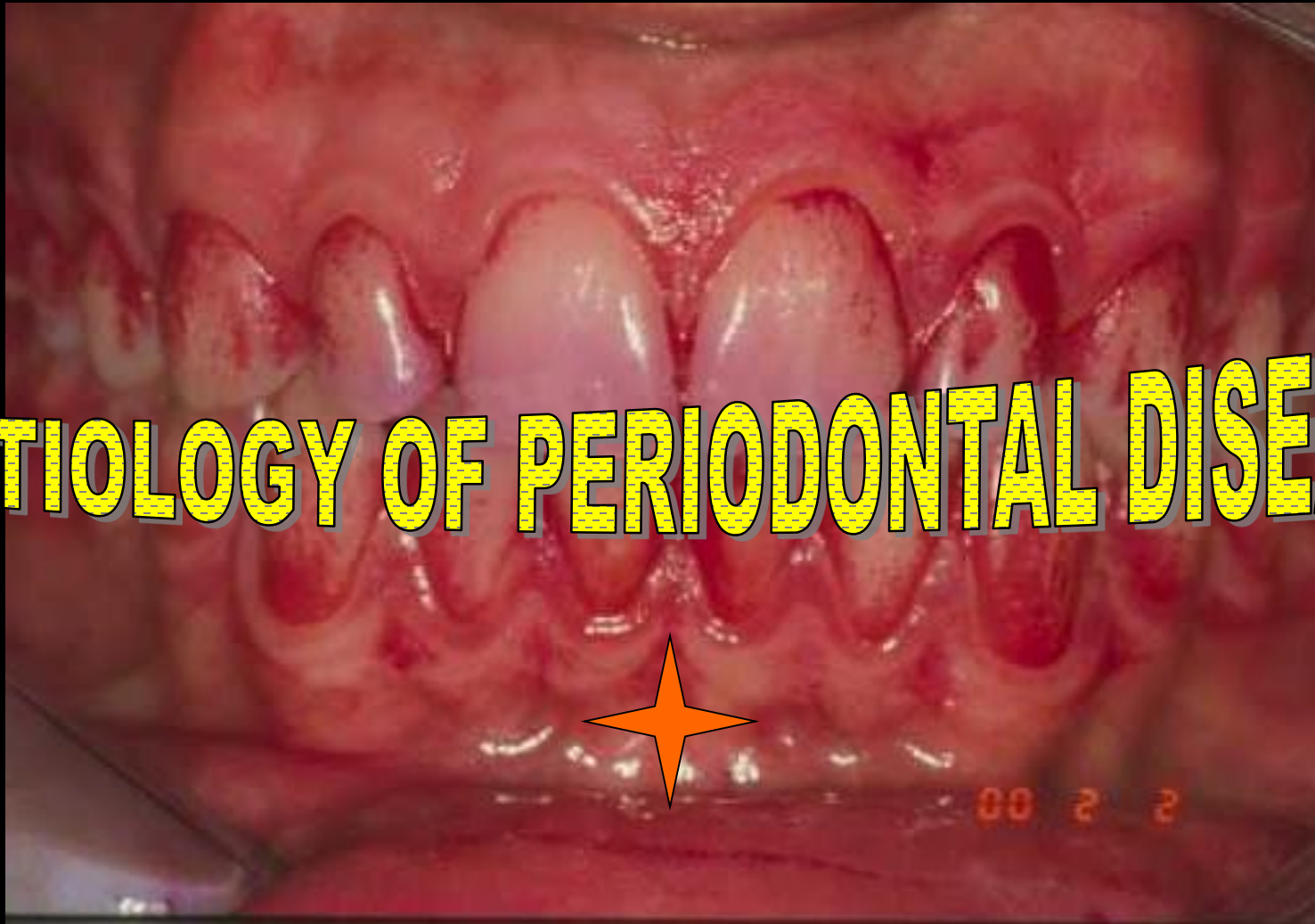


**DENTAL PLAQUE - THE MAJOR ETIOLOGIC FACTOR
OF ALL KIND OF PERIODONTAL DISEASES**

ETIOLOGY OF PERIODONTAL DISEASE







WHY DEVELOPS GINGIVITIS OR PERIODONTITIS???



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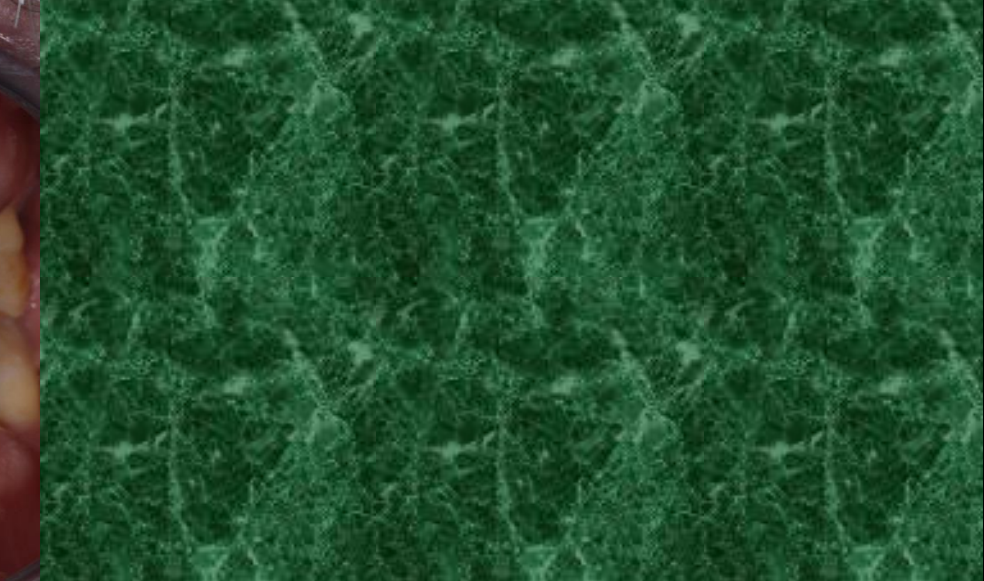
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PLAQUE FORMATION

Gingivitis/periodontitis are hardly exogenous infections

The normal indigenous bacterial flora is the source of the "endogenous" infection.

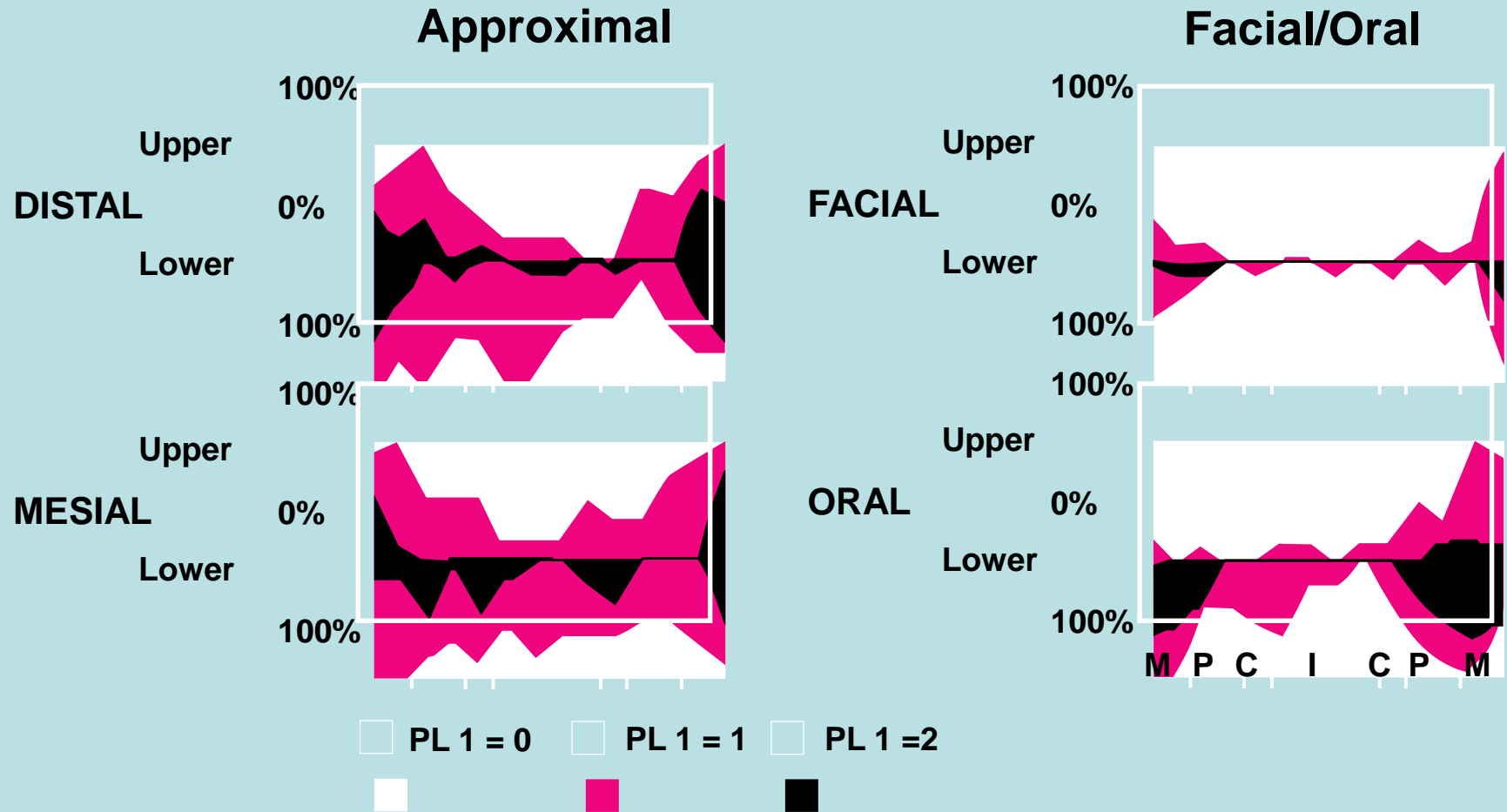
It is a true opportunistic infection

PLAQUE FORMATION

Dental plaque accumulation starts supragingivally but later the bacteria spread into the gingival sulcus and into the pathologically deepened sulcus - ie. periodontal pocket.

The composition and morphology of the subgingival plaque are totally different from the supragingival one.

Plaque distribution after 12 hours of no oral hygiene



THE MECHANISM OF PLAQUE ACCUMULATION

Initially bacteria binds to pellicle by physico-chemical forces (ionic bonds, Van der Waals force). Later bacteria bind to pellicle by protein-protein and protein - carbohydrate interactions.

Several virulence factors can enhance their adhesion. The natural bacterial glycoprotein coat (glycocalix), contains a great amount of polysaccharides that many times organized in surface fibrils.

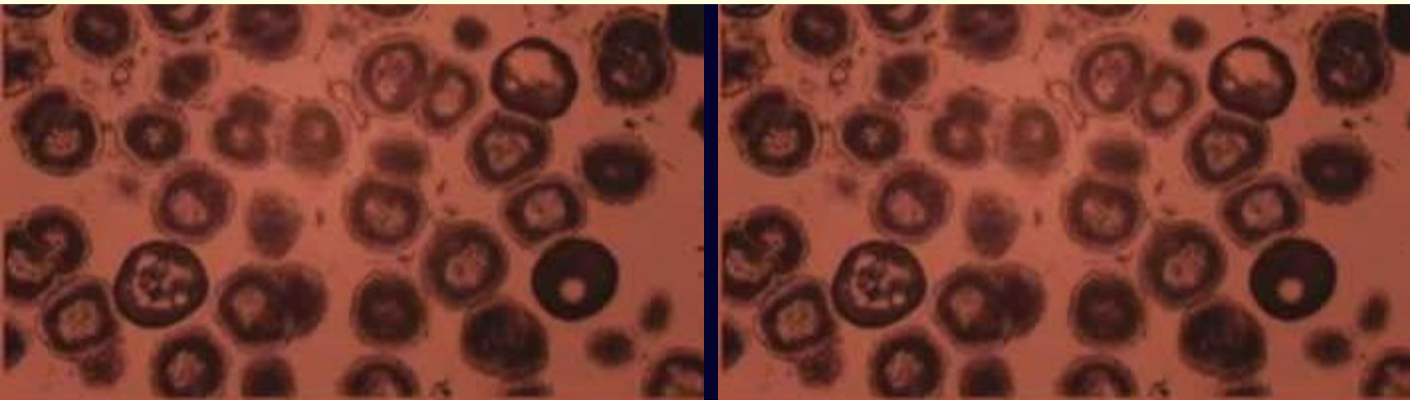
Certain bacteria can synthesize extracellular polysaccharides by glycosyl transferase enzyme.

De Jong HP, DeBoer P, Busscher HJ et al.: Surface free energy changes of human enamel during pellicle formation: an in vivo study Caries Res 18: 408-415. 1984.

THE MECHANISM OF PLAQUE ACCUMULATION

The non-soluble extracellular polysaccharides form the interbacterial matrix of the dental plaque while the soluble part serves as energy reservoir.

The attached bacterial mass grows very rapidly partly by cell division partly bacterial coaggregation



THE MECHANISM OF PLAQUE ACCUMULATION

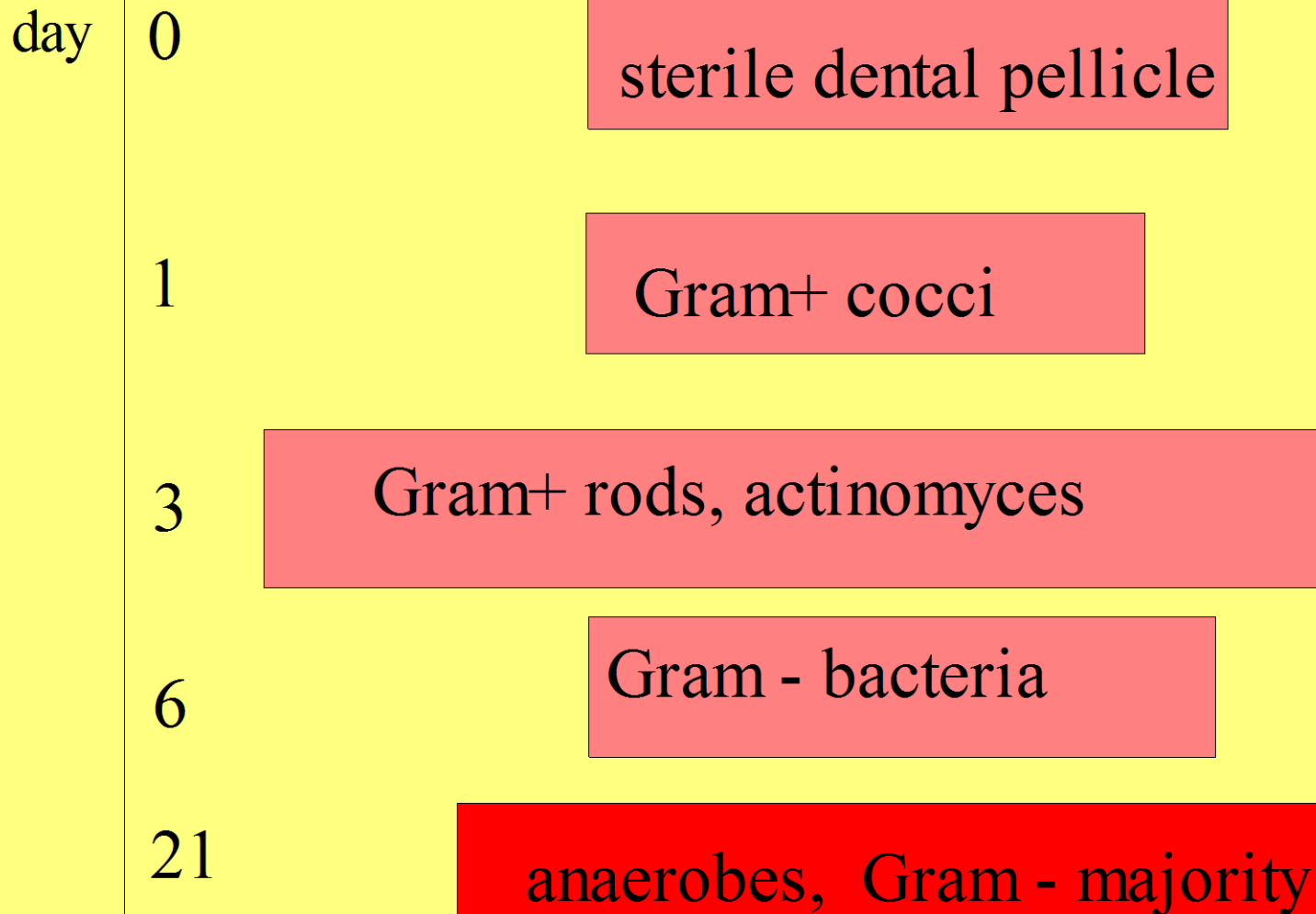
In the depth - anaerobes

on the surface - aerobe or facultative anaerobes

In the depth bacteria cannot utilize nutrients originating from the saliva they use the sulcular fluid and blood as energy source.

In the depth of the sulcus no saccharolytic bacteria occur.

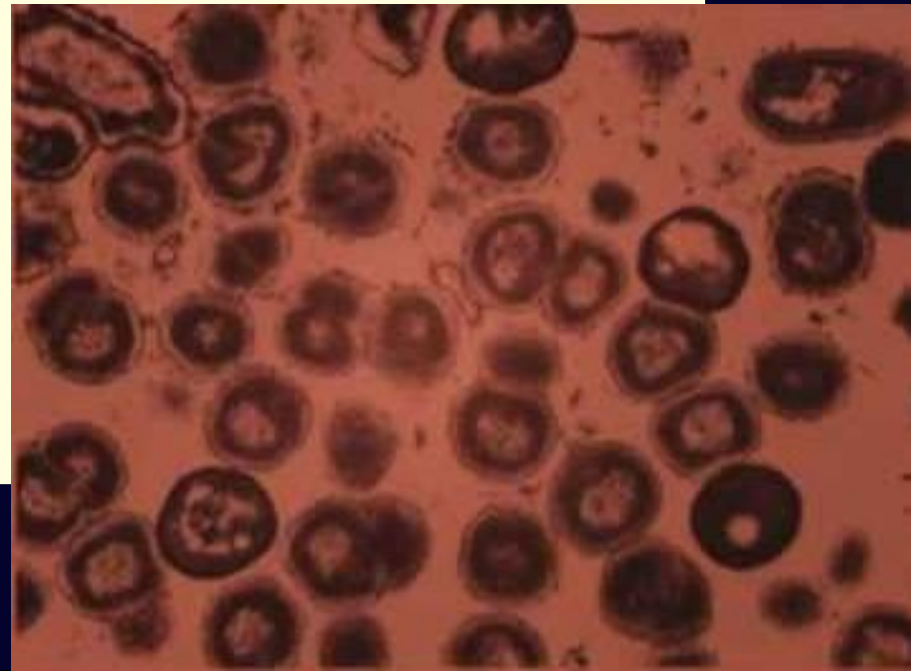
THE MECHANISM OF PLAQUE ACCUMULATION



Dental plaque as a biofilm

The nutrients diffuse through the soluble interbacterial matrix into the depth of the biofilm.

In the biofilm a well defined oxygen and nutrient gradient develops from the surface downwards into the depth.

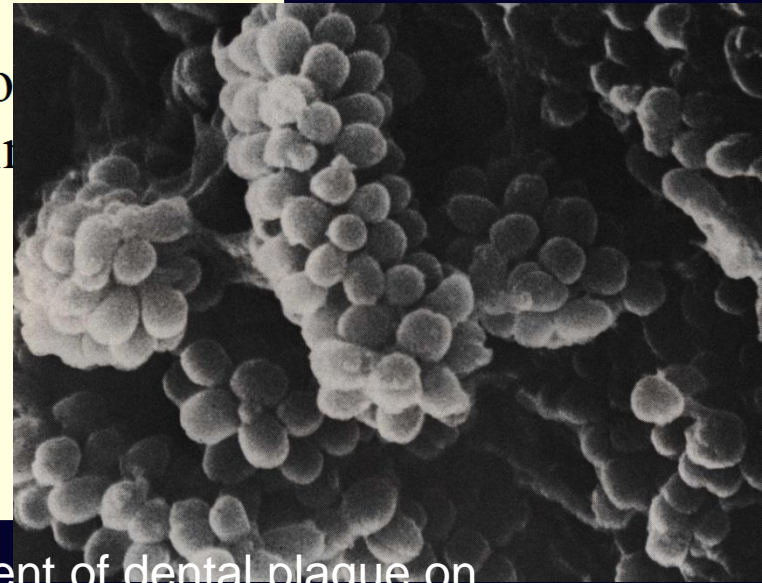


Supragingival plaque

When Van Lavenhook in the 17th century fabricated his first microscope and discovered light microscopy he first investigated and described dental plaque.

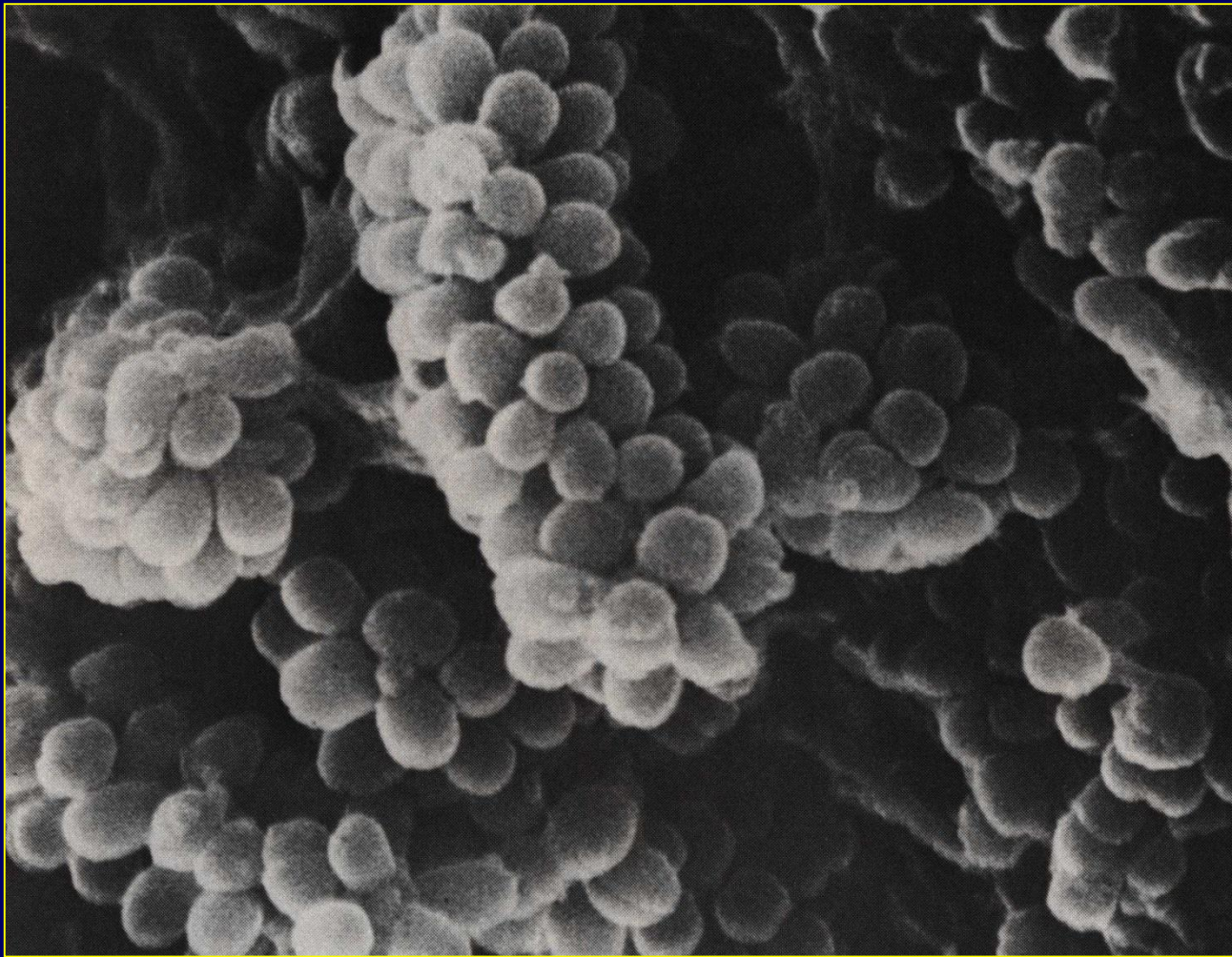
The ultrastructure the light and EM microscopy of plaque is well defined and varies in different clinical stages and maturation stages.

Corn-cob formation



Listgarten MA, Mayo HE, Tremblay R.: Development of dental plaque on epoxy resin crowns in man. A light and electron microscopic study J Periodontol 46: 10-26. 1975,

Listgarten MA: Structure of the microbial flora associated with periodontal health and disease in man. A light and electron microscopic study J Periodontol 47: 1 -18. 1976,



Corn-cob formation

Subgingival plaque

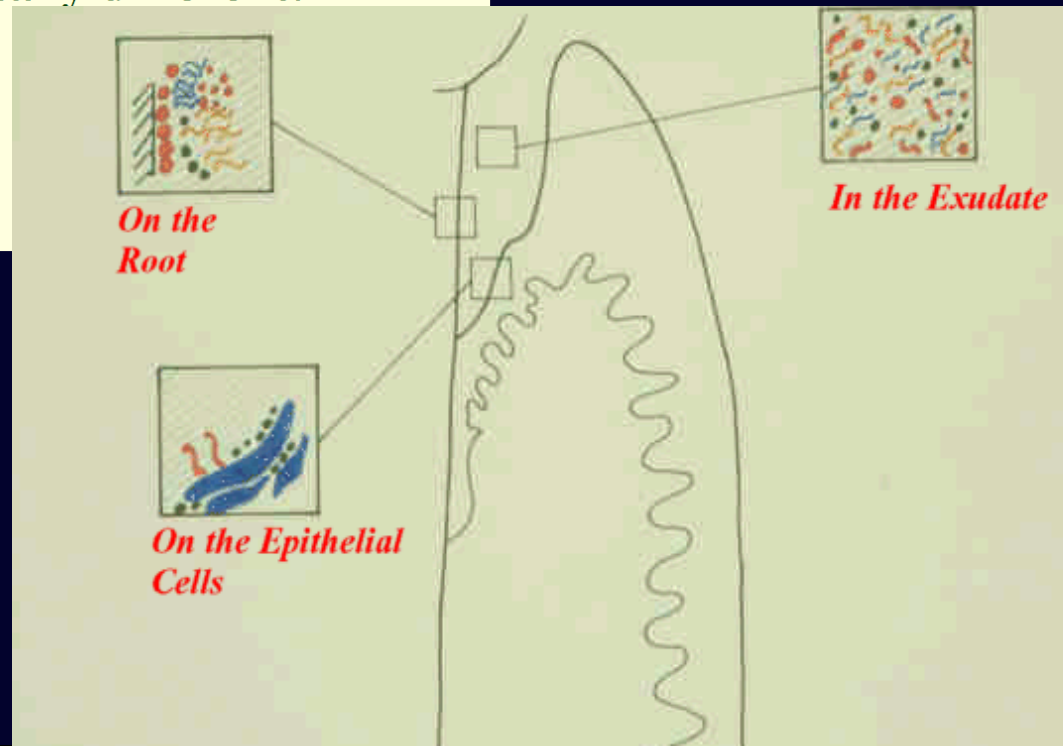
Subgingivally can be distinguished :

hard tissue associated plaque

dispersed bacteria in the sulcular fluid

epithelia associated plaque

The composition of subgingival dental plaque in superficial and deep pockets are totally different.



Subgingival plaque

In gingivitis the subgingival plaque is the simple continuation of the supragingival plaque

Its structure is similar to the supragingival plaque but its bacterial composition is substantially different.

Subgingival plaque

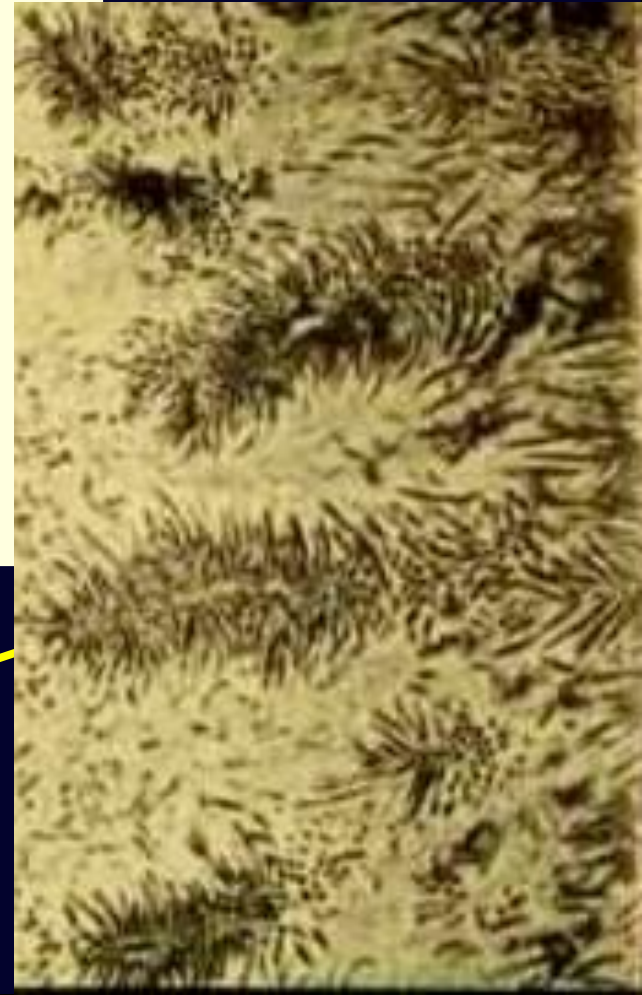
Deep periodontal pockets:

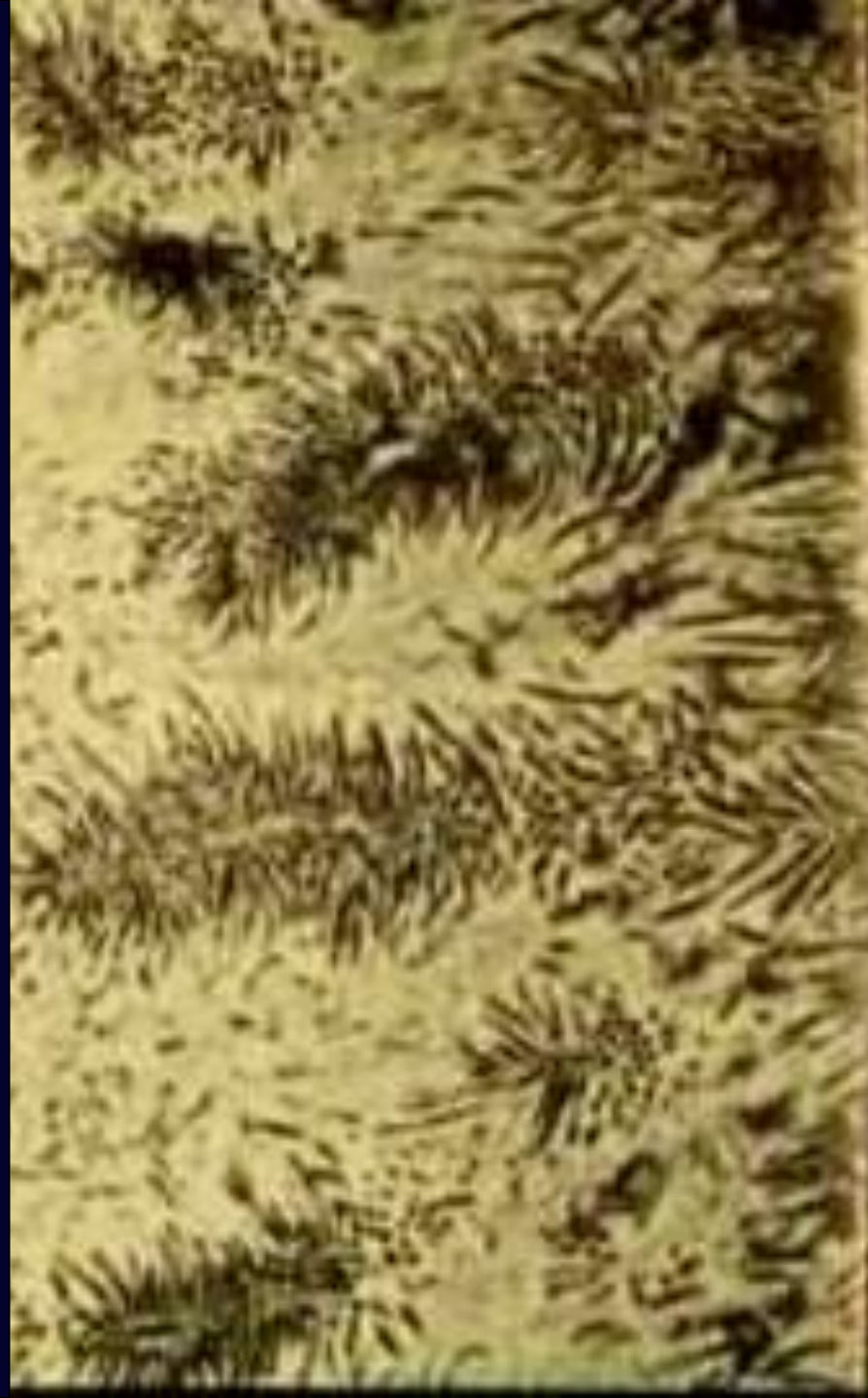
Anaerobe filaments, few cocci and rods numerous spirochaetes The subgingival plaque is predominantly motile.

Well organized bacterial colonies and bacterial configurations

Characteristic subgingival plaque formations - **" test tube brush formation"** , in which a central filament is coated by a mass of anaerobe rods.

Testube brush formation





Subgingival plaque

In the bottom of the pocket the plaque organization is not so complex. The filaments are missing, the majority of plaque is built up by small densely packed anaerobic bacteria without any characteristic organization.

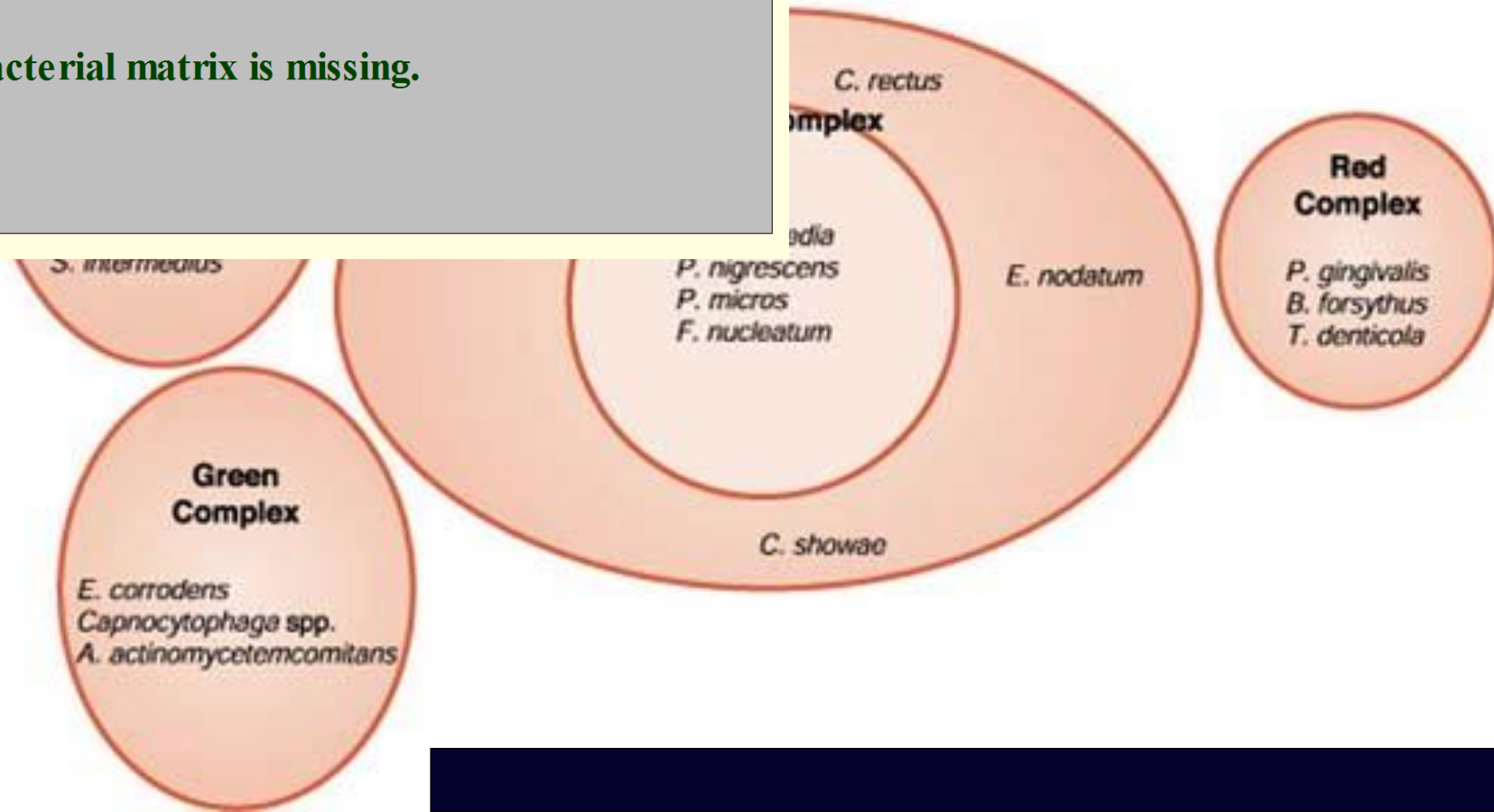
1. *Listgarten MA: Structure of the microbial flora associated with periodontal health and disease in man. A light and electron microscopic study J Periodontol 47: 1 -18. 1976,*

Subgingival plaque

The structure of the soft tissue associated plaque is totally different from the hard tissue associated one. The main component are spirochaetes and, other motile flagellated bacteria.

There are numerous cocco-bacilli and short rods.

The interbacterial matrix is missing.



Dental calculus

Normally develops from the adherent old dental plaque after its mineralization. Its formation is totally dependent on the individual oral hygiene.

Nevertheless some calculus formation was detected in gnatobiotic animals under sterile conditions.

Similarly to dental plaque, also supragingival and subgingival calculus can be distinguished.



Dental calculus

Supragingival calculus

Develops coronally from the gingival margin. Its color is white or yellow. But tobacco smoking or other dietary pigments can create brownish shade.







Dental calculus

Subgingival calculus

Subgingival calculus never develops in healthy, non inflamed sulcus.

Though it plays an important role in the development and sustain of inflammation, it is rather considered as a symptom of inflammation than the primary etiologic factor.



Dental calculus

Subgingival calculus

Many times it can be detected by naked eyes.
If the gingiva is thin the dark grey calculus can be seen as a blue discoloration on the gum line.

The superficial subgingival calculus can be visualized by pressing down the gingival margin or by blowing air from air syringe into the sulcus.



Dental calculus

Subgingival calculus

Its mineral content originates from the sulcular fluid and from the serum.

The is no predilection for its deposition. It occurs in every periodontal pockets, extending from the cemento-enamel junction apically to the bottom of the pocket.

Dental calculus

Subgingival calculus

The calculus, especially the old one attaches to the tooth very hard.

Subgingivally also the acquired dental pellicle is mineralized.

Subgingivally the adhesion of calculus is also enhanced by the surface irregularities



Dental calculus

ITS CLINICAL RELEVANCE

The calculus per se is not a primary etiologic factor.

In the practice the calculus deposition is always an important factor in the development and especially in the sustain of destructive periodontitis.

Dental calculus always develops on those surfaces where the plaque can accumulate undisturbed.

It is a plaque retentive factor. Its surface is always covered by fresh bacterial plaque and it can also guide the plaque bacteria subgingivally







The chemical composition of dental calculus

The recent and old calculus consists of four different crystals of calcium phosphate

1. $\text{CaH}(\text{PO}_4) \times 2\text{H}_2\text{O}$ = Brushite (B)
2. $\text{Ca}_4\text{H}(\text{PO}_4)_3 \times 2(\text{H}_2\text{O})$ = octa calcium phosphate (OCP)
3. $\text{Ca}_5(\text{PO}_4)_3 \times \text{OH}$ = Hydroxyapatite (HA)
4. $\beta\text{-Ca}_3(\text{PO}_4)_2$ = tricaciumphosphate Whitelockte



Microbiology of periodontal disease

More than 700 bacterial species can be cultured

One single tooth can harbor 10^9 microorganisms

The sulcus is not sterile.

The healthy, shallow sulcus contains at least 10^3

The inflamed pocket might harbor at least 10^8 microorganisms



Culture of subgingival sample

LÖE CLASSIC EXPERIMENTAL GINGIVITIS STUDIES



DENTAL PLAQUE

GINGIVITIS

PERIODONTITIS



WHY NOT NECESSARILY ALL GINGIVITIS PROGRESSES TO DESTRUCTIVE PERIODONTITIS????

DENTAL PLAQUE

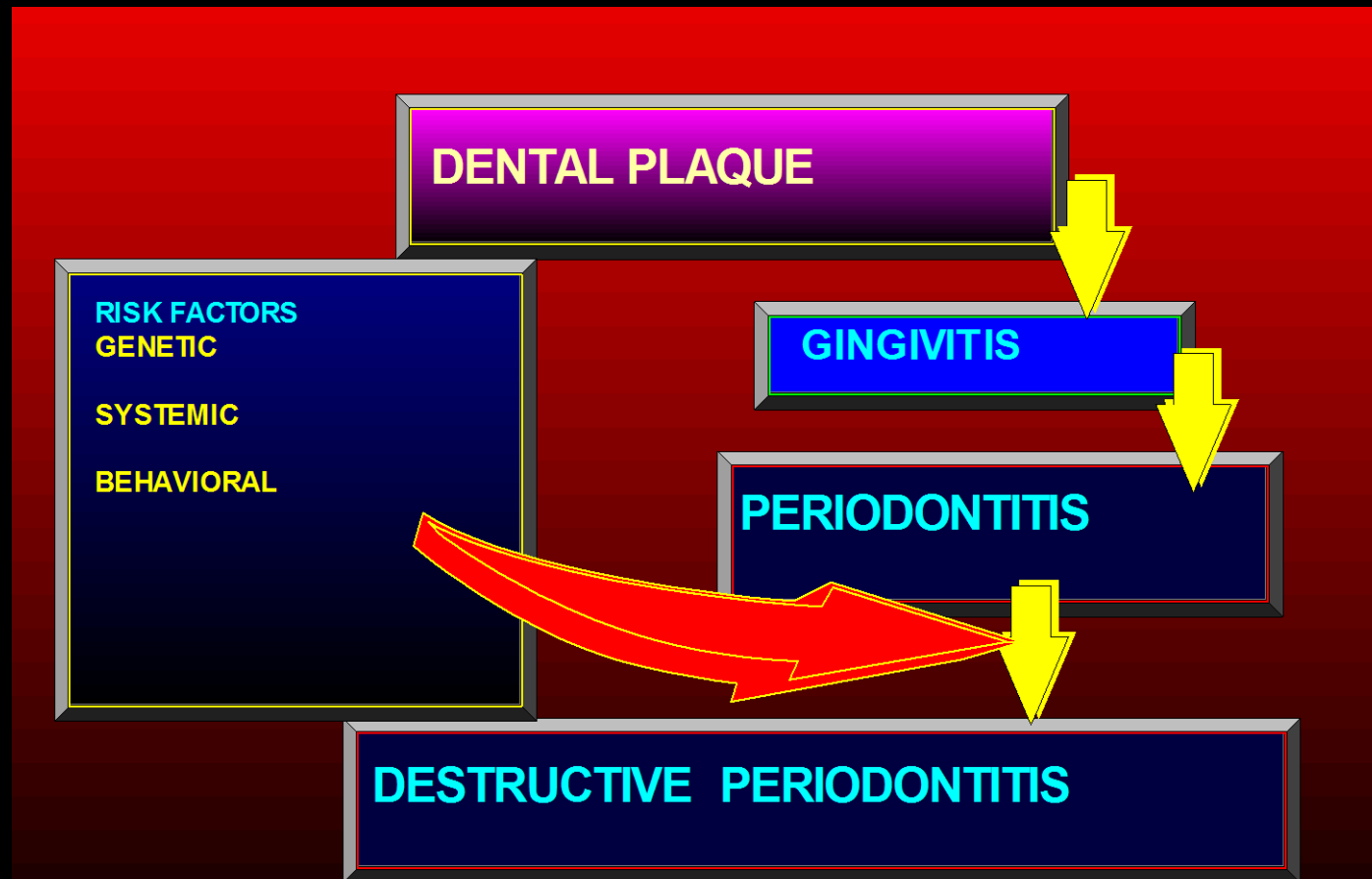


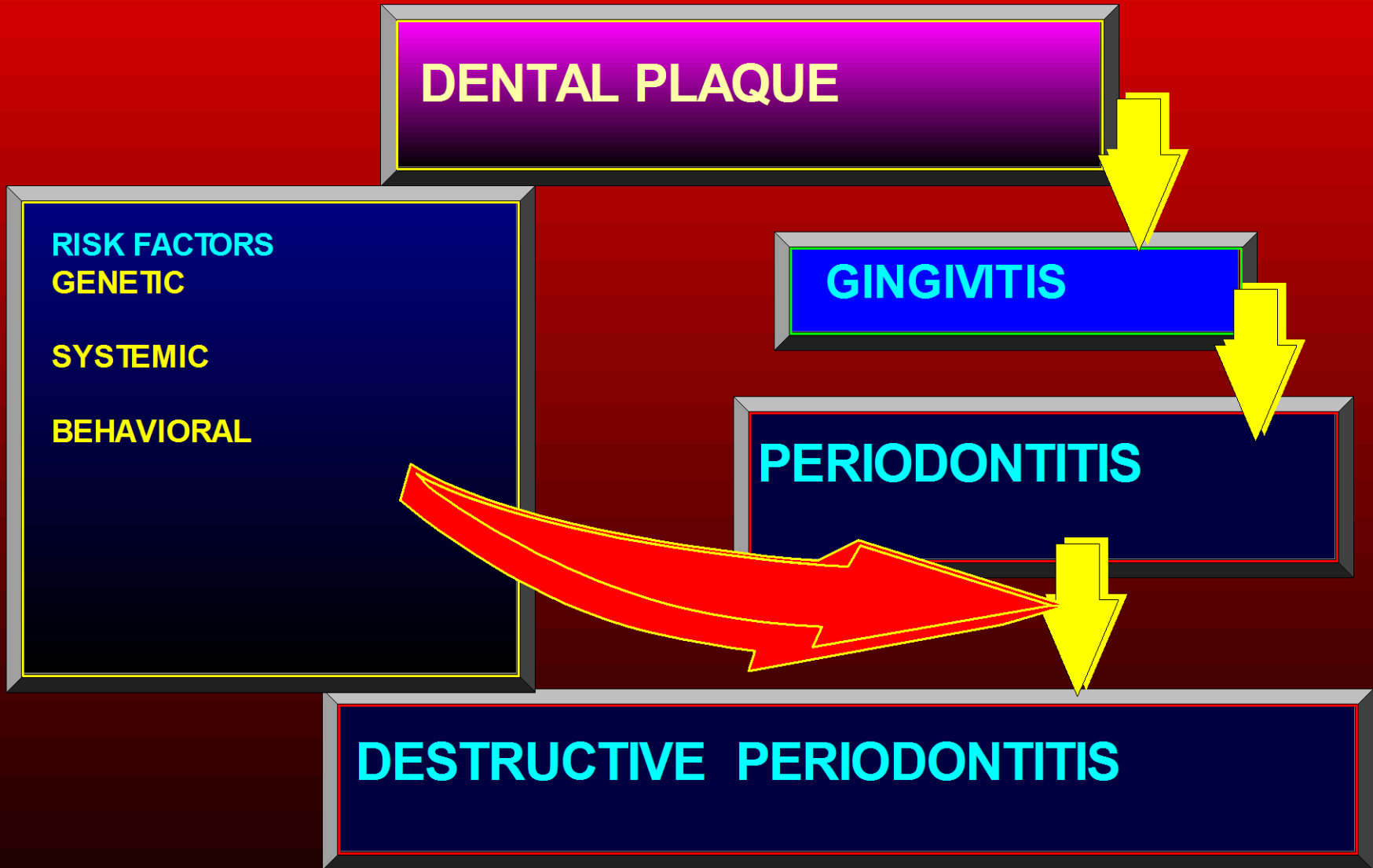
GINGIVITIS



PERIODONTITIS

DENTAL PLAQUE IS NECESSARY BUT NOT SUFFICIENT ETIOLOGIC FACTOR OF DESTRUCTIVE PERIODONTITIS





RISK FACTORS:

GENETICS

1(IL-1)

TNF

SYSTEMIC

ENDOCRINE

CARDIOVASCULAR

IMMUNOLOGICAL

BEHAVIORAL

STRESS

DIET

SMOKING

WAY OF LIFE

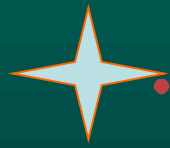
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ETIOLOGIC FACTORS

- Oral hygiene
 - Local plaque retentive factors
 - bacterial specificity
 - systemic immune status
 - Diabetes mellitus
 - Tobacco smoking
 - Osteoporosis
 - Ethnic background
 - Age
 - Diet
 - Genetics
- Stress
 - Socio-economics

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**SEVERAL SYSTEMIC
AND LOCAL
MODIFYING
FACTORS**





**HEAVY
SUPRAGINGIVAL
DENTAL CALCULUS
DEPOSITION
MECHANICALLY
SEPARATING FRONT
TEETH**



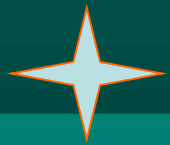


**HEAVY SUPRAGINGIVAL
PLAQUE AND DENTAL
CALCULUS DEPOSITION**





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FAULTY RESTAURATIONS



LOCAL PLAQUE RETENTIVE FACTORS

ETIOLOGIC FACTORS

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**LARGE MASS OF SUPRAGINGIVAL
PLAQUE AND CALCULUS**

THE SUBGINGIVAL PLAQUE IS TOTALLY
INDEPENDENT OF THE
SUPRAGINGIVAL ORAL ENVIRONMENT
FORMS A BIOFILM, THAT CAN ONLY BE
REMOVED BY PROFESSIONAL
MECHANICAL DEBRIDEMENT



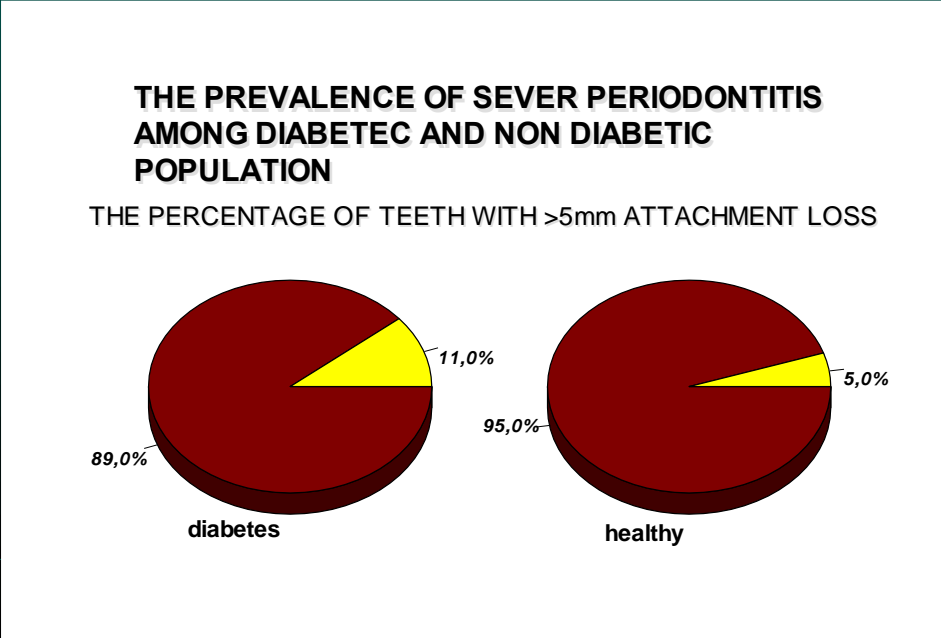
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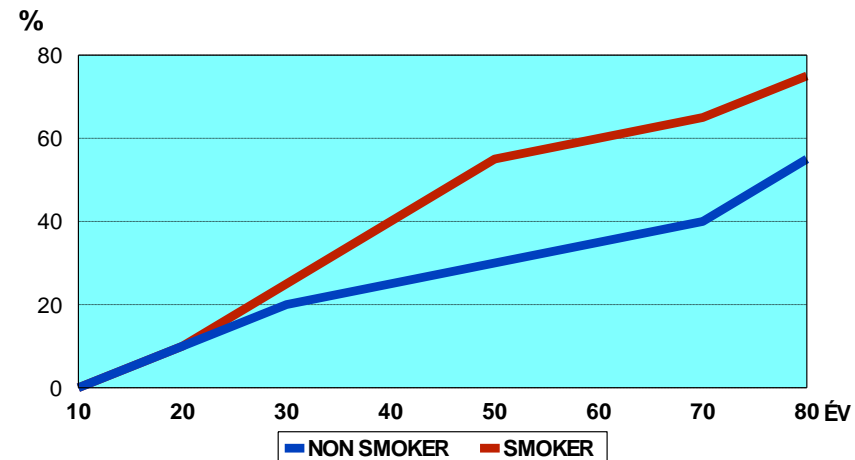


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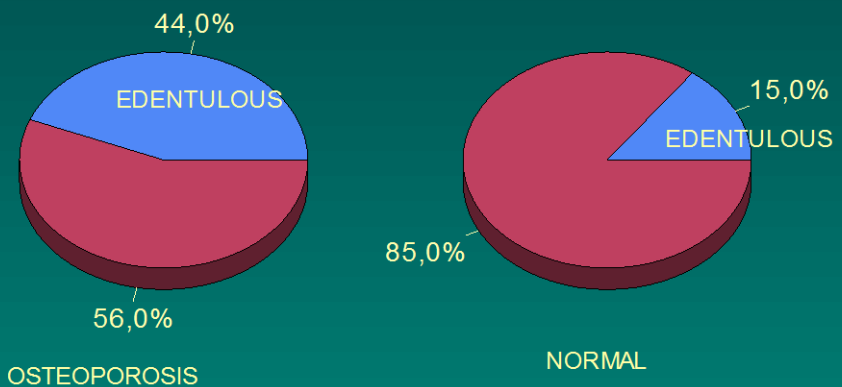
THE RATE OF ALVEOLAR BONE LOSS RELATED TO THE AGE



ETIOLOGIC FACTORS

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TOTAL EDENTULOUSNESS IN OSTEOPOROTIC AND NORMAL AGE MATCHED POPULATION



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THERE IS NO
CORRELATION
BETWEEN LOCAL
ETIOLOGIC FACTORS
AND THE SEVERITY
OF TISSUE
DESTRUCTION



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CPITN scores in Hungary in 1985 and 1991.

WHO pathfinder studies

CPITN scores	12 year old		35-44 year old	
	1985	1991	1985	1991
Deep pocket (CPITN 4)	0,1	0	8	2,3
3-5mm Pocket (CPITN 3)	4,1	0	26,4	15,41
Calculus (CPITN 2)	30,9	30,1	50,8	71,3
Initial gingivitis (CPITN 1)	38,5	30,8	7,6	6,8
Healthy (CPITN 0)	26,2	39,1	5	4,3

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**VERY SEVERE
ALVEOLAR BONE LOSS**

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Many studies have indicated that lower income groups have a much higher prevalence of gingivitis and periodontitis than people living on a much higher living standards *(Oliver et al. 1998, Micheelis & Bauch 1996)*.

These can be attributed to the inferior oral hygiene, the lack of sophisticated oral hygienic aids, the inferior standards in dental care and the limited access to dental services

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According to a survey from eastern Germany **62,4% of all people** with a qualification equivalent to **junior high school certificate** had advanced periodontitis and none of them had healthy periodontium, while **only 37.7%** of the participants with **university degree** had advanced periodontitis (Mengel et al., 1993)

CARDIOVASCULAR DISEASES

DIABETES MELLITUS

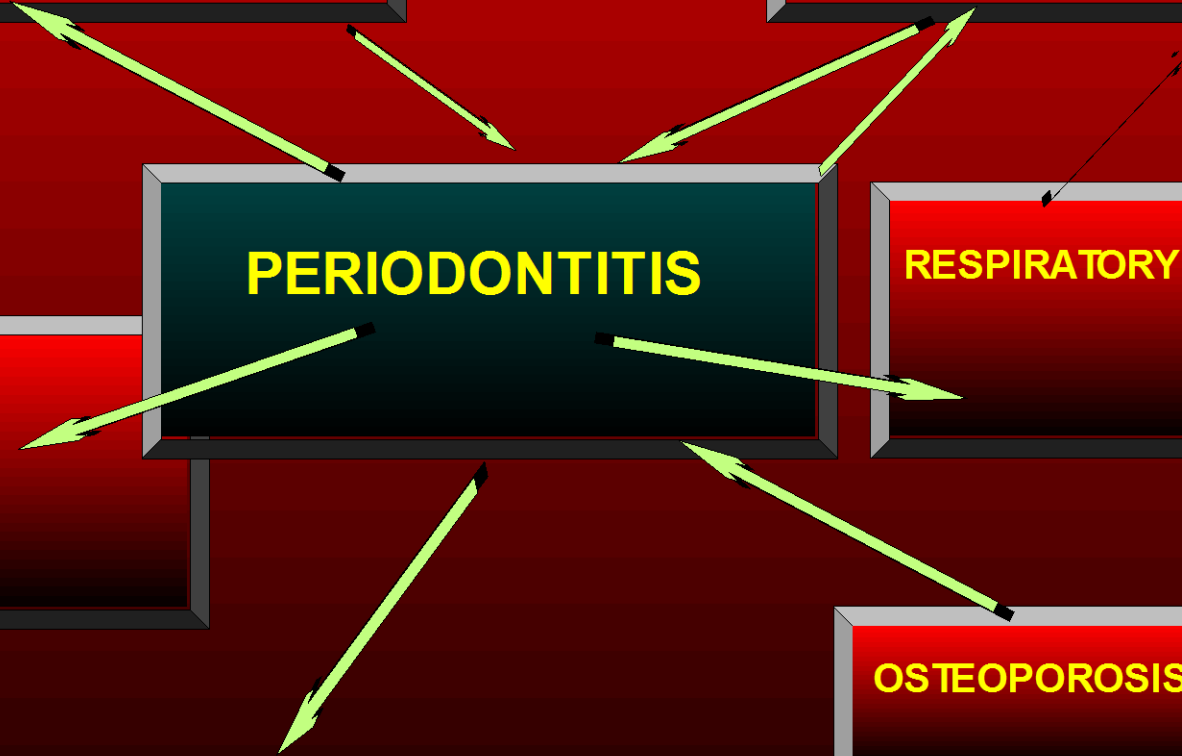
PERIODONTITIS

RESPIRATORY DISEASES

**GASTRO-
INTESTINAL
DISEASES**

OSTEOPOROSIS

PRETERM LW BIRTH



**SEVERAL SYSTEMIC
AND LOCAL
MODIFYING
FACTORS**





**THE INFECTED PERIODONTAL
POCKET CAN BE A DENTAL
FOCUS**

CARDIOVASCULAR DISEASES

DIABETES MELLITUS

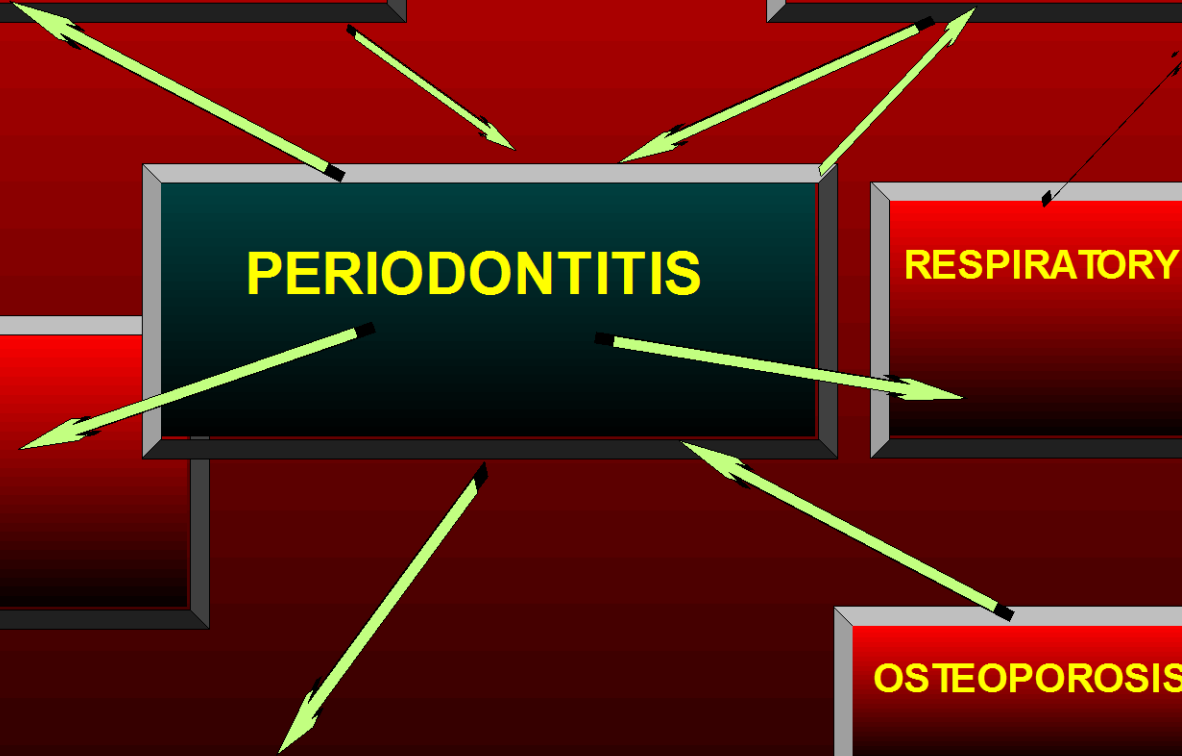
PERIODONTITIS

RESPIRATORY DISEASES

**GASTRO-
INTESTINAL
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PRETERM LW BIRTH





THE MAJOR GOAL
OF ANY CAUSE
RELATED
PERIODONTAL
TREATMENT IS:

TO CLEAN TEETH
AND RESTORE
ORAL HYGIENE

