PARADIGM CHANGES IN THE ETIOLOGY OF PERIODONTAL DISEASE

RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

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DEPT. PERIODONTOLOGY
BUDAPEST
RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

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BACTERIAL PLAQUE IS A NECESSARY BUT NOT SUFFICIENT ETIOLOGIC FACTOR IN DESTRUCTIVE PERIODONTITIS.

Destructive periodontitis affects only a relatively small percentage of adult population.

There is a relatively weak correlation between supragingival plaque and the severity of periodontal attachment loss.

Identical twin studies indicated that the manifestation of periodontal disease can be contributed to genetic determinants at least in 50%.
The risk factor can be defined as a factor that indicates the odds ratio of the manifestation of disease if the given factor is present comparing to those where the factor is missing.
Absolute risk - the probability of the manifestation of the disease in a subject who is exposed to a given factor

Relative risk factor expresses the probability of the manifestation of disease in a population exposed to a given factor relative to those where the factor is not present

ODDS RATIO

The calculated relative risk is the ODDS RATIO
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- **Stress**
  - Social-economics
Behavioral, systemic, and local risk factors in the etiology of periodontal disease
Oral hygiene

Classic experimental studies by Löe et al. (1965) highlighted the importance of oral hygiene and the role of dental plaque in the etiology of gingivitis. A 15 year follow-up study by Axelson & Lindhe indicated that the professional oral hygienic programs can anticipate the development of gingivitis and control caries.
Oral hygiene

The strictest oral hygienic programs were not effective in controlling aggressive periodontitis and could not prevent the progression of attachment loss in those individuals being susceptible to aggressive disease.

In the developed industrialized countries the prevalence of aggressive periodontitis is not significantly different from those in the developing nations.
Oral hygiene

There is only a weak correlation between the level of oral hygiene and severity of aggressive periodontitis.

The oral hygiene is a weak predictor for the occurrence of aggressive periodontitis in a given population or in a given subject.
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Local plaque retentive factors

- The shape of the tooth,
- Orthodontic disorders,
- The shape and the tightness of contact point
- The thickness and shape of the alveolar bone,
- The shape of the roots,
- The shape and position of the furcation area
- The quality of the cemento-enamel junction,
- Enamel developmental disorders
- Dental calculus
- Faulty restorations
Local plaque retentive factors

The sterile calculus will not cause gingivitis

The rough surface of the calculus is a plaque retentive factor and will always be a bacterial reservoir the amount of calculus will correlate with the severity of gingivitis
Local plaque retentive factors

A subgingivally placed or approximal crown margin - even with the best marginal adaptation - will provide a niche in which bacterial accumulation takes place.

All restorations are considered as a plaque retentive factors

Consequently the quality of restorations are a decisive factor in periodontal health.
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Bacterial specificity

Periodontitis will not develop without the presence of subgingival bacterial biofilm.

The composition of the subgingival biofilm is decisive in the course of periodontitis.
Bacterial specificity

**Periodontal infection is an opportunistic infection**

*There are certain well defined bacterial strains that do not normally occur in healthy sulcus and healthy oral cavity*

*They colonization strictly correlates with the occurrence or progression of disease*
A. actinomycetemcomitans is closely associated with early onset aggressive periodontitis.

The presence of P. gingivalis and Bacterioides forsythus is a reliable indicator for periodontal attachment loss.
# The Prevalence of Periodontopathogen Positive and Negative Pockets in Chronic Periodontitis and in Healthy Controls

## The Absolute Number and Relative Proportion (%) of Positive Pockets

<table>
<thead>
<tr>
<th>Bacterium</th>
<th>Chronic Periodontitis Active Pocket (n=116)</th>
<th>Chronic Periodontitis Inactive Pocket (n=28)</th>
<th>Healthy Controls (n=100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treponema sp.</td>
<td>114 (98.3)</td>
<td>13 (46.4)</td>
<td>22 (22)</td>
</tr>
<tr>
<td>A.A.</td>
<td>86 (74.1)</td>
<td>8 (28.6)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>P. gingivalis</td>
<td>113 (97.4)</td>
<td>14 (50)</td>
<td>18 (18)</td>
</tr>
<tr>
<td>Fusobacterium sp.</td>
<td>116 (100)</td>
<td>20 (71.4)</td>
<td>58 (58)</td>
</tr>
<tr>
<td>B. forsythus</td>
<td>112 (96.6)</td>
<td>9 (32.1)</td>
<td>18 (18)</td>
</tr>
<tr>
<td>P. intermedia</td>
<td>82 (70.7)</td>
<td>5 (17.9)</td>
<td>2 (2)</td>
</tr>
<tr>
<td>P. micros</td>
<td>95 (81.9)</td>
<td>10 (35.7)</td>
<td>8 (8)</td>
</tr>
</tbody>
</table>

**THE ABSOLUTE NUMBER AND RELATIVE PROPORTION OF PERIODONTOPATHOGENIC POSITIVE INDIVIDUALS AMONG PATIENTS WITH PERIODONTITIS AND HEALTHY CONTROLS**

**THE NUMBER AND % OF INDIVIDUALS**

<table>
<thead>
<tr>
<th>BACTERIUM</th>
<th>CHRONIC PERIODONTITIS (n=29)</th>
<th>HEALTHY (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treponema sp.</td>
<td>29 (100)</td>
<td>8 (40)</td>
</tr>
<tr>
<td>A.A.</td>
<td>26 (89.7)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>P. gingivalis</td>
<td>29 (100)</td>
<td>6 (30)</td>
</tr>
<tr>
<td>Fusobacterium sp.</td>
<td>29 (100)</td>
<td>17 (85)</td>
</tr>
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<td>28 (96.9)</td>
<td>11 (55)</td>
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<td>1 (5)</td>
</tr>
<tr>
<td>P. micros</td>
<td>28 (96.6)</td>
<td>6 (30)</td>
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Bacterial specificity

periopathogenic microorganisms

*Actinobacillus actinomycetemcomitans*,
*Bacterioides forsythus (Tannerale forshytia)*,
*Campylobacter rectus*,
*Eubacterium nodatum*,
*Fusobacterium nucleatum*,
*Peptostreptococcus micros*,
*Porphyromonas gingivalis*,
*Prevotella intermedia*,
*Prevotella nigrescans*,
*Streptococcus intermedius*
*Treponema denticola*
According to several cross sectional and longitudinal studies, the presence of A. actinomycetemcomitans, P. gingivalis, P. intermedia, B. forsythus (Tannerella forsythia), impose the highest risk for periodontal attachment loss.
Bacterial specificity

bacterial risk analysis

Each bacterial strain imposes risk just above a certain threshold of concentration in sulcus to develop tissue destruction.

In case of A. actinomycetemcomitans, $3 \times 10^3$ bacteria in a periodontal pocket are sufficient to sustain permanent inflammation and to cause additional 2 mm attachment loss with a short period of time.
Bacterial specificity

bacterial risk analysis

Though periodontitis is not an exogenous infection the oral flora develops and evolves due to interpersonal contacts

In this way the periodontitis develops due to an infection transmitted from one of our acquaintances
# Risk Factors in the Etiology of Destructive Periodontal Disease

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Systemic immune status

Though the innate and adapted immune reactions are responsible for the majority of periodontal tissue destructions, the individuals with systematically healthy immune system can tolerate and neutralize the majority of the side effects of the inflammation protecting the periodontium against plaque bacteria.
Systemic immune status

In HIV positive individuals very severe destructive periodontitis, ulcerative periodontitis can frequently occur.

The number of CD4+ lymphocytes are significantly decreased. There is a positive correlation between the number of CD4+ lymphocytes in the peripheral blood and the severity of periodontal disease.

a
Patients with inherited or acquired immune deficiencies develop very severe periodontitis in the very early childhood.
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### III. NHANES in the USA (more than 31000 people) showed the following prevalence of periodontitis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Diabetes %</th>
<th>Control %</th>
</tr>
</thead>
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<tr>
<td>&gt;5mm attachment loss</td>
<td>30</td>
<td>20</td>
</tr>
<tr>
<td>&gt;5 mm- deeper pocket</td>
<td>21</td>
<td>8,8</td>
</tr>
<tr>
<td>&gt;3mm- gingival recession</td>
<td>31</td>
<td>22</td>
</tr>
<tr>
<td>positive bleeding on probing</td>
<td>63</td>
<td>50</td>
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Diabetes mellitus

- In diabetics 11% while in non diabetics only 5.8% of all the teeth showed advanced attachment loss
- 12.5% of the severe periodontitis patients had diabetes while 6% of patients without periodontal disease had diabetes
The occurrence of severe periodontitis in diabetic and non diabetic population

the prevalence of attachment loss >5mm

89.0% for diabetes
11.0%

95.0% for healthy
5.0%
The prevalence of Diabetes among patients with severe periodontitis and those with healthy periodontium

- Periodontitis: 13.0% (87.0%)
- Healthy periodontium: 6.0% (94.0%)
Diabetes mellitus

- The hyperglycaemia associated with diabetes will produce protein-glycates that can bind to high affinity membrane receptors on the phagocytes.

  - this will increase the interleukin production in the cells.

  - The permeability of endothelial cells are increased.
  - The expression of adhesion molecules are enhanced.
  - The collagen production by fibroblasts are inhibited.
Diabetes mellitus

- Diabetic patients are responding with an elevated pro-inflammatory cytokine production against bacterial stimuli.
- The regenerative potential of the PDL mesenchymal cells are decreased. Cells are more resistant to anabolic and local growth factors.
Diabetes mellitus

The overall tissue damage is due to two mechanisms

- The glucose enzymatically converted to sorbitol which in turn exert toxic effect on the tissues
- In hyperglycemia the hexose can bind to different protein molecules forming protein-glycat

This end product will change the function of many important proteins (collagen, hemoglobin, albumin, lipoproteins)
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Tobacco smoking

In the past - we believed that only the inferior oral hygiene of smokers was responsible for the worse periodontal status.

Now - several evidences are indicating a direct metabolic effect of smoking on periodontal health state.
Smoking → Gingivitis

- Close correlation between smoking and ANUG
  Humane experimental gingivitis studies showed a marked reduction in inflammatory response in the gingiva to plaque accumulation in smokers. Much less severe marginal gingivitis develops in smokers than in non-smokers.
- Cross sectional studies also indicated that smokers have less severe gingivitis than non-smokers.
THE RATE OF ALVEOLAR BONE LOSS RELATED TO THE AGE

% 

0 20 40 60 80 

10 20 30 40 50 60 70 80 ÉV 

NON SMOKER
SMOKER
THE ATTRIBUTABLE RELATIVE RISK FOR SMOKING IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTITIS
Severe destructive periodontitis is more common in smokers than in matched non-smokers.

The age matched smoker population shows a significantly higher average periodontal index score than non-smokers.

The extent and severity of periodontal bone loss is more severe in smokers than in age matched non-smokers.
Smoking → Periodontitis

The average attachment loss in smokers correlates with the amount of cigarettes smoked during the previous years:

- < 10 cigarettes/day, 2.79 - times higher
- > 30 cigarettes/day, 6 - times higher risk to develop periodontitis

In those who quitted smoking in the past, the odds ratio is lower, but still much higher than among the virgin non-smokers - 1.5
Smoking has negative effect on the periodontium even in the teenagers.

The juvenile generalized aggressive periodontitis is more common and more severe among smokers than in non-smokers adolescents.

The relative risk to develop periodontitis in the 19-30 age group is 3.8-times higher in smokers, than in non-smokers.
Smoking Periodontitis

- The smoking targets several mechanisms in the gingiva that determine the balance between dental plaque and periodontal defensive factors

MICROBIOLOGY
IMMUNOLOGY
PHYSIOLOGY
Smoking  Microbiology

- Has no effect on the speed of plaque accumulation.

- Enhances the accumulation of periodonto-pathogenic microorganisms in relatively shallow pockets (>4mm)

- The relative and absolute number of anaerobic microorganisms are increased in deep pockets by smoking.
PMN leukocytes chemotactic activity is decreased

Increased local TNFα, PGE production

Increased MMP-8 production by PMN cells

Responding to LPS stimuli the monocytes produces more PGE
- Decreased vascular responses
- Decreased subgingival temperature
- Slower tissue responses
The attachment gain after periodontal flap surgery is significantly smaller in smokers than in non-smokers.

The differences are much more marked around the front teeth where the direct effects of smoking is stronger than around the molars.
The effect of smoking on the outcome of periodontal therapy and periodontal regeneration

Compromised wound healing capacity and the regenerative potential is inferior in smokers after free gingival grafting and GTR surgery. 80 to 90% of all therapy refractor cases occur among smokers.
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Osteoporosis

There is a positive correlation between the severity of generalized osteoporosis and the rate of tooth loss.

45% of women with severe generalized osteoporosis were edentulous while only 15% were edentulous among non-osteoporotic matched controls.
Osteoporosis

According to the Third National Health and Nutrition Examination Survey (NHANES) in the USA among 11000 women aged 20-90 year there was a positive correlation between the severity of periodontal disease and the bone density measured in the hip.
Osteoporosis

The oestrogen hormone plays an important role in the regulation of the "coupled bone remodelling" in the bone. It controls the local IL-1b and TNF-a production and modulates the osteoclasts stimulated by bone resorbers (PTH, PGE etc.).

The oestrogen hormones exert certain "bone protective" effects.
Osteoporosis

In Oestrogen deficiency the osteoblasts respond with increased IL-6 production to local stimuli and consequently promotes the osteoclasts differentiation from precursor cells.

The oestrogen/progesteron substitution blocks the increased IL-1 production.

Among severe osteoporotic women there were significantly more common the total edentulousness than in non osteoporotic matched controls.
The negative bone effects by smoking is dependent on age, sex and oestrogen level.

In males, the smokers' average bone mass is 15% less than non-smokers. In premenopausal women, there is no marked difference between smokers and non-smokers.
In premenopausal women there is no marked difference between smokers and non-smokers in the bone density.

In postmenopausal women the bone mass and bone density more rapidly decrease in smokers than in non-smokers. Presumably in premenopausal women the oestrogen protective effects can negotiate the negative bone effect of smoking. This will be ceased after menopause.
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Ethnic group

The effect of race can easily be studied in the USA.

The periodontal attachment loss is significantly higher in American blacks than in age matched white controls.
According to another US study the occurrence of 5mm deep pocket was 330% more frequent among blacks than in whites.

The prevalence of the juvenile aggressive periodontitis is 10% among black teenagers while only 5% among Caribbeans and only 1.3 among Caucasians.
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Age

The average attachment loss is increasing parallel with the ages.
The average pocket depth does not follow this trend.
This is due to the gingival recession.

The prevalence of the individuals with >4mm pocket depth does not increase with the 50-80 age period.
RUSSELL'S PERIODONTAL INDEX ACCORDING TO AGE GROUPS

USA 1971-1974

FEMALE
MALE

YEARS

PI

0 0,5 1 1,5 2 2,5

6-11 12-17 18-44 45-64 65-74
PERCENTAGE OF TEETH WITH $\geq$ 3 mm ATTACHMENT LOSS BY AGE

PERCENTAGE OF PERSONS WITH > 4mm POCKETS BY AGE

TOOTH LOSS DUE TO CARIES OR PERIODONTAL DISEASE IN AGE GROUPS

YEARS

PERIODONTITIS
CARIES
Age

Due to the gerophysiological changes occurring in the periodontium the aged periodontal tissue is more susceptible to plaque effect and inflammatory tissue damages than that of the younger tissue.

The regenerative potential of the periodontium is decreasing with ages.
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Diet

According to the USA NHANES data, those who consumed less food products with high Ca content, are more susceptible to osteoporosis and are more prone to periodontal disease.

The increased Ca supplementation will increase bone density both in the childhood and in adults and can in certain extent prevent the progression of osteoporosis.
Diet

The alimentary phosphate, protein, vitamin-D, fluoride and coffein intake has effect on bone metabolism and bone mass.

The fluoride significantly increase bone density and trabecular bone mass.

The coffein consumption has a negative effect on bone mass.
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Genetic factors

The human genom is made up of 35,000 genes.

The phenotype is determined by the genes and the environmental influences.

Certain disease are monogenic disorder with a single gene mutations and the presence of defected gene will be a sufficient causative factor.

Many times the gene mutations are only susceptibility or severity factors.
Genetic factors

Any kind of gene combination that might have effect on the development of periodontal tissues or influence the innate or specific immune reactions can be a major susceptibility risk factor in the etiology of periodontal disease.
Genetic factors

The familiar occurrence of localized or generalized aggressive periodontitis has long been known. Frequently some PMN leukocyte functional disorder can be responsible for the disease.

Frequently the PMN leukocytes' Fc receptors have been involved that can severely damage the phagocytotic capability of the cells.
Genetic factors

The role of genetic factors can easily be studied among identical twins.

According to twin studies the clinical manifestation of periodontal disease, the severity, the rate of progression is determined by genetic factors in 70-80%.
Genetic factors

A true chromosomal disorder is the Down syndrome (21 chromosomal trisomy), in which very severe destructive periodontitis occurs.

The hypophosphatasia is a rare inherited disease in which the tissue alkaline phosphatase (1p 36.1-p34) gene mutation leads to impaired alkaline phosphatase activity in the tissues with severe bone developmental disorders, cementum hypoplasia and periodontal disease.
Down-syndrome (21-es chromosoma trisomy),
Genetic factors

Papillon-LeFevre syndrome
inherited by autosomal recessive pattern
The palmoplantar hyperkeratosis syndrome is associated with early onset aggressive generalized periodontitis.

The cause is the cathepsin C gene mutation on the locus of the #11 chromosome (11q14-q21)

In PLS patients the proportion of the Actinobacillus actinomycetemcomitans are significantly increased.
11 chromosom (11q14-q21) cathepsin C gene mutation
Genetic factors

*leucocyte adherence deficiency LAD*
Impaired the PMN cells and monocytes emigration from the capillaries and the migration towards the chemotactic stimulus
Genetic factors

Individual variability in the pro inflammatory cytokine production

Monocytes from different individuals secrete different quantity of pro inflammatory cytokines (IL-1, TNF) and PGE against bacterial endotoxin stimulation
Genetic factors

Those individual variability is determined by certain genetic polymorphisms.

In humans the genes coding the IL-1α, IL-1β and IL-1ra synthesis are located on the long arm of the 2nd chromosome.

On these loci multiple polymorphism can occur.
In humans the genes coding the IL-1α, IL-1β and IL-1ra synthesis are located on the long arm of the 2nd chromosome.
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Stress factors

The role of stress in the pathogenesis of periodontal disease is rather controversial.

It is well known that stress can alter the corticosteroid production in the adrenal cortex, which in turn can modulate the cellular and humoral immune reactions.
Stress factors

In certain type of depression patients lost their motivation they neglect oral hygiene and dental office attendance.

The antidepressants will cause xerostomia, that promote plaque accumulation and consequently periodontal disease.
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Socio-economic status plays a significant role in the etiology of destructive periodontal disease.
Socio-economic factors

Epidemiological studies show a positive correlation between the socio-economic status and the prevalence and severity of periodontal disease.

The social status also determine some other behavioral risk factors (smoking, diet, oral hygiene, systemic diseases, stress).
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