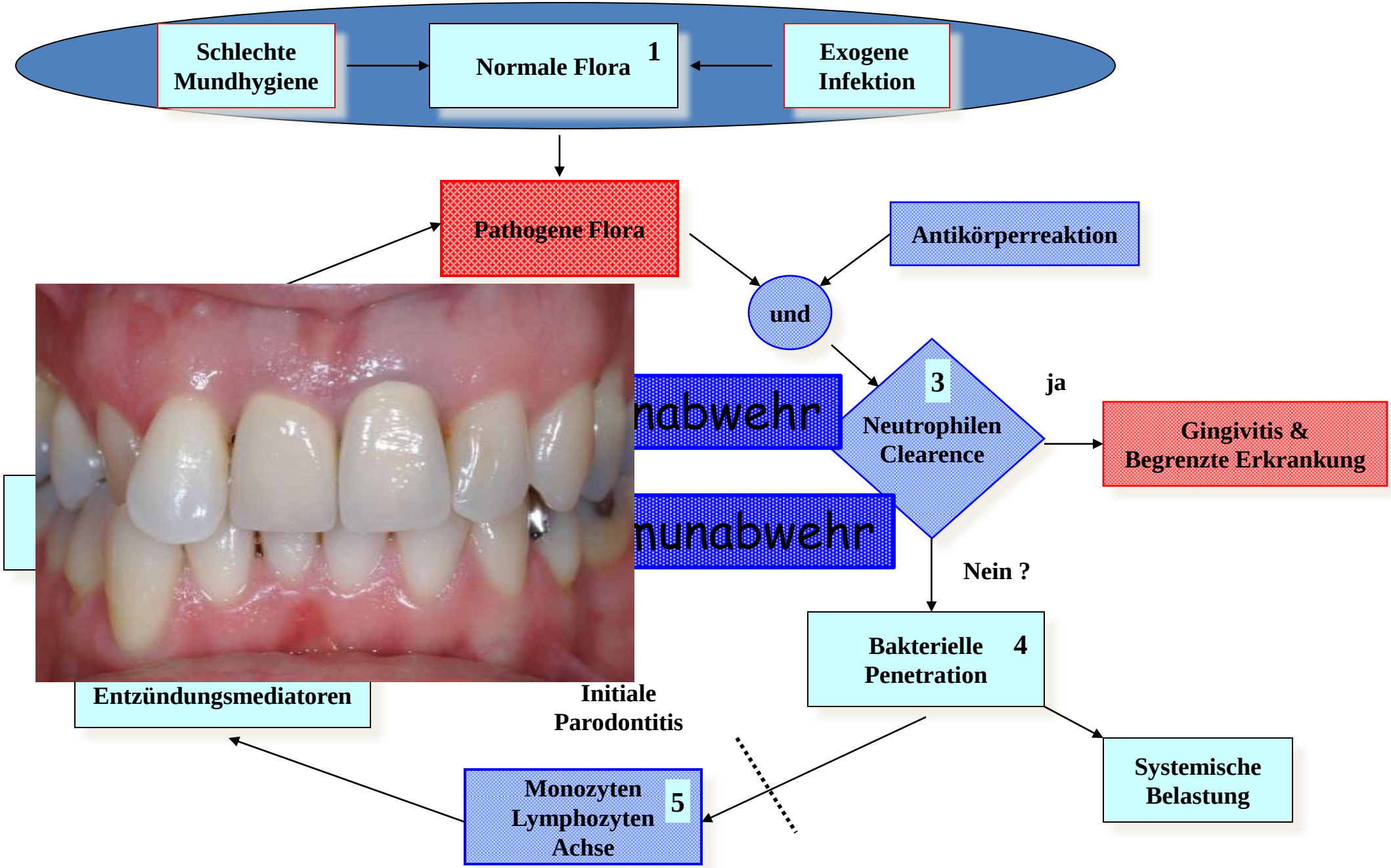

Schlechte Mundhygiene
Normale Flora 1
Exogene Infektion
Pathogene Flora
Antikörperreaktion
und
3 Neutrophilen Clearence
ja
Gingivitis & Begrenzte Erkrankung
Nein ?
Bakterielle Penetration 4
Systemische Belastung
Initiale Parodontitis
Monozyten Lymphozyten Achse 5
Zytokine & Entzündungsmediatoren 6
Entzündung & Gewebedestruktion 7
Taschenbildung & Knochenabbau 8
Critical Pathway Model of Pathogenesis
S. Offenbacher Ann Periodontol 1996; Vol 1: 821-878.


Schlechte Mundhygiene
Normale Flora 1
Exogene Infektion
Pathogene Flora 2
Antikörperreaktion
und
Taschenbildung & Knochenabbau 8
Angeborene Immunabwehr
3 Neutrophilen Clearence
ja
Gingivitis & Begrenzte Erkrankung
Entzündung & Gewebedestruktion 7
Nein ?
Bakterielle Penetration 4
Zytokine & Entzündungsmediatoren 6
Initiale Parodontitis
Systemische Belastung
Monozyten Lymphozyten Achse 5

Toll like receptoren






Schlechte Mundhygiene
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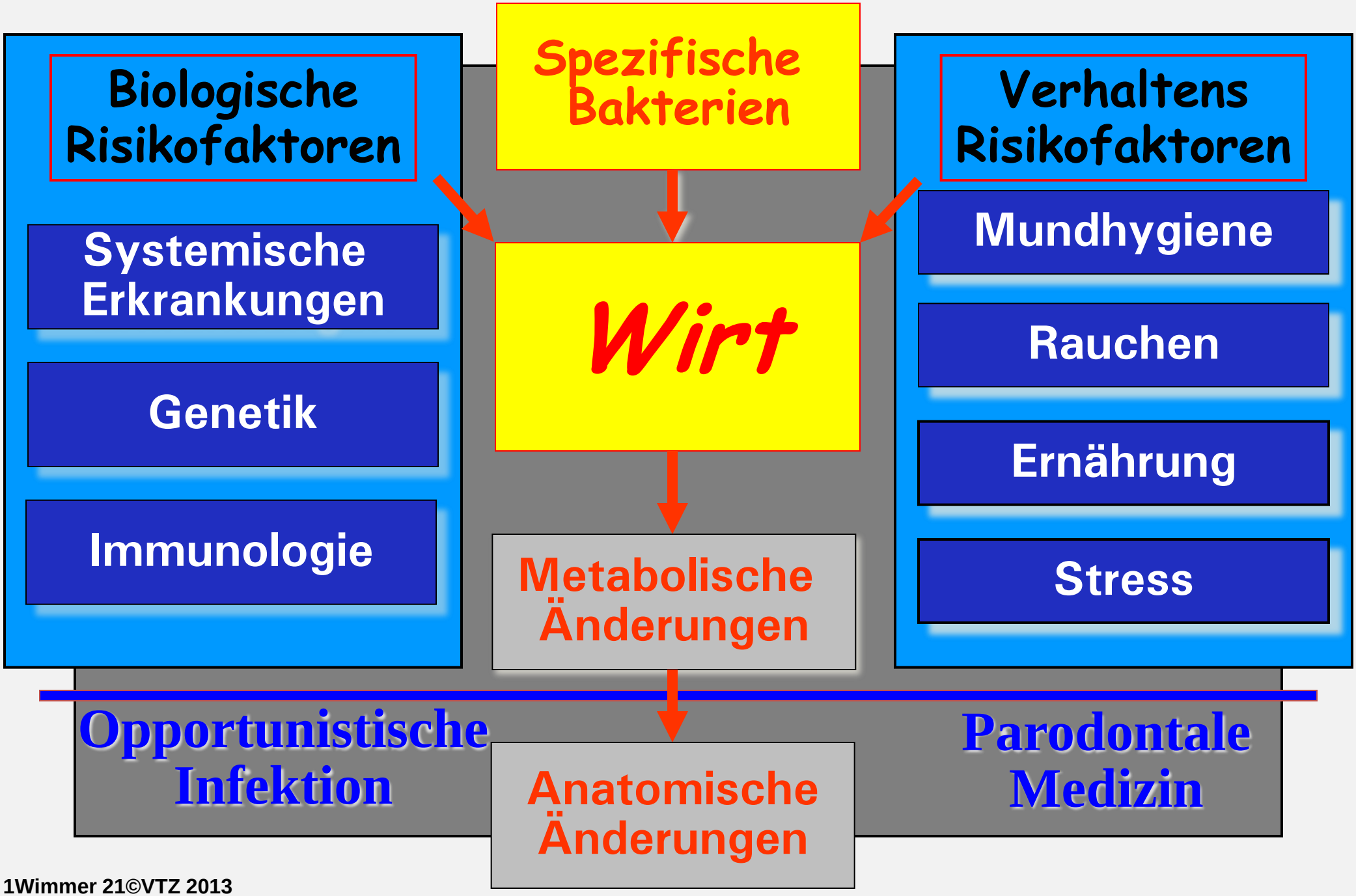
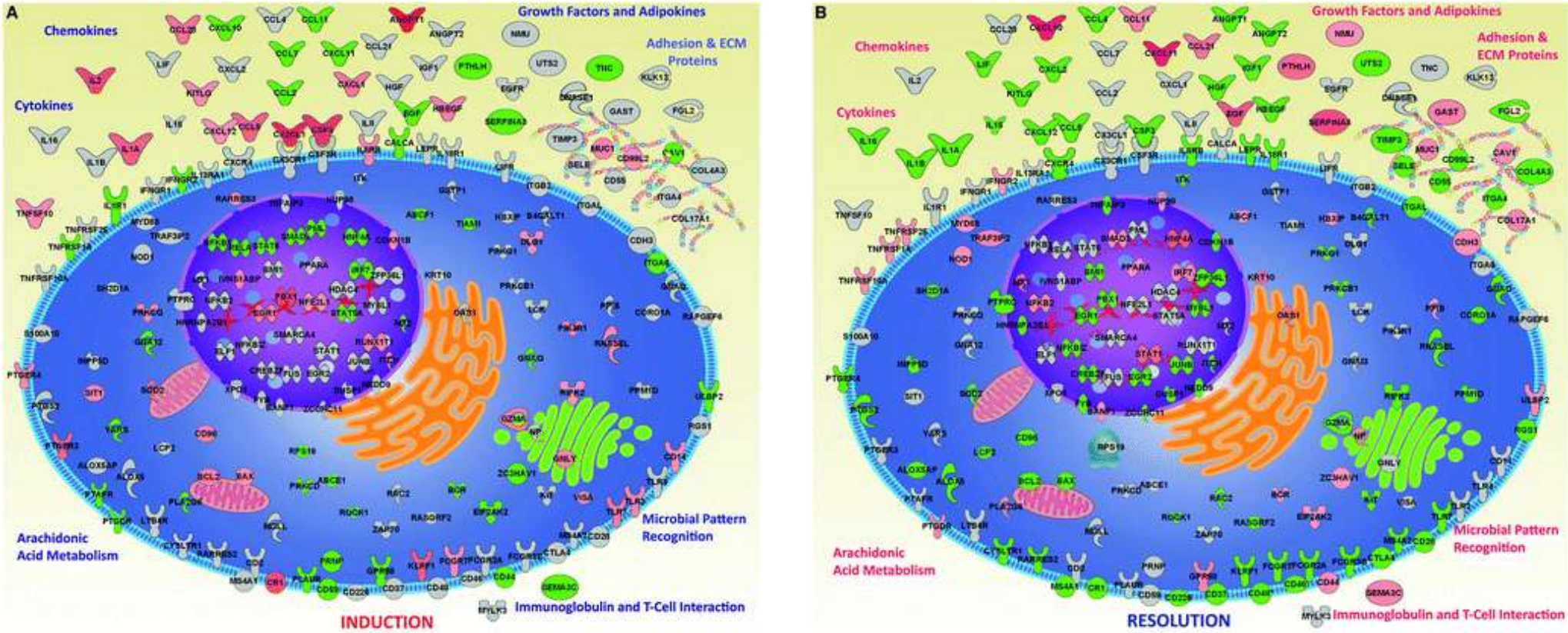

Biologische Risikofaktoren
Systemische Erkrankungen
Genetik
Immunologie
Spezifische Bakterien
Wirt
Metabolische Änderungen
Anatomische Änderungen
Verhaltens Risikofaktoren
Mundhygiene
Rauchen
Ernährung
Stress
Opportunistische Infektion
Parodontale Medizin

Offenbacher S. et al.

Gingival Transcriptome Patterns During Induction and Resolution of Experimental Gingivitis in Humans. *JP* 2009;80:1963-1982.

JOURNAL OF Periodontology

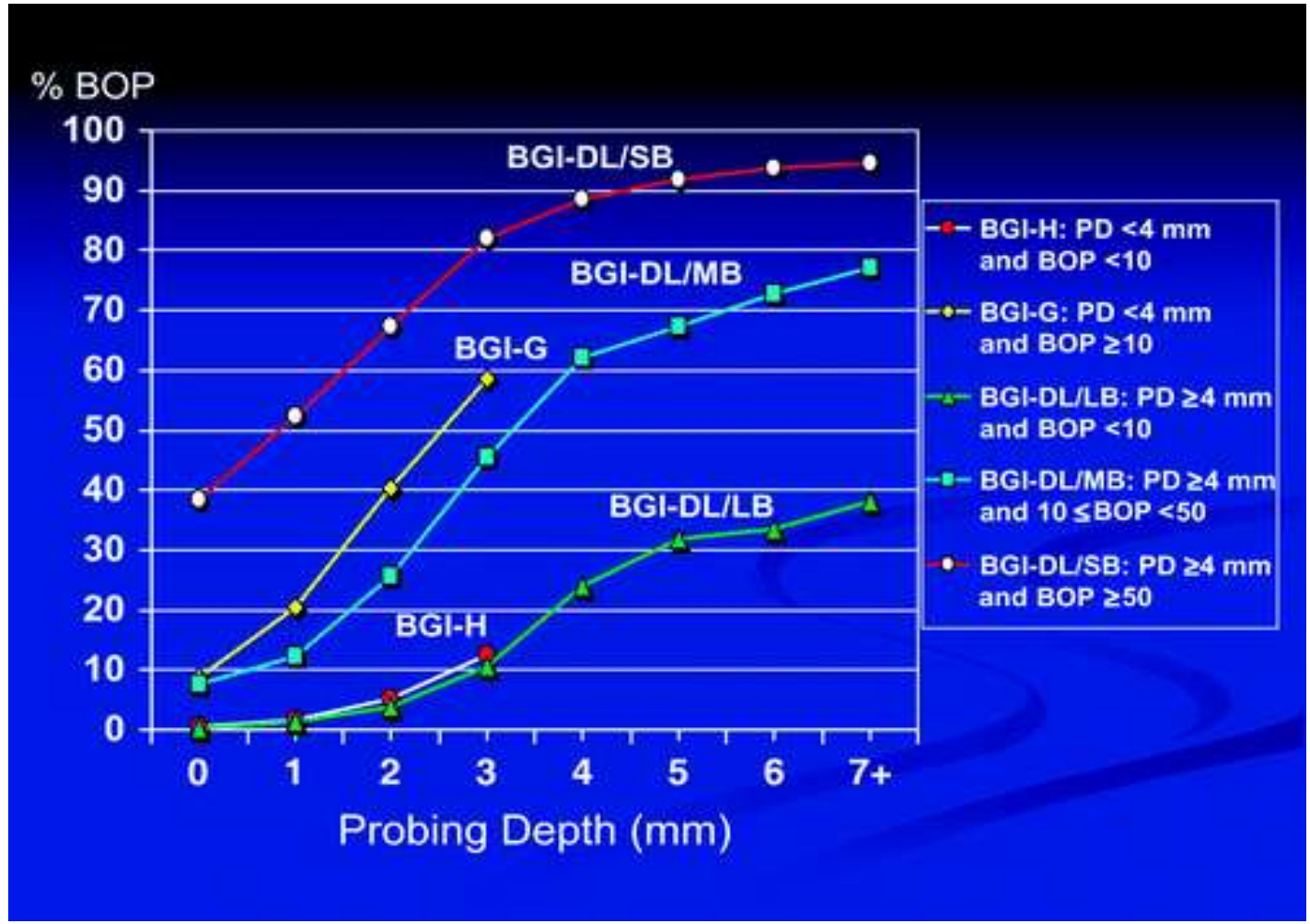


Changes in the patterns of immune response gene expression during induction and resolution of gingivitis.

A) Induction phase. **B)** Resolution.

Color reflects changes in gene expression during induction (day 28 versus day 0) and resolution (day 35 versus day 28).

S. Offenbacher, S.P. Barros, R.E. Singer, K. Moss, R.C. Williams, J.D. Beck·
Periodontal Disease at the Biofilm–Gingival Interface. Journal of Periodontology 2007, 78(10): 1911.



New BGI classifications create categories with distinct biologic phenotypes.

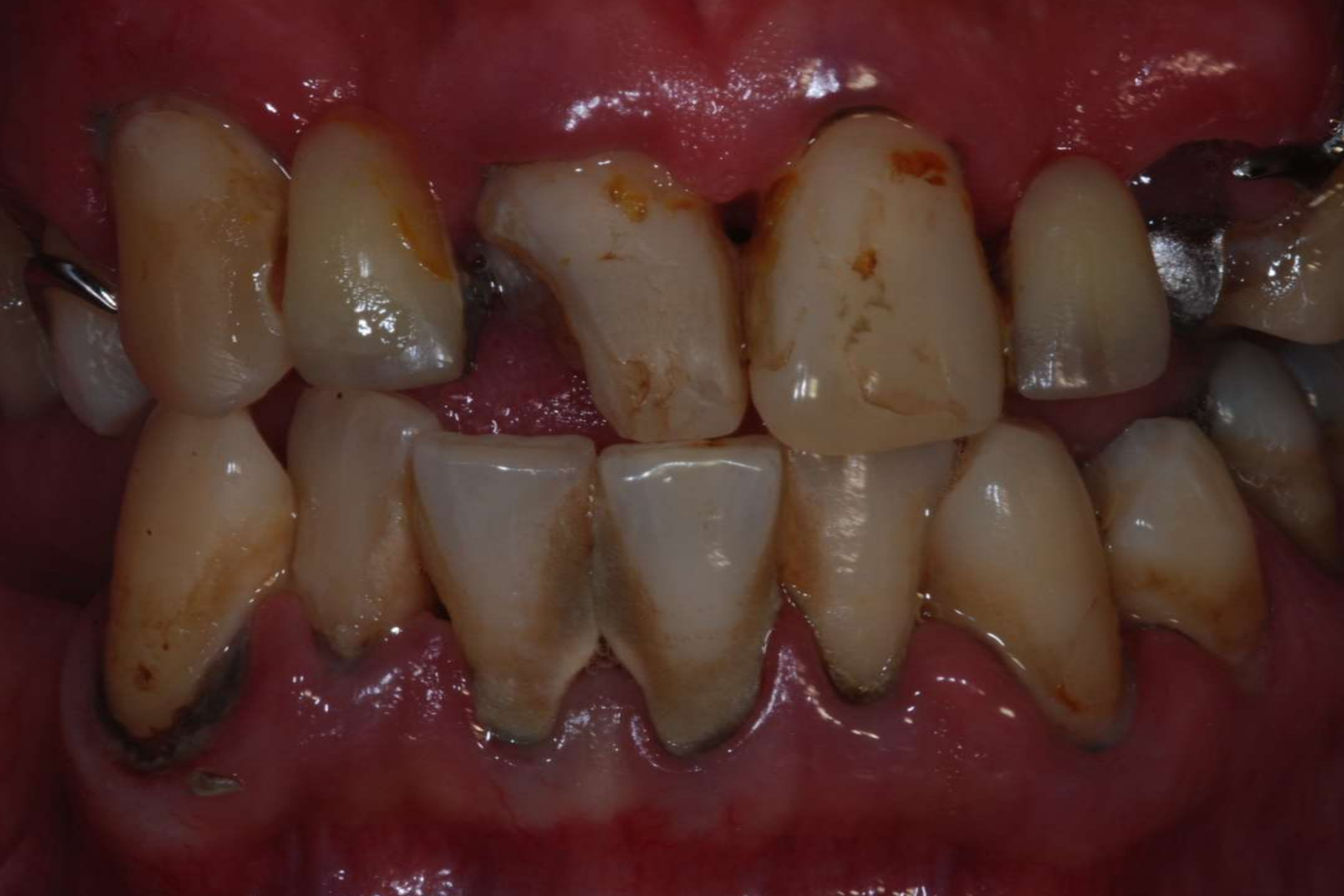
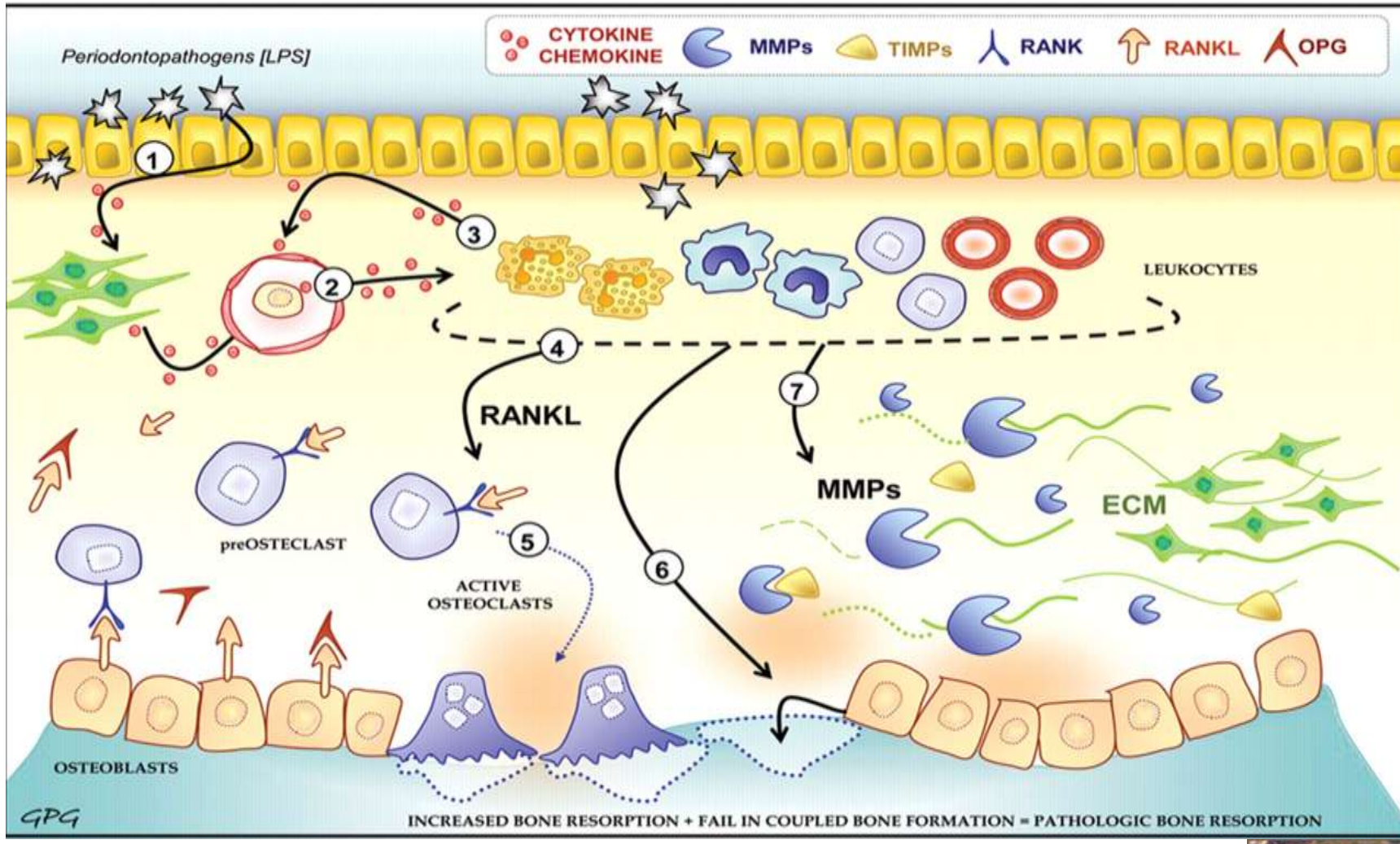
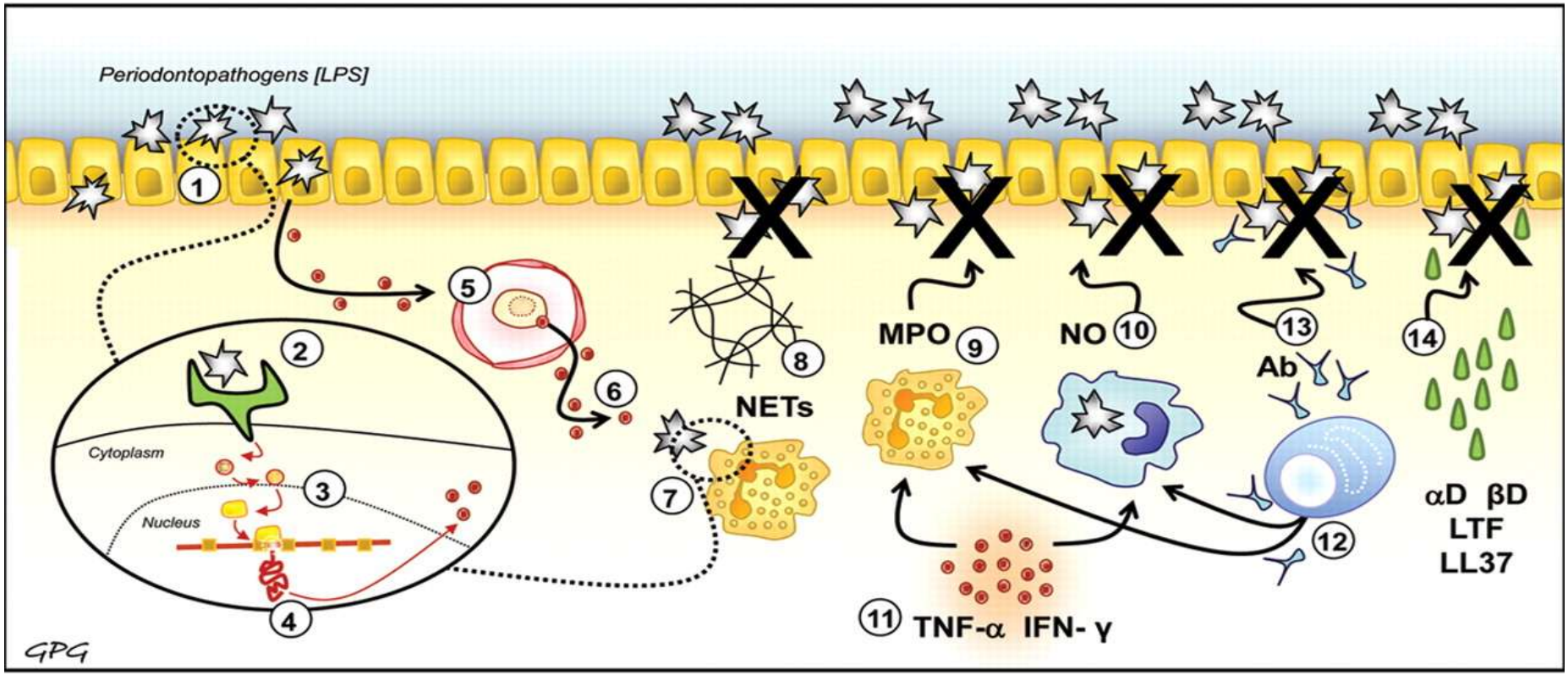


Figure 1. The cellular and molecular pathways linking host inflammatory immune response to periodontal disease progression.



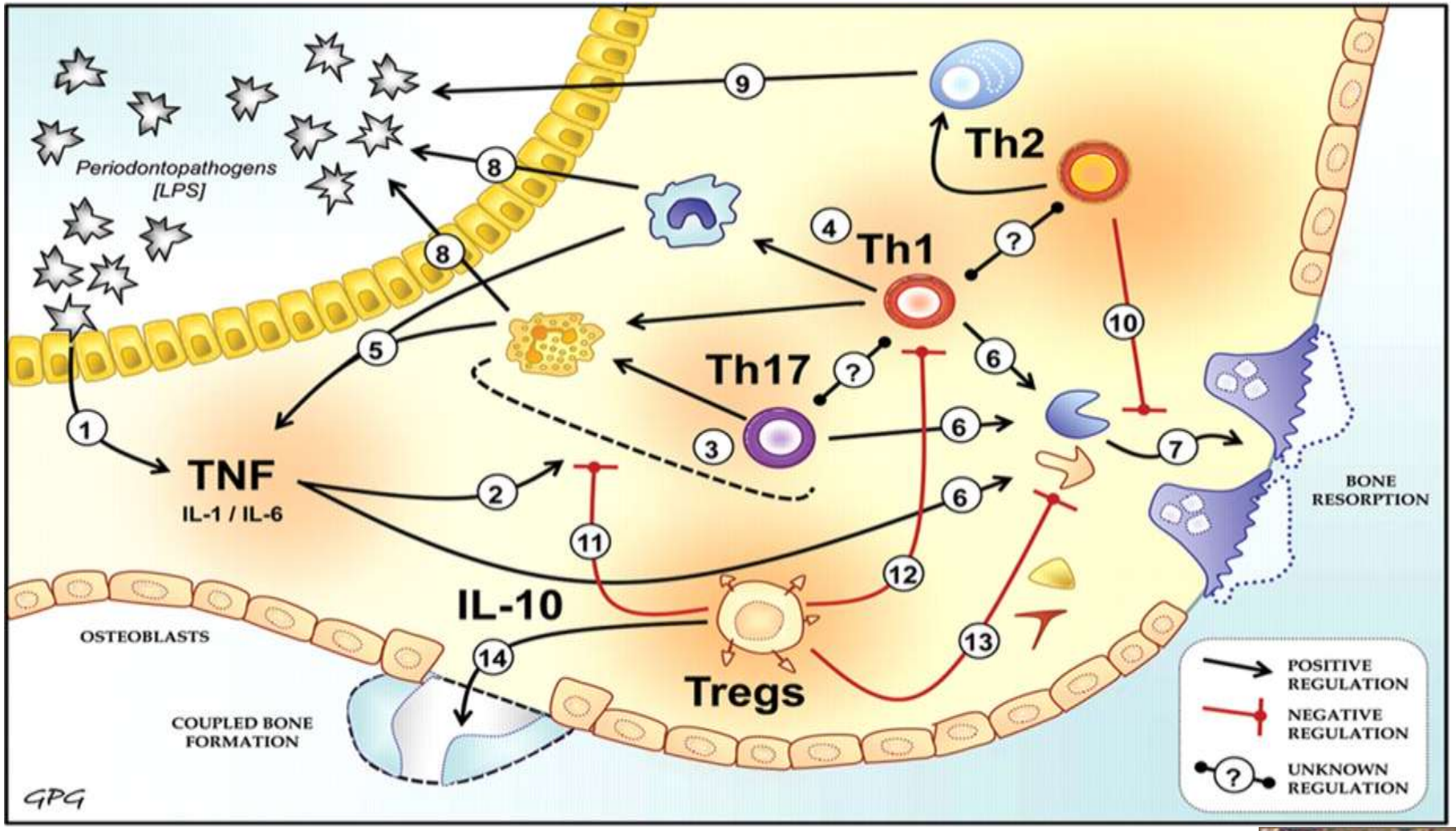
Garlet G J DENT RES 2010;89:1349-1363

Figure 2. The cellular and molecular pathways linking host inflammatory immune response to the control of periodontal infection.

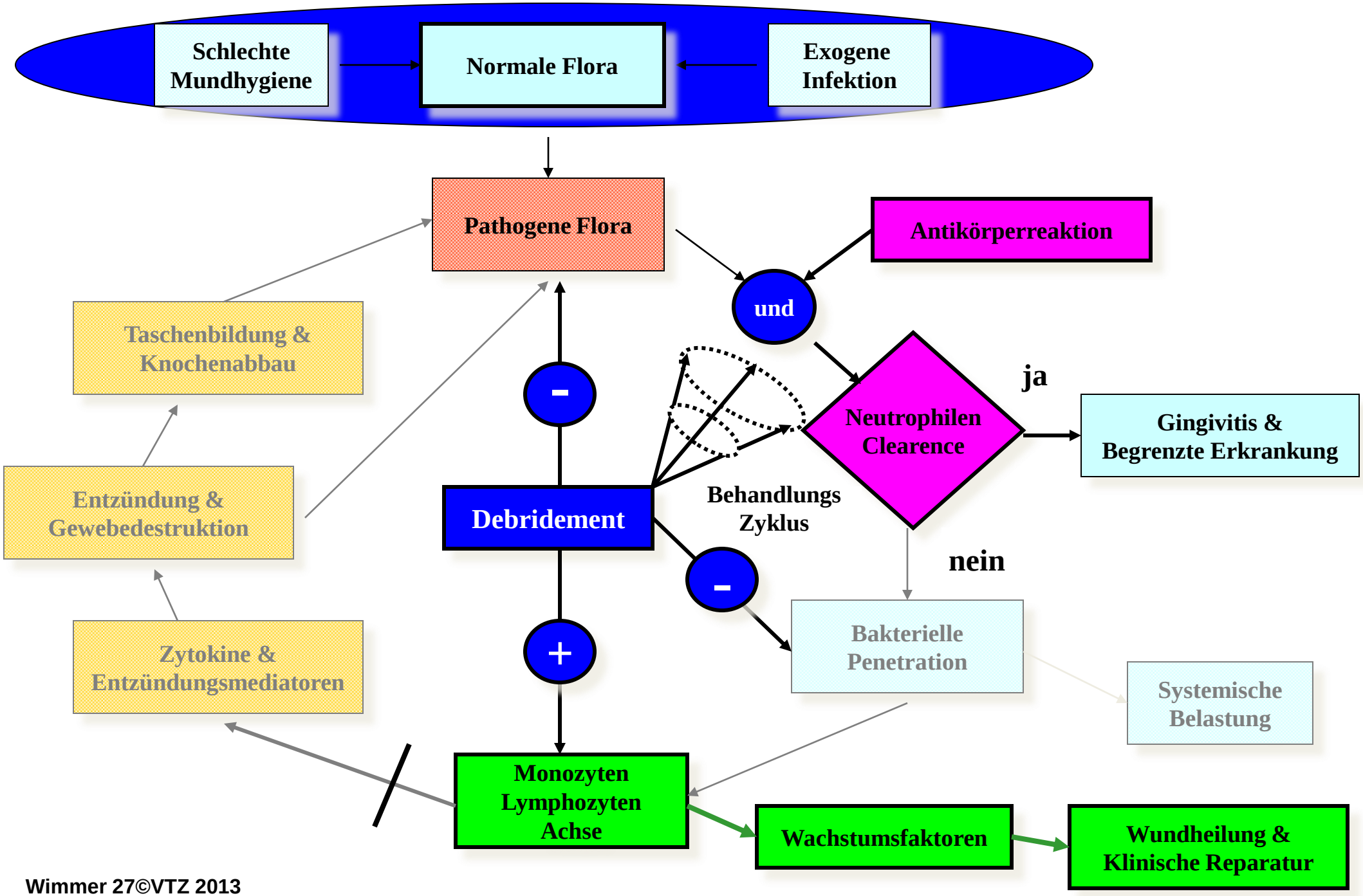


Garlet G J DENT RES 2010;89:1349-1363

Figure 3. The cytokine crosstalk and its destructive and protective roles in periodontal disease: a re-appraisal from host defense and tissue destruction viewpoints.



Garlet G J DENT RES 2010;89:1349-1363



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Parodontale Medizin

- Allgemeine und systemische Umstände erhöhen die Anfälligkeit für parodontale Erkrankungen.
- Parodontale Infektionen sind jedoch auch als entzündliche metastasierende Erkrankungen für die allgemeine Gesundheit ein Risikofaktor !

Privatdozent Dr. G. Wimmer
Medizinische Universität Graz

Parodontale Medizin

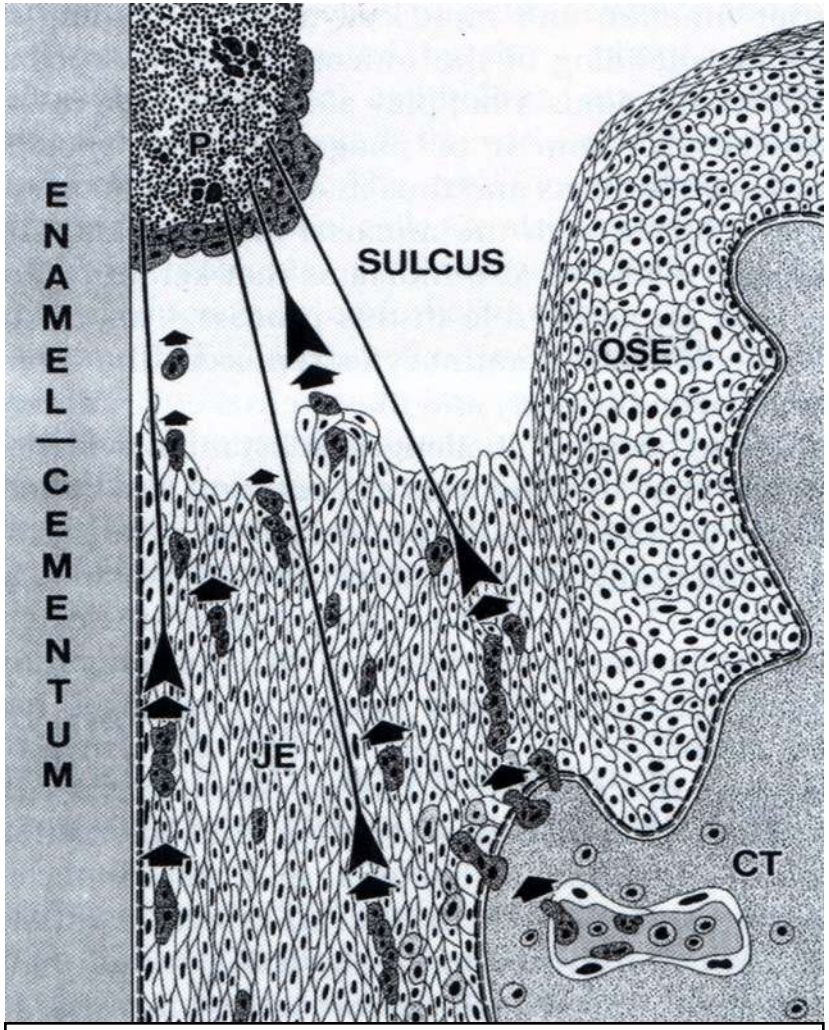
Mechanismen non-oraler Manifestationen oraler Erkrankungen:

- **Metastatische Infektionen via transienter Bakteriämien**
- **Metastatische Dissemination und Zirkulation mikrobieller Toxine**
- **Metastatische Inflammation durch immunologische Schädigung**

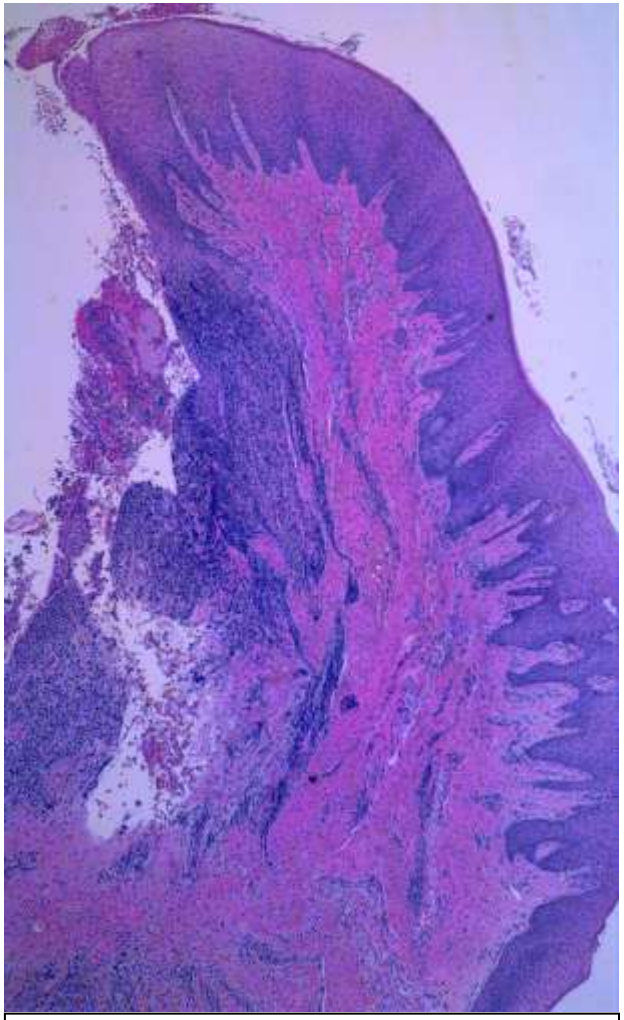
Kinane D. et al. Bacteraemia following periodontal procedures. J Clin Perio 2005 (32): 708.

Chiang A. & Massague J. Molecular basis of metastasis. N Engl J med 2008 (359): 2814.

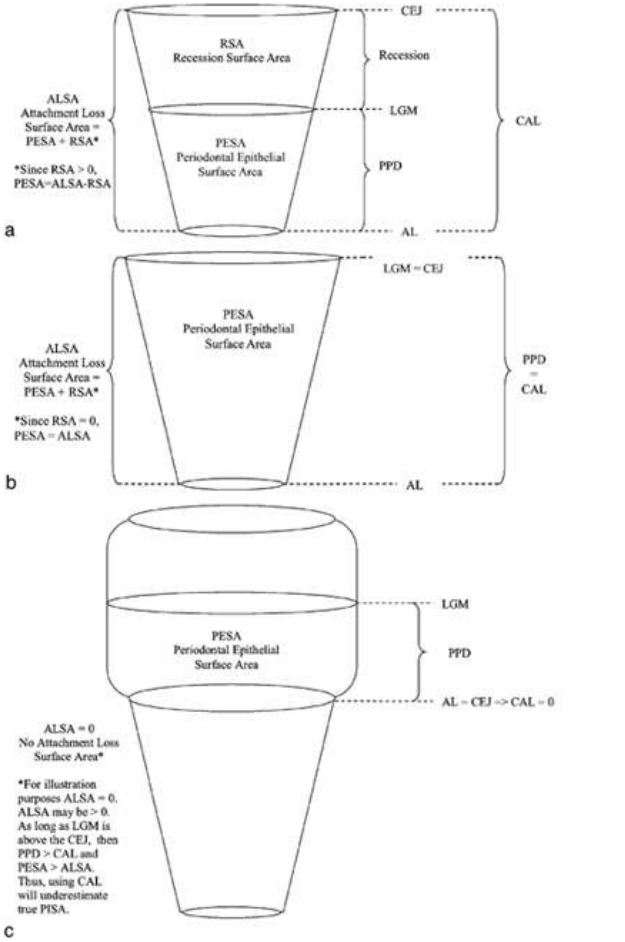
Rams TE, Slots J. Systemic manifestations of oral infections. St. Louis, Mosby; 1992. 500.



Schroeder HE, Attström R. Pocket formation: an hypothesis.



Wimmer G, Kresse AD.



List of Abbreviations:
AL= Attachment Level; ALSA= Attachment Loss Surface Area; CAL= Clinical Attachment Level;
CEJ= Cemento-Enemal Junction; LGM= Location of Gingival Margin;
PESA= Periodontal Epithelial Surface Area; PISA= Periodontal Inflamed Surface Area;
PPD= Probing Pocket Depth, RSA= Recession Surface Area

Nesse W. et al. Periodontal inflamed surface area: quantifying inflammatory burden. J Clin Perio 2008 (35): 668.

A freely clinical downloadable spreadsheet is available calculate PISA using CAL, recession and BOP measurements. This spreadsheet can be used to show patients their surface area of bleeding pocket epithelium, illustrating the inflammatory burden periodontitis potentially poses to their body.

www.parsprototo.info

	tooth	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28	tooth	
CAL	buccal		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		buccal	CAL
	palatinal		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		palantinal	
	lingual		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		lingual	
CAL	buccal		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		buccal	CAL
	tooth	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38	tooth	

	tooth	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28	tooth	
LGM	buccal		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		buccal	LGM
	palatinal		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		palantinal	
	lingual		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		lingual	
LGM	buccal		0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0		buccal	LGM
	tooth	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38	tooth	

tooth	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28	tooth
ALSA (mm2)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	ALSA (mm2)
(mm2) ALSA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	(mm2) ALSA
tooth	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38	tooth

tooth	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28	tooth
RSA (mm2)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	RSA (mm2)
(mm2) RSA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	(mm2) RSA
tooth	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38	tooth

tooth	PESA	nr of sites with BOP	PISA (mm2)
18	0		0
17	0		0
16	0		0
15	0		0
14	0		0
13	0		0
12	0		0
11	0		0
21	0		0
22	0		0
23	0		0
24	0		0
25	0		0
26	0		0
27	0		0
28	0		0

tooth	PESA	nr of sites with BOP	PISA (mm2)
38	0		0
37	0		0
36	0		0
35	0		0
34	0		0
33	0		0
32	0		0
31	0		0
41	0		0
42	0		0
43	0		0
44	0		0
45	0		0
46	0		0
47	0		0
48	0		0

Total **Periodontal Epithelial Surface Area** (mm2)
0

Total **Periodontal Inflamed Surface Area** (mm2)
0

CAL = Clinical Attachment Level relative to CEJ
LGM = Location of Gingival Margin relative to CEJ
ALSA = Attachment Loss Surface Area
RSA = Recession Surface Area
PESA = Periodontal Epithelial Surface Area
PISA = Periodontal Inflamed Surface Area

Nesse W. et al. Periodontal inflamed surface area: quantifying inflammatory burden. J Clin Perio 2008 (35): 668.

Parodontitis als Risiko für andere Erkrankunkungen

- **Kardiovaskuläre Erkrankungen**
- **Diabetes mellitus**
- **Untergewichtige Frühgeburten**
- **COPD, Pneumonie, CKD, RA, CI, Fettsucht,**

  Metabolisches Syndrom, Cancer,

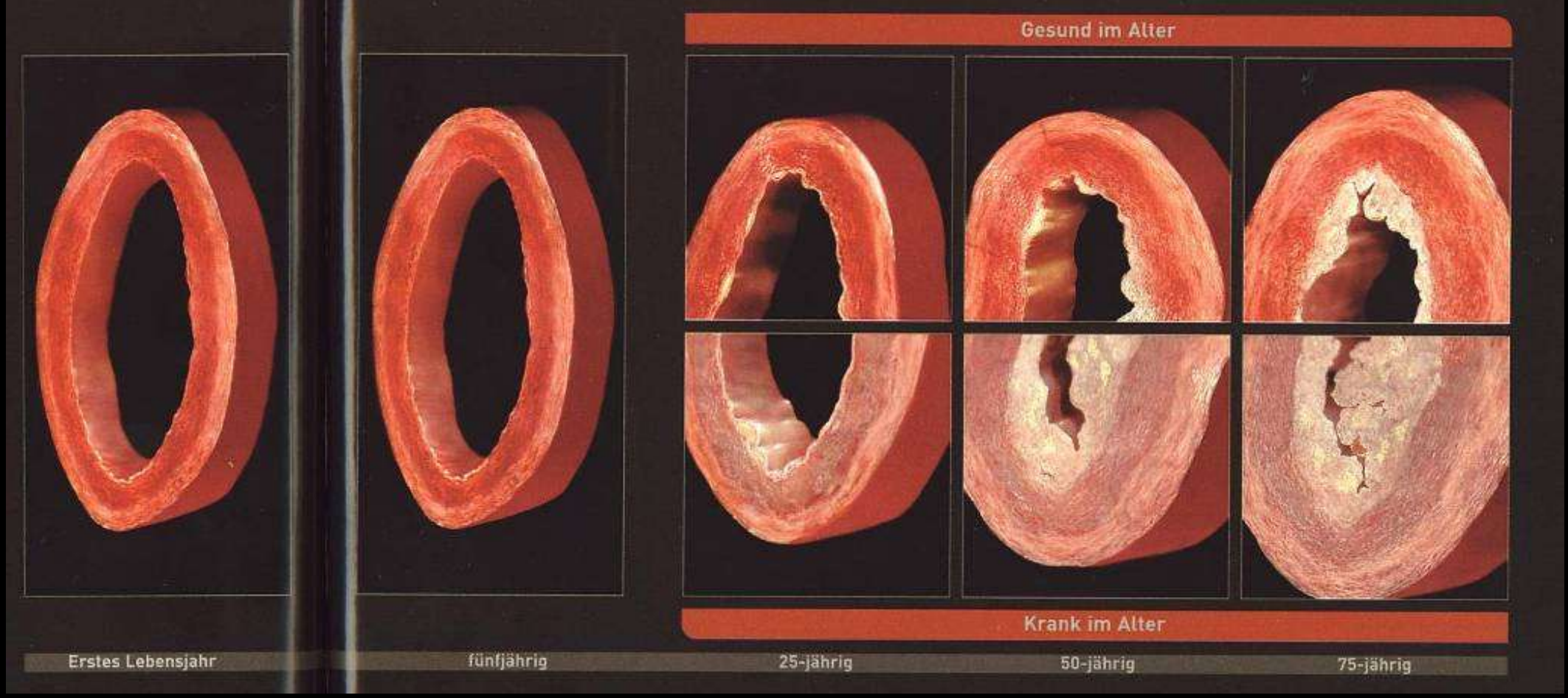
1st Joint EFP/AAP Workshop on Periodontal and Systemic Diseases, LaGranja 11/1212

Journal of Clinical Periodontology and Journal of Periodontology Feber/March 2013

Parodontitis & kardiovaskuläre Erkrankungen

Atherosklerotische CVD umfassen eine Gruppe von Erkrankungen inklusive nicht-fataler und fataler ***koronaler*** Herzerkrankungen (Angina pectoris, Myokardinfarkt), ischämische ***cerebrovasculäre*** Erkrankungen (Schlaganfall/ TIA) und ***peripherarteriale*** Erkrankungen. Kardiovaskuläre Erkrankungen sind in den westlich industrialisierten Ländern die häufigste Todesursache. (WHO 1995)

GEO



Parodontitis und kardiovaskuläre Erkrankungen haben eine Reihe gemeinsamer Charakteristika

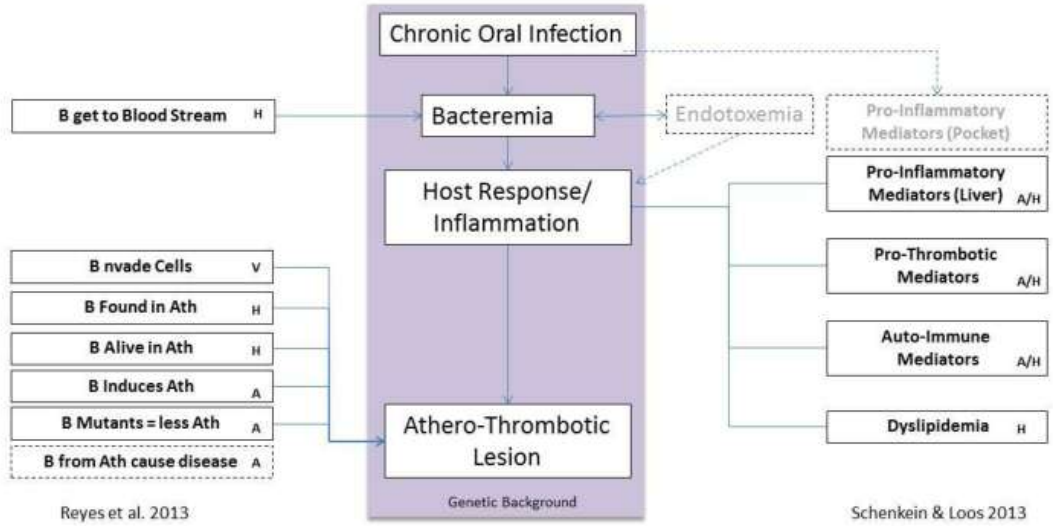
Sind infektiöse parodontale Erkrankungen ein Risikofaktor für artherosklerotisch kardiovaskuläre Erkrankungen?

Kritische Fragen:

1. To assess **biological plausibility** of mechanisms underpinning the relationship between periodontitis and cardiovascular diseases.
2. To review the available **epidemiological evidence** with an emphasis on longitudinal studies allowing measures of excess cardiovascular risk attributable to periodontitis
3. To review the results of initial **intervention trials** on the benefits of periodontal therapy on surrogate cardiovascular outcomes
4. To critically evaluate the available evidence spanning biological plausibility of mechanisms, epidemiological evidence and initial intervention trials
5. To identify key issues for the design of future trials
6. To provide **reasonable action/recommendations** for the public, the dental and medical profession at this stage of incomplete knowledge

Consensus Report of the Joint European Federation of Periodontology and American Academy of Periodontology Workshop on Periodontitis and Systemic Diseases, J Clin Perio 2013.

Biologische Plausibilität



Tomás I, Diz P, Tobías A, Scully C, Donos N. (2012) Periodontal health status and bacteraemia from daily oral activities: systematic review/meta-analysis. Journal of Clinical Periodontology 39:213-28.

Reyes L, Kozarov E, Herrera D, Roldan S, Progulske-Fox A. (2013). Periodontal Bacterial Invasion and Infection: Contribution to Cardiovascular Disease. Journal of Clinical Periodontology in press.

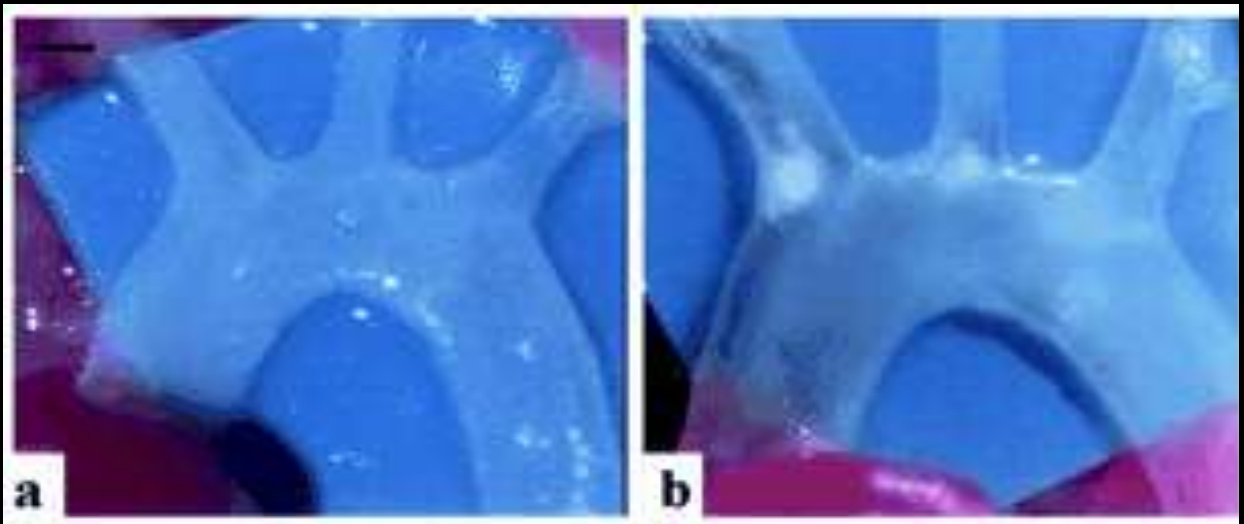
Schenkein HA, Loos BG. (2013) Inflammatory Mechanisms Linking Periodontal Diseases to Cardiovascular Diseases. Journal of Clinical Periodontology in press.

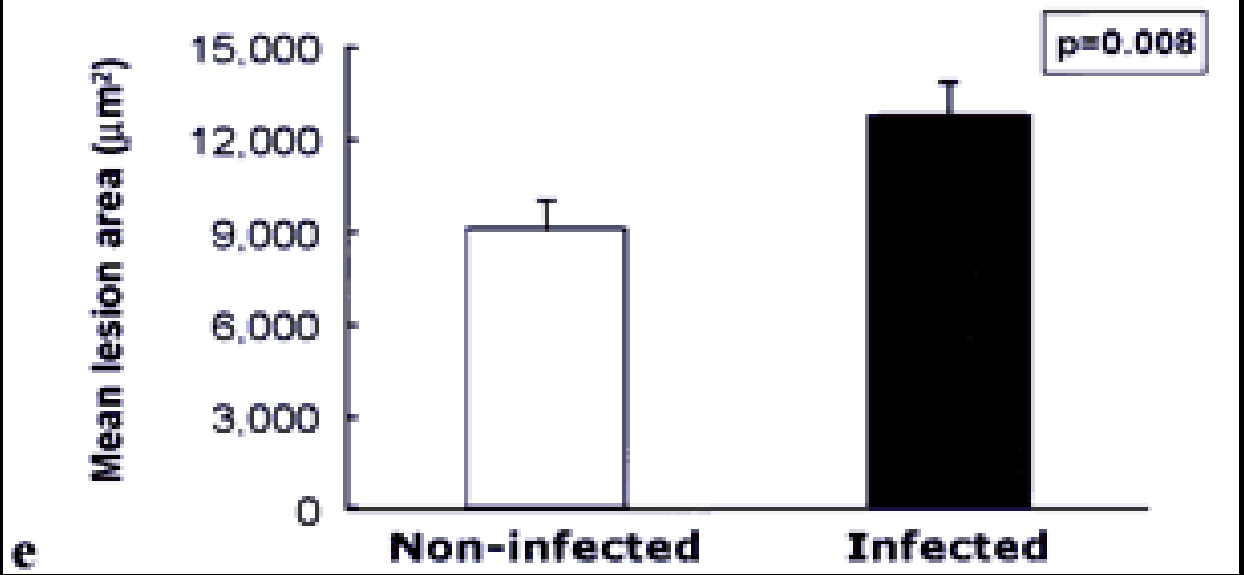
Parodontitis & kardiovaskuläre Erkrankungen

Existieren Plausibilitäts-Hinweise?

Studie	Hintergrund
Herzberg 1998 Ann Periodontol	PAAP^{+}S. sanguis interagiert in vitro mit zirkulierenden Plättchen und führt zu Aggregationen mit pulmonären und cardialen Abnor.
Chiu 1999 Am Heart J	Nachweis von *Porphyromonas gingivalis* und *Streptococc. sanguis* in humaner artherosclerotischer Plaque (Carotis-Endarterektomie)
Miyakawa 2003 J Periodontol Res	*Porphyromonas gingivalis kann* durch Anhäufung von LDL infolge Proteolyse von Apo B-100 die Schaumzellformation stimulieren.
Ishihara 2004 J Clin Microbiol	Wiederfindungsraten von P.g., A.a.,T.f.,T.d.,C.r. in stenosierender Koronararterienplaque korreliert mit subgingivaler Verteilung.
Li L. et al. 2002 Circulation	Systemische Langzeitbelastung mit *Porphyromonas gingivalis* kann im Mausmodell eine atherogene Plaqueprogression beschleunigen.

Oral Infections With a Periodontal Pathogen Accelerates Early Atherosclerosis in Apolipoprotein E-Null Mice





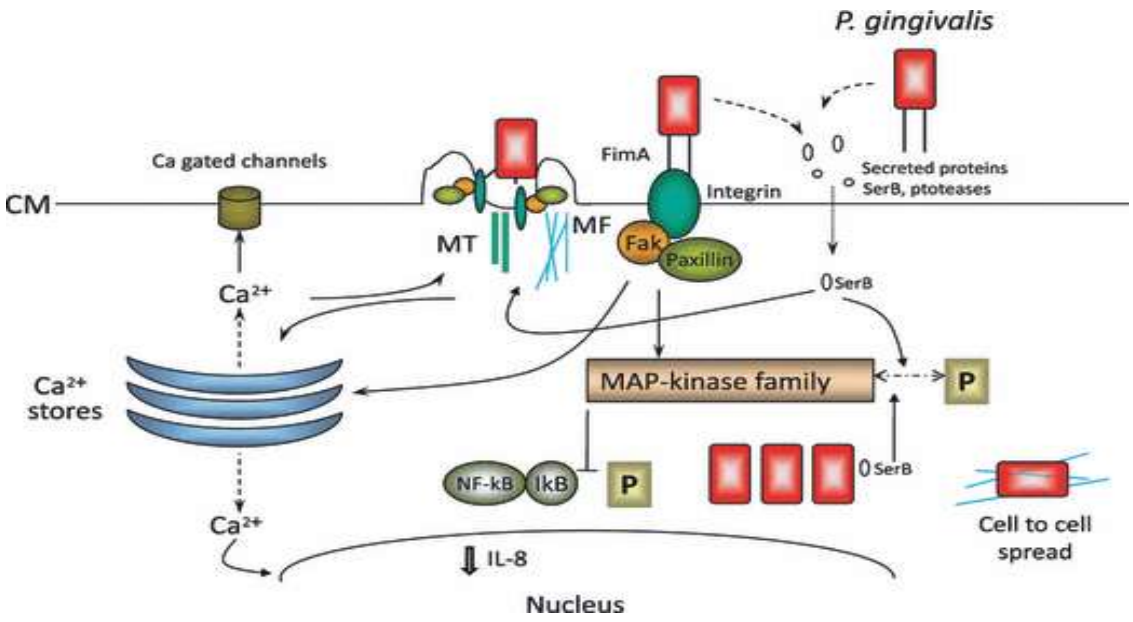
Lalla E. et al.,
Arterioscler Thromb Vasc Biol
2003; 23: 1404-1411,

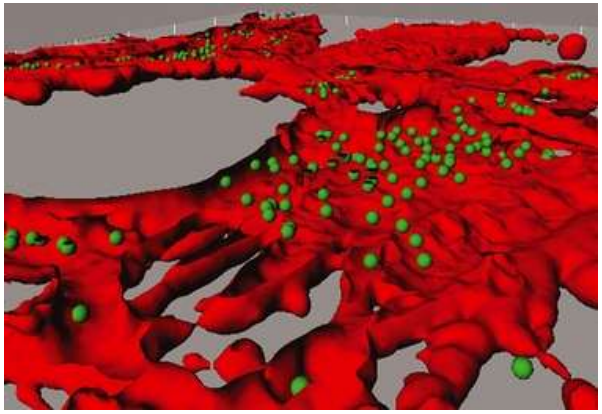
Quantification of atherosclerotic lesion at the aortic sinus

Non-infected vs.
***P. gingivalis*-infected mice**

Gibson E. et al.,
Circulation 2004
Innate immune recognition of invasive bacteria accelerates atherosclerosis in apolipoprotein E-deficient mice.

Gena D. Tribble & Richard J. Lamont. Bacterial invasion of epithelial cells and spreading in periodontal tissue. Periodontology 2000, Volue 52 (1): 68





P. gingivalis **entering to host cells and rapidly locating in the perinuclear area where they replicate and utilize microfilaments to spread to adjacent gingival epithelial cells**

Detection of Periodontal Bacteria in Atheromatous Plaque by Nested Polymerase Chain Reaction

Elena Figuero,*† María Sánchez-Beltrán,† Susana Cuesta-Frechoso,‡ Jose María Tejerina,‡ Jose Antonio del Castro,§ Jose María Gutiérrez,§ David Herrera,*‖ and Mariano Sanz*‖

Methods: Atheromatous plaque from endarterectomies from carotid arteries were scraped and homogenized, and bacterial DNA was extracted. To obtain a representative concentration of amplicons, two amplifications of the bacterial 16S ribosomal-RNA gene were carried out for each sample with universal eubacteria primers by a polymerase chain reaction (PCR). A nested PCR with specific primers for the target bacteria was performed next. Statistical tests included the χ^2 test.

Results: Forty-two atheromatous plaque were analyzed. All of them were positive for ≥1 target bacterial species. The bacterial species most commonly found was *Porphyromonas gingivalis* (78.57%; 33 of 42), followed by *Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans*) (66.67%; 28 of 42), *Tannerella forsythia* (previously *T. forsythensis*) (61.90%; 26 of 42), *Eikenella corrodens* (54.76%; 23 of 42), *Fusobacterium nucleatum* (50.00%; 21 of 42), and *Campylobacter rectus* (9.52%; four of 42). The simultaneous presence of various bacterial species within the same specimen was a common observation.

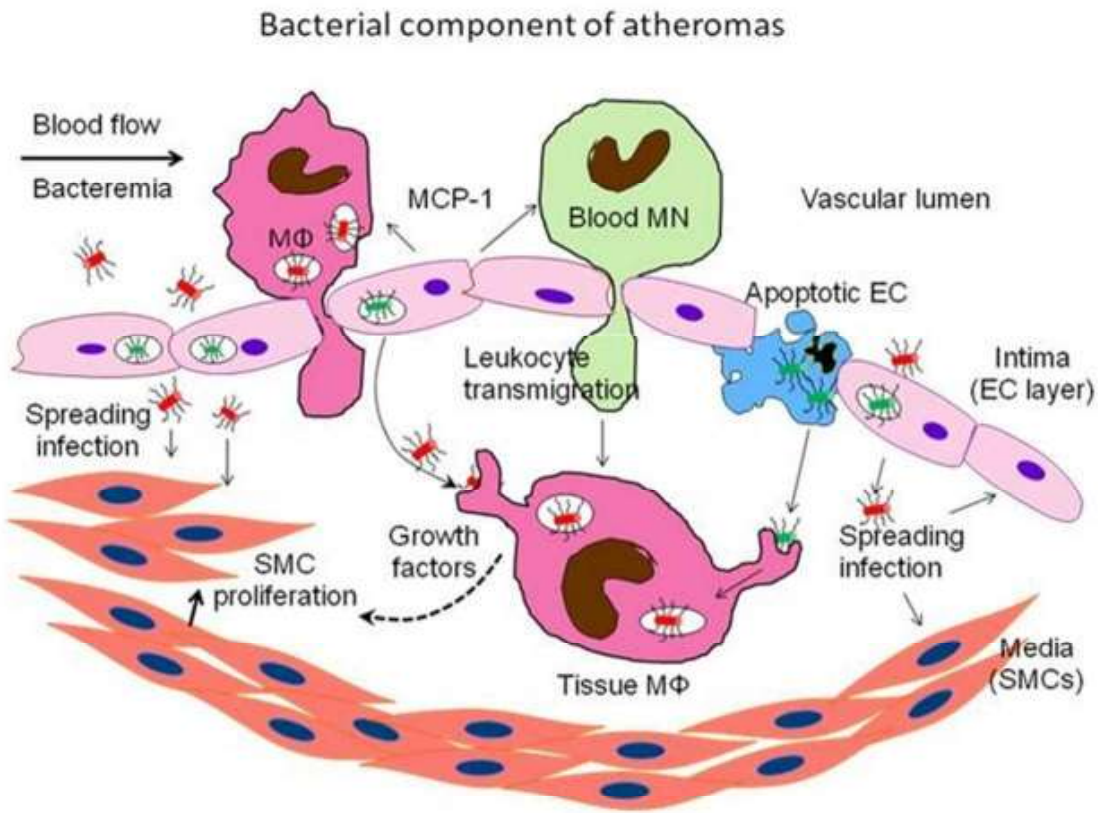
Conclusion: Within the limitations of this study, the presence of DNA from periodontitis-associated bacteria in carotid artery atheromatous plaque retrieved by endarterectomy is confirmed. *J Periodontol 2011;82:1469-1477.*

Simultaneous Detection of Bacterial DNA in the Same Atheromatous Plaque

Bacteria	Prevalence (% [number of plaque])
Aa and *Pg*	61.90 (26 of 42)
Pg and *Tf*	50.00 (21 of 42)
Aa and *Tf*	50.00 (21 of 42)
Pg, *Aa*, and *Tf*	47.62 (20 of 42)
Pg, *Aa*, *Tf*, and *Ec*	35.71 (15 of 42)
Pg, *Aa*, *Tf*, *Ec*, and *Fn*	28.57 (12 of 42)

Journal of Clinical Periodontology

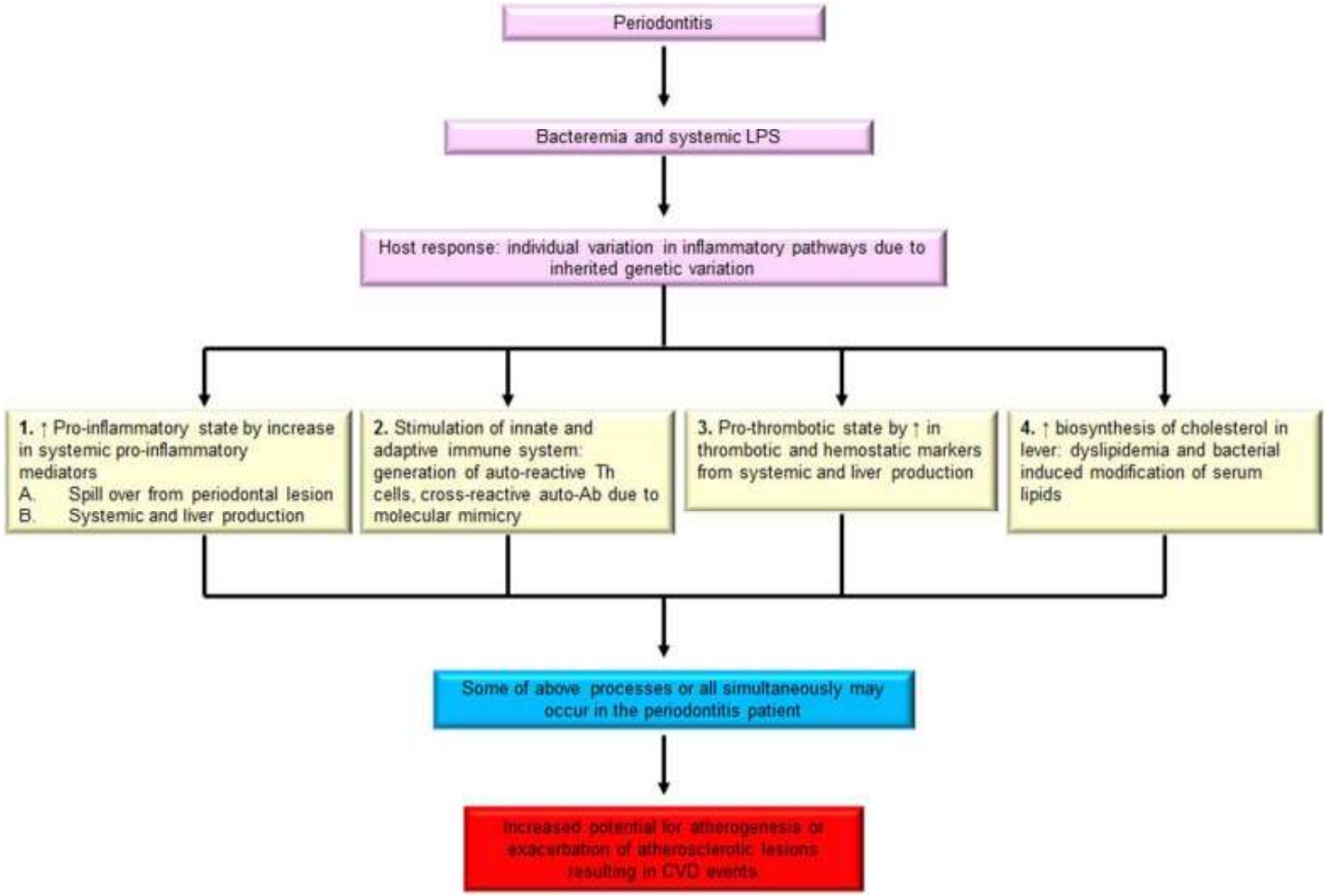
Leticia Reyes, Emil Kozarov, David Herrera, Silvia Roldán & Ann Progulske-Fox. Periodontal Bacterial Invasion and Infection: Contribution to Cardiovascular Disease. Journal of Clinical Periodontology, in press.



Direkte und indirekte bakterielle Gefäßwandinvasion

Journal of Clinical Periodontology

Harvey Schenkein & Bruno Loos.
Inflammatory Mechanisms Linking Periodontal Diseases to Cardiovascular Diseases.
Journal of Clinical Periodontology, in press.



Schematische Übersicht möglicher entzündlicher Mechanismen einer Verbindung parodontaler und kardiovaskulärer Erkrankungen

Clinical studies in periodontitis suggesting a link to inflammation in CVD:

- ***the role of biomarkers and increased systemic mediators of inflammation***

  (C-reactive protein, Fibrinogen, Interleukin 4,6, 18, Haptoglobin, Serum amyloid A,

  Alpha 1 anti-chymotrypsin, Matrix-Metalloproteinase 12, (MMP)-9,

  Platelet activating factor (PAF) and PAF- acetylhydrolase (AH)

- ***the role of thrombotic and hemostatic mediators and markers***

  (Fibrinogen, Plasminogen-activator inhibitor (PAI)-1,von Willebrand Faktor,

  P-selectin, P- selectin, CD18, activated glycoprotein IIb/IIIa (platelet activation markers)

- ***antibodies***

  (Anti-Heat Shock Proteins (HSPs), Anti-cardiolipin, Anti- phosphorylcholine (PC)

  Anti-oxLDL,

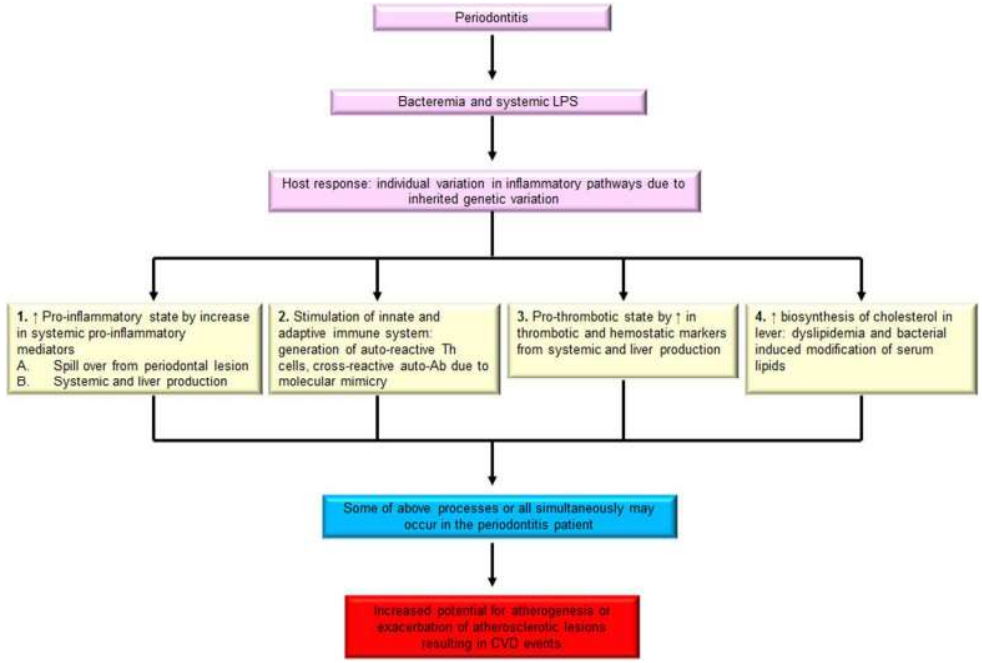
- ***the role of serum lipids***

  (Total serum cholesterol, Serum Lipid Profil, Low-density lipoprotein (LDL), High-density lipoprotein

  (HDL), Triglyzeride, Very (v)LDL, Intermediate density lipoprotein (IDL)

Journal of Clinical Periodontology

Harvey Schenkein & Bruno Loos.
Inflammatory Mechanisms Linking Periodontal Diseases to Cardiovascular Diseases.
Journal of Clinical Periodontology, in press.



However, proof that the increase in systemic inflammation attributable to periodontitis impacts inflammatory responses during atheroma development, thrombotic events, or myocardial infarction or stroke is lacking.

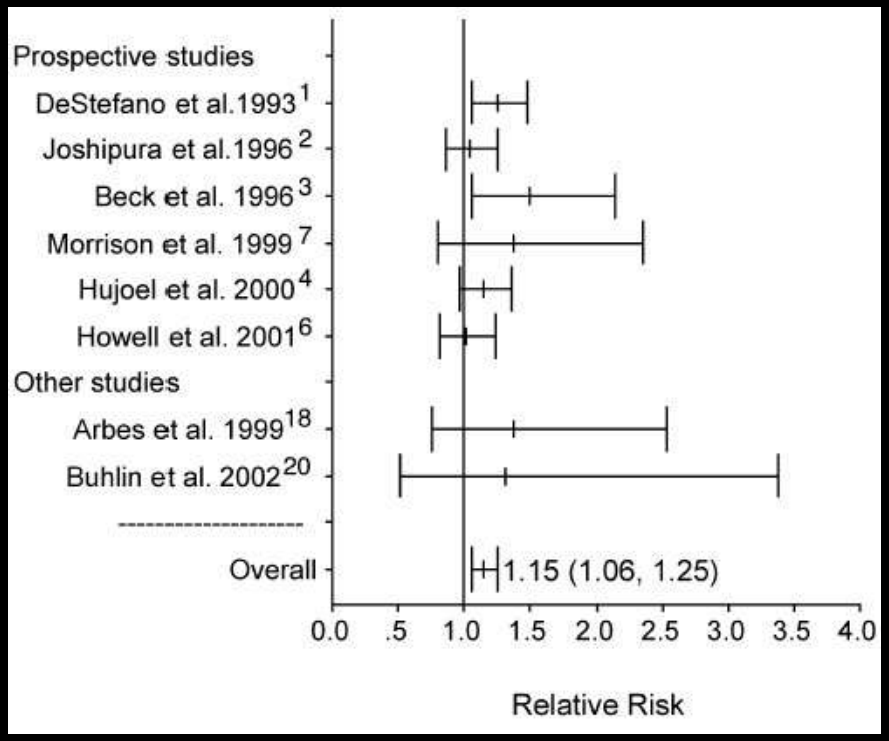
Kritische Fragen:

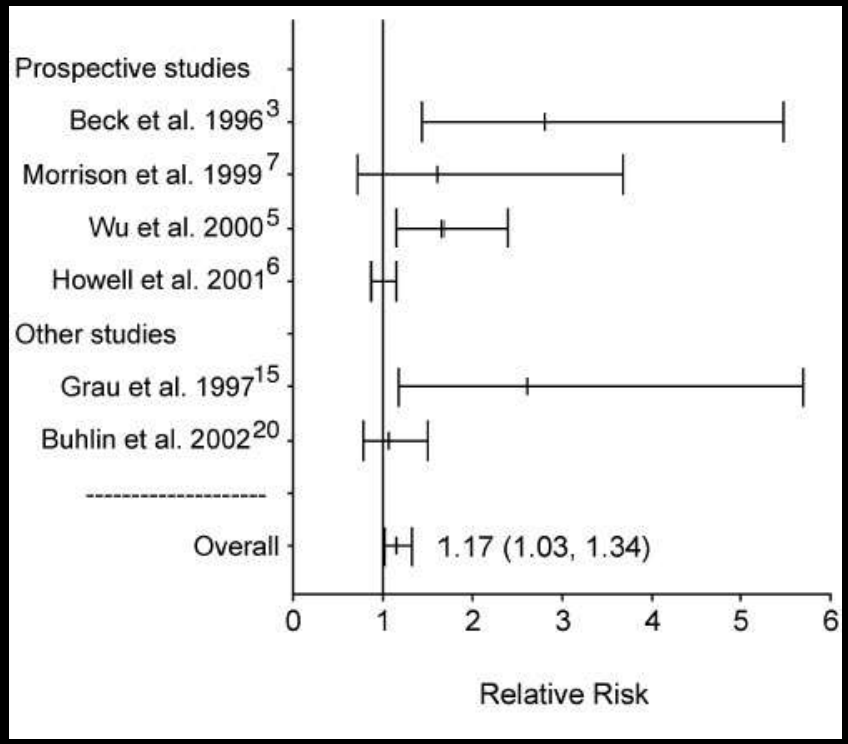
- ✓ Welche klinischen Parodontitis- Charakteristika sind mit CVD assoziert?
- ✓ Wie stark sind die Belastungswerte für ein zusätzliches Risiko?
- ✓ Gibt es Confounder von Bedeutung für eine Assoziation?
- ✓ Klinische und „public health“ Implikationen
- ✓ Wissenslücken und zukünftige Forschungsfragen/Studiendesigns

Consensus Report of the Joint European Federation of Periodontology and American Academy of Periodontology Workshop on Periodontitis and Systemic Diseases, J Clin Perio 2013.

Parodontitis & kardiovaskuläre Erkrankungen

Wie konsistent und stark sind die Assoziationen?



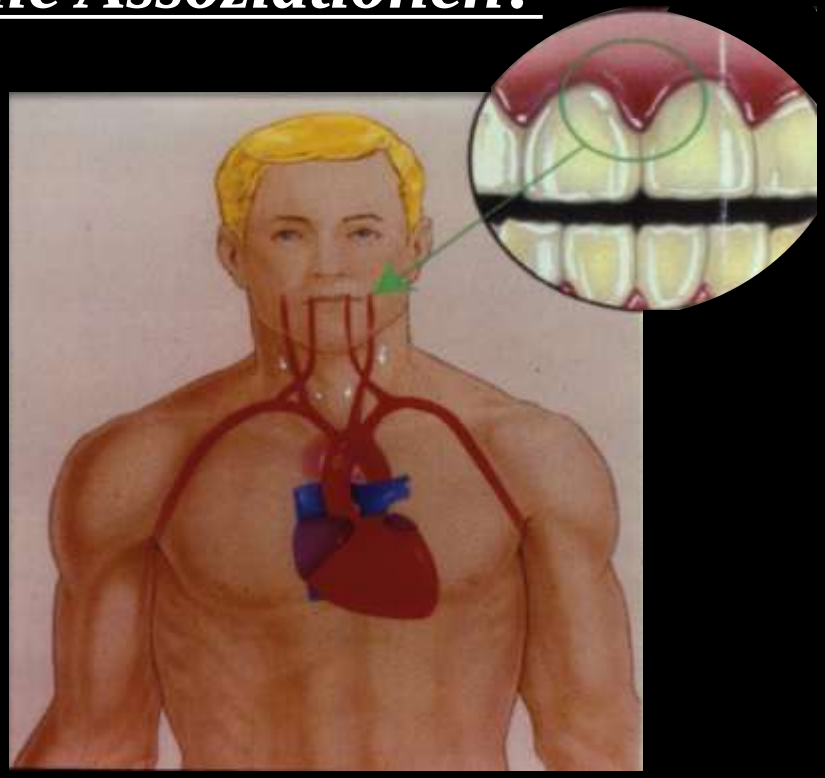


Risk Ratios für CHD und CVD in Assoziation mit Parodontitis

Khader Y. et al.
Periodontal Diseases and the Risk of Coronary Heart and Cerebrovascular Diseases: A Meta-Analysis *J Periodontol 2004;75:1046-1053*.

Parodontitis & kardiovaskuläre Erkrankungen

Wie konsistent und stark sind die Assoziationen?



Die Analysen zeigen, dass parodontale Infektionen das Risiko an CHD zu erkranken um das Doppelte für CVD um das Vierfache erhöhen !

Risk Ratios für CHD und CVD in Assoziation mit Parodontitis

Khader Y. et al.
Periodontal Diseases and the Risk of Coronary Heart and Cerebrovascular Diseases: A Meta-Analysis *J Periodontol 2004;75:1046-1053*.

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Journal of General Internal Medicine
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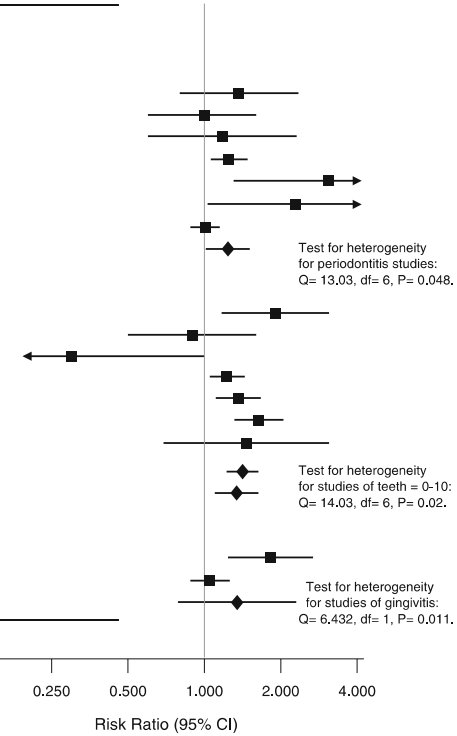
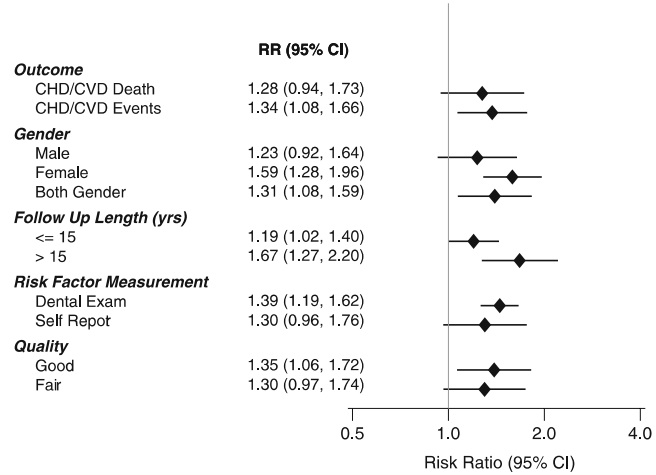
Periodontal disease is a risk factor or marker for CHD that is independent of traditional CHD risk factors, including socioeconomic status. Summary relative risk estimates for different categories of periodontal disease (including periodontitis, tooth loss, gingivitis, and bone loss) ranged from 1.24 (95% CI 1.01–1.51) to 1.34 (95% CI 1.10–1.63).

	Outcome	Number of Subjects*	Risk Factor Measurement	Quality	Gender	Followup (in yrs)	RR (95% CI)
Risk Factor = Periodontitis							
Morrison, 1999	CHD Death	2297	Dental exam	Fair	B**	21	1.37 (0.80, 2.35)
Tuominen, 2003 (M)	CHD Death	2518	Dental exam	Fair	M**	12	1.00 (0.61, 1.63)
Tuominen, 2003 (F)	CHD Death	2392	Dental exam	Fair	F**	12	1.18 (0.60, 2.31)
DeStefano, 1993	CHD Events	5041	Dental Exam	Good	B	14	1.25 (1.06, 1.48)
Beck, 1996	CHD Events	1221	Dental Exam	Fair	M	18	3.10 (1.30, 7.40)
Ajwani, 2003	CVD Death	175	Dental Exam	Fair	B	5	2.28 (1.03, 5.05)
Howell, 2001	CVD Events	22037	Self report	Fair	M	12	1.01 (0.88, 1.15)
Periodontitis Studies Combined							1.24 (1.01, 1.51)
Risk Factor = 0-10 teeth							
Morrison, 1999	CHD Death	3330	Dental exam	Fair	B	21	1.90 (1.17, 3.10)
Tuominen, 2003 (M)	CHD Death	1510	Dental exam	Fair	M	12	0.90 (0.50, 1.60)
Tuominen, 2003 (F)	CHD Death	1479	Dental exam	Fair	F	12	0.30 (0.10, 1.00)
DeStefano, 1993	CHD Events	5398	Dental Exam	Good	B	14	1.23 (1.05, 1.44)
Hung, 2004 (M)	CHD Events	41407	Self report	Good	M	12	1.36 (1.11, 1.67)
Hung, 2004 (F)	CHD Events	58974	Self report	Good	F	20	1.64 (1.31, 2.05)
Ajwani, 2003	CVD Death	175	Dental Exam	Fair	B	5	1.46 (0.69, 3.09)
Combined excluding Tuominen							1.41 (1.22, 1.63)
Studies of 0-10 Teeth Combined							1.34 (1.10, 1.63)
Risk Factor = Gingivitis							
Morrison, 1999	CHD Death	3921	Dental exam	Fair	B	21	1.82 (1.24, 2.68)
DeStefano, 1993	CHD Events	5537	Dental Exam	Good	B	14	1.05 (0.88, 1.26)
Gingivitis Studies Combined							1.35 (0.79, 2.30)

* Number of subjects included in the analysis
** Abbrevations: B, Both gender; M, Male; F, Female.





Humphrey L. et al. **Periodontal Disease and Coronary Heart Disease Incidence: A Systematic Review and Meta-analysis.**
J Gen Intern Med. 2008 December; 23(12): 2079–2086.

Journal of Clinical Periodontology

Epidemiologische Evidenz

Thomas Dietrich, Praveen Sharma, Clemens Walter, Paul Weston, James Beck

The epidemiological evidence behind the association between periodontal disease and incident cardiovascular disease? Journal of Clinical Periodontology, in press.

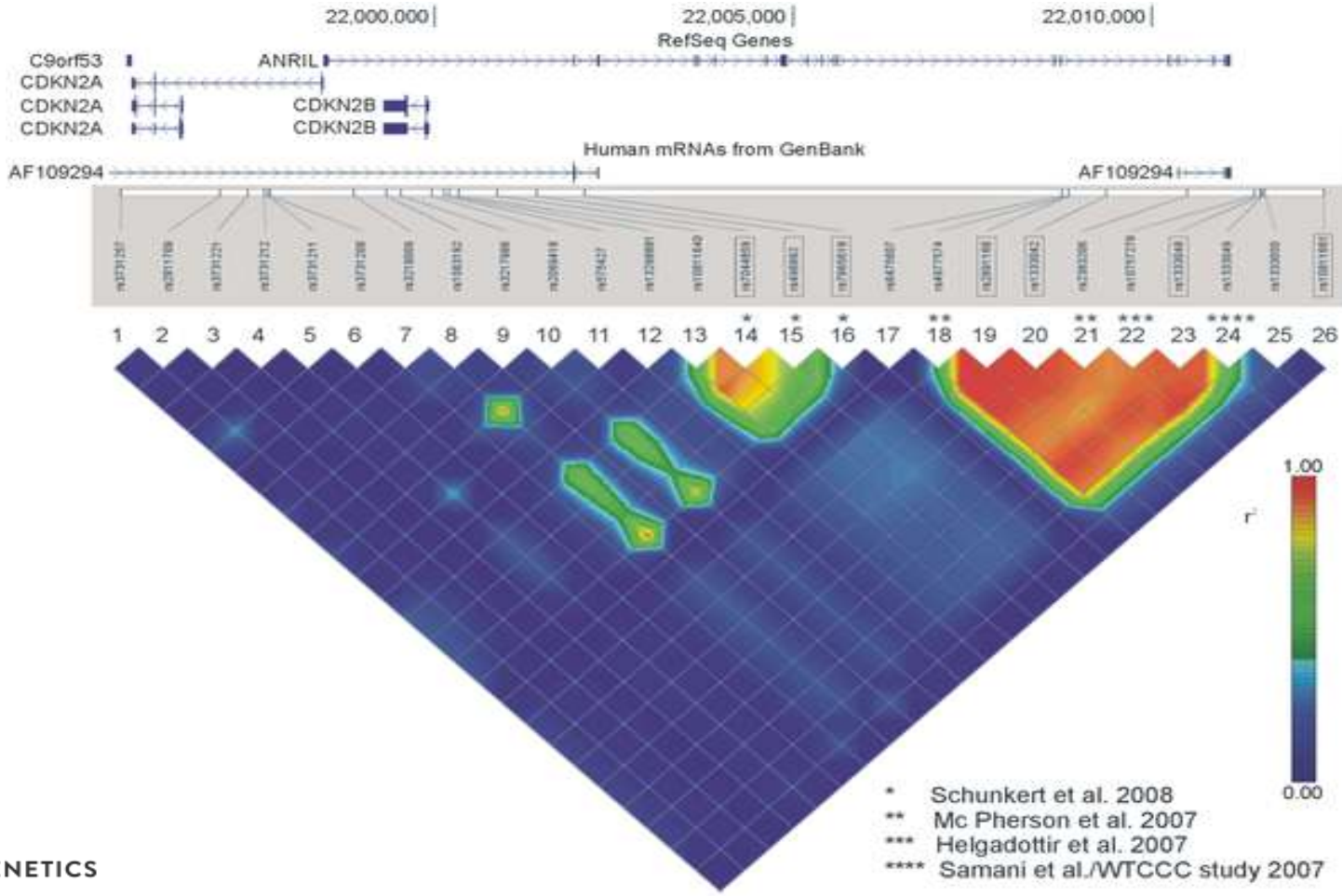
- Statistically significant excess risk for CVD in individuals with periodontitis was reported to be independent of established cardiovascular risk factors. However varies by type of cardiovascular outcome and across populations by age and gender.
  Specifically, the risk is greater with cerebrovascular disease, and greater males and younger individuals.
- There is insufficient evidence to indicate whether or not periodontitis is associated with the incidence of secondary (a second CVD event after the original event) cardiovascular events.
- Even low to moderate excess risk reported in studies is enough to be important from a public health perspective because of the high prevalence of periodontitis.

✓ Gibt es Confounder von Bedeutung für eine Assoziation?

- There are many potentially important confounders of the association between periodontitis and ACVD risk, including co-morbidities such as diabetes and lifestyle factors such as smoking.
- However, established cardiovascular risk factors do not completely explain the excess cardiovascular risk in subjects with periodontitis.
- However, excess risk could be due to unknown confounders. Recent findings from the ENCODE project, the deep sequencing project (1000 Genomes Project) indicate that there are common genetically determined pathways underpinning various complex inflammatory diseases.

Schaefer AS, et al. (2009) Identification of a Shared Genetic Susceptibility Locus for Coronary Heart Disease and Periodontitis. PLoS Genet 5(2): e1000378. doi:10.1371/journal.pgen.1000378

Figure 1. LD Structure (r2) of the Chromosome 9p21.3 Locus in HapMap CEU.



PLOS | GENETICS

Several genome studies have identified a region of the human genome near the *CDKN2A* and *CDKN2B* genes as having an influence on CHD. We show that this genetic region, being the most important susceptibility locus for CHD to date, is also associated with a substantial risk increase of aggressive periodontitis.

✓ Klinische und „public health" Implikationen

- Periodontal treatment requires individual professional intervention. Therefore, ***primary prevention*** becomes more important and novel strategies to prevent (and treat?) disease at the population level would be highly desirable.
- A ***diagnosis of periodontitis*** may contribute to cardiovascular risk stratification, if shown to improve cardiovascular risk prediction over and above currently established prediction models (e.g. Framingham score).

Periodontal treatment improves endothelial dysfunction in patients with severe periodontitis

Gerald Seinost, MD,[a] Gernot Wimmer, MD,[b] Martina Skerget, MD,[a]
Erik Thaller,[b] Marianne Brodmann, MD,[a] Robert Gasser, MD,[c]
Rudolf O. Bratschko, MD,[b] Ernst Pilger, MD[a]

Am Heart J, 2005;149:1050-1054.

[a] Division of Angiology, Department of Medicine, University Hospital, Graz, Austria
[b] Department of Prosthetics and Periodontology, School of Dentistry, Graz, Austria
[c] Division of Cardiology, Department of Medicine, University Hospital, Graz, Austria

Endotheliale Dysfunktion

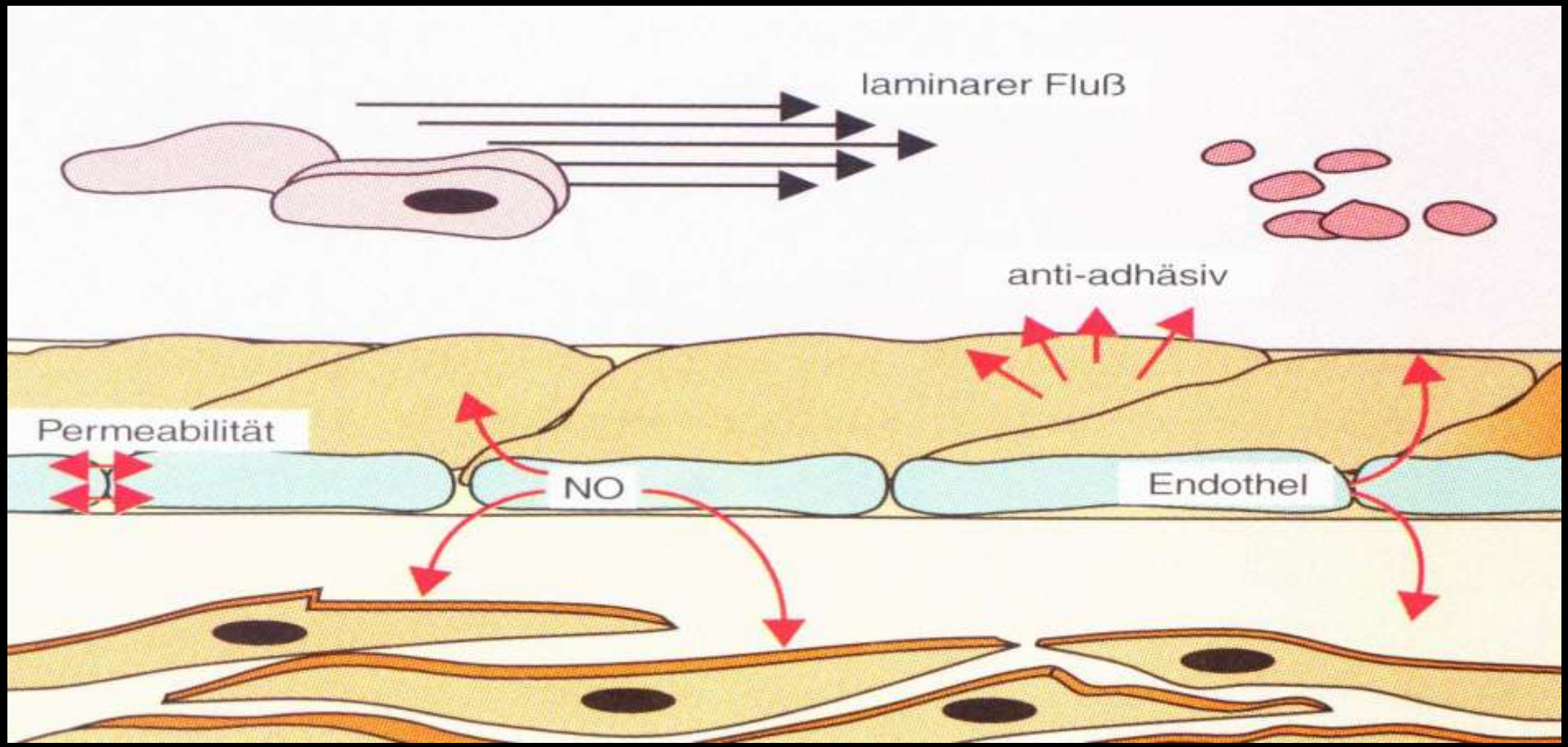
A perspective on the potential cardioprotective benefits of periodontal therapy

Steven Offenbacher, DDS, PhD, MMSc, and James D. Beck, PhD ***Chapel Hill, NC***

Am Heart J 2005;149:950-4.(Editorial)

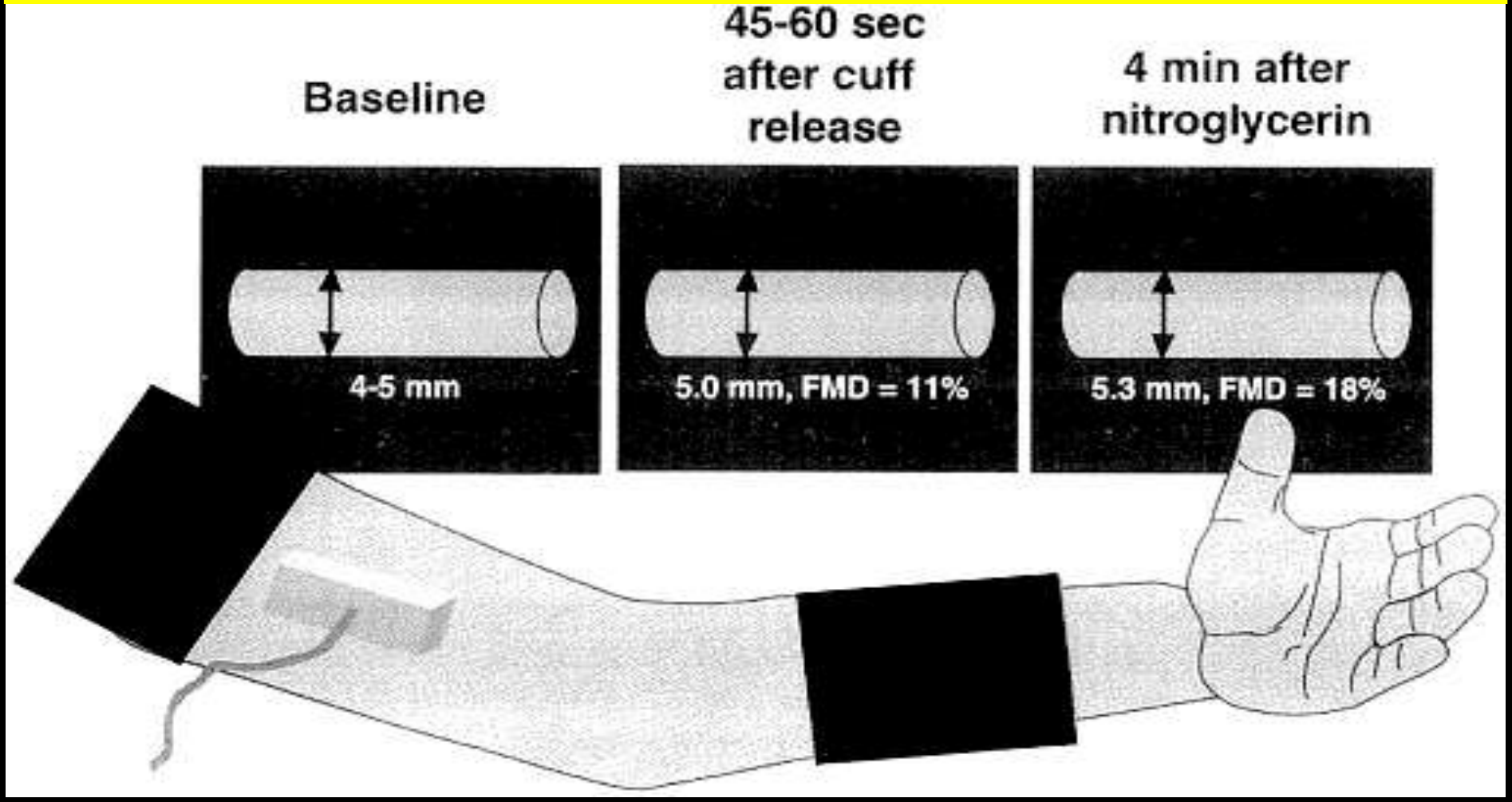
. . . . "The report of Seinost et al. is significant because it provides the first evidence that treating periodontal disease results in a functional improvement in cardiovascular status."

Endotheliale Dysfunktion



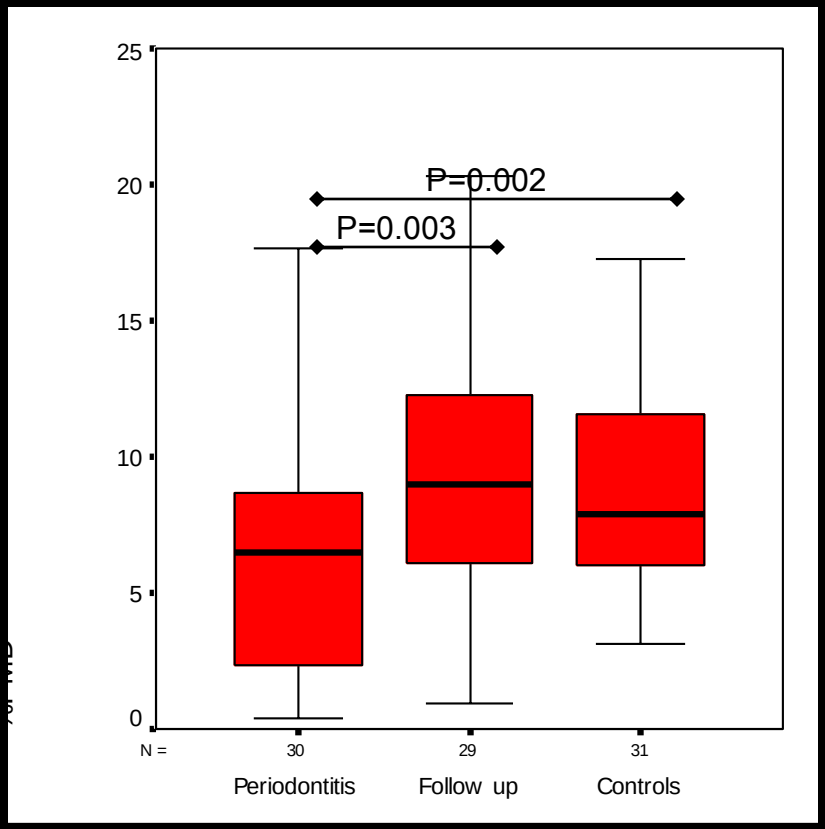
Gestörte endotheliale Verfügbarkeit von biologisch aktivem Stickstoffmonoxyd (NO) führt zu einer verminderten endothel-abhängigen Vasodilatation.

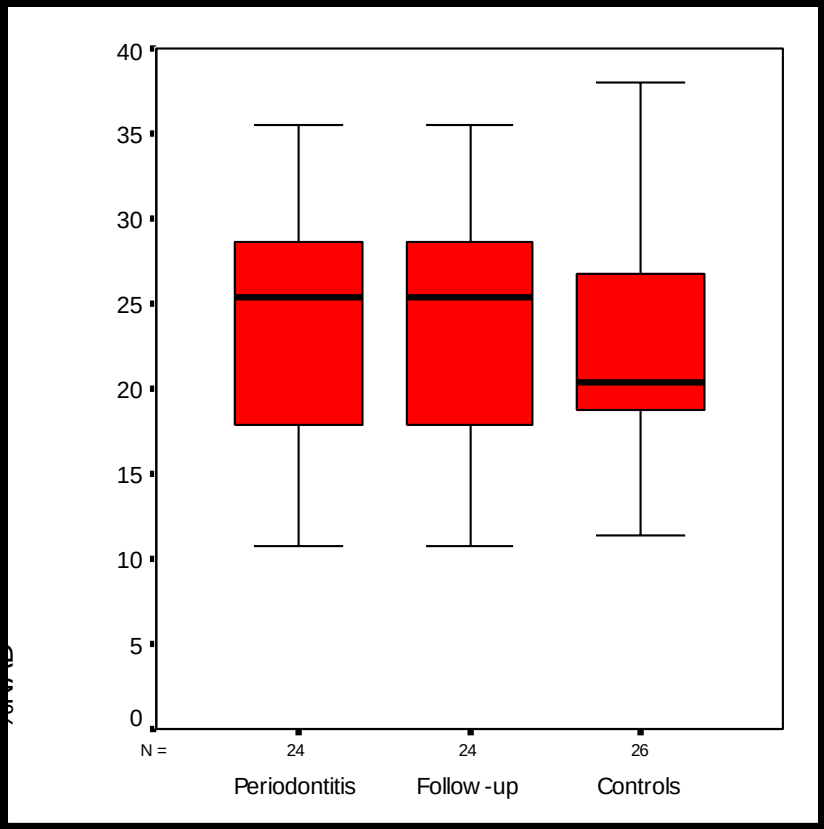
Endotheliale Dysfunktion



Messung der Endothelialen Dysfunktion im Leitungsgefäß

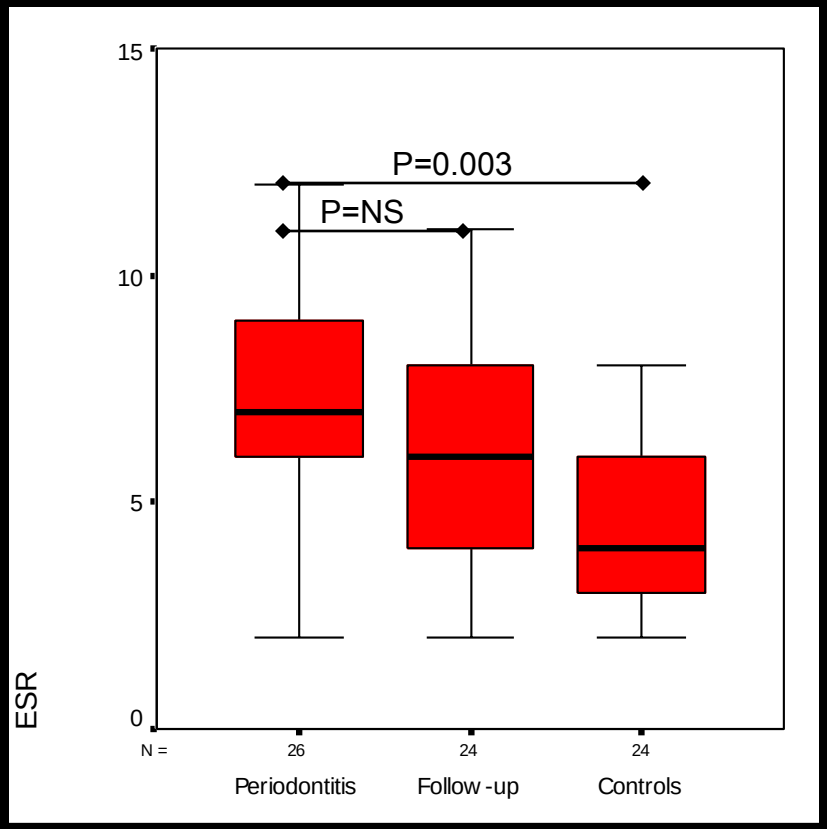
Endotheliale Dysfunktion

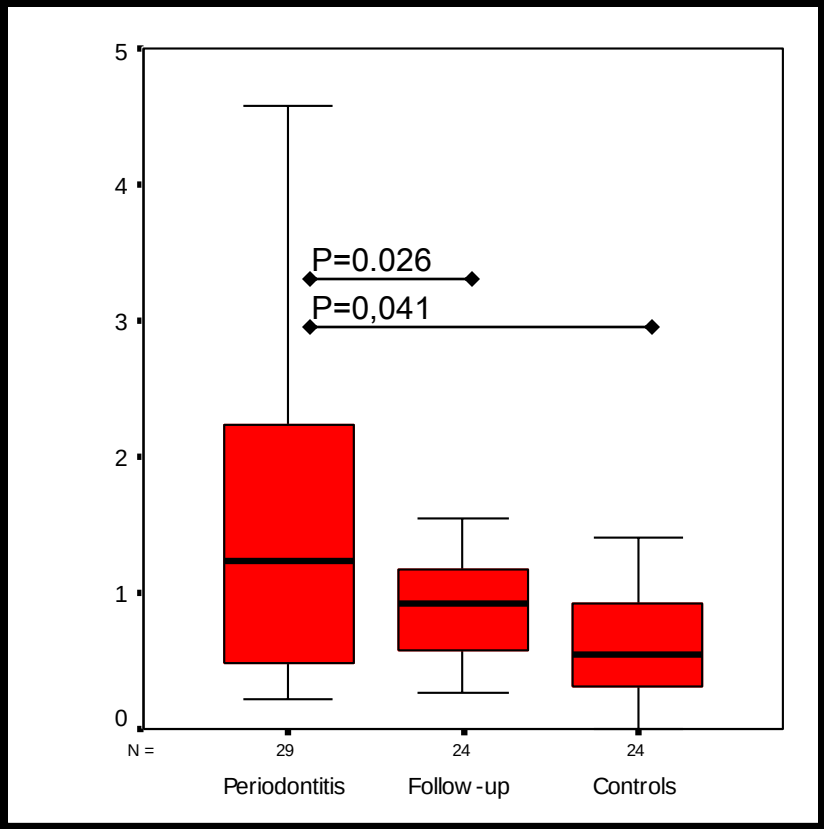




Relative flow-mediated (%FMD) and nitroglycerin-associated (%NAD) dilation of the brachial artery of patients with severe periodontitis before and after periodontal treatment in comparison with matched control subjects.

Endotheliale Dysfunktion

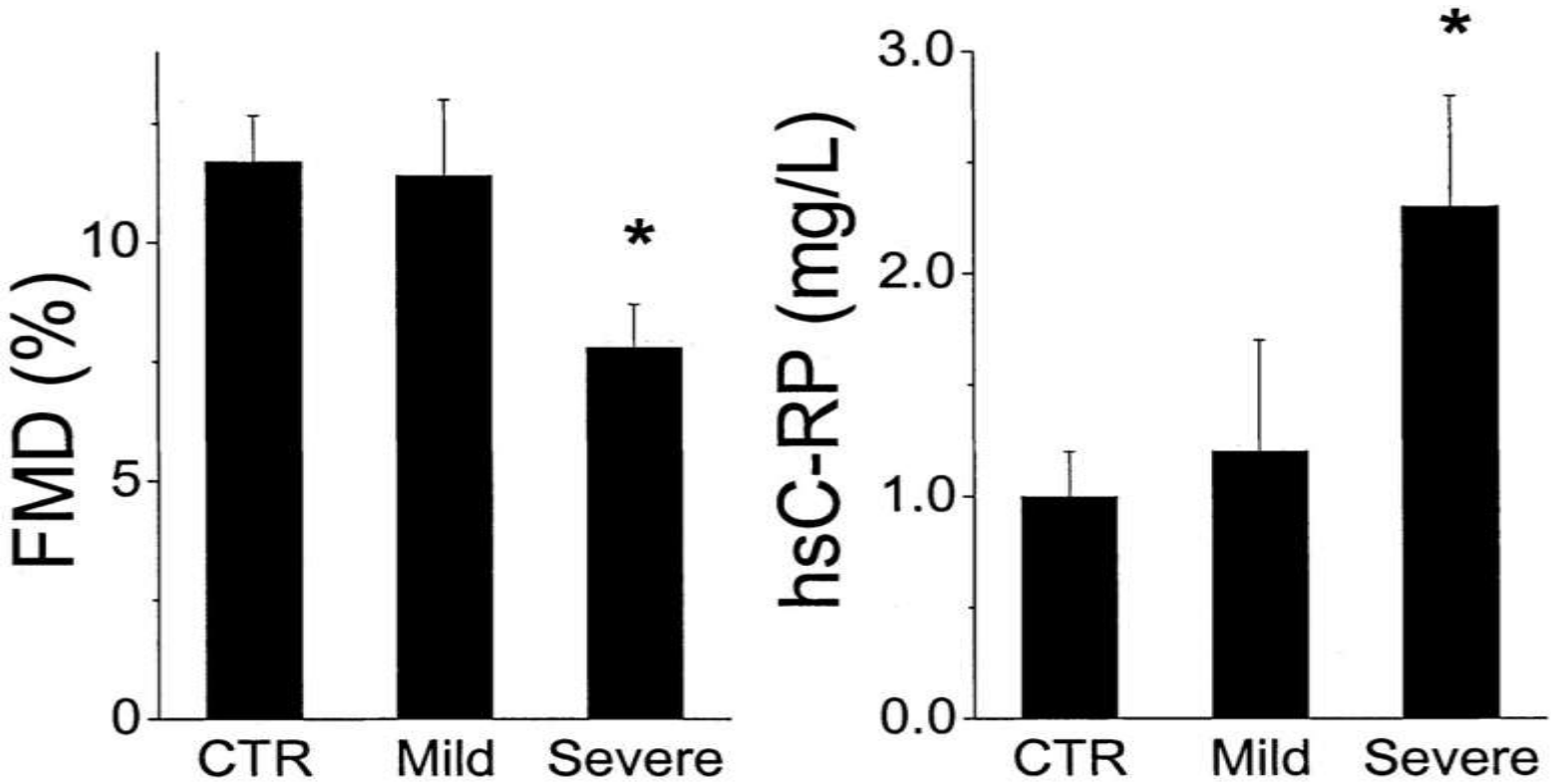




Markers of systemic inflammation of patients with severe periodontitis before and after periodontal treatment and of control subjects.

ESR = *erythrocyte sedimentation rate*, hs-CRP = *high sensitive C-reactive protein*

Brachial artery flow-mediated dilation (left) and C-reactive protein (right) were assessed as described in Methods



Amar, S. et al. Arterioscler Thromb Vasc Biol 2003;23:1245-1249

Gerald Seinost, Gernot Wimmer et al.

Periodontal treatment improves endothelial dysfunction in patients with severe periodontitis.

Am Heart J 2005;149:1050-4.

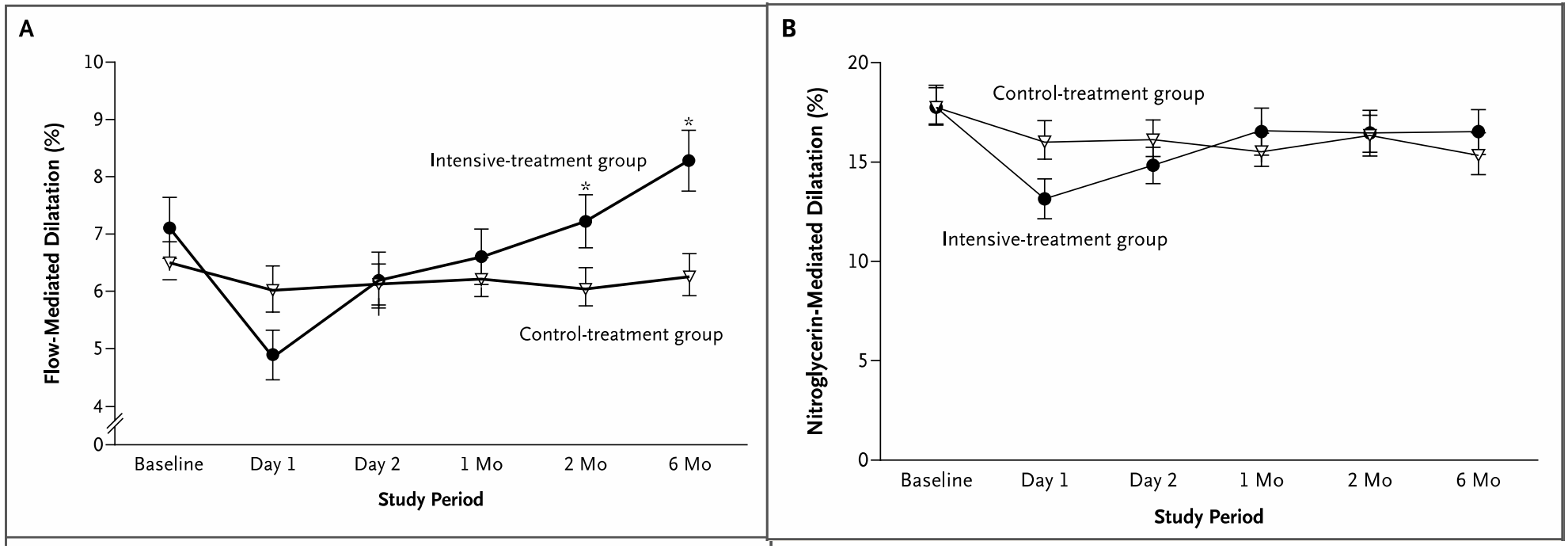
Konklusion

- Patienten mit fortgeschritten parodontalen Erkrankungen zeigen eine endotheliale Dysfunktion und Zeichen systemischer Entzündung. Das könnte Patienten zu einem erhöhtes Risiko für eine weitere Entwicklung kardiovaskulärer Erkrankungen prädisponieren.
- Die therapeutische Kontrolle parodontaler Erkrankungen verbessert die endotheliale Dysfunktion und Anzeichen systemischer Entzündung.

The NEW ENGLAND JOURNAL of MEDICINE

Maurizio S. Tonetti, Francesco D'Aiuto et al.

Treatment of Periodontitis and Endothelial Function.N Engl J Med 2007; 356:911-920.

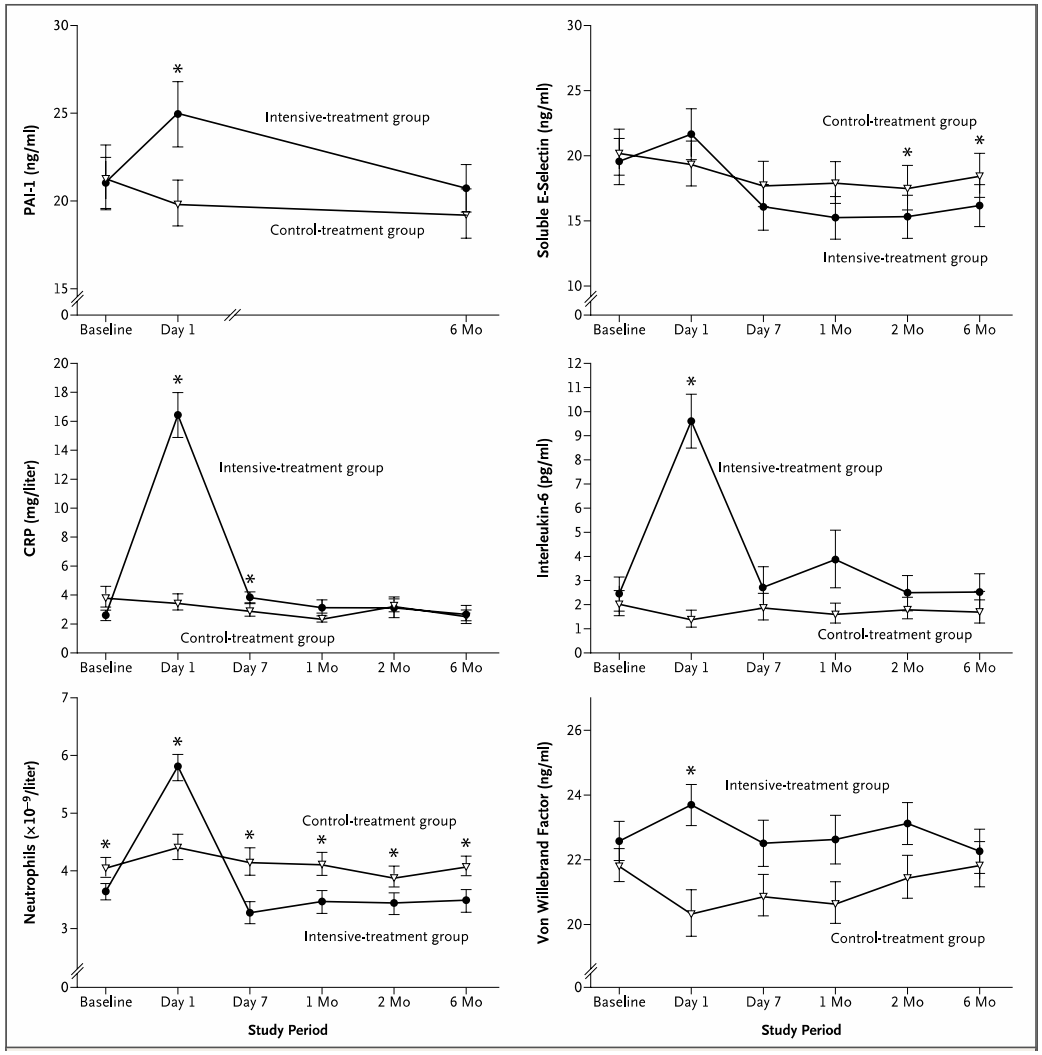


Flow-Mediated Dilatation and Nitroglycerin-Mediated Dilatation during the 6-Month Study Period.

The NEW ENGLAND JOURNAL of MEDICINE

Maurizio S. Tonetti, Francesco D'Aiuto et al.

Treatment of Periodontitis and Endothelial Function.N Engl J Med 2007; 356:911-920.



Circulating Biomarkers during the 6-Mo Study Period.

CONCLUSIONS

Intensive periodontal treatment resulted in an acute, endothelial dysfunction and short-term systemic inflammation. However, 6 months after therapy, the benefits in oral health were associated with improvement in endothelial function as well as systemic inflammation.

Journal of Clinical Periodontology

Interventions-Studien

Francesco D'Aiuto, Marco Orlandi, John C. Gunsolley.

Evidence that periodontal treatment improves biomarkers and CVD outcomes.

Journal of Clinical Periodontology, in press. c

Konklusion:

There is moderate evidence that periodontal treatment:

i) reduces systemic inflammation as evidenced by reduction in CRP and improvement of both clinical and surrogate measures of endothelial function; but

ii) has a negligible effect on lipid profiles – supporting specificity.

Limited evidence shows improvements in coagulation, biomarkers of endothelial cell activation, arterial blood pressure and subclinical atherosclerosis after periodontal therapy.

The available evidence is consistent and speaks for a contributory role of periodontitis to CVD.

Periodontitis and Atherosclerotic Cardiovascular Disease

Konklusion:

1. Es bestehen konsistente und starke epidemiologische Evidenz, dass Parodontitis ein erhöhtes Risiko für zukünftige kardiovaskuläre Erkrankungen verleiht.
2. Die Beeinflussung der Parodontitis auf CVD ist biologisch plausibel; disseminierte und zirkulierende orale Mikroorganismen können direkt oder indirekt eine systemische Entzündung herbeiführen, die die Pathogenese der Atherothrombogenese unterstützt.
3. Während in vitro, Tier- und klinische Studien die Interaktion als auch biologische Mechanismen unterstützen, zeigen Interventionsstudien bis jetzt noch nicht ausreichend weitere Schlüsse zu ziehen.

Maurizio Tonetti & Tom Van Dyke.
Consensus Report of the Joint European Federation of Periodontology and American Academy of Periodontology Workshop on Periodontitis and Systemic Diseases, J Clin Perio in press.

Parodontitis &

Diabetes mellitus

Schädigungen der Gingiva und des Zahnhalteapparates sind bei Diabetikern ausgeprägter als bei Nicht-Diabetiker.

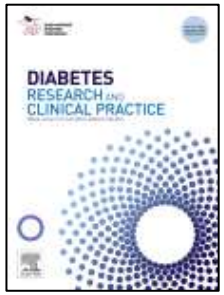
- ✓ Diabetes ist ein eigenständiger Parodontitis-Risikofaktor.
- ✓ Die Parodontitis gilt als 6. diabetische Komplikation.

Parodontitis &

Diabetes mellitus

Parodontalerkrankungen und Diabetes mellitus:

→ Eine Wechselbeziehung



Monik Jimenez, Frank B. Hu, Miguel Marino, Yi Li, Kaumudi J. Joshipura.

Type 2 diabetes mellitus and 20 year incidence of periodontitis and tooth loss.

Diabetes Research and Clinical Practice) 98 (3) Dezember 2012: 494.

Aims

Evaluate the prospective associations between type 2 diabetes mellitus (T2DM) and the risk of periodontitis and tooth loss.

Methods

35,247 male participants who were dentate, free of periodontitis at baseline, were followed from 1986 to 2006. Data on self-reported diabetes, periodontitis, tooth loss and potential confounders were collected at baseline and biennially.

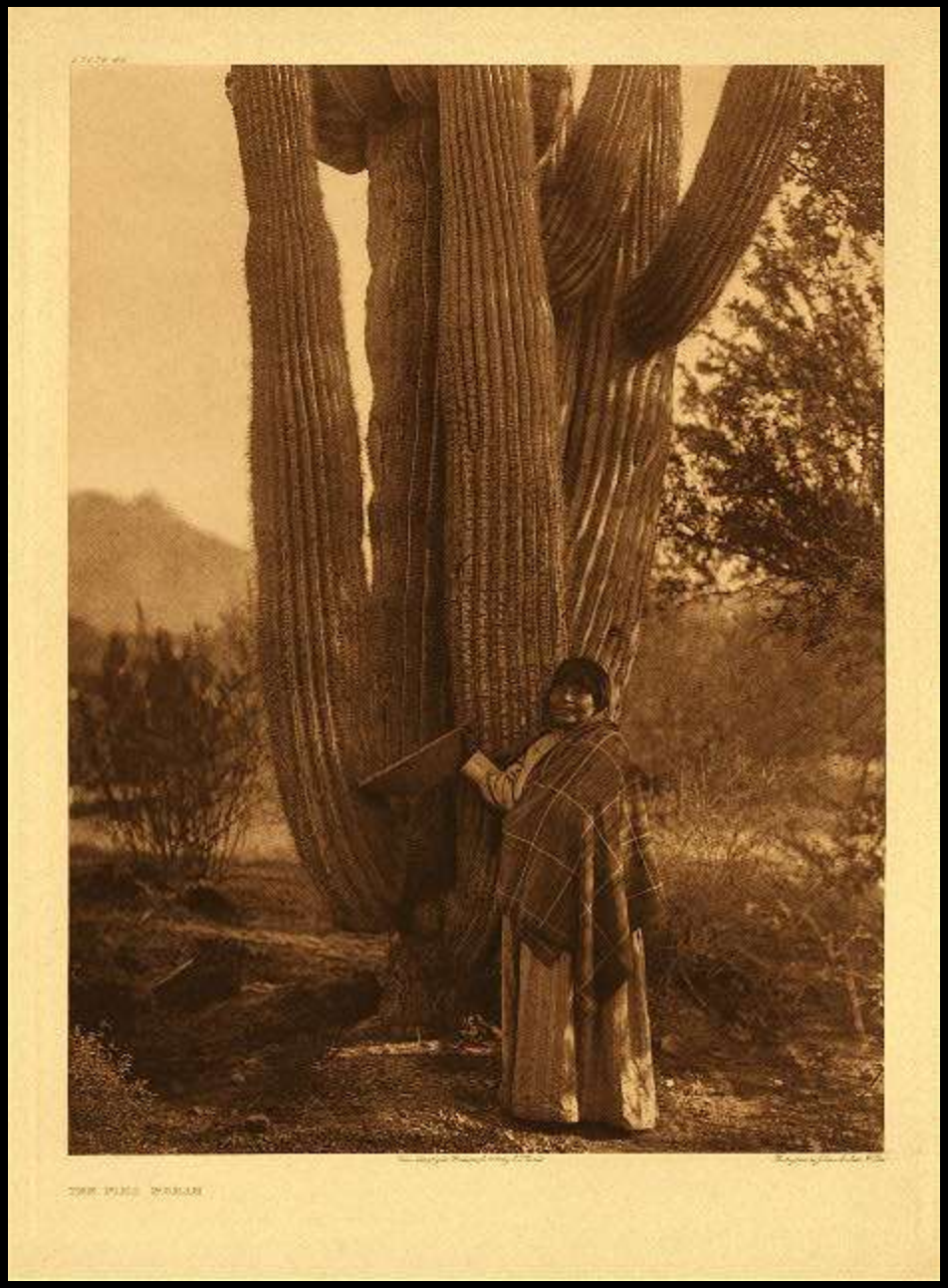
Results

Men with T2DM showed a 29% (HR = 1.29; 95% CI: 1.13–1.47) increased risk of periodontitis compared to those without, (adjusted for age, race, smoking, BMI, fruit/vegetable intake, physical activity, alcohol consumption and dental profession)

Conclusions

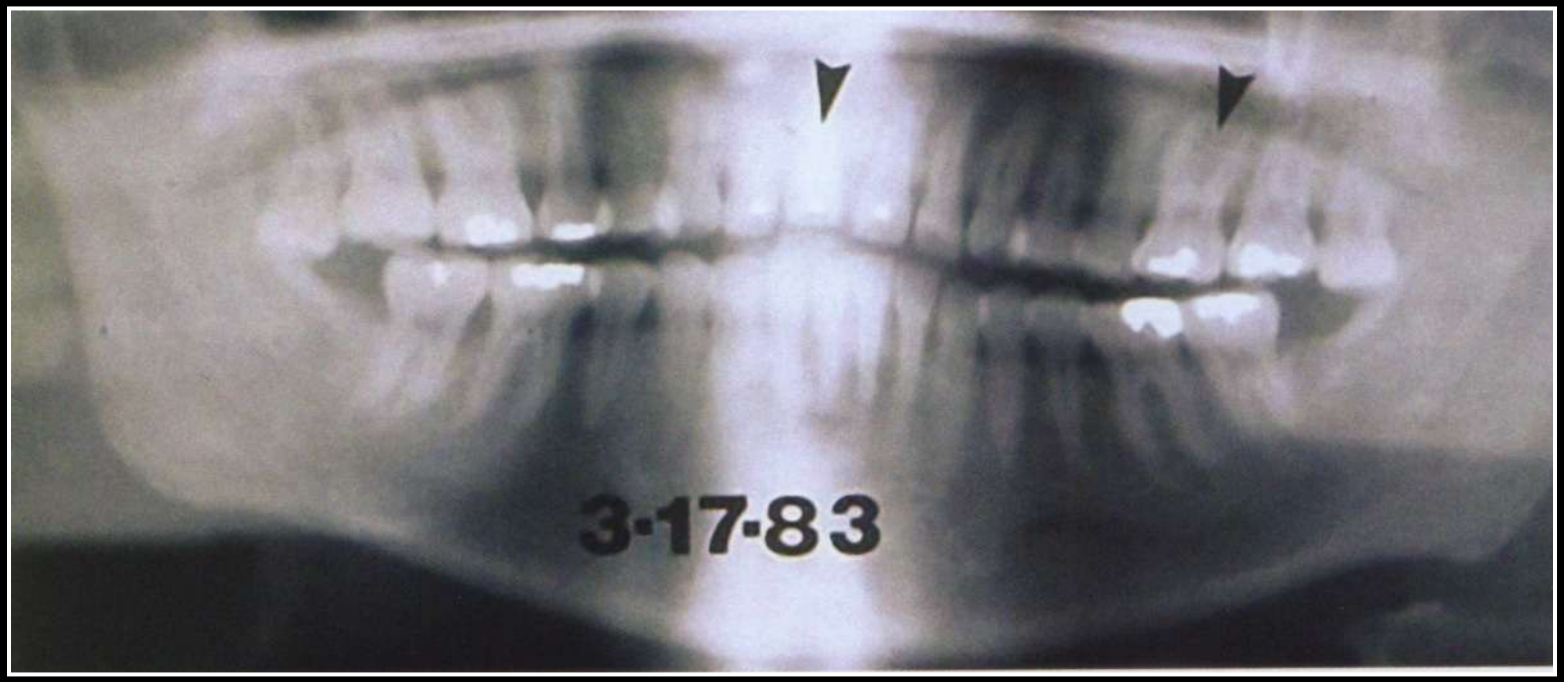
Type 2 diabetes mellitus was associated with a significantly greater risk of self-reported periodontitis.

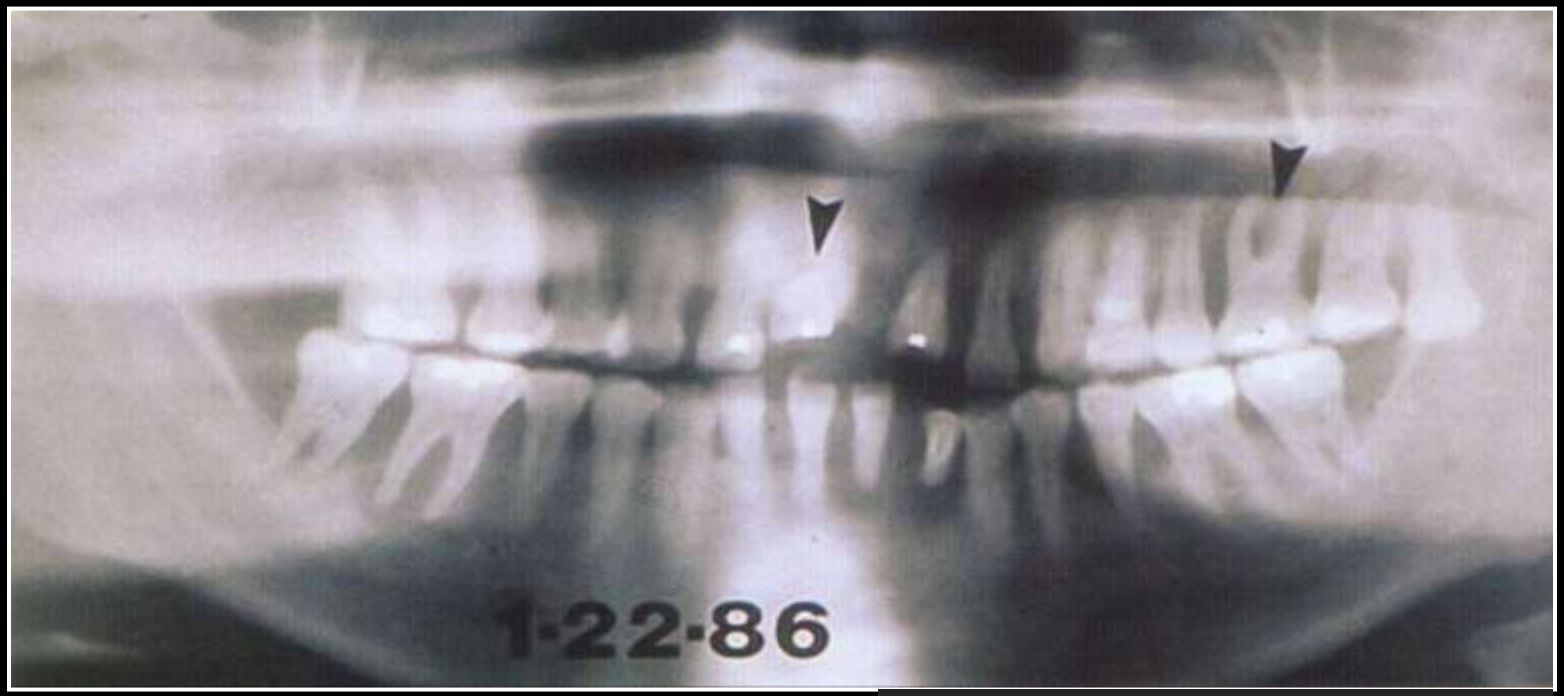
Pima Indian



Almost half of adults over 35 developed type 2 diabetes –
and also contracted severe periodontal disease at twice the normal incidence.

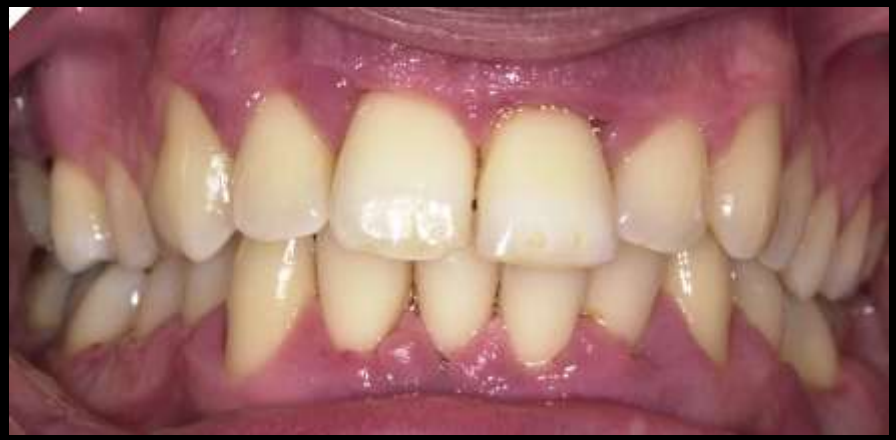
NORTHWESTERN UNIVERSITY LIBRARY, *EDWARD S. CURTIS'S* "THE NORTH AMERICAN INDIAN" 2003

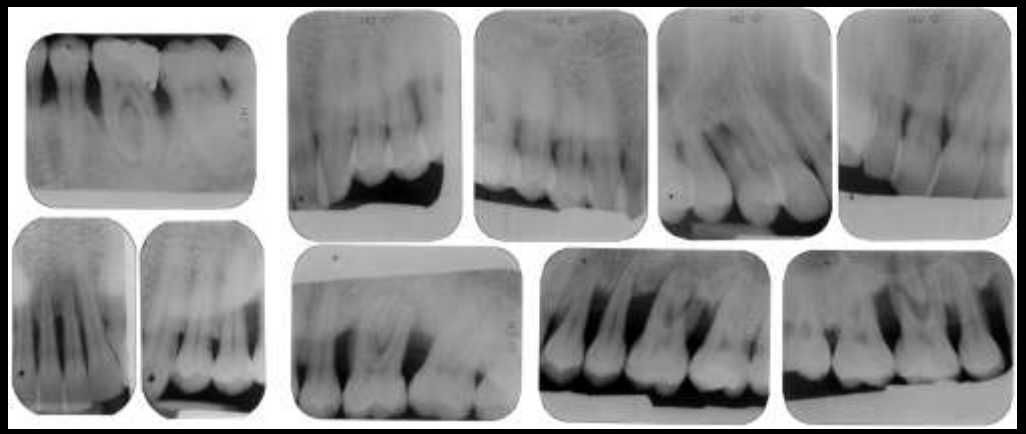




Courtesy by Robert J. Genco

Parodontalerkrankungen bei Diabetikern:

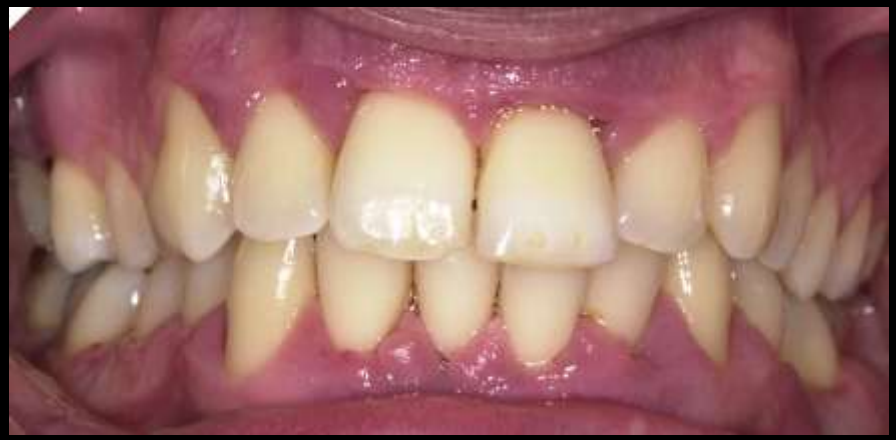


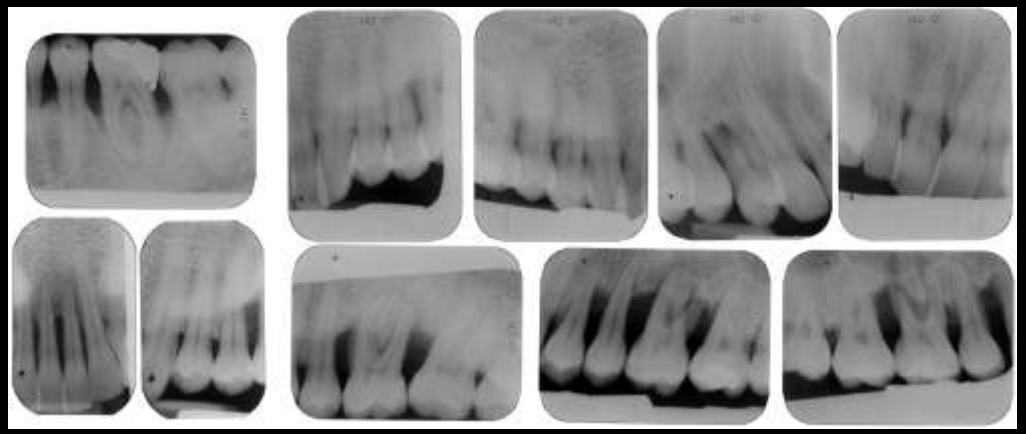


Typ-I-Diabetiker haben schwerere Parodontitisverläufe als Nicht-Diabetiker, besonders bei schlechter Einstellung.

Bei Typ-II-Diabetiker treten Parodontalerkrankungen zwar häufiger auf, der Verlauf aber scheint ähnlich.

Parodontalerkrankungen als diabetische Komplikation?





Parodontale Komplikationen scheinen beim Diabetes genauso früh zu entstehen, wie Retinale. Viele diabetische Komplikationen scheinen auf einen gesteigerten Spiegel an Entzündungsfaktoren zurückzuführen zu sein und sind im Verlauf parodontalen Erkrankungen

Parodontalerkrankungen und Diabetes: Eine Wechselbeziehung

PARODONTALERKRANKUNG

Parodontales Pathogen

↓

Endotoxin, toxische Zellwandprodukte

Bakteriämien dentalen Ursprungs stehen in direkter Beziehung zur Schwere der gingivalen Entzündung

Infektions-vermitteltes Pathogenese-Modell der Parodontitis

Parodontalerkrankungen und Diabetes: Eine Wechselbeziehung

PARODONTALERKRANKUNG

Parodontales Pathogen

↓

Endotoxin, toxische Zellwandprodukte

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Proinflammatorische Kaskade

↓

Sekretion von TNF-α + IL-1ß

↓

Bindegewebezerstörung Knochenresorption

Infektions-vermitteltes Pathogenese-Modell der Parodontitis

Bakteriämien dentalen Ursprungs stehen in direkter Beziehung zur Schwere der gingivalen Entzündung

Die bakteriellen Stoffwechsel-Produkte lösen eine Kaskade von Entzündungsmediatoren aus

Parodontalerkrankungen und Diabetes: Eine Wechselbeziehung

Die bei Hyperglykämie vermehrt entstehenden AGEs sind wohl für die meisten diabetischen Komplikationen verantwortlich

Diabetes Mellitus

AGE -Protein

↓

Makrophage AGE-Rezeptor

AGE-vermitteltes Pathogenese-Modell des Diabetes mellitus

Parodontalerkrankungen und Diabetes: Eine Wechselbeziehung

Die bei Hyperglykämie vermehrt entstehenden AGEs sind wohl für die meisten diabetischen Komplikationen verantwortlich

Die übermäßige AGE-Bildung beim Diabetes regt Monozyten mit speziellen AGE-Rezeptoren an und setzt eine Kaskade von Bindegewebeabbau, Proliferation und Thrombusbildung in Gang

Diabetes Mellitus

AGE -Protein

↓

Makrophage AGE-Rezeptor

Synthese und Sekretion von TNF-α + IL-1ß

↓

Abbauende Kaskade

↓

Hydrolase, MMP, Kollagenasesekretion

↓

Bindegewebeabbau

AGE-vermitteltes Pathogenese-Modell des Diabetes mellitus

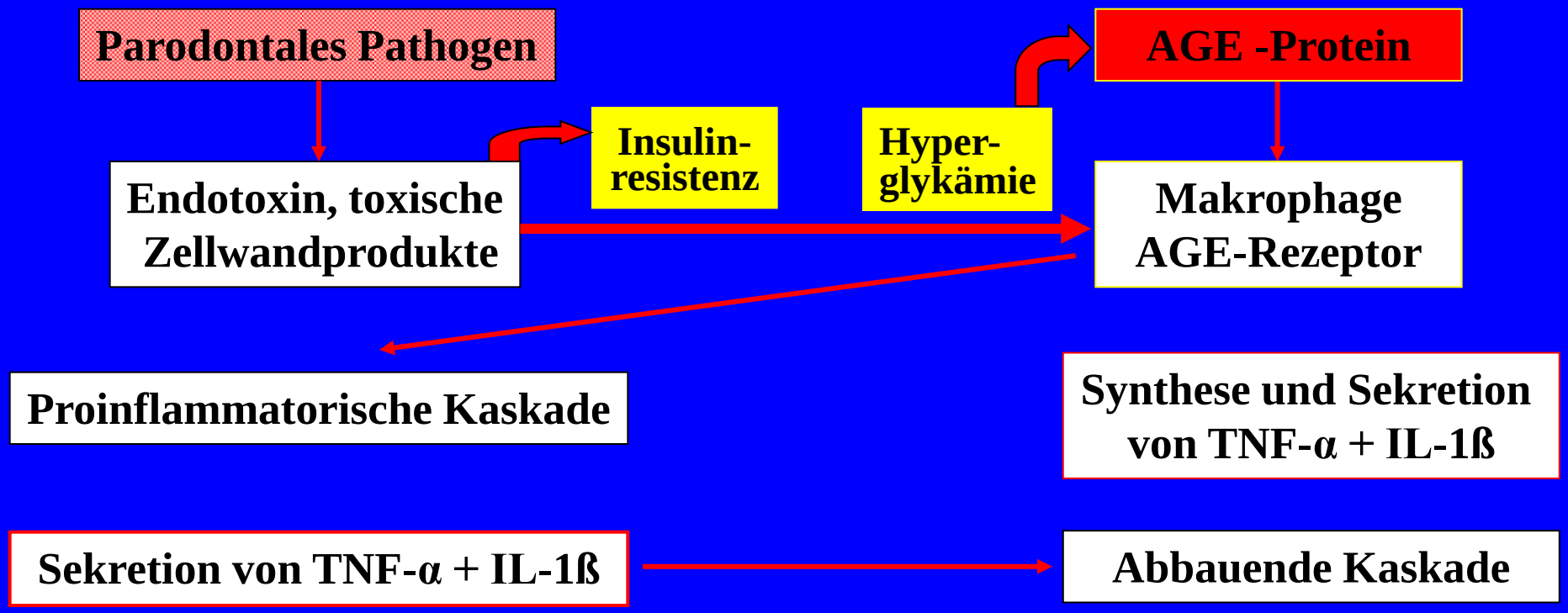
Parodontalerkrankungen und Diabetes: Eine Wechselbeziehung

Infektionen erhöhen die Insulinresistenz des Körpers und erschweren demzufolge die glykämische Kontrolle

Die AGE-Akkumulation hat wohl auch negativen Einfluss auf die Zusammensetzung der subgingivalen Flora

Hypothetisches Zwei-Wege-System von Parodontitis und Diabetes mellitus

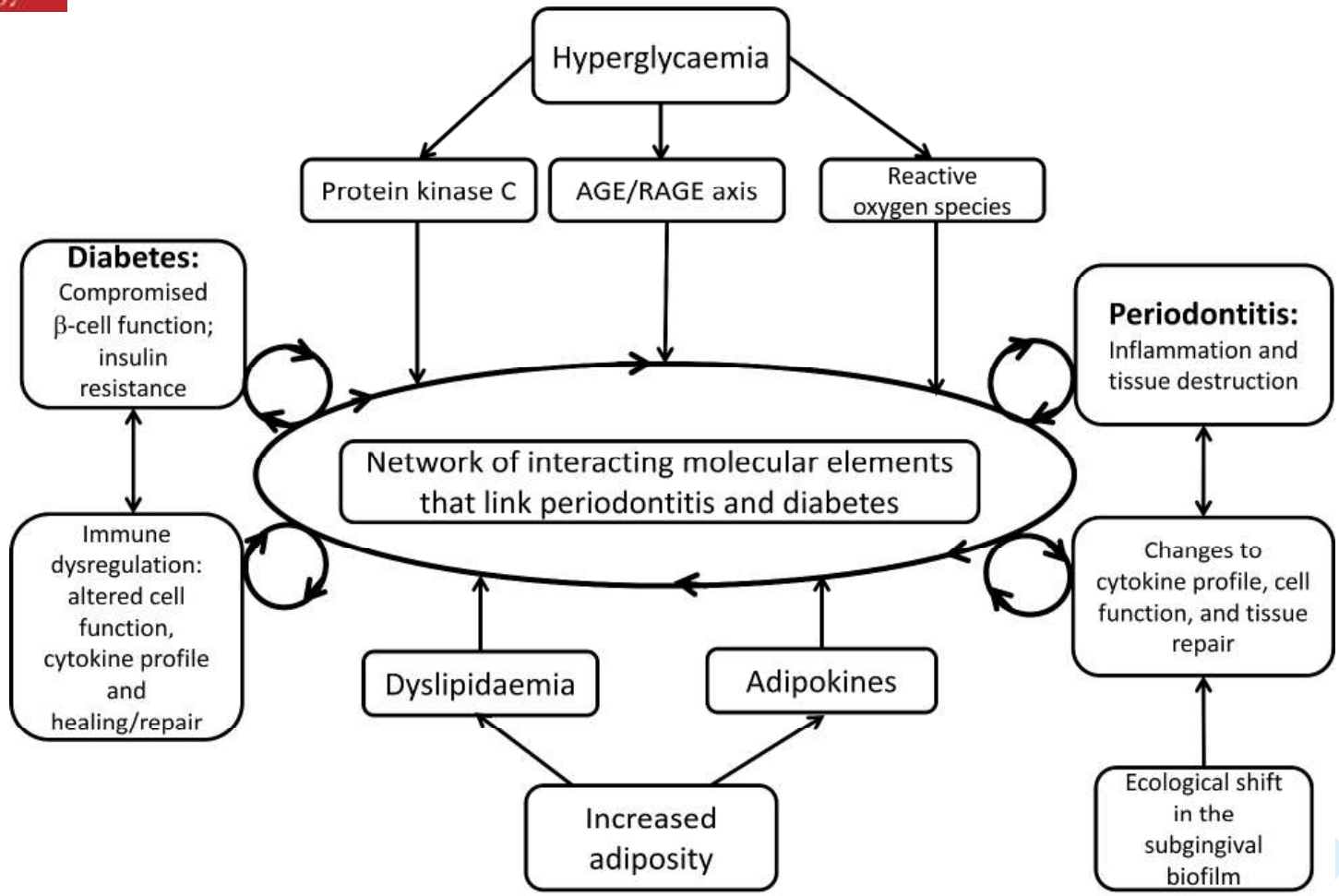
Parodontalerkrankungen und Diabetes: Eine Wechselbeziehung



Parodontale Infektion und AGE-vermittelte Zytokin-Antwort verstärken sich gegenseitig

Hypothetisches Zwei-Wege-System von Parodontitis und Diabetes mellitus

Journal of Clinical Periodontology

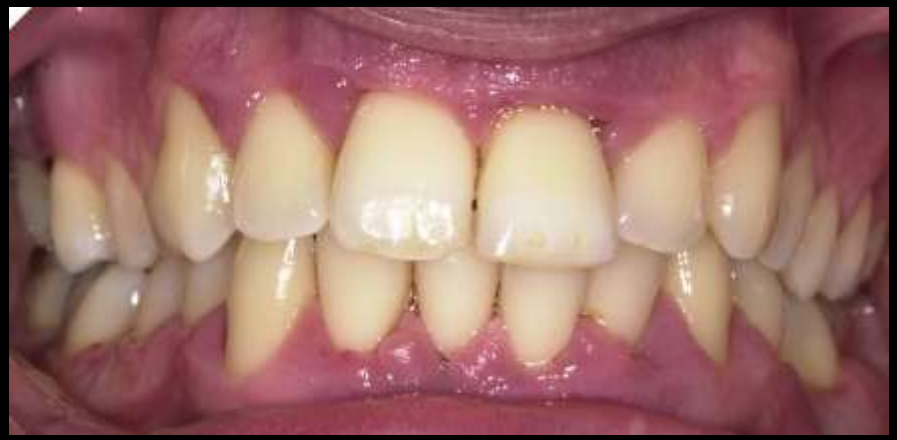


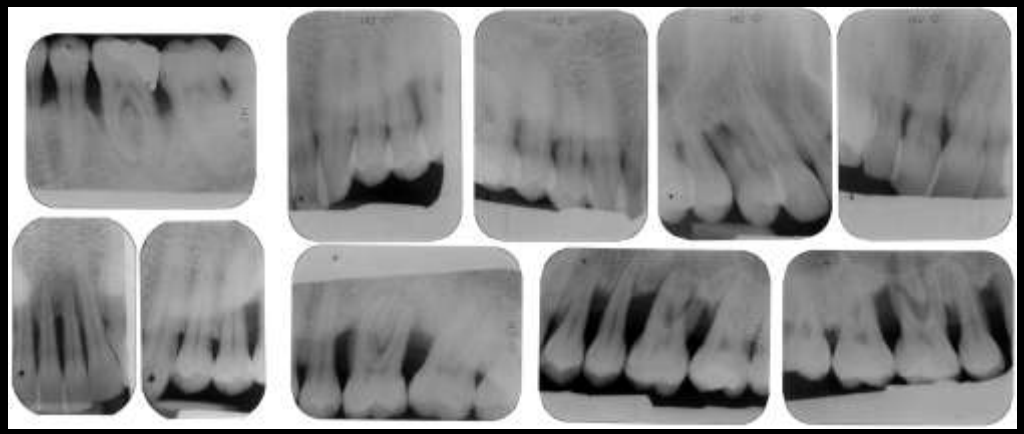
John J Taylor, Philip M Preshaw & Evanthia Lalla.

A review of the evidence for pathogenic mechanisms that may link periodontitis and diabetes.

Journal of Clinical Periodontology, in press. c

Parodontale Therapie bei Diabetikern





- Die Parodontitis ist eine chronisch bakterielle systemische Belastung, die den Diabetes mellitus kompliziert.
- Diabetiker mit chronischen Parodontalinfektionen sollten eine umfassende parodontale Therapie mit adäquater Eigenhygiene bekommen.
- Neben der Kontrolle des Diabetes (HbA1c Einstellung bis 6,5%) ist eine regelmäßige unterstützende parodontale Betreuung imperativ .

Interventions-Studien

IMPACT OF PERIODONTAL TREATMENT ON DIABETES OUTCOMES

Wijnand J.Teeuw, Victor E.A. Gerdes & Bruno Loos.
Effect of Periodontal Treatment on Glycemic Control of Diabetic Patients.
A systematic review and meta-analysis. Diabetes Care 33, 421-427.

OBJECTIVE

This study was assigned to explore that periodontal therapy leads to the improvement of glycemic control in diabetic patients.

RESEARCH DESIGN AND METHODS

Selection of publications was

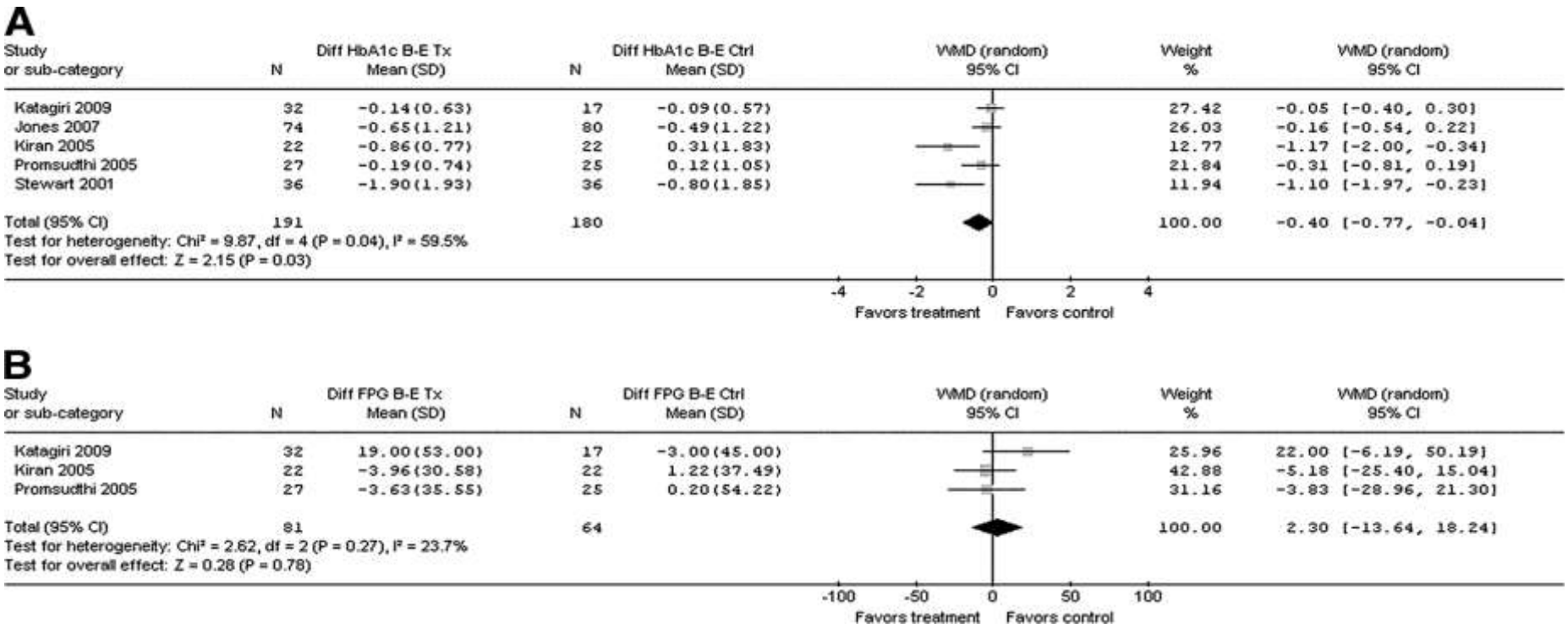
2) controlled periodontal intervention studies where the diabetic control group received no periodontal treatment, and

3) study duration of ≥3 months.

RESULTS

A total of 371 patients were included in this analysis with periodontitis as predictor and the actual absolute change in A1C (ΔA1C) as the outcome. The duration of follow-up was 3–9 months. The studies in a meta-analysis demonstrated a weighted mean difference of ΔA1C before and after therapy of −0.40% (95% CI −0.77 to −0.04%, $P = 0.03$) favoring periodontal intervention in type 2 diabetic patients.

Forest plots presenting WMD of Δ baseline–end %A1C levels (A) and Δ baseline–end FPG levels in mg/dl (B) between the treatment groups and control groups, heterogeneity and overall effect for treatment studies. diff, difference; B, baseline; E, end; Ctrl, control group; Tx, treatment group.



CONCLUSIONS

The present meta-analysis suggests that periodontal treatment leads to an improvement of glycemic control in type 2 diabetic patients for at least 3 months.

Teeuw W J et al. Dia Care 2010;33:421-427

American Diabetes Association.

IMPACT OF PERIODONTAL TREATMENT ON DIABETES OUTCOMES

Journal of Clinical Periodontology

Engebretson, Steven & Kocher, Thomas.

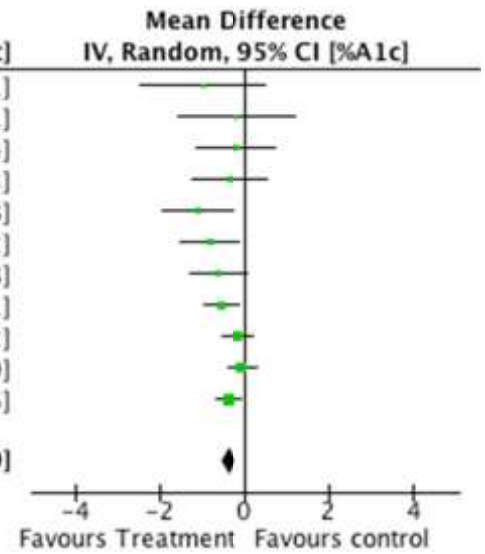
Evidence that periodontal treatment improves diabetes outcomes: a Systematic Review and Meta-analysis

Journal of Clinical Periodontology, in press.

	Treatment			Control				Mean Difference
Study or Subgroup	Mean [%A1c]	SD [%A1c]	Total	Mean [%A1c]	SD [%A1c]	Total	Weight	IV, Random, 95% CI [%A1c]
Moeintaghavi 2012	-0.74	1.67	22	0.25	2.87	18	1.3%	-0.99 [-2.49, 0.51]
Yun	-0.77	2	23	-0.58	2.77	23	1.5%	-0.19 [-1.59, 1.21]
Chen 2012 B	0.14	2.18	41	0.34	2.14	41	3.3%	-0.20 [-1.14, 0.74]
Chen 2012 A	-0.01	1.94	42	0.34	2.14	41	3.7%	-0.35 [-1.23, 0.53]
Kiran	-0.8	0.77	22	0.31	1.83	22	4.1%	-1.11 [-1.94, -0.28]
Singh 2008 B	-0.8	0.92	15	0.02	1.02	15	5.8%	-0.82 [-1.52, -0.12]
Singh 2008 A	-0.6	0.92	15	0.02	1.02	15	5.8%	-0.62 [-1.32, 0.08]
Koromantzos 2011	-0.73	0.91	30	-0.18	0.82	30	13.4%	-0.55 [-0.99, -0.11]
Jones	-0.65	1.21	74	-0.49	1.22	80	16.8%	-0.16 [-0.54, 0.22]
Katagiri	-0.14	0.63	32	-0.09	0.57	17	19.7%	-0.05 [-0.40, 0.30]
Sun 2011	-0.5	0.99	82	-0.14	0.94	75	24.5%	-0.36 [-0.66, -0.06]
Total (95% CI)			**398**			**377**	**100.0%**	**-0.36 [-0.54, -0.19]**

Heterogeneity: $Tau^2 = 0.01$; $Chi^2 = 11.01$, df = 10 (P = 0.36); $I^2 = 9\%$

Test for overall effect: Z = 4.13 (P < 0.0001)



Reduction in HbA1c is an established outcome measure of successful diabetes treatment. Evidence derived from RCTs shows that periodontal treatment results in a mean reduction in HbA1c of 0.36% (95% CI 0.19, 0.54) at 3 months.

IMPACT OF PERIODONTAL TREATMENT ON DIABETES OUTCOMES

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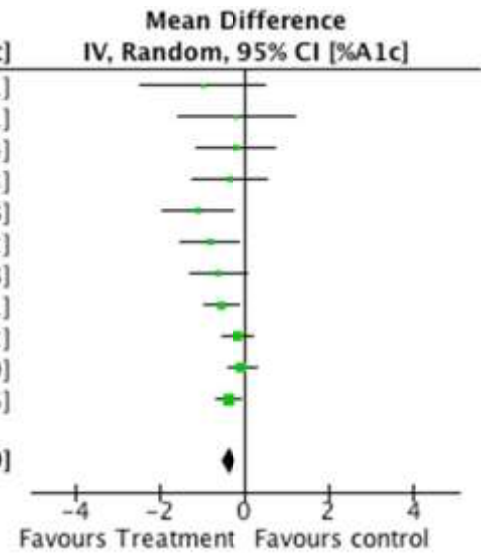


Reduction in HbA1c is an established outcome measure of successful diabetes treatment. Evidence derived from RCTs shows that periodontal treatment results in a mean reduction in HbA1c of 0.36% (95% CI 0.19, 0.54) at 3 months.

Guideline A

Guidelines for physicians and other medical health professions for Use in Diabetes Practice

Because(of(the(increased(risk(for(developing(periodontitis(in(patients(with(diabetes(the(following(recommendations(are(made:!

1. Patients!with!diabetes!should!be!told!that!periodontal!disease!risk!is!increased!by! diabetes.!They!should!also!be!told!that!if!they!suffer!from!periodontal!disease!their! glycemic! control! may! be! more! difficult,! and! they! are! at! higher! risk! for! diabetic! complications!such!as!cardiovascular!and!kidney!disease.!
   !
2. As! part! of! their! initial! evaluation,! patients! with! type! 1,! type! 2! and! gestational! diabetes! (GDM)! should! receive! a! thorough! oral! examination! which! includes! a! comprehensive!periodontal!examination.!
   !
3. For!all!newly!diagnosed!type!1!and!type!2!diabetes!patients,!subsequent!periodontal! examinations!should!occur!(as!directed!by!the!dental!professionals)!as!part!of!their! onZgoing! management! of! diabetes.! Even! if! no! periodontitis! is! diagnosed! initially,! annual!periodontal!review!is!recommended.!!
4. Diabetes! patients! presenting! with! any! overt! signs! and! symptoms! of! periodontitis,! including!loose!teeth!not!associated!with!trauma;!spacing!or!spreading!of!the!teeth;! and/or! gingival! abscesses! or! gingival! suppuration,! require! prompt! periodontal! evaluation.!
5. Patients! with! diabetes! who! have! extensive! tooth! loss! should! be! encouraged! to! pursue!dental!rehabilitation!to!restore!adequate!mastication!for!proper!nutrition.!
6. Oral!health!education!should!be!provided!to!all!patients!with!diabetes.!
7. For! children! and! adolescents! diagnosed! with! diabetes,! annual! oral! screening! is! recommended!from!the!age!of!6Z7!years!by!referral!to!a!dental!professional.!
8. Patients! with! diabetes! should! be! advised! that! other! oral! conditions! such! as! dry! mouth!and!burning!mouth!may!occur,!and!if!so!they!should!seek!advice!from!their! dental!practitioner.!!Also!patients!with!diabetes!are!at!increased!risk!of!oral!fungal! infections! and! experience! poorer! wound! healing! than! those! who! do! not! have! diabetes.!

Guideline B

Suggested guidelines for use in dental practice

My patient with diabetes

- Patients with diabetes should be told that they are at increased risk for periodontitis. They should also be told that if they suffer from periodontal disease their glycemic control may be more difficult, and they are at higher risk for other complications such as cardiovascular and kidney disease.
- Patients presenting with a diagnosis of type 1, type 2 or gestational diabetes should receive a thorough oral examination, which includes a comprehensive periodontal evaluation.
- If periodontitis is diagnosed, it should be properly managed. If no periodontitis is diagnosed initially, patients with diabetes should be placed on a preventive care regime and monitored regularly for periodontal changes.
- Patients with diabetes presenting with any acute oral/periodontal infections require prompt oral/periodontal care.
- Patients with diabetes who have extensive tooth loss should be encouraged to pursue dental rehabilitation to restore adequate mastication for proper nutrition.
- Oral health education should be provided to all patients with diabetes.
- Patients with diabetes should also be evaluated for other potential oral complications, including dry mouth, burning mouth, and candida infections.
- For children and adolescents diagnosed with diabetes, an annual oral screening for early signs of periodontal involvement is recommended starting at the age of 6 years.
- Patients who present without a diabetes diagnosis, but with obvious risk factors for type 2 diabetes and signs of periodontitis should be informed about their risk for having diabetes, assessed using a chairside HbA1c test, and/or referred to a physician for appropriate diagnostic testing and follow-up care.

Guideline C

Recommendations for patients with diabetes at the physician's practice/office

Why should I have my gums checked?

!

If your physician has told you that you have diabetes, you should make an appointment with a dentist to have your mouth and gums checked. This is because people with diabetes have a higher chance of getting gum disease. Gum disease can lead to tooth loss and may make your diabetes harder to control.

You may have gum disease if you have ever noticed:

- red, bleeding or swollen gums
- pus from the gums
- foul taste
- longer looking teeth
- loose teeth
- increasing spaces between your teeth
- calculus (tartar) on your teeth

If you have noticed any of these problems, it is important to see a dentist as soon as possible. Gum disease may be present and get worse with no apparent symptoms to you, so even if you do not think you have gum disease now, you should still get regular dental check-ups as part of managing your diabetes. Your dentist will be able to pick up early signs of gum disease.

You also need to clean your teeth and gums very carefully at home.

If you have diabetes, you may also suffer from dry mouth, burning mouth, yeast infections of the mouth or poor healing of mouth wounds.

It is important to keep your mouth and your whole body as healthy as possible with regular dental and medical care.

Guideline D

Recommendations for patients at the dental surgery/office who have diabetes or are found to be at-risk for diabetes]

You(have(diabetes(or(you(have(been(told(by(your(dentist(you(are(at(risk(for(diabetes(

!

People!with!diabetes!have!a!higher!chance!of!getting!gum!disease.!If!you!have!been!told!by! your!dentist!that!you!have!gum!disease,!you!must!follow!up!with!necessary!treatment!as! advised.!This!may!require!several!appointments.!Like!diabetes,!gum!disease!is!a!chronic! condition!and!requires!lifelong!maintenance.!You!also!need!to!clean!your!teeth!and!gums! very!carefully!at!home.!If!left!untreated,!gum!disease!can!lead!to!tooth!loss!and!may!also! make!your!diabetes!harder!to!control.!

Gum!disease!may!be!present!and!get!worse!with!no!apparent!symptoms!to!you,!so!if!your! dentist!told!you!that!you!do!not!have!gum!disease!now,!you!should!still!get!regular!dental! checkups!as!part!of!managing!your!diabetes.!Your!dentist!will!be!able!to!pick!up!early!signs! of!gum!disease.!!

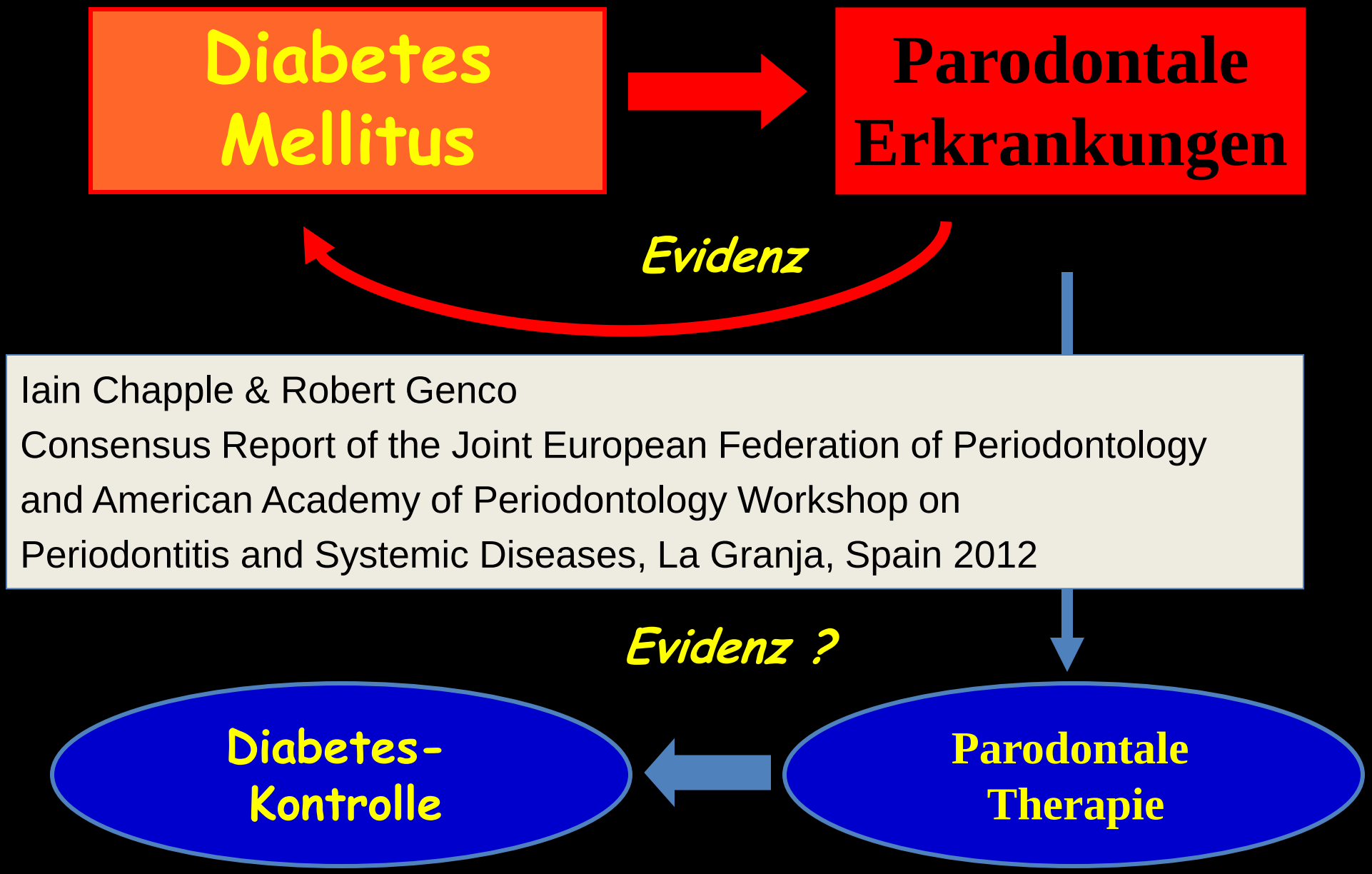
You!may!have!gum!disease!if!you!ever!notice:!!

- red,!bleeding!or!swollen!gums!!
- pus!from!the!gums!
- foul!taste!!
- longer!looking!teeth!!
- loose!teeth!!
- increasing!spaces!between!your!teeth!!
- calculus!(tartar)!on!your!teeth!

People!with!diabetes!may!also!suffer!from!dry!mouth,!burning!mouth,!yeast!infections!of! the!mouth!or!poor!healing!of!mouth!wounds.!

If!you!do!not!have!diabetes,!but!your!dentist!identified!some!risk!factors!for!diabetes! including!signs!of!gum!disease,!it!is!important!to!get!a!medical!checkup!as!advised.!Your! medical!doctor!can!order!blood!tests!to!see!if!you!have!diabetes!and!not!know!it,!and!can! provide!proper!advice!and!care!based!on!the!results.!Make!an!appointment!to!see!you! medical!doctor!as!soon!as!possible.!And!remember!to!inform!your!dentist!about!the! outcome!of!your!visit!to!the!medical!doctor.!!

It!is!important!to!keep!your!mouth!and!your!whole!body!as!healthy!as!possible!with!regular! dental!and!medical!care.!!


Diabetes Mellitus
Parodontale Erkrankungen
Evidenz
Iain Chapple & Robert Genco
Consensus Report of the Joint European Federation of Periodontology and American Academy of Periodontology Workshop on Periodontitis and Systemic Diseases, La Granja, Spain 2012
Evidenz ?
Diabetes-Kontrolle
Parodontale Therapie

Parodontitis & Untergewichtige Frühgeburten

Während die Neugeborenen-Sterblichkeit in den letzten 40 Jahren deutlich abgenommen hat, konnte die Zahl der Frühgeburten und untergewichtigen Neugeborenen nicht reduziert werden.

Frühgeburt ist eine Geburt < 37 SSW

Vermindertes Geburtsgewicht < 2500 g

Preterm birth

- **Incidence is increasing**

  (USA 12-13%, Europe 5-9%): (e.g. Denmark about 22% between 1995-2004, USA about 21% between 1990-2006)

  WHO (2010) Neonatal and perinatal Mortality: Country, regional and Global Estimates. Geneva.

  Martin et al. Births: final data for 2006. Natl Vital Stat rep 57: 1-102.

- Due to ARTS increased number of medical indications

  (**a**rtificial **r**eproduction **t**echnologies cause more multiple births)

  Reddy et al. Infertility, assisted reproductive technology, and adverse pregnancy outcomes: executive summary of a National Institute of Child Health and Human Development Workshop. Obstetrics and Gynecology 2007.

- To be held responsible for three-quarter of all neonatal deaths

  and more than half of long term morbidity

  ACOG Practice Bulletin (2003). Management of preterm labor. Int J Gynaecology and Obstetrics.

- 40% of all survivors with weight at birth under 750g exhibit forms of moderate to severe disabilities

  Hack et al. School-age outcomes in children with birth weights under 750g. New Engl J Medicine, 1994.

Preterm birth: terminal of different etiologies

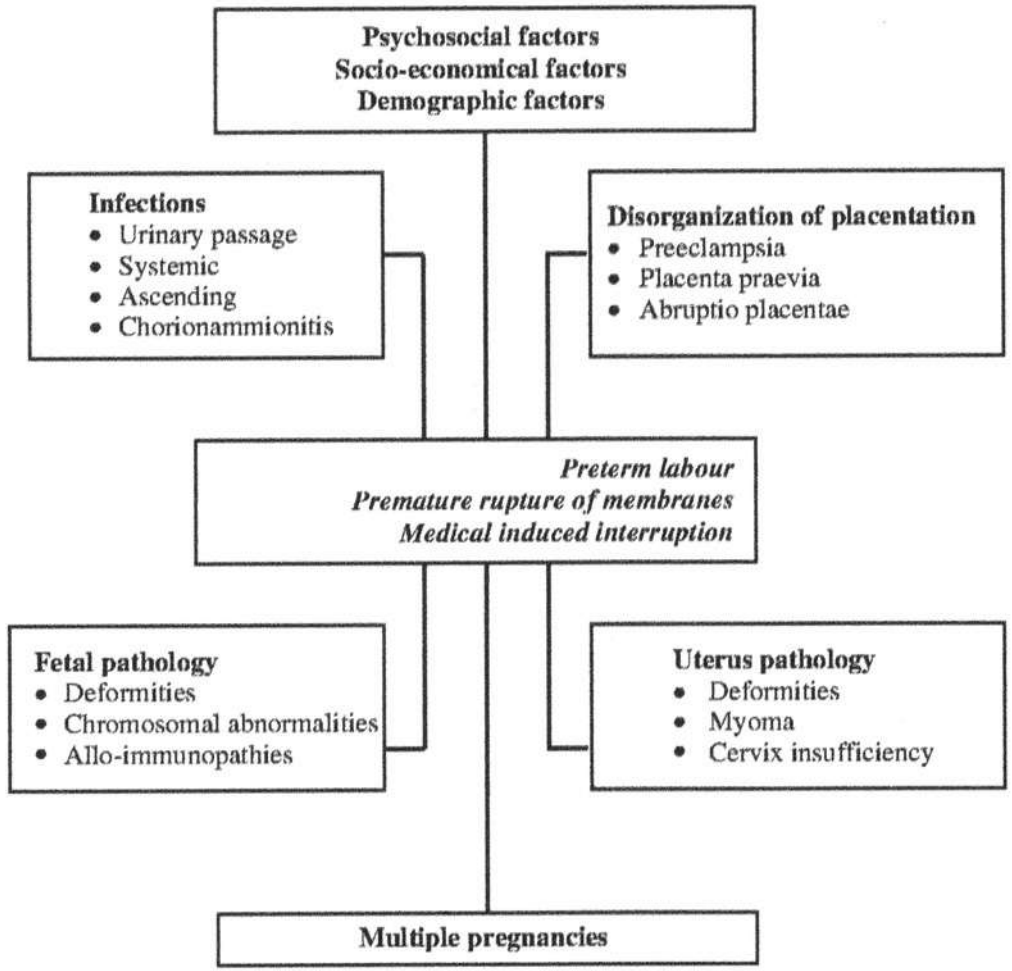


Fig. 1. Pathogenesis of pre-term birth (Schneider et al. 2006).

Wimmer G. & Pihlstrom BL. **A critical assessment of adverse pregnancy outcome and periodontal disease. J Clin Perio 2008;35(Suppl. 8): 380-397**

Periodontal disease during pregnancy and obstetric outcomes

- **Woman during pregnancy are susceptible to poor oral health (higher risk of suffering various dental problems such as gingivitis/periodontitis).**

  Mills & Moses. Oral health during pregnancy. MCN Am J Mater Child Nurs 2002.

  Silk et al. Oral health during pregnancy. Am Fam Physician 2008.

- **Periodontitis is a relatively common clinical condition; it has a prevalence in pregnant woman up to 75% for gingivitis, 5% and 20% for periodontitis.**

  Laine. Effect of pregnancy on periodontal and dental health. Acta Odontol Scand 2002.

  Stefanac. Treatment planing in Dentistry. Mosby, 2001.

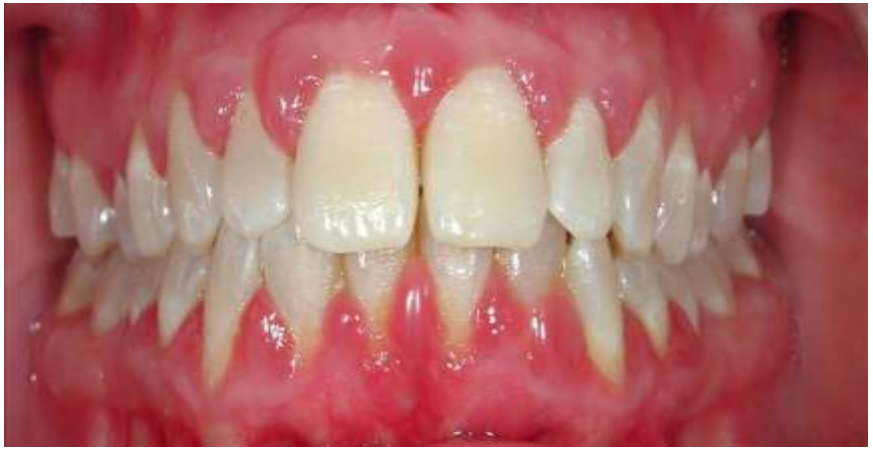
- **There is mounting evidence high-lightening the potential impact of poor maternal oral health on the health outcomes of mothers and their infants .**

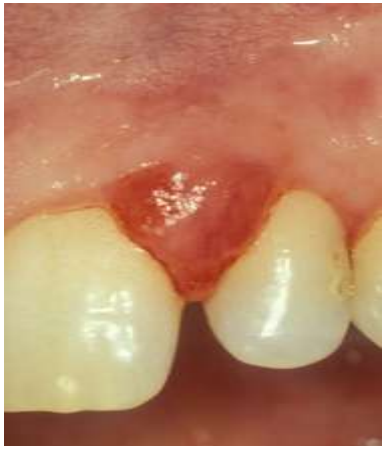
  Gussy et al. Early childhood caries: current evidence for aetiology and prevention. J Paediatr Child Health 2006.

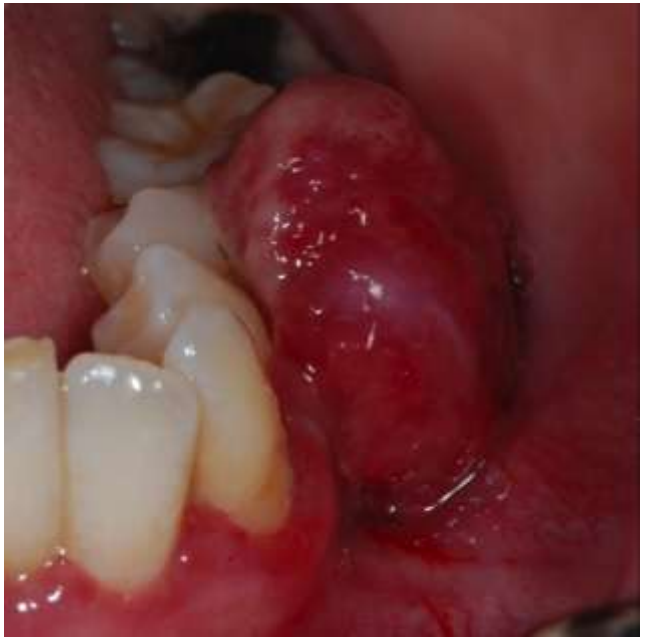
Periodontal disease during pregnancy and obstetric outcomes

In der Schwangerschaft kommt zu immunologischen Veränderungen durch die zunehmende Progesteronkonzentration im ökologischen Milieu der Mundhöhle:

- Verstärkung einer vorausgehenden Gingivitis
  - Ödematöse Schwellung mit starker Blutungsneigung
  - Schwangerschaftsepulis







Frühgeburt – Infektion/Entzündung

- **Einziger pathologischer Prozess, bei dem ein kausaler Link zur Frühgeburt besteht und der molekulare pathophysiologische Mechanismus bekannt ist**
- **25-40% intrauterine Infektion**
  - 13% bei vorzeitiger WT & intakten Membranen, 32% bei PROM
  - Am häufigsten Mycoplasmen (Ureaplasma urealyticum) im Fruchtwasser
  - Fraglich: Mikrobiale Kolonisation zwischen Amnion und Chorion, Kultivierung nicht von allen Keimen möglich
  - Muss nicht immer Wehen auslösen, Infektion in den Membranen prädisponiert zur vorzeitigen Wehentätigkeit

Periodontal infections as a possible risk factor for preterm low birth weight

Offenbacher S. et al. 1996

Jeffcoat 2001, 2003, Bunduneli 2005, Lopez 2002, 2005 (pregnancy-associated gingivitis), Huyoel 2006

Fall-Kontroll-Studie mit 124 Frauen

kontrolliert für andere Risikofaktoren:

Rauchen, Alkohol und Drogenabusus
Ernährung und Ausmaß der pränatalen Obsorge
Infektionen des Urogenitaltraktes
vorangegangene Fehlgeburten, andere medizinische Probleme

Parodontal erkrankte Schwangere zeigten ein signifikant erhöhtes Risiko (OR = 7.9) zu untergewichtigen Frühgeburten.

Periodontal disease during pregnancy and obstetric outcomes

- **Periodontal disease causes the release of pathogens or inflammatory products (which may then affect embryonic tissue or amniotic fluid through hamatogenous transport).**

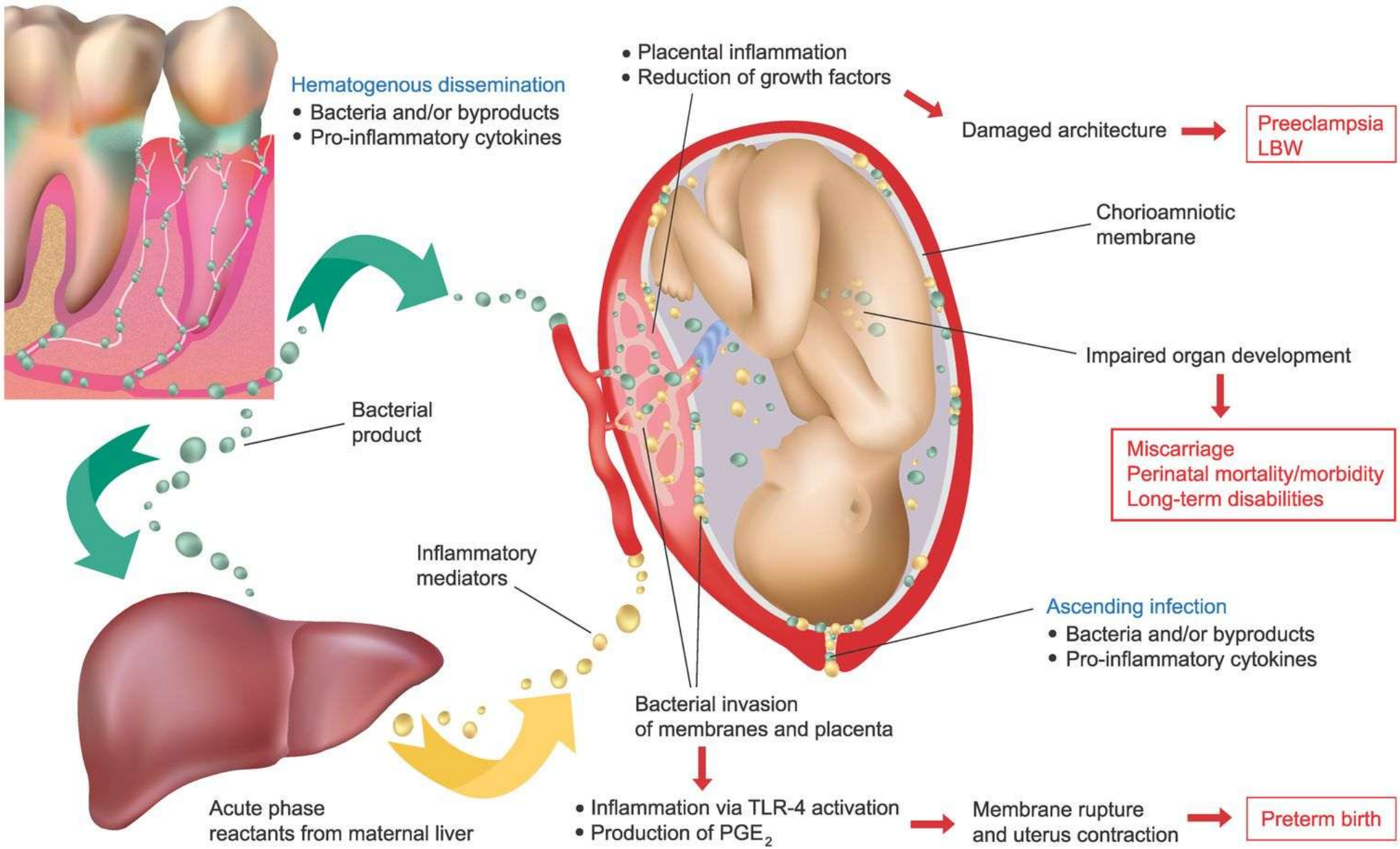
  Offenbacher et al. Potential pathogenic mechanism of periodontitis associated pregnancy complications. Ann Periodontol 1998.

- **There are indications of an association between periodontal disease and adverse pregnancy outcomes (such as preterm birth and low birth weight) in some populations.**

  Wimmer, Pihlstrom. A critical assessment of adverse pregnancy outcome and periodontal disease. J Clin Perio 2008.

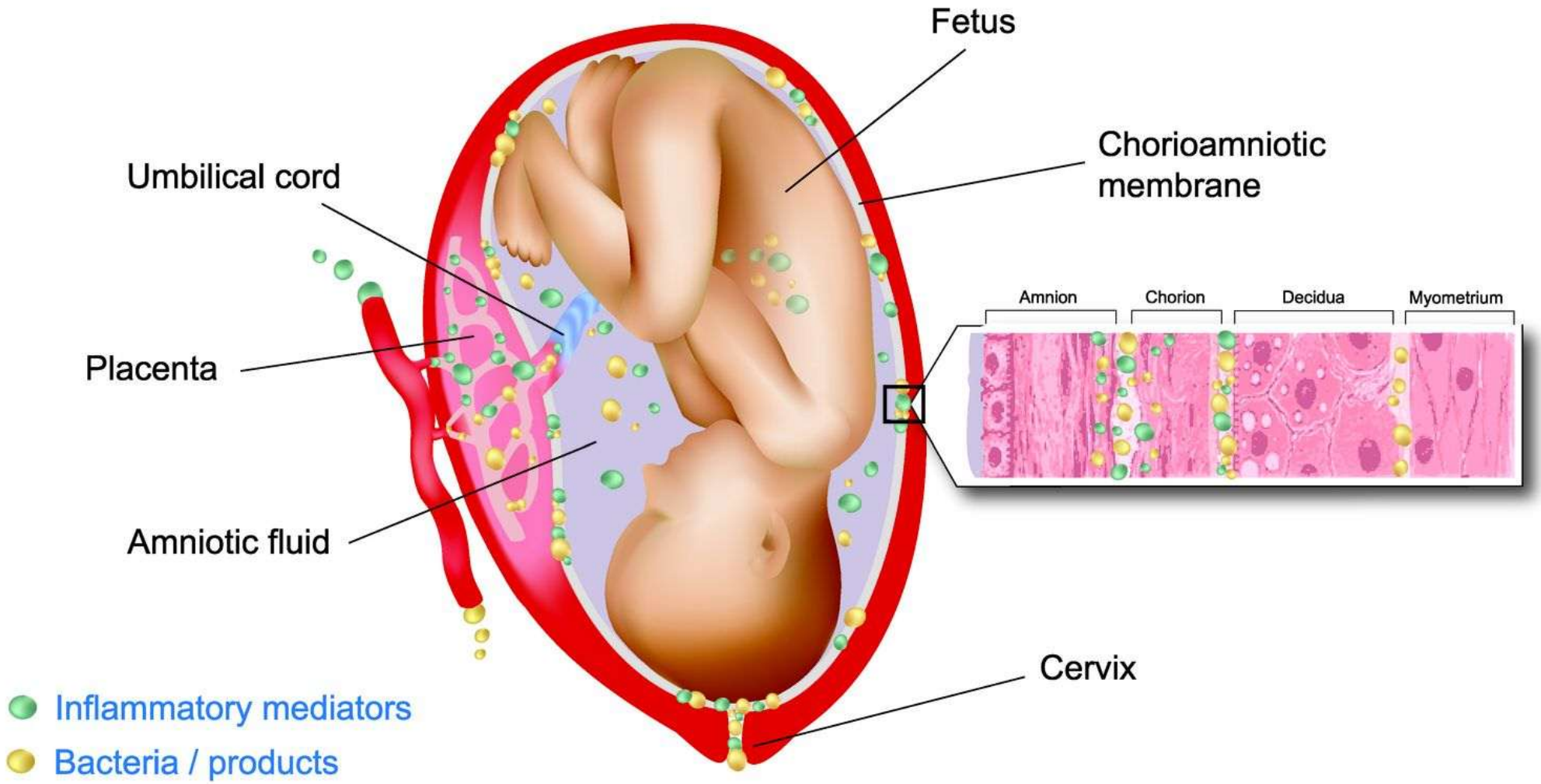
  Chambrone et al. Evidence grade associating periodontitis to preterm birth and/or low birth weight: I.

  A systematic review of prospective cohort studies. J Clin Perio 2011.


Hematogenous dissemination
• Bacteria and/or byproducts
• Pro-inflammatory cytokines
• Placental inflammation
• Reduction of growth factors
Damaged architecture
Preeclampsia
LBW
Chorioamniotic membrane
Impaired organ development
Miscarriage
Perinatal mortality/morbidity
Long-term disabilities
Bacterial product
Inflammatory mediators
Ascending infection
• Bacteria and/or byproducts
• Pro-inflammatory cytokines
Bacterial invasion of membranes and placenta
Acute phase reactants from maternal liver
• Inflammation via TLR-4 activation
• Production of PGE2
Membrane rupture and uterus contraction
Preterm birth

Journal of Clinical Periodontology

Phoebus N. Madianos, Yiorgos A. Bobetsis, Steven Offenbacher.
Adverse Pregnancy Outcomes (APOs) and Periodontal Disease: Pathogenic Mechanisms.
Journal of Clinical Periodontology, in press.

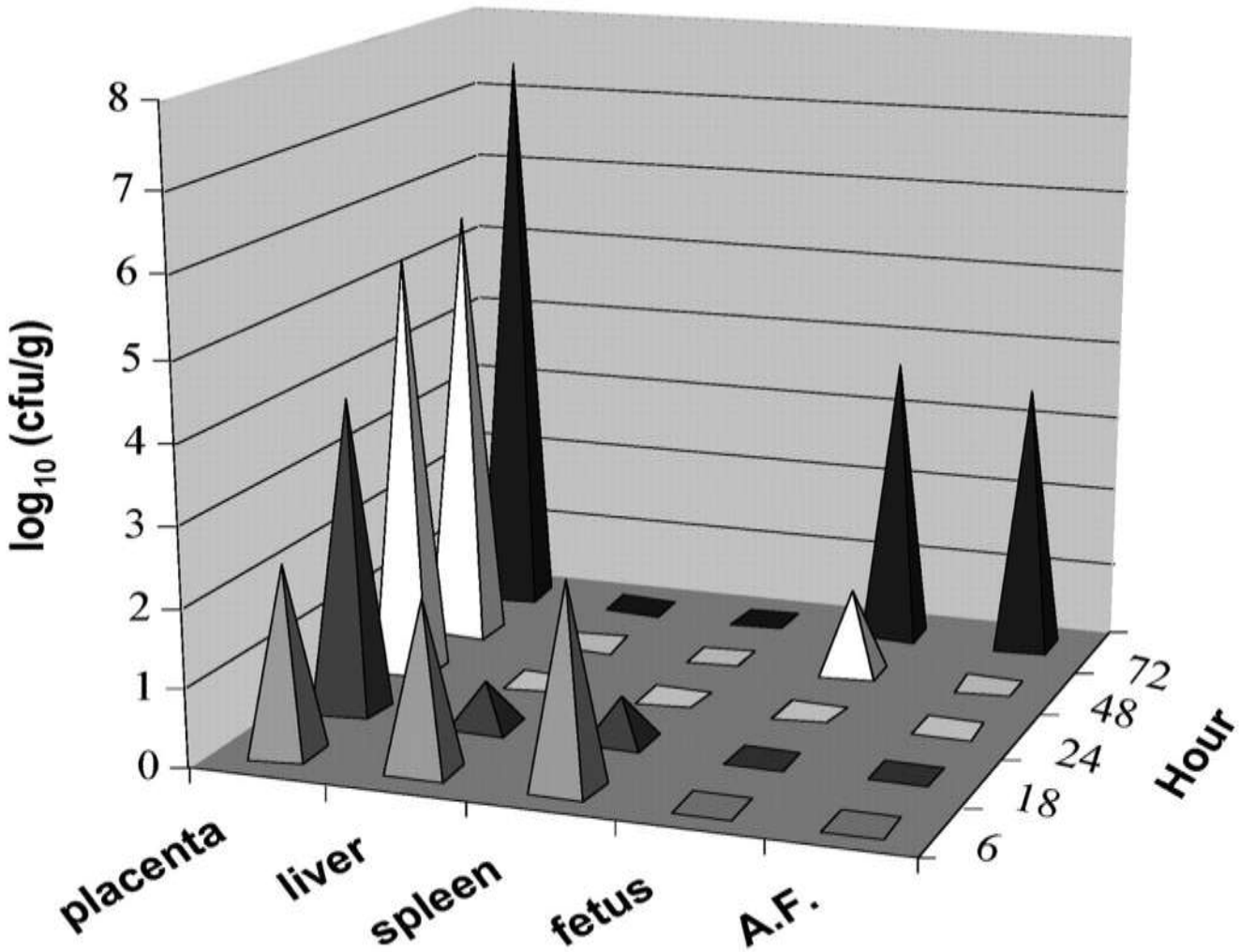


Periodontal disease and adverse pregnancy outcomes

Han YW, Redline RW,Li M, Yin L, Hill GB, McCormick TS**.**
***Fusobacterium nucleatum* induces premature and term Stillbirth in Pregnant Mice: Implication of Oral Bacteria in Preterm Birth.**
Infection and Immunity 72 (2004); 4:2279-2004

This study examined the possible mechanism underlying the link between these diseases. To test this hypothesis, we intravenously injected *F. nucleatum* into pregnant CF-1 mice. The injection resulted in premature delivery, stillbirths, and nonsustained live births. The bacterial infection was restricted inside the uterus, without spreading systemically.

Kinetic study of intravenous injection of F. nucleatum into pregnant mice.



Han Y W et al. Infect. Immun. 2004;72:2272-2279

Periodontal disease and adverse pregnancy outcomes

Han YW, Redline RW,Li M, Yin L, Hill GB, McCormick TS**.**
***Fusobacterium nucleatum* induces premature and term Stillbirth in Pregnant Mice: Implication of Oral Bacteria in Preterm Birth.**
Infection and Immunity 72 (2004); 4:2279-2004

F. nucleatum strains isolated from amniotic fluids and placentas along with those isolated from orally related sources invaded both epithelial and endothelial cells.

The invasive ability may enable F. nucleatum to colonize and infect the pregnant uterus.

This study represents the first evidence that Fusobakterium nucleatum may be transmitted hematogenously to the placenta and cause adverse pregnancy outcomes.

Evidence associating periodontitis to preterm birth and/or low birth weight

There are indications of an association between periodontal disease and adverse pregnancy outcomes (such as preterm birth and low birth weight) in some populations.

- *Wimmer G, Pihlstrom BL.* A critical assessment of adverse pregnancy outcome and periodontal disease. J Clin Perio 2008.
- *Chambrone et al.* Evidence grade associating periodontitis to preterm birth and/or low birth weight:

  I. A systematic review of prospective cohort studies. J Clin Perio 2011.

Periodontal Diseases and Adverse pregnancy Outcomes (Wimmer & Pihlstrom, J Clin Perio 2008)

Systematic Reviews

Authors	Studies Included	Outcomes	Conclusions
Scannapieco et al. (2004)	12 (6 case-control, 3 cross-sectional and longitudinal and 3 intervention studies)		PD may be a risk factor for PTB/LBW It is not yet clear if PD has causal role in APO.
Xiong et al. (2006)	25 (13 case-control, 9 cohort studies and 3 controlled trials)	18 Positive (ORs ranging from 1.10 to 20.0) 7 Negative (ORs ranging from 0.78 to 2.54) 3 Trials: Periodontal Intervention can lead to 57% reduction in PLBW (RR 0.43; 0.24-0.78) and a 50% reduction PTB (RR 0.5; 0.20-1.30).	PD may be associated with an increased risk of APO.
Vettore et al. (2006)	36 (27 case-control, 6 cohort studies and 3 RCTs)	26 Positive	Methodological limitations of studies didn't allow conclusions concerning the effects of PD on APO.

Meta-analysis

Authors	Studies Included	Outcomes	Conclusions
Khader & Ta'ani (2005)	5 (2 case-control, 3 cohort studies)	PTB: OR 4.28 (2.62 to 6.99) PLBW: OR 5.28 (2.21 to 12.62) PTB or LBW 2.30 (1.21 to 4.38)	PD in pregnants significantly increases the risk of subsequent PTB birth or LBW.
Vergnes & Sixou (2007)	17 (11 case-control, 4 cohort, 2 cross-sectional studies)	OR 2.83 (1.95 to 4.10) Of 7151 women, 1056 delivered PLBW	The findings indicate a likely association.
Xiong et al: (2007)	44 (26 case-control, 13 cohort studies, and 5 controlled trials)	29 Positive OR (ranging from 1.10 to 20.0) 15 Negative OR (ranging from 0.78 to 2.54) 5 Trials: Periodontal intervention may reduce PLBW: RR: 0.53 (0.30-0.95) NSD reduction of PTB: RR 0.79 (0.55-1.11) or LBW: RR 0.86 (0.58 to 1.29).	The authors conclude that PD may be associated with increased risk of APO.

Evidence associating periodontitis to preterm birth and/or low birth weight

Wimmer G, & Pihlstrom BL.
A critical assessment of adverse pregnancy outcome and periodontal disease.
J Clin Periodontol. 2008 Sep;35(8 Suppl):380-97.

CONCLUSION

- **Variability in definitions** among studies of periodontal disease/adverse pregnancy outcomes and
- **Inadequate control for confounding factors** is widespread and possible effect modifications make it difficult to base **meaningful conclusions** on published data.

There are **indications of an association** between periodontal disease and **increased risk** of adverse pregnancy outcome in **some** populations.

However there is no conclusive evidence that treating periodontal disease improves birth outcome.

Evidence associating periodontitis to preterm birth and/or low birth weight

Chambrone L, Guglielmetti MR, Pannuti CM, Chambrone LA.
Evidence grade associating periodontitis to preterm birth and/or low birth weight: I. A systematic review of prospective cohort studies. J Clin Perio 2011;38 (9): 795-808.

CONCLUSION

The results of this SR of prospective cohort studies provide evidence to support the hypothesis that periodontitis is associated with the risk of PB and/or LBW.

Evidence associating periodontitis to preterm birth and/or low birth weight

Chambrone L, Guglielmetti MR, Pannuti CM, Chambrone LA.
Evidence grade associating periodontitis to preterm birth and/or low birth weight: I. A systematic review of prospective cohort studies. J Clin Perio 2011;38 (9): 795-808.

Practical implications:

Despite the positive findings linking PB and/or LBW to periodontitis, such outcomes have to be interpreted with prudence as it was not possible to explain the reasons of high heterogeneity detected by the pooled estimates. Overall, pregnant women should be informed about the risks of PD and undergo a periodontal examination. Key methodological aspects should be evaluated and reported by future studies

- Most of studies reported a high methodological quality, but none fulfilled all methodological domains.
- Key domains such as sample size calculation, management of cofounders, training/calibration of examiners, examiner blinding, unity of analysis and assessment of periodontal conditions and APOs were not adequately reported/appraised.

Periodontal disease during pregnancy and obstetric outcomes

- **Well established maintenance of oral health during pregnancy (dental exam and appropriate preventive or therapeutic treatment) will reduce the risk of pregnancy associated oral diseases.**

  American Academy of Periodontology. Task force on periodontal treatment of pregnant woman. J Periodontol 2004.

  Meyer et al. An early health oral care program starting during pregnancy. Clin Oral Investig 2010.

- **However, there is still inconclusive data on whether periodontal treatment during pregnancy (scaling and root planing) will improve adverse pregnancy outcomes (preterm birth and low birth weight incidence).**

  Polyzos et al. Effect of periodontal diseas treatmnet during pregnancy on preterm birth incidence: a metaanalysis of randomized trials. Am J Obstetrics & Gynecology 2009.

  Polyzos et al. Obstetric outcomes after treatment of periodontal disease during pregnancy: systematic review and meta-analysis. BMJ 2010.

  George et al. Periodontal treatment during pregnancy and birth outcomes: a meta-analysis of randomised trials. Int J Evid Based Healthcare 2011.

Periodontal treatment during pregnancy and adverse pregnancy outcomes

Michalowicz BS, Hodges JS, DiAngelis AJ, Lupo VR, Novak MJ, Ferguson JE, Buchanan W, et al.
Treatment of Periodontal Disease and the Risk of Preterm Birth.
***N Engl J Med* 2006; 355:1885-1894.**

BACKGROUND

We studied the effect of nonsurgical periodontal treatment on preterm birth.

METHODS

We randomly assigned women between 13 and 17 weeks of gestation to undergo scaling and root planing either before 21 weeks (413 patients in the treatment group) or after delivery (410 patients in the control group). Patients in the treatment group also underwent monthly tooth polishing and received instruction in oral hygiene.

The gestational age at the end of pregnancy was the prespecified primary outcome. Secondary outcomes were birth weight and the proportion of infants who were small for gestational age.

No. at Risk

Control group	410	410	409	408	403	398	395	393	393	389	387	385	372	353
Treatment group	413	413	413	410	406	404	401	401	400	399	397	390	378	358

Figure 2. **Kaplan–Meier Curve for the Cumulative Incidence of Pregnancies Ending before 37 Weeks.**

Periodontal treatment during pregnancy and adverse pregnancy outcomes

Michalowicz BS, Hodges JS, DiAngelis AJ, Lupo VR, Novak MJ, Ferguson JE, Buchanan W, et al.
Treatment of Periodontal Disease and the Risk of Preterm Birth.
***N Engl J Med* 2006; 355:1885-1894.**

Conclusion:

Treatment of periodontitis in pregnant women improves periodontal disease and **is safe** but does not significantly alter rates of preterm birth, low birth weight, or fetal growth restriction.

Periodontal treatment during pregnancy and adverse pregnancy outcomes

Chambrone L, Cláudio Mendes Pannuti CM, Guglielmetti MR, Chambrone LA.
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.
J Clinical Periodontology 2011 (38);10: 902-914.

Aim:

The aim of this systematic review was to evaluate whether maternal periodontal disease treatment (MPDT) can reduce the incidence of preterm birth (PB) and/or low birth weight (LBW).

Methods:

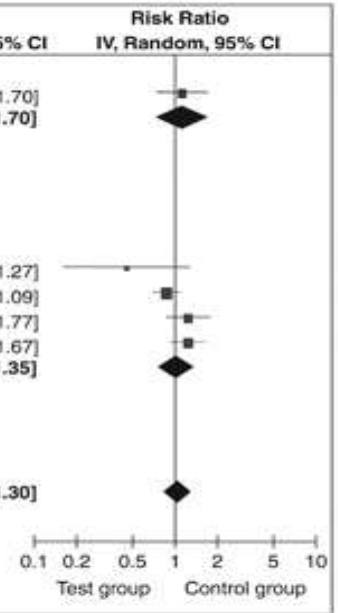
The Cochrane Central Register of Controlled Trials, MEDLINE and EMBASE were searched for entries up to October 2010 without restrictions regarding the language of publication.

Only randomized-controlled clinical trials (RCTs) that evaluated the effect of MPDT on birth term and birth weight were included. The search was conducted by two independent reviewers and random-effects meta-analyses were conducted methodically.

Forest plots of random-effects meta-analyses including only studies considered to be at a low risk of bias. Outcome: preterm birth.

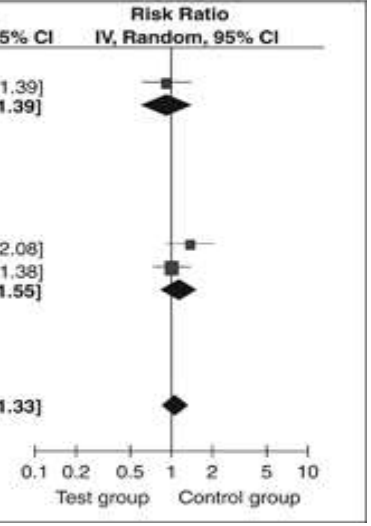
4.1 Preterm birth < 37 weeks of gestation

Study or Subgroup	Test group Events	Test group Total	Control group Events	Control group Total	Weight	Risk Ratio IV, Random, 95% CI
4.1.1 PD defined by PPD and CAL						
Michalowicz et al.(2006)	44	402	38	391	17.8%	1.13 [0.75, 1.70]
Subtotal (95% CI)		**402**		**391**	**17.8%**	**1.13 [0.75, 1.70]**
Total events	44		38			
Heterogeneity: Not applicable						
Test for overall effect: Z = 0.57 (P = 0.57)						
4.1.2 PD defined by CAL alone						
Jeffcoat et al. (2003)	5	123	11	123	4.2%	0.45 [0.16, 1.27]
Jeffcoat et al. (2010)	73	160	85	162	31.4%	0.87 [0.70, 1.09]
Macones et al. (2010)	58	359	47	361	21.0%	1.24 [0.87, 1.77]
Offenbacher et al. (2009)	91	874	73	871	25.5%	1.24 [0.93, 1.67]
Subtotal (95% CI)		**1516**		**1517**	**82.2%**	**1.02 [0.78, 1.35]**
Total events	227		216			
Heterogeneity: Tau² = 0.04; Chi² = 7.23, df = 3 (P = 0.06); I² = 59%						
Test for overall effect: Z = 0.17 (P = 0.87)						
Total (95% CI)		**1918**		**1908**	**100.0%**	**1.05 [0.84, 1.30]**
Total events	271		254			
Heterogeneity: Tau² = 0.03; Chi² = 7.44, df = 4 (P = 0.11); I² = 46%						
Test for overall effect: Z = 0.40 (P = 0.69)						
Test for subgroup differences: Chi² = 0.21, df = 1 (P = 0.65), I² = 0%						



4.2 Low birth weight < 2,500 g

Study or Subgroup	Test group Events	Test group Total	Control group Events	Control group Total	Weight	Risk Ratio IV, Random, 95% CI
4.2.1 PD defined by PPD and CAL						
Michalowicz et al.(2006)	40	406	43	403	27.6%	0.92 [0.61, 1.39]
Subtotal (95% CI)		**406**		**403**	**27.6%**	**0.92 [0.61, 1.39]**
Total events	40		43			
Heterogeneity: Not applicable						
Test for overall effect: Z = 0.38 (P = 0.70)						
4.2.2 PD defined by CAL alone						
Macones et al. (2010)	48	357	35	359	27.3%	1.38 [0.91, 2.08]
Offenbacher et al. (2009)	72	872	71	866	45.1%	1.01 [0.74, 1.38]
Subtotal (95% CI)		**1229**		**1225**	**72.4%**	**1.14 [0.85, 1.55]**
Total events	120		106			
Heterogeneity: Tau² = 0.01; Chi² = 1.42, df = 1 (P = 0.23); I² = 30%						
Test for overall effect: Z = 0.88 (P = 0.38)						
Total (95% CI)		**1635**		**1628**	**100.0%**	**1.07 [0.86, 1.33]**
Total events	160		149			
Heterogeneity: Tau² = 0.00; Chi² = 2.11, df = 2 (P = 0.35); I² = 5%						
Test for overall effect: Z = 0.62 (P = 0.54)						
Test for subgroup differences: Chi² = 0.69, df = 1 (P = 0.41), I² = 0%						



Periodontal treatment during pregnancy and adverse pregnancy outcomes

Chambrone L, Cláudio Mendes Pannuti CM, Guglielmetti MR, Chambrone LA.
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.
J Clinical Periodontology 2011 (38);10: 902-914.

Implications for practice

Despite the lack of an association between MPDT and the incidence of PB and/or LBW,

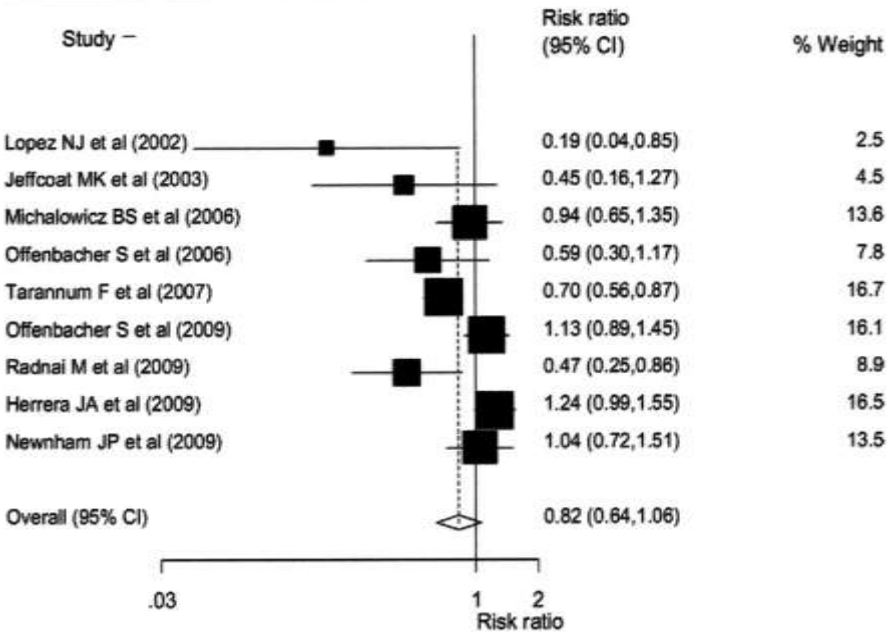
pregnant women with PD should be instructed about the importance of periodontal health and undergo proper treatment.

Moreover, obstetricians could be advised to refer their patients for a periodontal examination (as part of routine prenatal evaluations).

Periodontal disease and adverse pregnancy outcomes

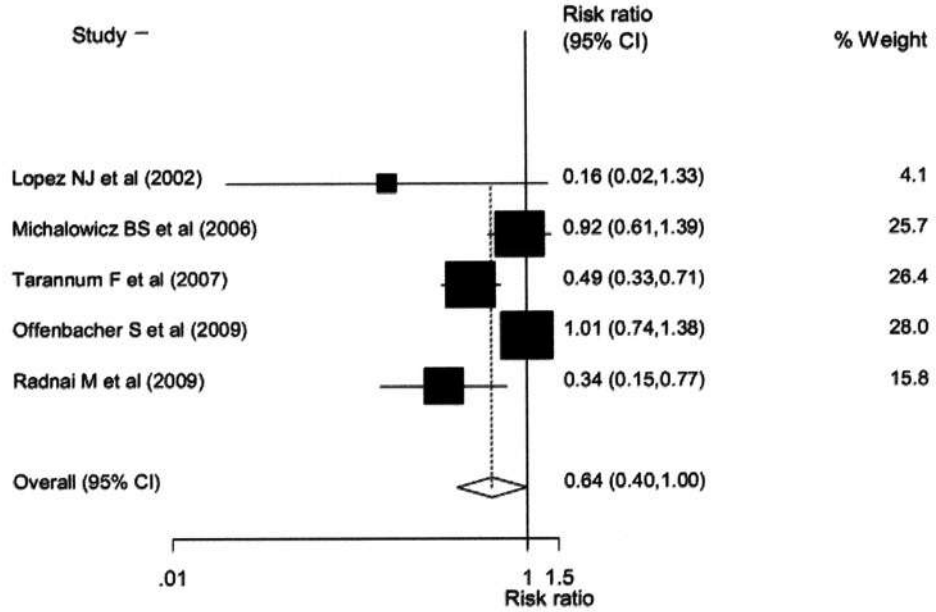
Xiong X, Buekens P, Goldenberg R, Offenbacher S, Qian X.
Optimal timing of periodontal disease treatment for prevention of adverse pregnancy outcomes: before or during pregnancy?
American Journal of Obstetrics & Gynecology 2011;205:111.e1-6.

FIGURE 1
Periodontal disease and preterm birth



Heterogeneity χ^2 = 28.48 (d.f. = 8) P = .000.
Estimate of between-study variance Tau-squared = 0.0850.
Test of relative risk = 1 : z = 1.51 P = .131.
CI, confidence interval.
Xiong. Timing of periodontal disease treatment for prevention of pregnancy outcomes. Am J Obstet Gynecol 2011.

FIGURE 2
Periodontal disease and low birthweight



Heterogeneity χ^2 = 14.88 (d.f. = 4) P = .005.
Estimate of between-study variance Tau-squared = 0.1653.
Test of relative risk = 1 : z = 1.95 P = .051.
CI, confidence interval.
Xiong. Timing of periodontal disease treatment for prevention of pregnancy outcomes. Am J Obstet Gynecol 2011.

Periodontal disease and adverse pregnancy outcomes

What other explanations need to be explored before a conclusion can be drawn?

- ***Timing of periodontal treatment?***

  (may be too late, inflammation cascade is activated, interventions ineffective)

- ***Oral mechanical manipulation may even initiate the pathway?***

  (can cause bacteremia, trigger a systemic inflammatory cascade, dilute beneficial tx effects)

- ***Periodontal treatment provided during pregnancy is not always effective?***

  (maintenance throughout gestation, pregnancy may not appropriate period for perio tx.

- Maybe **periodontal treatment before pregnancy** may reduce the rates of adverse pregnancy outcomes. Future randomized controlled trials are needed to test if treating periodontal disease in pre-pregnancy period reduces the rate of adverse pregnancy outcomes.

Periodontal disease and adverse pregnancy outcomes

Han, Y; Fardini, Y; Chen, C; Iacampo, K; Peraino, V.; Shamonki, J; Redline, RW.
Term Stillbirth Caused by Oral *Fusobacterium nucleatum*
Obstetrics & Gynecology 115(2, Part 2) Supplement, February 2010, pp 442-445

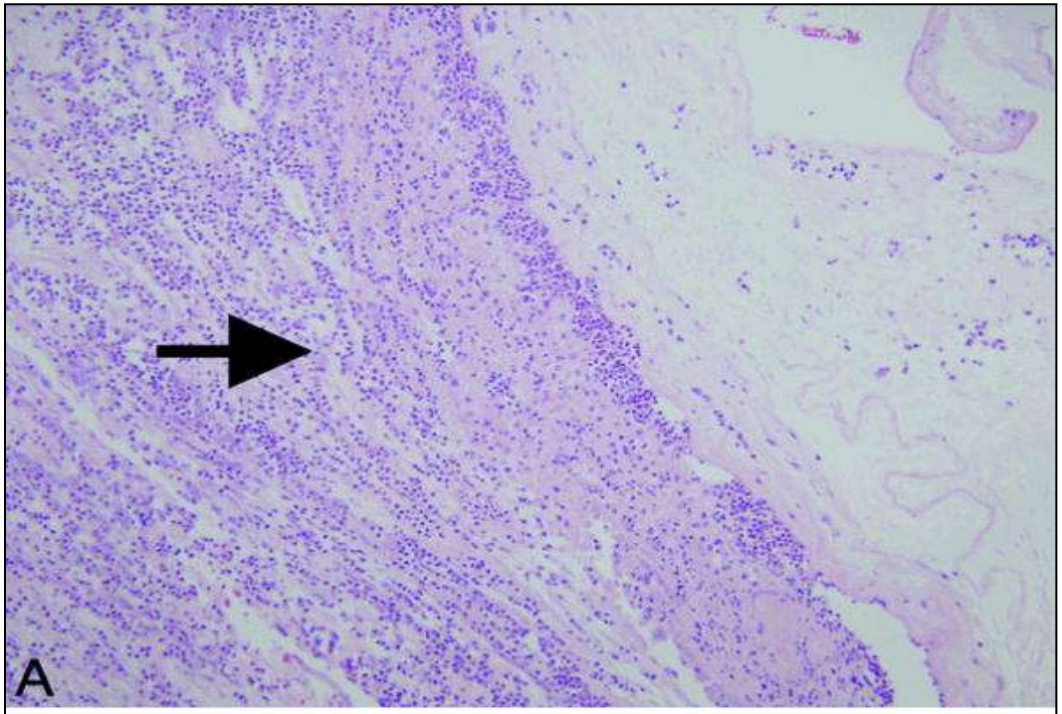
Intrauterine infection is a recognized cause of adverse pregnancy outcome, but the source of infection is often undetermined. We report a case of stillbirth caused by Fusobacterium nucleatum that originated in the mother's mouth.

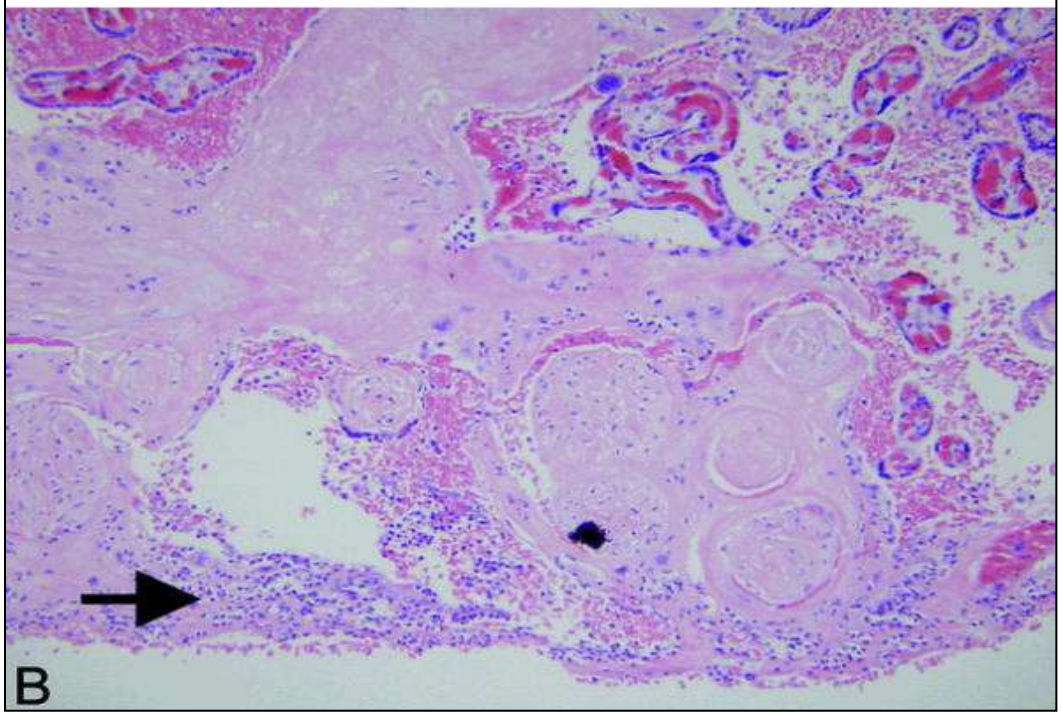
CASE:

A woman with pregnancy-associated gingivitis experienced an upper respiratory tract infection at term, followed by stillbirth a few days later. F. nucleatum was isolated from the placenta and the fetus. Examination of different microbial floras from the mother identified the same clone in her subgingival plaque but not in the supragingival plaque, vagina, or rectum.

CONCLUSION:

F. nucleatum may have translocated from the mother's mouth to the uterus when the immune system was weakened during the respiratory infection.





Term Stillbirth Caused by Oral Fusobacterium nucleatum.
Han, Yiping; Fardini, Yann; Chen, Casey; DDS, PhD; Iacampo, Karla; Peraino, Victoria; Shamonki, Jaime; Redline, Raymond

Obstetrics & Gynecology. 115(2, Part 2) Supplement:442-445, February 2010.
DOI: 10.1097/AOG.0b013e3181cb9955

Accentuated decidual inflammatory response in acute chorioamnionitis caused by F. nucleatum.

A. Decidua capsularis: there is an unusually extensive and intense neutrophilic infiltrate in the decidua capsularis (arrow).

B. Decidua basalis: focus of acute deciduitis in the decidua basalis underlying the intervillous space (arrow). These foci are rarely seen in typical cases of acute chorioamnionitis.

Parodontitis &
Untergewichtige Frühgeburten

Parodontal erkrankte Frauen, die eine Schwangerschaft planen, sollten eine Erkrankung vorzeitig behandeln lassen, um diese möglichst zu vermeiden.

Diese Strategie hat den zusätzlichen Vorteil, dass in der für Mutter und Fötus heikelsten Zeit weniger aktive Behandlungen durchgeführt werden müssen.

Parodontitis &
Untergewichtige Frühgeburten

Jede schwangere Frau sollte aus Gründen einer möglichen ungünstigen Beeinflussung ihrer Schwangerschaft auf parodontale Erkrankungen untersucht werden.

Bei bestehender Parodontitis muss diese und kann auch gefahrlos in der Schwangerschaft behandelt werden.

Parodontitis als Risiko für andere Erkrankunkungen

- **Kardiovaskuläre Erkrankungen**
- **Diabetes mellitus**
- **Untergewichtige Frühgeburten**
- **COPD, Pneumonie, CKD, RA, CI, Fettsucht,**

  Metabolisches Syndrom, Cancer,

1st Joint EFP/AAP Workshop on Periodontal and Systemic Diseases, LaGranja 11/1212

Journal of Clinical Periodontology and Journal of Periodontology Feber/March 2013

Comparative microbiological analyses of biofilms in patients with prosthetic joint infections (PJI) and periodontitis.

V. Clar, A. Heschl, M. Haas , H. Clar, R. Windhager, R. Persson, G. Wimmer

Medizinische Universität Graz

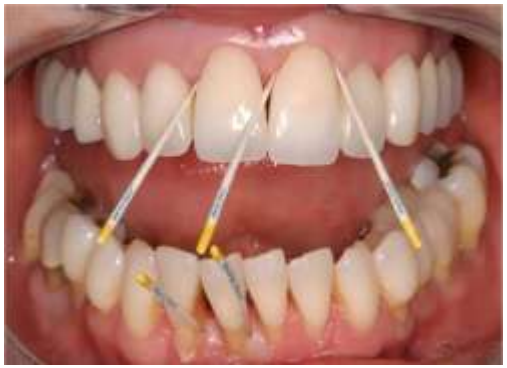


Figure 1. microbiological elevation with paper points in the mouth

Figure 2. microbiological elevation with paper points of the prosthesis

Patient 5 f / 75 tumor + HTEP

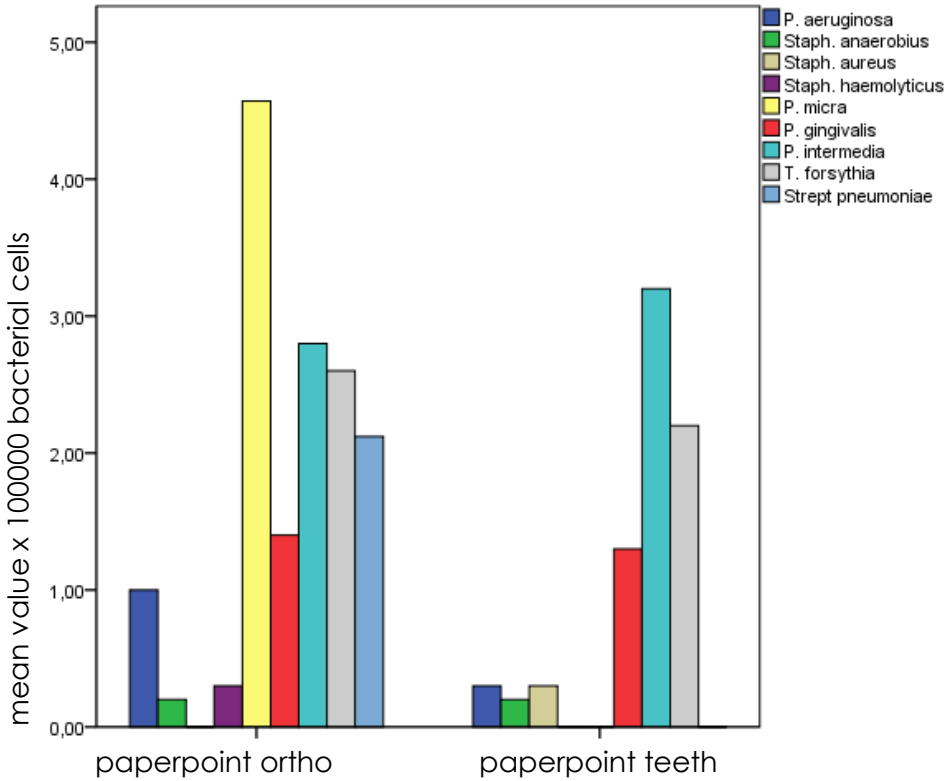
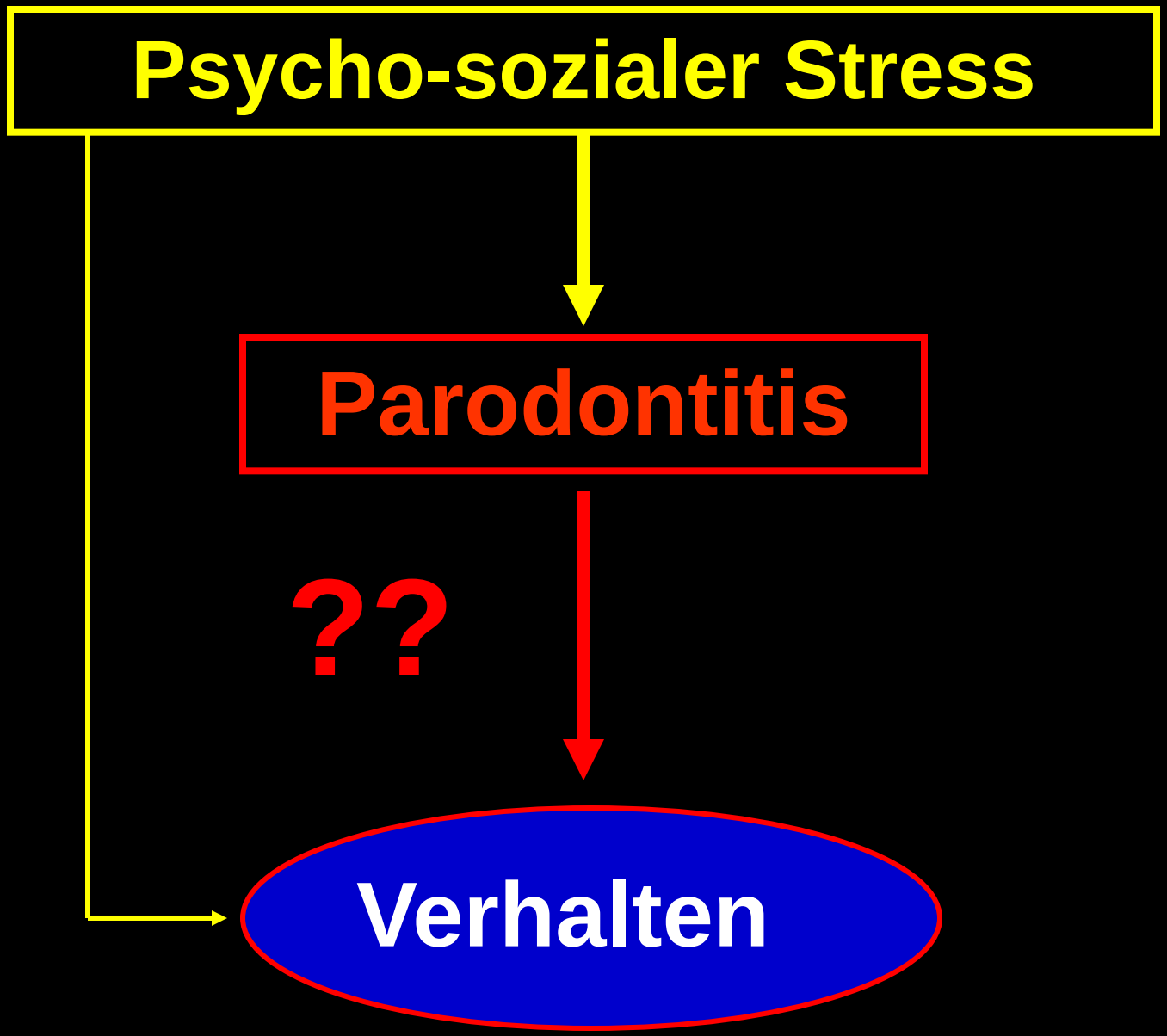
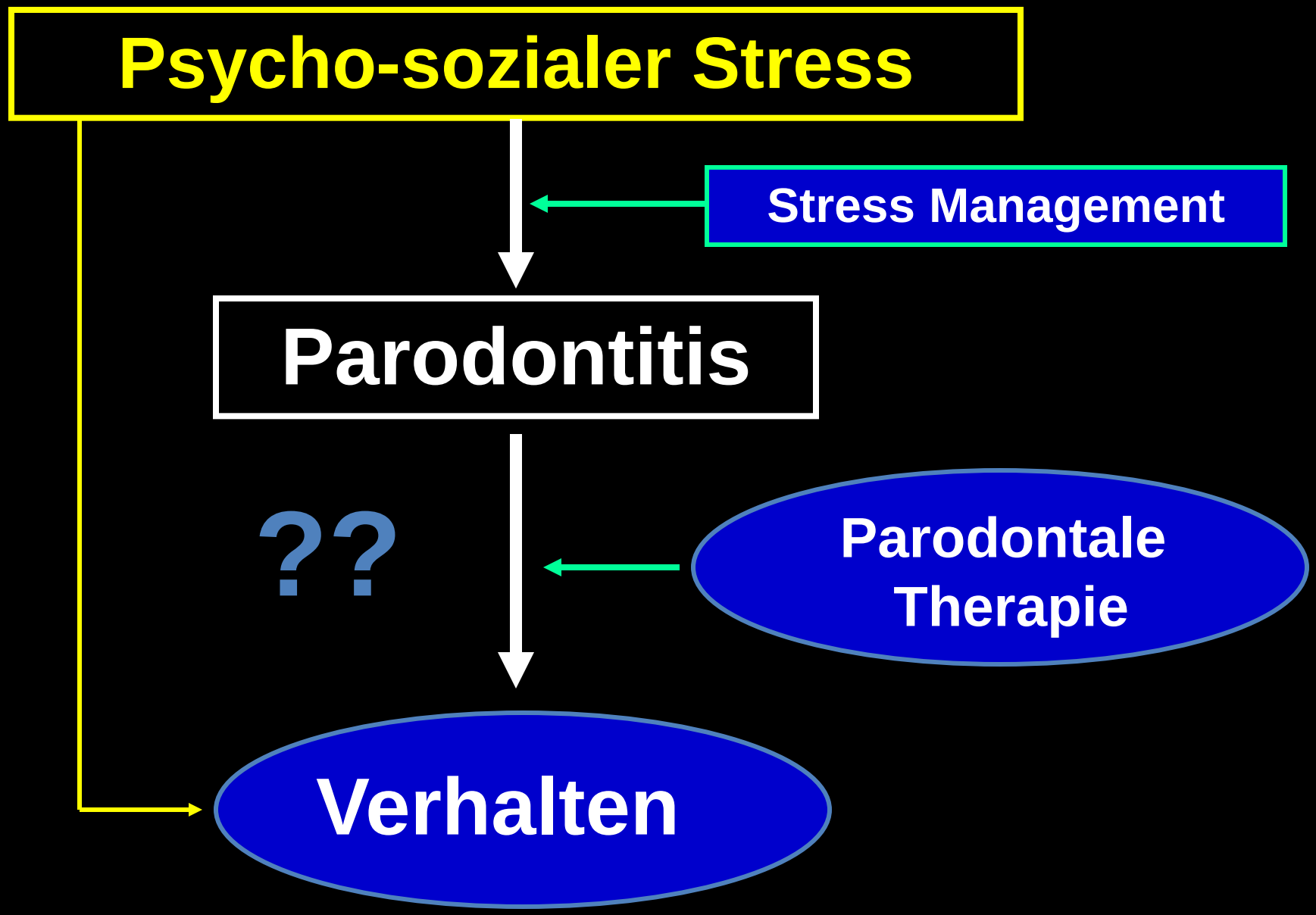


Figure 6. Identification of bacteria on endoprothesis and periodontal pocket in patient 5 (strong correlation)


Psycho-sozialer Stress
Parodontitis
??
Verhalten


Psycho-sozialer Stress
Stress Management
Parodontitis
??
Parodontale Therapie
Verhalten

Parodontale Medizin

G. Wimmer, G. Wieselmann

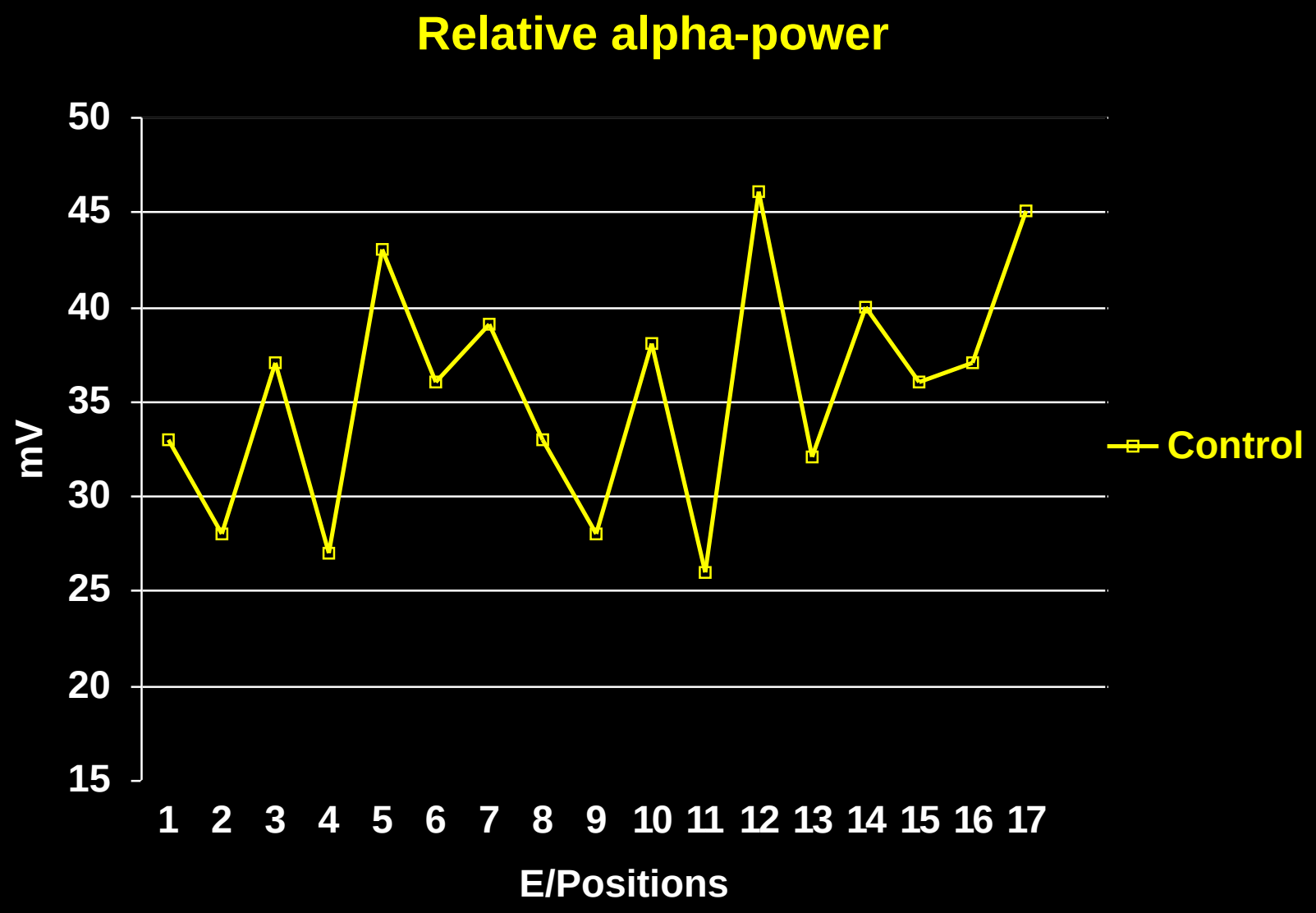
Electro-bio-physiological investigations on the influence of periodontal diseases to the central nervous system.

In prep. for Journal of Dental Research

Medical University Graz

Electroencephalogram (EEG)

30 Patients, 30 Controls before and after non-surg. Perio-Therapy



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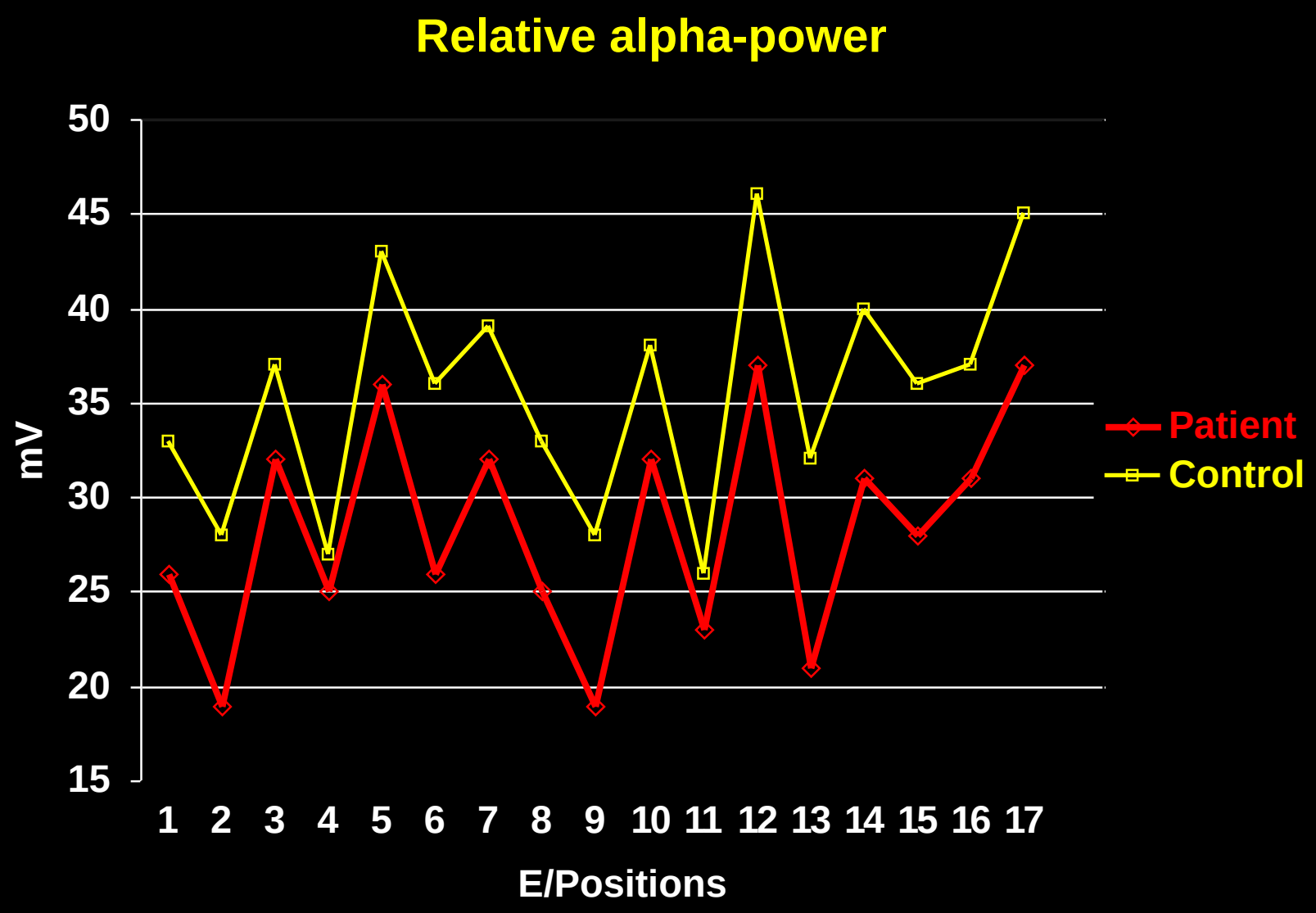

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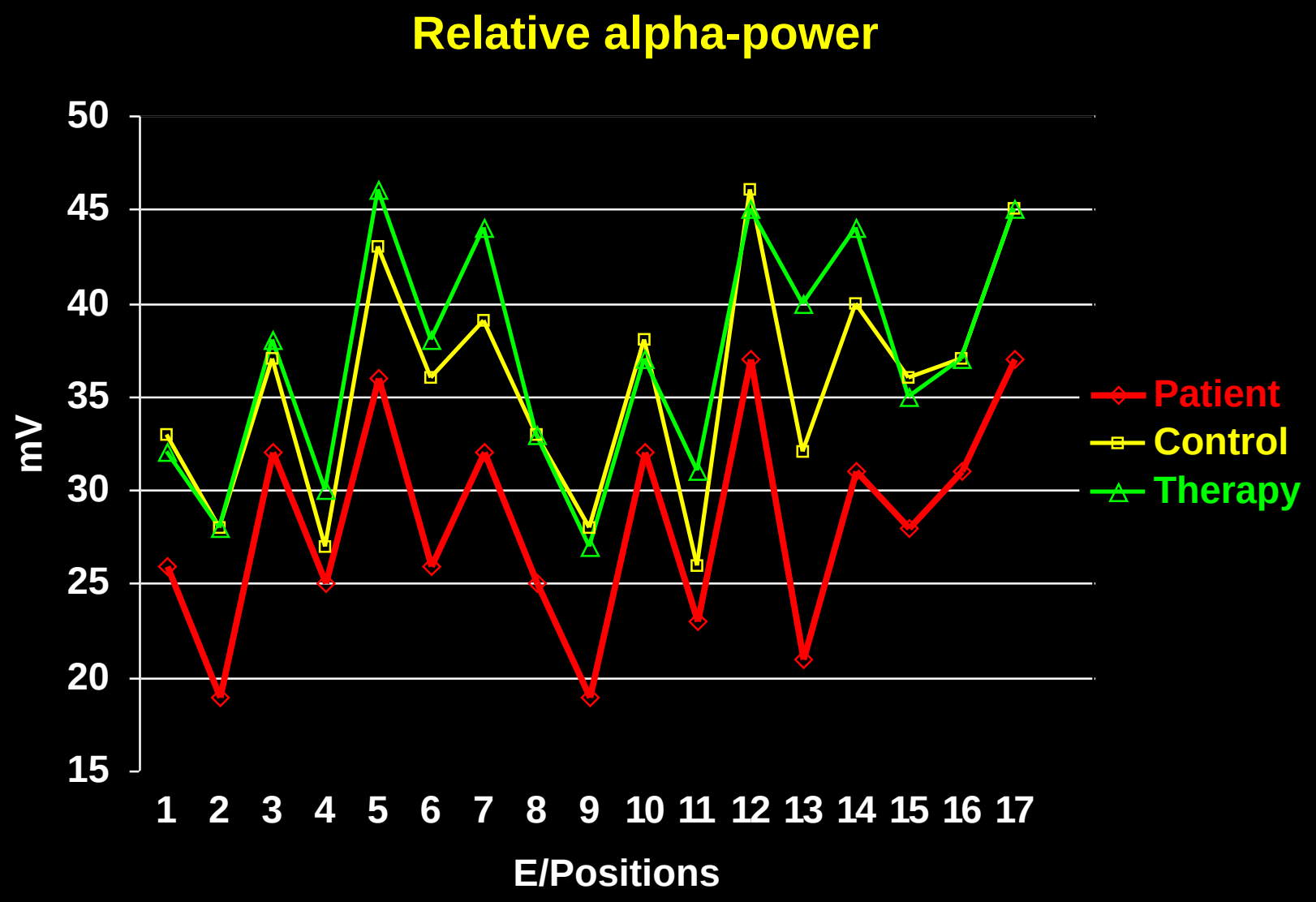

Parodontitis & Mortalität

Personen mit Parodontalerkrankungen haben ein erhöhtes Risiko für verschieden **medizinische Komplikationen**.

Chronische orale Infektionen können wichtige **systemische Langzeitschäden** nach sich ziehen zu können.

Auch wenn vorliegende Studienergebnisse signifikant sind, geben sie noch keinen Beweis für die Verknüpfung von Parodontitis und „vorzeitiger Sterblichkeit“. Auch kann zur Zeit bei anderen Verbindungen nicht von Kausalität gesprochen werden, jedoch sind die bestehenden Beweise für eine **existierende Beeinflussung** überzeugend!

Danke für Ihre Aufmerksamkeit!