DENTAL PLAQUE
The oral micro-ecological system
PLAQUE FORMATION

The organism harbour many billions of bacteria on its surfaces. Nevertheless the desquamation of the epithelial cells anticipates the long lasting bacterial coexistence in the body.

In the oral cavity the non shedding surfaces, like enamel, root cementum, restorations can promote permanent, long lasting bacterial adhesion and survival on the surfaces.
PLAQUE FORMATION

dental plaque is a bacterial aggregation that are tenaciously attached to the teeth and other non-sediment features. One mm$^3$-dental plaque contains more than $10^9$ microorganisms.
PLAQUE FORMATION

Löe - experimental gingivitis model (1965), proved that plaque accumulation can lead to gingivitis, and its removal can reverse the disease.

Similarly experimentally was proven that plaque accumulation can cause peri-implantalis kifejlődése (Pontoriero 1994).

Non-specific plaque theory (Theilade 1986)

Only the quantity of bacteria mass is important, and the abundant amount of plaque can cause disease. There are no individual differences between plaques and the healthy and diseased sites show only differences in the quantity of the plaque and not in its composition.

PLAQUE FORMATION

It was difficult to explain:

Why some patients have plaque and calculus accumulation for several years, but they develop only gingivitis without any attachment loss? Others show severe attachment loss with relatively good oral hygiene.

Why the severity and speed of progression differ from tooth to tooth in the same individual or even show different pattern around different surface at the same tooth?
Longitudinal studies showed that the presence of certain bacterial strains in the dental plaque show good correlation with the disease activity and the magnitude of attachment loss. The total eradication of those bacteria can ensure long lasting therapeutic results.

The massive mixed bacterial colony has minimal pathogenic potential to initiate attachment loss. Only certain members of those colony should be eradicate to anticipate the attachment loss.
Those early studies established a base of the so called specific plaque theory (Loesche 1979).

Gingivitis/periodontitis are true infections

But this infection is not a classic infection in terms of the Koch's postulates

Loesche WJ. Clinical and microbiological aspects of chemotherapeutical agents used according to the specific plaque theory. J. Dent Res. 56: 2404-2414. 1979.
Gingivitis/periodontitis are hardly exogenous infections.

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

It is a true opportunistic infection.
To day only a couple of bacterial strains are known that are not detectable in healthy oral environment or they only occur in a very-very low rate. (ie. *Porphyromonas gingivalis, Actinobacillus Actinomycetemcomitans*)

They are very numerous in diseased periodontal pockets.

Those so called "periodonto-pathogenic microorganisms" can only be considered and true exogenous agents.
DENTAL PLAQUE
The oral micro-ecological system
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 FORMATION

SUPRAGINGIVAL PLAQUE

SUBGINGIVAL PLAQUE

HARD TISSUE ASSOCIATED
EPITHELIAL ASSOCIATED
DISPERSED
INVASIVE

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Dental plaque or certain specific bacterial components are necessary but not sufficient etiologic factors for the development of destructive periodontitis.

The complex equilibrium between the oral bacterial ecosystem and the host defensive mechanisms will determine the nature of the disease and the character of its progression.
THE MECHANISM OF PLAQUE ACCUMULATION

As soon as the cleaned and polished tooth surface is moisturized by saliva a monomolecular protein layer will cover all the surfaces. It binds to enamel by ionic, hydrophobic and van der Waals forces. This protein layer is called as "acquired pellicle".

The main component of acquired pellicle is salivary glycoprotein (mucin), and in a lesser extent some salivary immunoglobulins. Its thickness is less than 1μm. Early pellicle is bacteria free.
Plaque distribution after 12 hours of no oral hygiene

Lang et al. 1973
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.

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

As the bacterial plaque gets thicker the oxygen cannot reach the deepest bacterial layers partly because of simple physico-chemical rules, partly because of the oxygen consumption of the superficial bacteria.

In this way an oxygen gradient is to form from the surface to the depth with decreasing oxygen concentration. This eventually will reorganize the composition of the bacteria in the depth and shifting towards the anaerobes.
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

<table>
<thead>
<tr>
<th>day</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>sterile dental pellicle</td>
</tr>
<tr>
<td>1</td>
<td>Gram+ cocci</td>
</tr>
<tr>
<td>3</td>
<td>Gram+ rods, actinomyces</td>
</tr>
<tr>
<td>6</td>
<td>Gram - bacteria</td>
</tr>
<tr>
<td>21</td>
<td>anaerobes, Gram - majority</td>
</tr>
</tbody>
</table>
Dental plaque as a biofilm

Biofilm is a well organized bacterial coating on the non-shedding hard surfaces. In the depth of the biofilm, the microorganisms are densely packed embedded into sticky polysaccharide matrix.
LOCAL PLAQUE RETENTIVE FACTORS
Periodontal diseases are bacterial infections. Specific bacterial pathogens are the primary etiologic agents. These bacteria form a biofilm above and below the gingival margin.
Plaque is natural and might exist in harmony with the host.
• NO OVERT INFLAMMATORY REACTION
• OR
• INFLAMMATION
Disease is the consequence of breaking down this balanced relationship

- The magnitude or nature of the microbial challenge
- Nature of the host response

(Socransky et al. 1998).
DENTAL PLAQUE IS A NECESSARY BUT NOT SUFFICIENT ETIOLOGIC FACTOR OF DESTRUCTIVE PERIODONTAL DISEASE
RISK 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
- Supragingival irregularities
- crowding,
- calculus
- rough restorations

Enhance the retention of the supragingival biofilm and protect organisms from the action of oral hygiene measures.
LOCAL PLAQUE RETENTIVE FACTORS

1. ANATOMICAL ETIOLOGIC FACTORS

2. IATROGENIC ETIOLOGIC FACTORS
1 ANATOMICAL ETIOLOGIC FACTORS

- a) Palatine sulcus of upper incisors.
- b) Furcation areas.
- c) Cervical enamel projections
- d) Enamel pearls
- e) Crowding of the teeth in the dental arch.
- f) Mucogingival deformities
- g) Occlusal anomalies.
a) Palatine sulcus of upper incisors.

- **palatine sulcus** which starts from the palatine tubercle of the lingual surface accumulates dental plaque, and enhances the pocket formation.

b) Furcation areas

- The anatomy of the furcation:
- favors retention of bacterial deposit
- makes periodontal debridement,
- oral hygiene procedures difficult.

b) Furcation areas

- Furcations are difficult to instrument because of their gothic arch configuration.
b) Furcation areas

- The dental plaque accumulates in that region and causes faster periodontal destruction of the molars and premolars,
PSEUDOFURCATION (BUCCAL GROOVES) ON THE ROOTS OF CENTRAL INCISIORS
FURCATION LESIONS ARE ALSO THE CONSEQUENCE OF PERIODONTAL ATTACHMENT LOSS AND PERIODONTAL RESECTIVE SURGERY
c) Cervical enamel projections

- Ectopic deposits of enamel apical to the level of the normal cementoenamel junction
- Connective tissue does not attach to cervical enamel projections
- They can lead to furcation defects.
c) Cervical enamel projections

- 82.5% of molars with cervical enamel projections, exhibited furcation involvement,
- while only 17.5% of molars without cervical enamel projections had furcation involvement.

d) Enamel pearls

- Enamel pearls can lead to furcation involvements.
- The prevalence of enamel pearls are reported between 1.1–9.7%.
- Nearly three-quarters of enamel pearls are found on maxillary third molars.

Cervical enamel pearl on the maxillary first molar
Alveolar bone loss associated with this anomaly

That is not present in the contralateral tooth and no bone loss either
e) Crowding of the teeth in the dental arch.

- The close convergence of the roots of neighboring teeth promotes the plaque accumulation resulting in faster periodontal destruction, is important due to the difficulty of removing dental plaque.


f) Mucogingival lesions

- The clinical impression is that the attached gingiva will provide a protective barrier against inflammation and attachment loss.
- Several studies have challenged the view that a wide zone of attached gingiva is a more effective barrier against recession.
f) Mucogingival lesions

- It has been demonstrated that in the absence of attached gingiva, gingival health and attachment levels can be maintained.


f) Mucogingival lesions

- High frenum and muscle attachments,
- can cause the detachment of the free gingiva,
- promotes the spread of the dental plaque inside the gingival sulcus
f) Mucogingival lesions
g) Occlusal anomalies.

- Occlusal anomalies had been considered as causative factor for periodontal disease (Ramfjord et al. 1966).
- It has been proven by long term animal studies (Lindhe & Svanberg 1974), that the traumatic occlusion can not be regarded as an etiologic factor for periodontal disease.
- It does not cause pocket or attachment loss.

g) Occlusal anomalies.

- Occlusal traumatism can cause degenerative changes in the deep periodontal structures.
- Inflammatory process in the gingiva is allowed to spread apically more rapidly and result in more severe periodontal destruction.
g) Occlusal anomalies.

- Missing teeth can lead to mesial drifting, tilting and extrusion of teeth.
- These alterations can result in increased plaque retention, food impaction and vertical bony defect
2 IATROGENIC ETIOLOGIC FACTORS

- a) Dental caries
- b) Dental calculus
- c) Dental materials and plaque retention.
- d) Effect of bad restoration quality on periodontal health.
- e) The effect of the position of the crown margin to the periodontium.
- f) Pontic design and the edentulous mucosal area.
- g) Overconturing of restorations.
- h) Temporary restorations and their effect on the periodontium.
a) Dental caries

- Dental caries enhance plaque retention - promoting periodontal disease.
- **Ainamo** (1970) first noted a strong relation between the GI value and untreated dental caries.

a) Dental caries

Secondary caries in restored teeth and the relationship of its incidence to the location of the preparation margin

5 years follow-up study:
- 15.4% of the supragingivally located amalgam-restoration
- 30.4% of the subgingivally located amalgam-restoration
- exhibited secondary caries

Hammer and Hotz
b) Dental calculus

The calculus per se is not a primary etiologic factor.

- In the practice calculus is always an important factor in the development of periodontitis.
- It is a plaque retentive factor.
- Its surface is always covered by fresh bacterial plaque.
- It can also guide the plaque bacteria subgingivally.
Patient’s and dentist’s negligence
c) Dental materials and plaque retention.

- Dental materials possess a greater capacity to accumulate and retain plaque than do either enamel or dentin.
- Polymethyl-methacrylate accumulates plaque faster than gold and porcelain.
Dental gold, porcelain and composites irritate tissues hardly if at all. Porosity contribute to the plaque retentive potentials.
c) Dental materials and plaque retention.

- Especially the transition zone at restoration margins represents a predilection site for plaque accumulation.
- The "cement line" associated with seated crowns may approach several square millimeters.
- Histological investigations by Waerhaug have shown that the subgingival cement roughness enhances plaque accumulation in the gingival sulcus.
d) Effect of bad restoration quality on periodontal health.

- The World Workshop in Periodontics (1966) reported that the overhanging at the margins of a restoration are local plaque retentive factors promoting periodontitis.
d) Effect of bad restoration quality on periodontal health.

- Teeth with inadequate restorations had significantly more plaque, gingivitis and periodontal pocket formation than adequately restored teeth.
- For both amalgam and crown restorations, the health of the periodontium is adversely affected by the presence of a restoration.

d) Effect of bad restoration quality on periodontal health.

- Björn et al. reported a generally poor marginal fit in retainers for fixed partial dentures.
- Eighty percent of the radiographically studied restorations exhibited marginal defects on the proximal surfaces.
d) Effect of bad restoration quality on periodontal health.

- In a German survey, only 18.2% of crown margins were clinically perfect.
- Margins that were open by more than 0.2mm were always associated with alveolar bone loss.

# Incidence of bad restorations

<table>
<thead>
<tr>
<th>Reference</th>
<th>Diagnostic method for detection</th>
<th>% restored surfaces with overhangs (n = number of subjects)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gilmore &amp; Sheiham, 1971</td>
<td>Bitewing radiographs</td>
<td>25% (n = 1976)</td>
</tr>
<tr>
<td>Burch et al., 1976</td>
<td>Bitewing radiographs</td>
<td>30% (n = 825)</td>
</tr>
<tr>
<td>Hakkainen &amp; Ainamo, 1980</td>
<td>Orthopantograms</td>
<td>50% (n = 85)</td>
</tr>
<tr>
<td>Than et al., 1982</td>
<td>Calculus probe</td>
<td>60% (n = 240)</td>
</tr>
<tr>
<td>Lervik &amp; Riordan, 1984</td>
<td>Bitewing radiographs, microscope</td>
<td>25% (n = 175)</td>
</tr>
<tr>
<td>Keszthelyi &amp; Szabo, 1984</td>
<td>Bitewing radiographs, microscope</td>
<td>86% (n = 176)</td>
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<tr>
<td>Coxhead, 1985</td>
<td>Bitewing radiographs, mirror, probe</td>
<td>76% (n = 50)</td>
</tr>
<tr>
<td>Claman et al., 1986</td>
<td>Bitewing radiographs</td>
<td>27% (n = 826)</td>
</tr>
<tr>
<td>Jansson et al., 1994</td>
<td>Bitewing radiographs</td>
<td>18 % (n = 162)</td>
</tr>
</tbody>
</table>
d) Effect of bad restoration quality on periodontal health.

- Overhanging restorations disturb the ecological balance in the gingival sulcus.
- Allow the growth of a group of disease associated microorganisms.

d) Effect of bad restoration quality on periodontal health.

- Even an adequately restored tooth can lead to gingivitis and periodontal pocket formation.
- The larger the number of restorations, the more important the plaque control is.

OVERCONTOURED CROWN MARGIN WITH SEVERE OVERGANG

THE WHOLE DENTAL PROBE CAN BE PUT UNDER THE CROWN MARGINE!!!!!!
CLASS TWO FURCATION LAESION TOTALLY COVERED BY OVERHANGING CROWN MARGIN

OVERCONTOURED CROWN MARGIN WITH SEVERE OVERGANG
d) Effect of bad restoration quality on periodontal health.

- The early detection of overhanging dental restorations is an important part of preventive dental care.
- The removal of overhanging margins should be part of initial periodontal therapy.
BRAND NEW FULL ARCH BRIDGE WITH SEVERE OVERHANGS AND OVERCOUNTURED CROWNS AND PONTICS
The effect of the position of the crown margin to the periodontium.

- In the past, Black's theory dominated dentistry for decades.
- The concept of "extension for prevention" by Black (1908).
- It postulates that the cervical margins of all reconstructions should be placed subgingivally.
The effect of the position of the crown margin to the periodontium.

- **Bodecker and Applebaum** (1934) where the first to question Black's theory about extension of the cavity boarders in the gingival sulcus.
- **Waerhaug** (1967, 1968), stated that there is scientific proof that subgingival crown margins create periodontal destruction due to plaque retention.
- **Loe (1968), Zander and Kennedy** (1970) supported the position of the crown margins above the free gingiva
The effect of the position of the crown margin to the periodontium.

- Follow-up examinations of fixed reconstructions have demonstrated:
  - crown margins positioned subgingivally were associated with the highest
  - and supragingival crown margins with the lowest GI values.

The effect of the position of the crown margin to the periodontium.

- 102 patients with 108 bridges were studied over 15 years.

- Loss of the periodontal supporting apparatus was significantly higher around teeth with subgingivally located crown margins than around teeth with crown margins located supragingivally.

Valderhaug J., Birkland JM: Periodontal conditions in patients 5 years following insertion of fixed prosthesis. Pocket depth and loss of attachment. J. Oral Rehab. 1976; 3(3)
The effect of the position of the crown margin to the periodontium.

- A follow-up survey of 423 crown margins
- Gingival tissues tended to bleed 2.42 times more frequently with subgingival margins
- had a 2.65 times higher chance of gingival recession
- Crowns with supragingival margins did not differ significantly compared with the contra-lateral tooth,

The effect of the position of the crown margin to the periodontium.

- From a caries preventive point of view, the location of crown margins does not seem to be of great importance if patients maintain a satisfactory oral hygiene.

Valderhaug J, Loe H: Oral hygiene in a group of supervised patients with fixed prosthesis. J. Periodontol. 1977; 48:221-224
The effect of the position of the crown margin to the periodontium.

- Subgingival margins can also lead to gingival recession as a possible consequence of chronic irritation and the violation of biologic width

Valderhaug J, Loe H: Oral hygiene in a group of supervised patients with fixed prosthesis. J. Periodontol. 1977; 48:221-224
f) Pontic design and the edentulous mucosal area

- Badly designed pontics are very frequently the cause of tissue damage, gingival inflammation, hyperplasia of the underlying mucosa and bone resorption.
f) Pontic design and the edentulous mucosal area

- The distribution of *P. gingivalis*, *P. intermedia*, *T. forsythia* under the pontics adjacent to healthy and inflammed mucosa is different.

g) Overcontouring of restorations.

- Oral hygiene practices may be severely hampered by overcontouring restorations.
- It is more difficult for the patient to effectively clean the area.
g) Overcontouring of restorations.

- Interdental space should be kept wide,
- for the normal development of the gingival papilla,
- to make access to hygienic oral devices.
- access to interdental spaces is one of the most important factors for a long-time expectancy
h) Temporary restorations and their effect on the periodontium

THE QUALITY AND THE MARGINAL ADAPTATION OF A PROVISIONAL RESTORATION SHOULD ALSO BE CORRECT
Quality of restorations
From a periodontal point of view, a supragingival position of the crown margin is the most favorable.

The most expensive techniques and material can cause severe periodontal disease if the quality is questionable.