PARADIGM CHANGES IN THE ETIOLOGY OF PERIODONTAL DISEASE

RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

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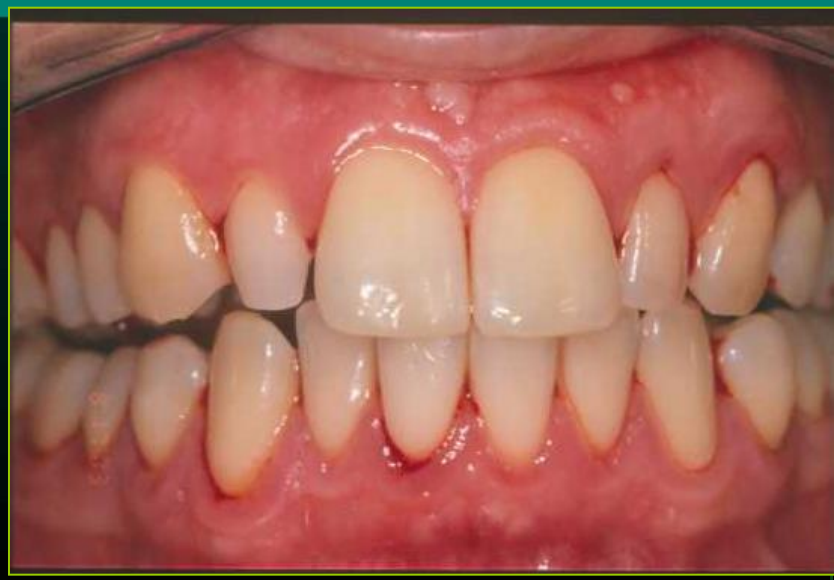
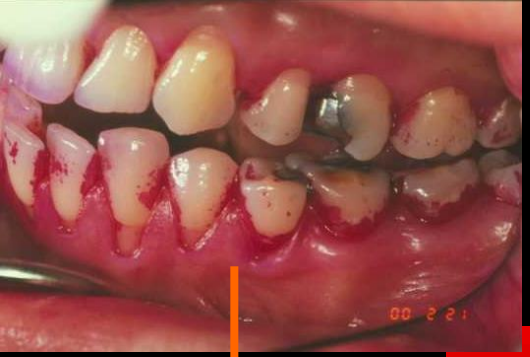
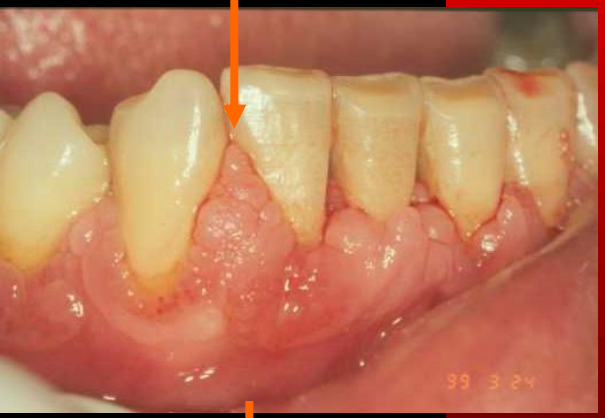
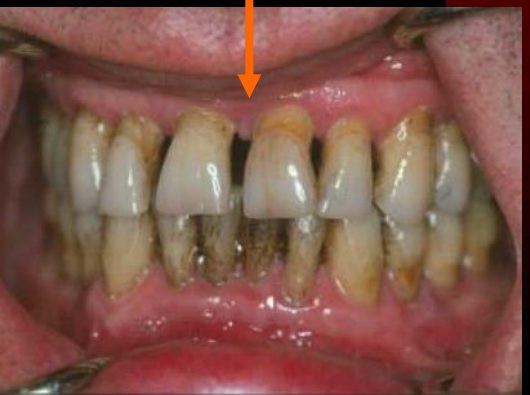
DEPT.PERIODONTOLOGY
BUDAPEST



LÖE CLASSIC EXPERIMENTAL GINGIVITIS STUDIES



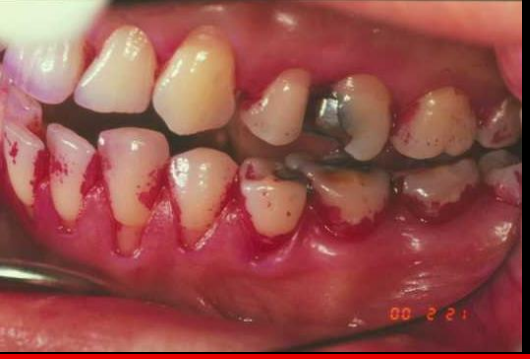




DENTAL PLAQUE

GINGIVITIS

PERIODONTITIS



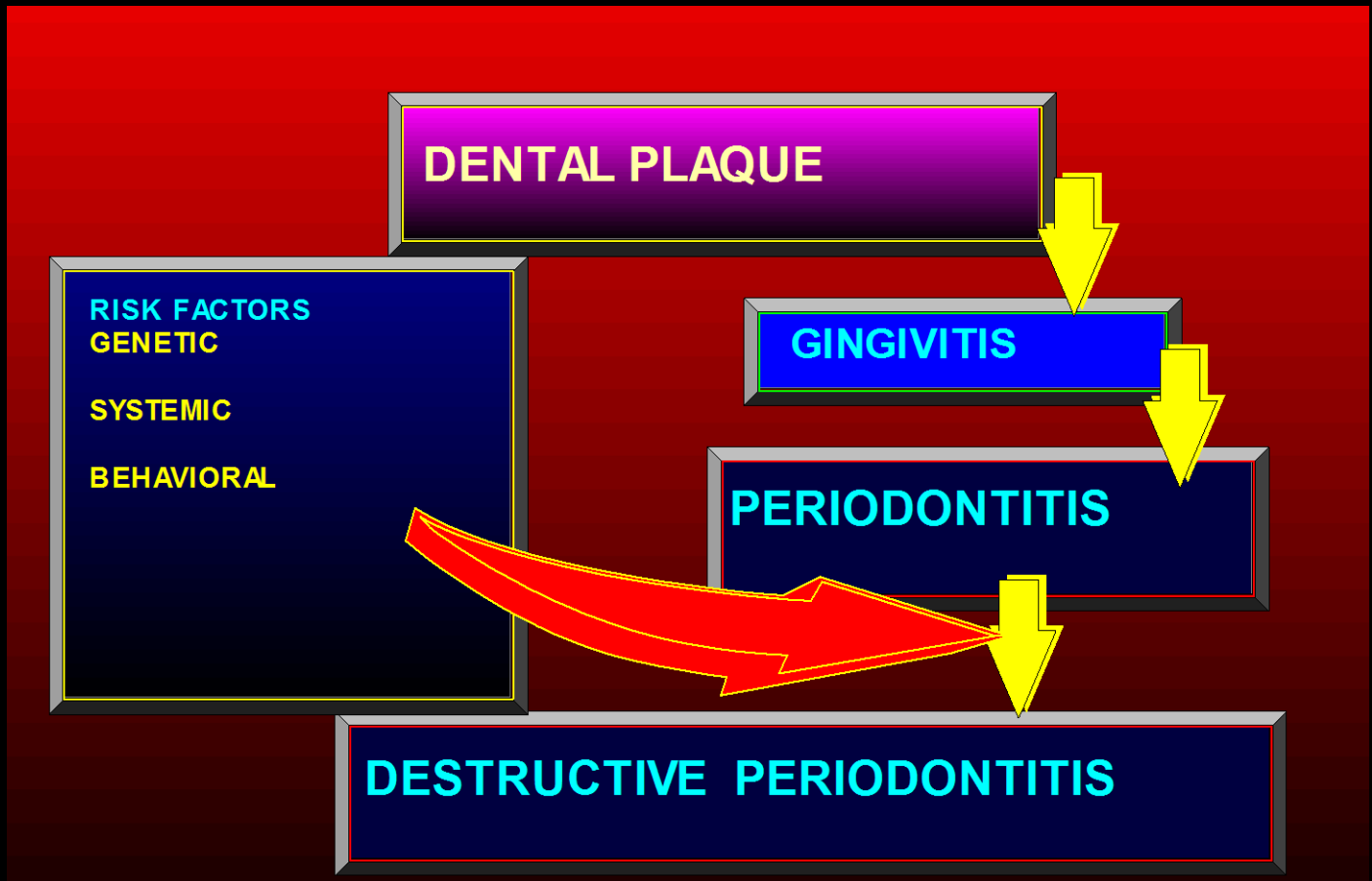
WHY NOT NECESSARILY ALL GINGIVITIS PROGRESSES TO DESTRUCTIVE PERIODONTITIS????

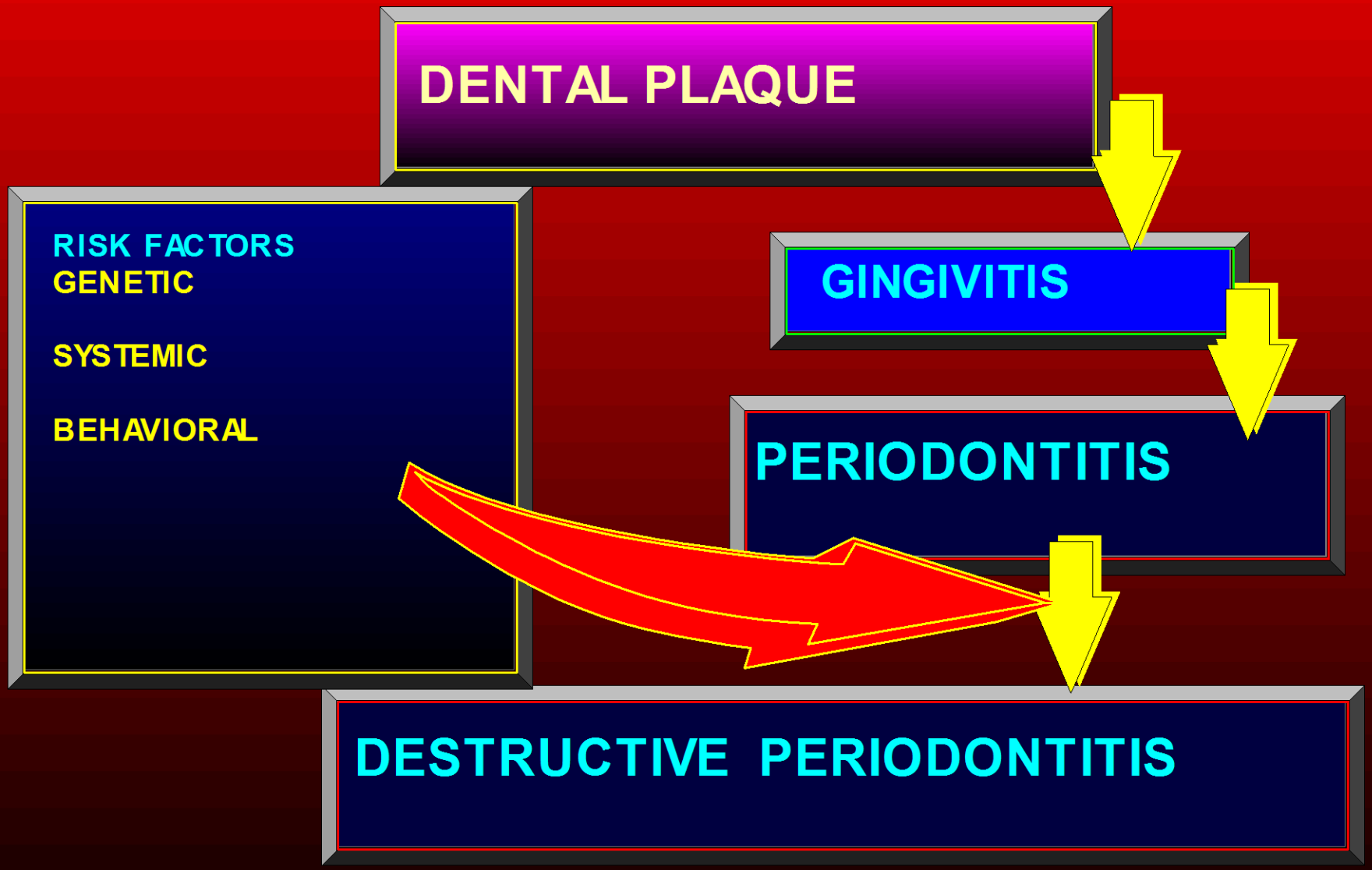
DENTAL PLAQUE

GINGIVITIS

PERIODONTITIS

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




DENTAL PLAQUE
RISK FACTORS
GENETIC
SYSTEMIC
BEHAVIORAL
GINGIVITIS
PERIODONTITIS
DESTRUCTIVE PERIODONTITIS

RISK FACTORS:

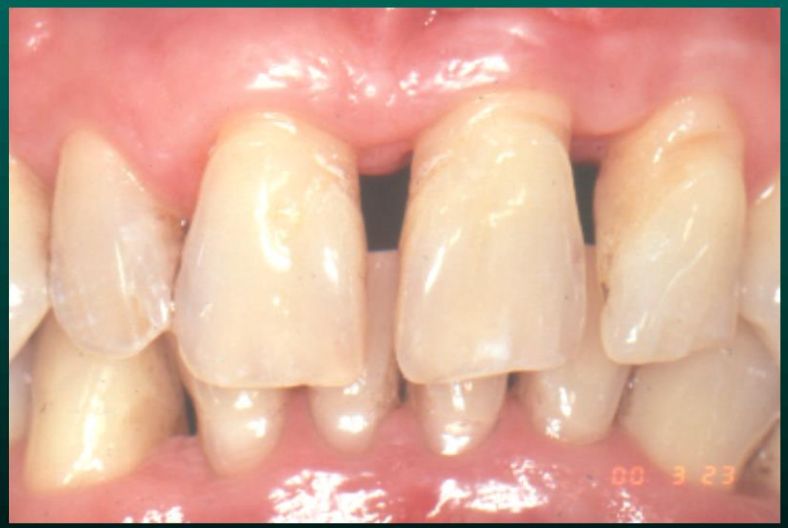
GENETICS
1(IL-1)
TNF

SYSTEMIC
ENDOCRINE
CARDIOVASCULAR
IMMUNOLOGICAL

BEHAVIORAL
STRESS
DIET
SMOKING
WAY OF LIFE

RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

- Oral hygiene
  Local plaque retentive factors
  Bacterial specificity
  Systemic immune status
  Diabetes mellitus
  Tobacco smoking
  Osteoporosis
  Etnic background
  Age
  Diet
  Genetics
- Stress
  Social-economics



RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

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 tween studies indicated that the manifestation of periodontal disease can be contributed to genetic determinant 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

RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

- **Oral hygiene**
  Local plaque retentive factors
  Bacterial specificity
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  Diabetes mellitus
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  Osteoporosis
  Etnic background
  Age
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- Stress
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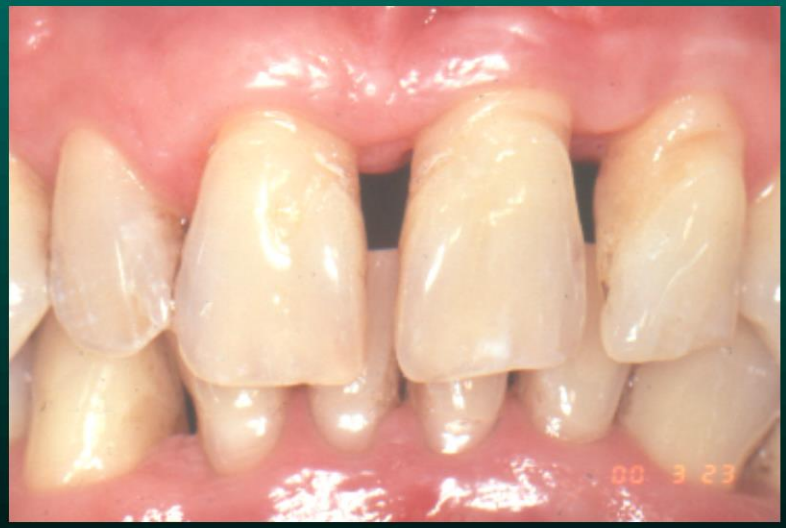
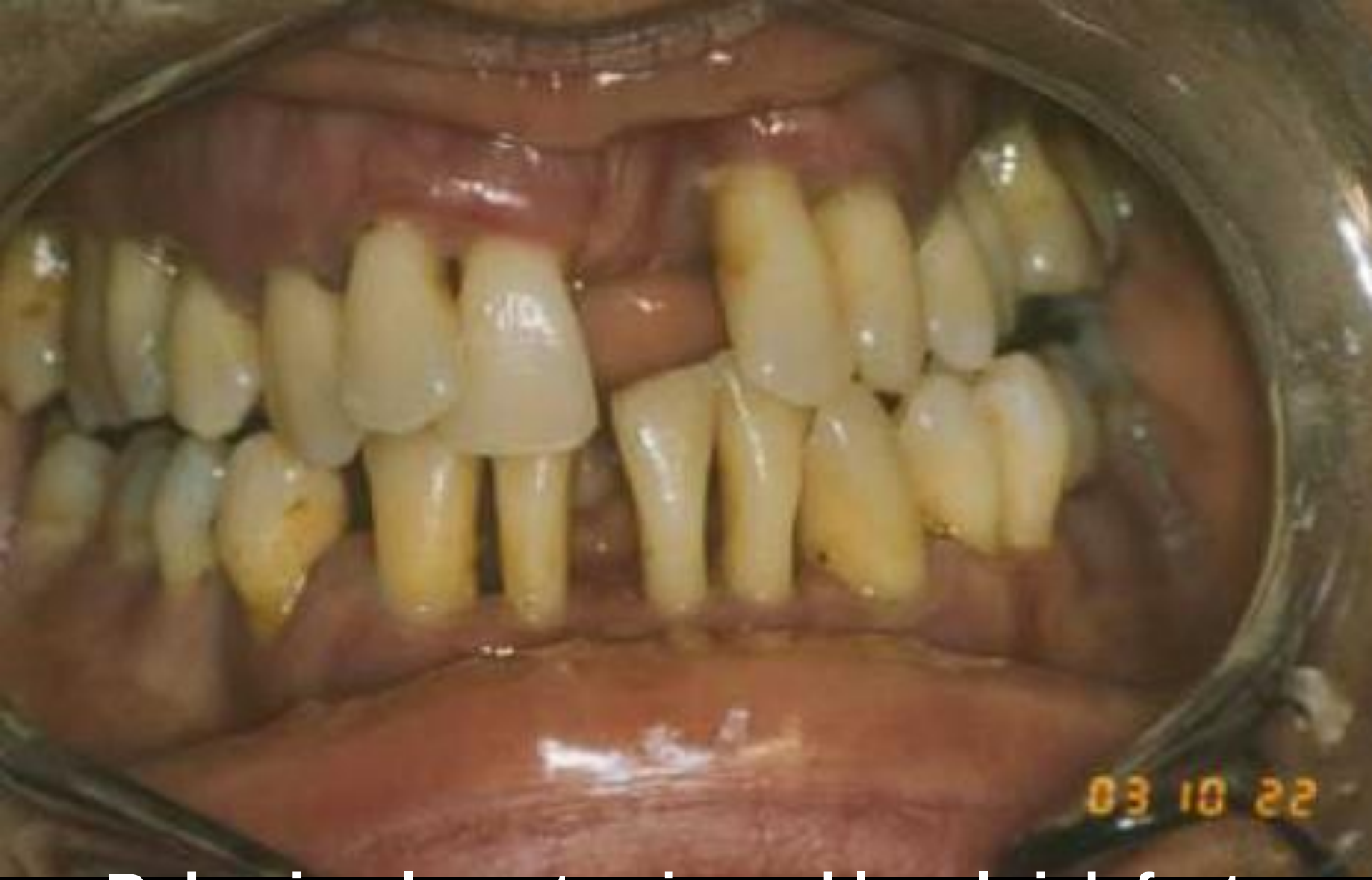


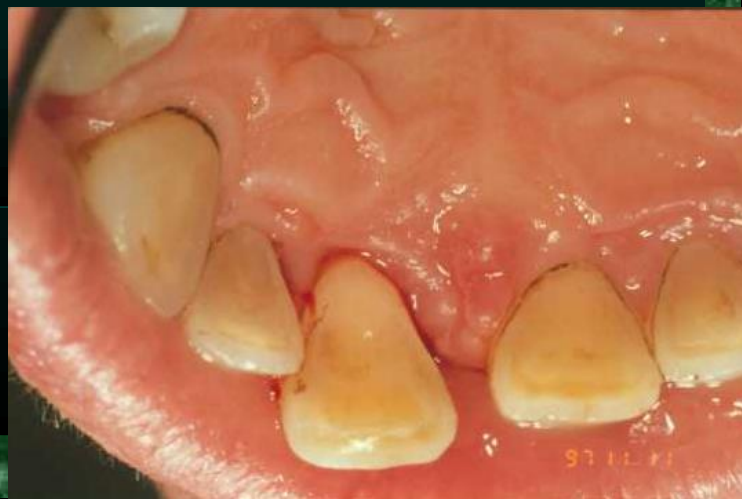


Behavioral systemic and local risk factors in the etiology of periodontal disease

Oral hygiene

There is only a weak correlation between the level o
oral hygiene and severity of aggressive periodontiti

The oral hygiene is a weak predictor for the occurrence of aggressive periodontitis in a given population or in a given subject



RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

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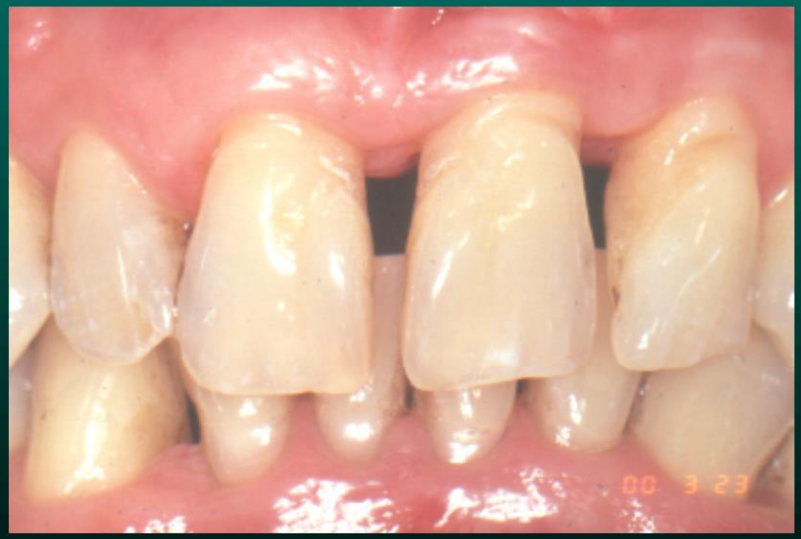
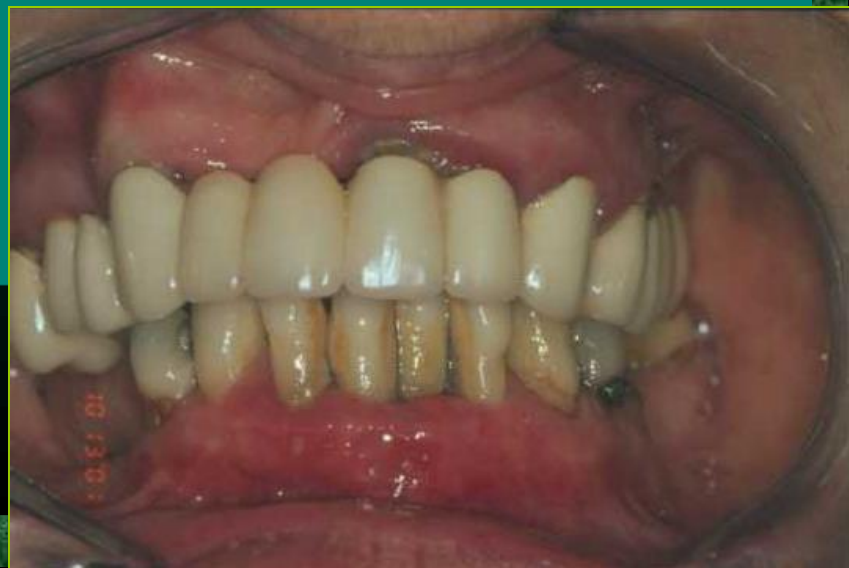


Local plaque retentive factors

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



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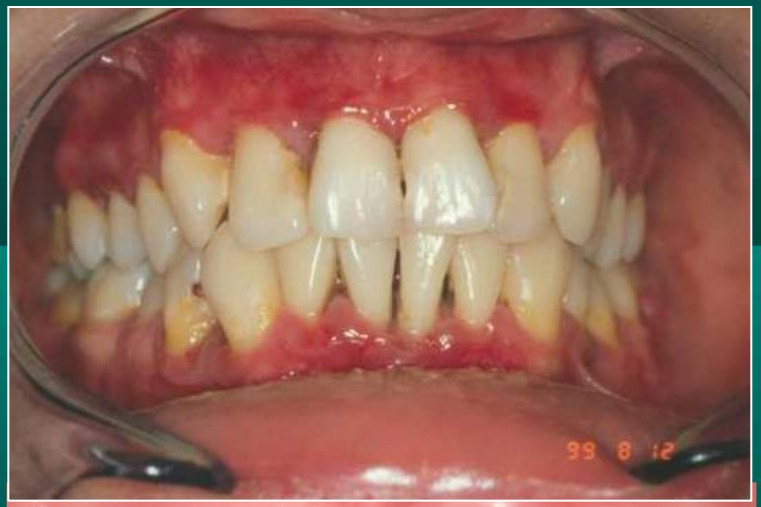

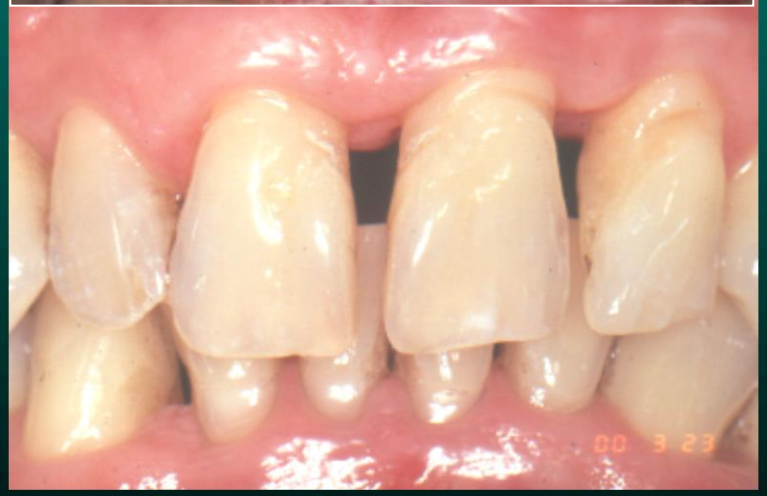

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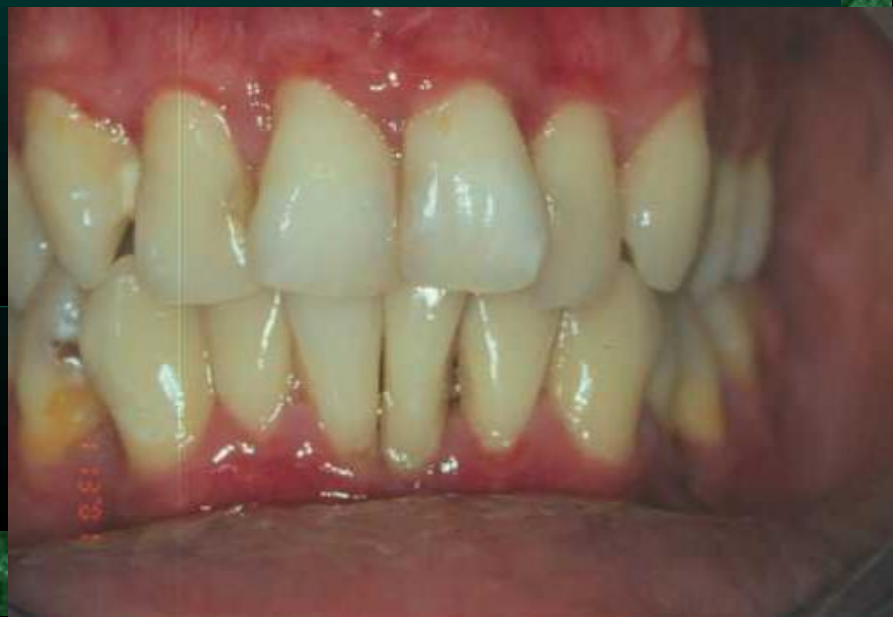
Bacterial specificity

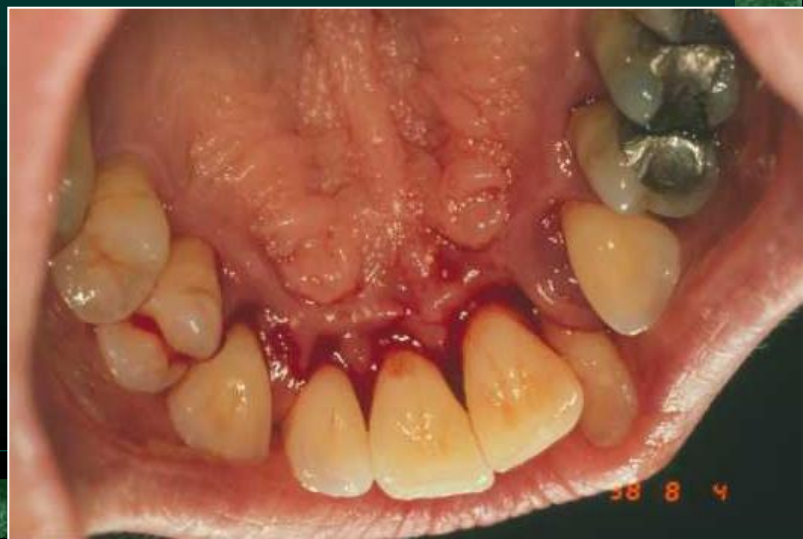
- Periodontitis will not develop without the presence of subgingival bacterial biofilm
- The composition of the subgingival biofilm is decisive in the course of periodontitis



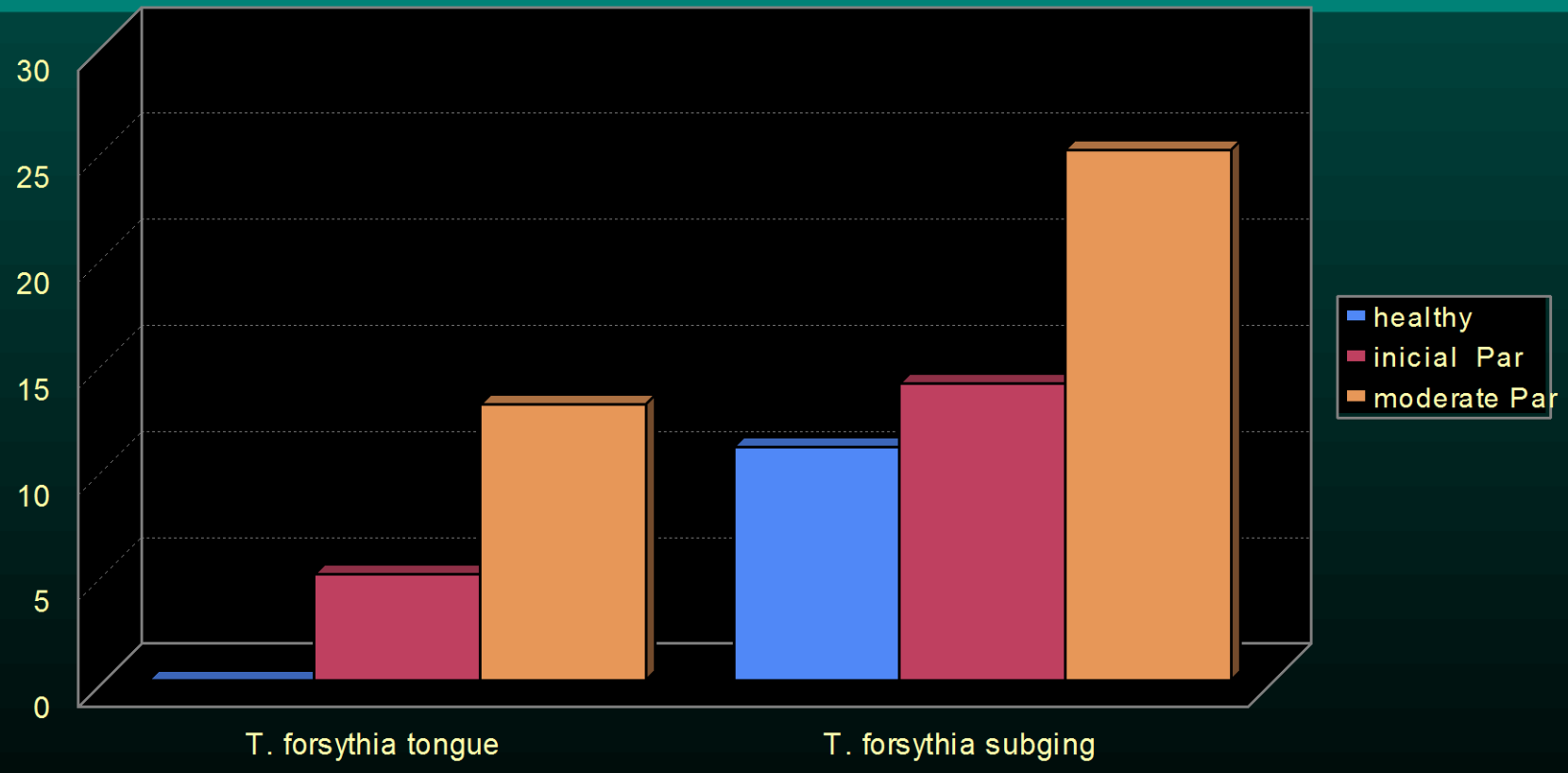
Bacterial specificity

periopathogenic microorganisms

Aggregatibacter actinomycetemcomitans,
Bacterioides forsythus (Tannerale forshytia),
Campylobacter rectus,
Eubacterium nodatum,
Fusobacterium nucleatum,
Peptostreptococcus micros,
Porphyromonas gingivalis,
Prevotella intermedia,
Prevotella nigrescans,
Streptococcus intermedius
Treponema denticola



The presence of Tannerela forsythia in healthy and periodontitis



Tanner AC et al Subgingival and tongue microbiota during early periodontitis J Dent Res 2006;85:318-323

RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

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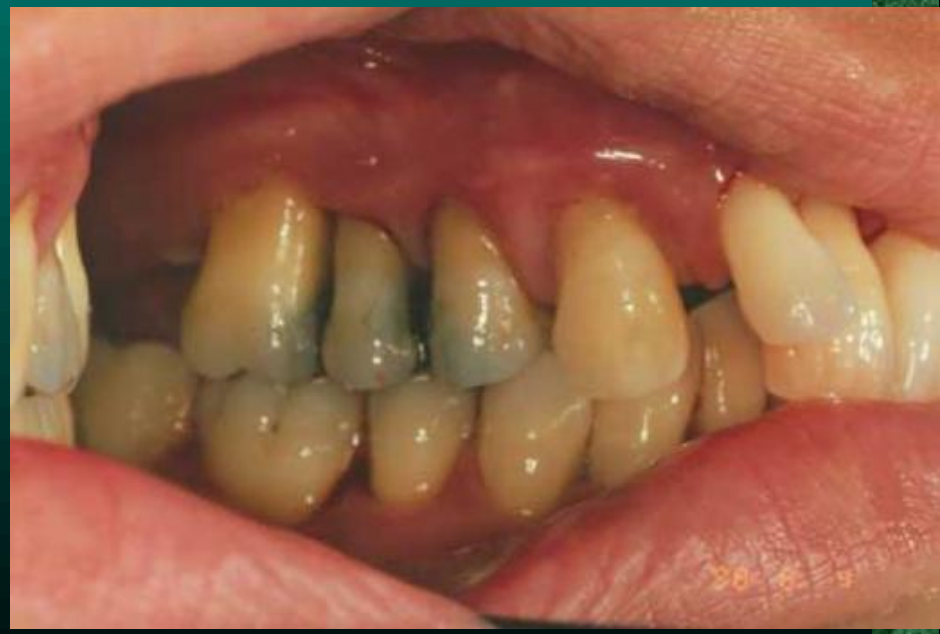


Systemic immune status

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

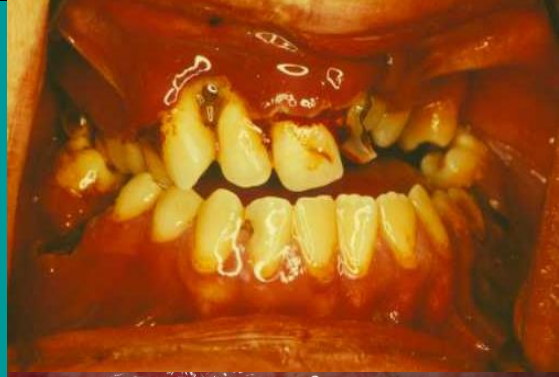
a

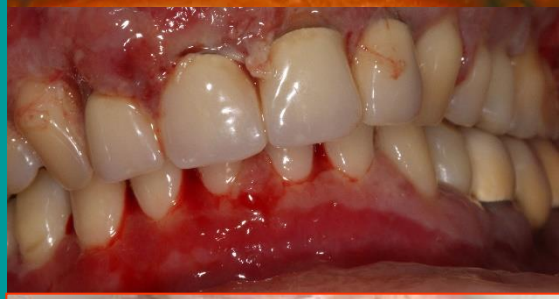
Systemic immune status

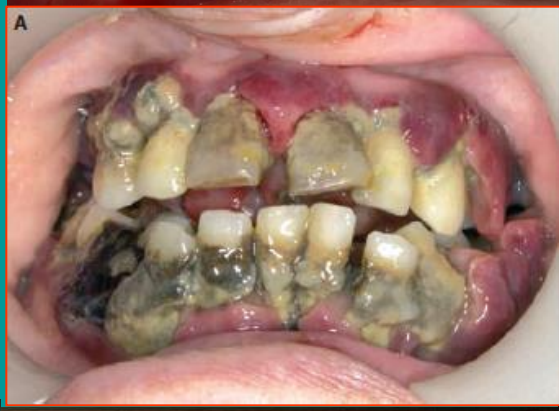
Patients with inherited or acquired immune deficiencies develop very sever periodontitis in the very early childhood.

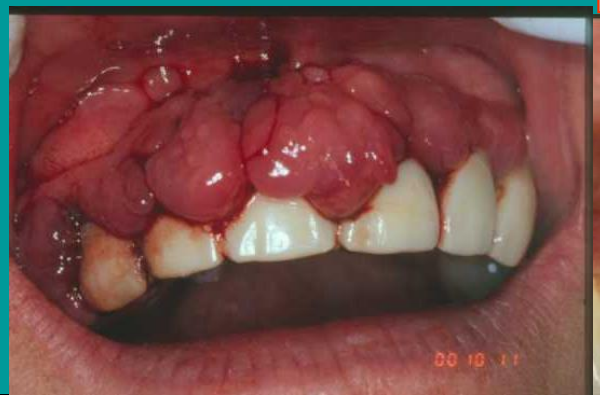
acquired immunological diseases

- HIV +
- AIDS
- HYPO-GAMMAGLUBULINAEMIA
- IgA deficiencies
- LEUKEMY
- CYTOSTATIC THERAPY
- DIABETES
- SMOKING









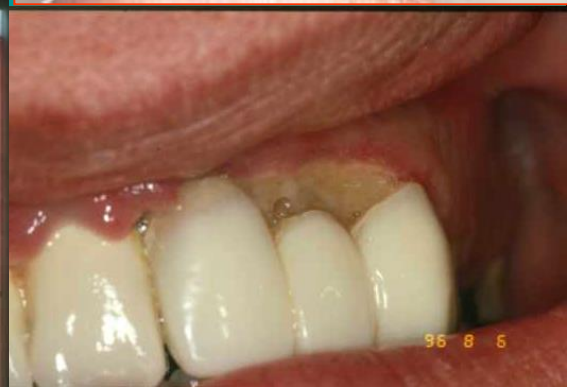


TABLE 1 Classification of systemic diseases and conditions that affect the periodontal supporting tissues (adapted from Albandar et al.[1])

Classification	Disorders	ICD-10 code
1.	**Systemic disorders that have a major impact on the loss of periodontal tissues by influencing periodontal inflammation**	
1.1.	**Genetic disorders**	
1.1.1.	**Diseases associated with immunologic disorders**	
	Down syndrome	Q90.9
	Leukocyte adhesion deficiency syndromes	D72.0
	Papillon-Lefèvre syndrome	Q82.8
	Haim-Munk syndrome	Q82.8
	Chediak-Higashi syndrome	E70.3
	Severe neutropenia	
	– Congenital neutropenia (Kostmann syndrome)	D70.0
	– Cyclic neutropenia	D70.4
	Primary immunodeficiency diseases	
	– Chronic granulomatous disease	D71.0
	– Hyperimmunoglobulin E syndromes	D82.9
	Cohen syndrome	Q87.8
1.1.2.	**Diseases affecting the oral mucosa and gingival tissue**	
	Epidermolysis bullosa	
	– Dystrophic epidermolysis bullosa	Q81.2
	– Kindler syndrome	Q81.8
	Plasminogen deficiency	D68.2
1.1.3.	**Diseases affecting the connective tissues**	
	Ehlers-Danlos syndromes (types IV, VIII)	Q79.6
	Angioedema (C1-inhibitor deficiency)	D84.1
	Systemic lupus erythematosus	M32.9
1.1.4.	**Metabolic and endocrine disorders**	
	Glycogen storage disease	E74.0
	Gaucher disease	E75.2
	Hypophosphatasia	E83.30
	Hypophosphatemic rickets	E83.31
	Hajdu-Cheney syndrome	Q78.8
1.2.	**Acquired immunodeficiency diseases**	
	Acquired neutropenia	D70.9
	HIV infection	B24
1.3.	**Inflammatory diseases**	
	Epidermolysis bullosa acquisita	L12.3
	Inflammatory bowel disease	K50, K51.9, K52.9
2.	**Other systemic disorders that influence the pathogenesis of periodontal diseases**	
	Diabetes mellitus	E10 (type 1), E11 (type 2)
	Obesity	E66.9
	Osteoporosis	M81.9
	Arthritis (rheumatoid arthritis, osteoarthritis)	M05, M06, M15-M19
	Emotional stress and depression	F32.9
	Smoking (nicotine dependence)	F17
	Medications	
3.	**Systemic disorders that can result in loss of periodontal tissues independent of periodontitis**	
3.1.	**Neoplasms**	
	Primary neoplastic diseases of the periodontal tissues	
	– Oral squamous cell carcinoma	C03.0 – 1
	– Odontogenic tumors	D48.0
	– Other primary neoplasms of the periodontal tissues	C41.0
	Secondary metastatic neoplasms of the periodontal tissues	C06.8
3.2.	**Other disorders that may affect the periodontal tissues**	
	Granulomatosis with polyangiitis	M31.3
	Langerhans cell histiocytosis	C96.6
	Giant cell granulomas	K10.1
	Hyperparathyroidism	E21.0
	Systemic sclerosis (scleroderma)	M34.9
	Vanishing bone disease (Gorham-Stout syndrome)	M89.5

(Continues)

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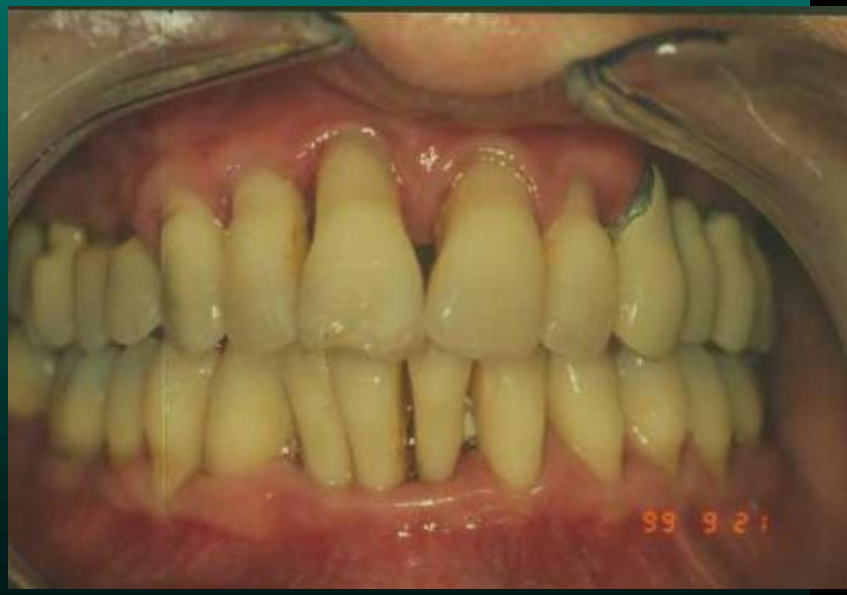
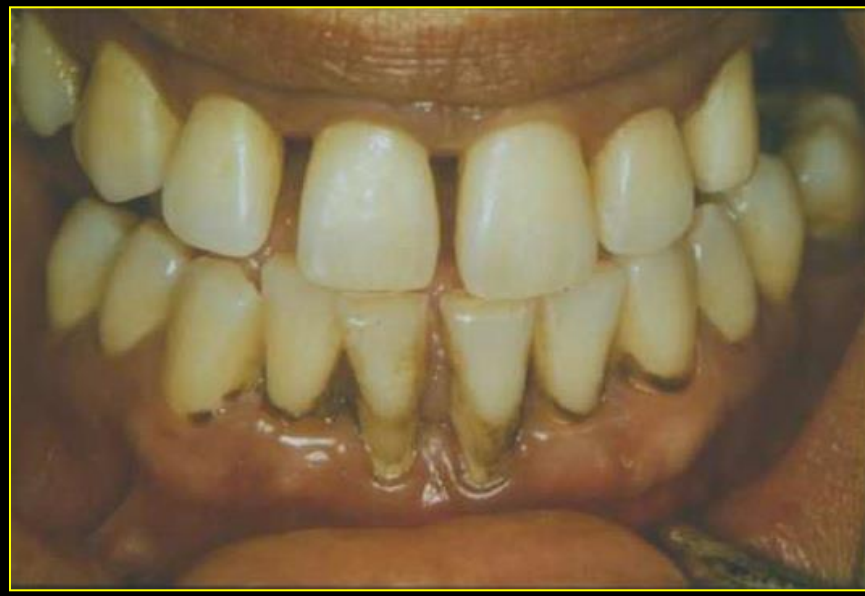


ASSOCIATED WITH DIABETES

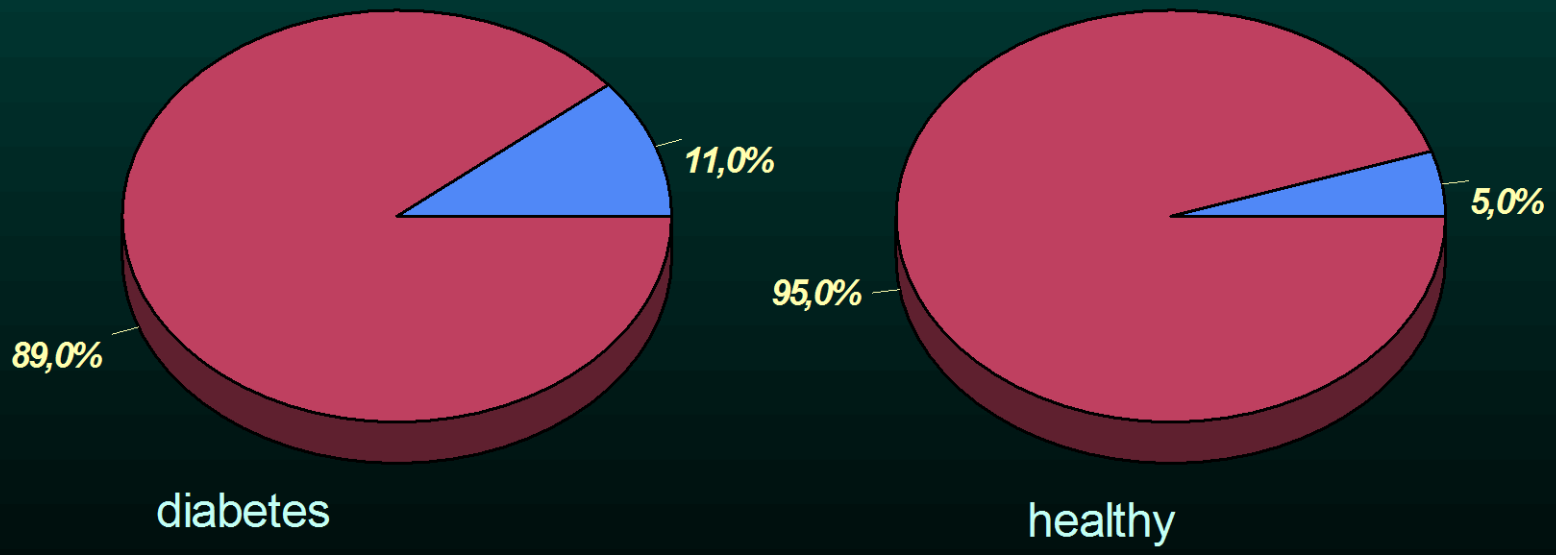


- retinopathy
- neuropathy,
- nephropathy
- angiopathy,
- atherosclerosis,
- Compromised wound healing
- ***periodontitis***
- .

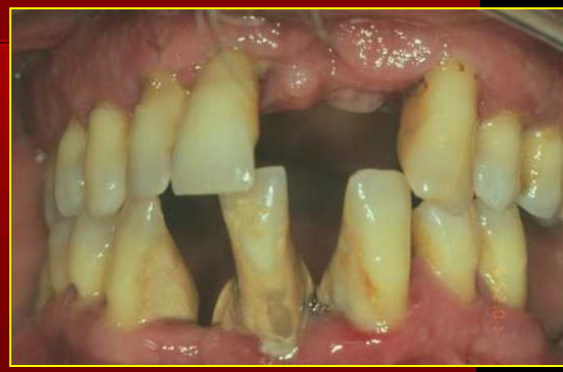
Löe H. Periodontal disease. The sixth complication of diabetes mellitus. Diabetes care 1993; 16: 329-334.

The occurrence of severe periodontitis in diabetic and non diabetic population

the prevalence of attachment loss >5mm

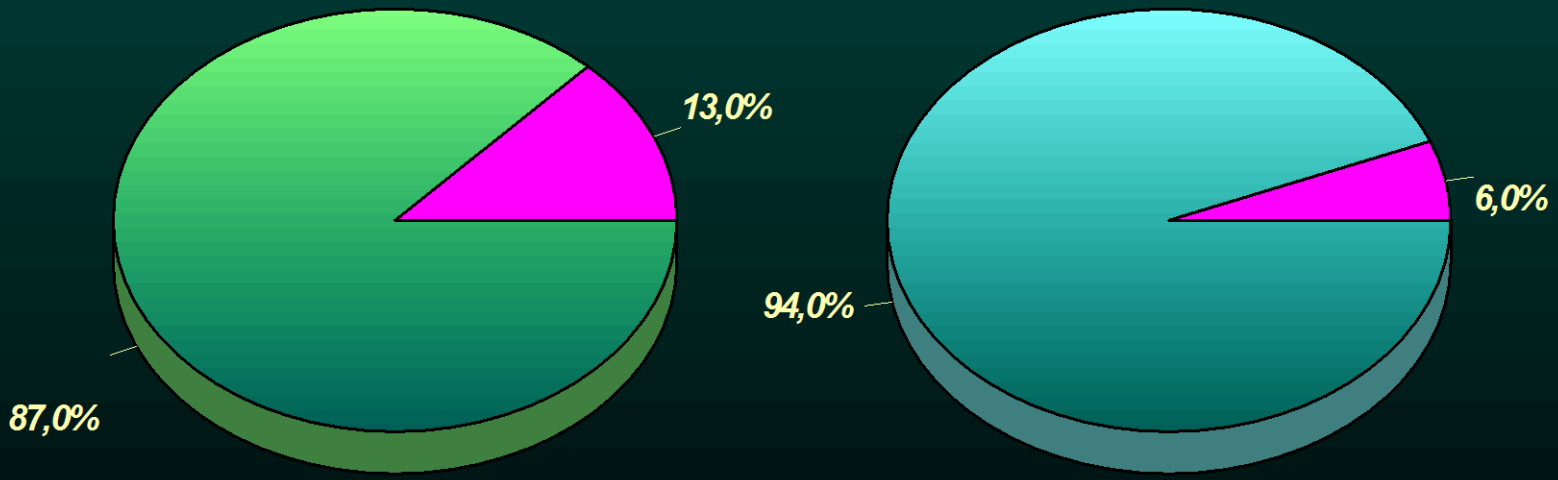


DIABETES A RISK FACTOR FOR PERIODONTITIS



Khader Y, Dauod A, El-Qaderi S, Alkafajei A, Batayha W. Periodontal status of diabetics compared with nondiabetics: a meta-analysis. *J Diabetes Complications* 2006: **20**: 59–68.

The prevalence of Diabetes among patients with severe periodontitis and those with healthy periodontium



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 enhance
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

TYPE II DM ABD PERIODODNTAL ATTACHMENT LOSS

- NHANES III USA
- 4343 subjects 45–90 years of age
- Significant correlation between the blood sugar level and attachment

- HbA1C>9% vs. HbA1C <9%.

Tsai, C., Hayes, C. & Taylor, G. W. (2002) Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population. *Community Dentistry and Oral Epidemiology* **30**, 182–192..

there is no difference in the composition of biofilm between diabeteic and non diabeteic groups

Zambon JJ, Reynolds H, Fisher JG, Shlossman M, Dunford R, Genco RJ. Microbiological and immunological studies of adult periodontitis in patients with non-insulin dependent diabetes mellitus. *J Periodontol* 1988: **59**: 23–31.

Sastrowijoto SH, Hillemans P, van Steenbergen TJ, Abraham-Inpijn L, de Graff J. Periodontal condition and microbiology of healthy and diseased periodontal pockets in type 1 diabetes mellitus patients. *J Clin Periodontol* 1989: **16**: 316–322.

Experimental gingivitis in diabetic and non-diabeteic subjects

- After 21 days of toothbrushing cessation in the type I diabetic groups 16 times higher BOP scores were detected A
- There was no difference between the bacterial sampling (Socransky et al. 1998)
- I type I diabetec group severe inflammation develops much more earlier

Salvi, G. E., Kandylaki, M., Troendle, A., Persson, G. R. & Lang, N. P. (2005) Experimental gingivitis in type 1 diabetics. A controlled clinical and microbiological study. *Journal of Clinical Periodontology* **32**, 310–316.

The primary cause is hyperglycaemia

USA III NHANES

Type II diabetes non controlled 2,9 ODDS

Type II controlled no differences

Type II diabetes non controlled+ obese 11 ODDS,

Two years follow-up

Non-controlled Within two years very severe bone loss

Controlled no differences

Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M, Knowler WC, Pettitt DJ. Non-insulin dependent diabetes mellitus and alveolar bone loss progression over 2 years. *J Periodontol* 1998: **69**: 76–83.

Tsai C, Hayes C, Taylor GW. Glycemic control of type 2 diabetes and severe periodontal disease in the US adult population. *Community Dent Oral Epidemiol* 2002: **30**: 182–192.

The primary cause is hyperglycaemia

hyperglycaemia

- protein-glycates
- binding to high affinity membrane receptors
- increase interleukin production
- phagocytes

Markedly enhanced

endothelial cell permeability

adhesion molecules expression

decreased fibroblast collagen production

Devaraj S, Venugopal SK, Singh U, Jialal I. Hyperglycemia induces monocytic release of interleukin-6 via induction of protein kinase C-α and -β. *Diabetes* 2005: **54**: 85–91.

The primary cause is hyperglycaemia

- Decreased PMN adhesion
- Disturbed PMN chemotaxis
- decreased bacterial clearance in the a sulcus
- Increased tissue damage
- Hyper reactive monocytes – enhanced pro-inflammatory cytokine production

Salvi GE, Yalda B, Collins JG, Jones BH, Smith FW, Arnold RR, Offenbacher S. Inflammatory mediator response as a potential risk marker for periodontal diseases in insulin-dependent diabetes mellitus patients. *J Periodontol* 1997: **68**: 127–135.

116.
Salvi GE, Collins JG, Yalda B, Arnold RR, Lang NP, Offenbacher S. Monocytic TNF-*α* secretion patterns in IDDM patients with periodontal diseases. *J Clin Periodontol* 1997: **24**: 8–16.

A hyperglycaemia has a negative effect on the bone metabolism

Compromised gene expression responsible for osteoblast differentiation

- Decreased growth hormone production
- Decreased extracellular matrix (collagen) production
- Consequently - decreased new bone formation

Bouillon R. Diabetic bone disease. *Calcif Tissue Int* 1991: **49**: 155–160.

Kawaguchi H, Kurokawa T, Hanada K, Hiyama Y, Tamura M, Ogata E, Matsumoto T. Stimulation of fracture repair by recombinant human basic fibroblast growth factor in normal and streptozotocin-diabetic rats. *Endocrinology* 1994: **135**: 774–781.

Santana RB, Xu L, Chase HB, Amar S, Graves DT, Trackman PC. A role for advanced glycation end products in diminished bone healing in type 1 diabetes. *Diabetes* 2003: **52**: 1502–1510.

Periodontitis and type II diabetes mellitus

SEVERE PERIODONTAL INFLAMMATION INCREASES TISSUE INSULIN RESISTANCE

IN PERIODONTITIS THE GRAM-NEGATIVE INFECTION AND CHRONIC ENDOTOXEMIA ELEVATE TISSUE INSULIN RESISTANCE AND WORSENS PATIENTS' METABOLIC CONTROLS

Grossi és mts. Response to periodontal therapy in diabetics and smokers *J Periodontol 1996; 67: 1094-1102*

Periodontitis and type II diabetes mellitus metabolic control

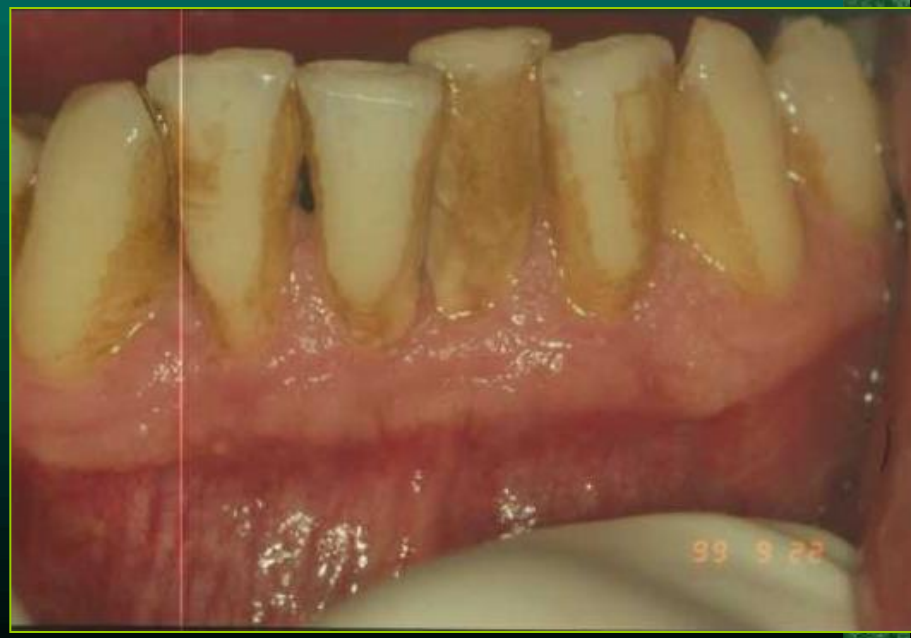
successful comprehensive periodontal therapy and regular periodontal maintenance in patients with type II DM potentiated the effect of anti-hyperglycamic therapy

Stewart et al. The effect of periodontal treatment on glycemic control in patients with ty diabetes mellitus. *J Clin Periodontol 2001; 28: 306-310*

Iwamoto Y et al: The effect of antimicrobial periodontal treatment on circulating tumor necrosis factor alpha and glycated hemoglobin level in patients with Type 2 diabetes *Periodontol 2001;72: 774-778.*

RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

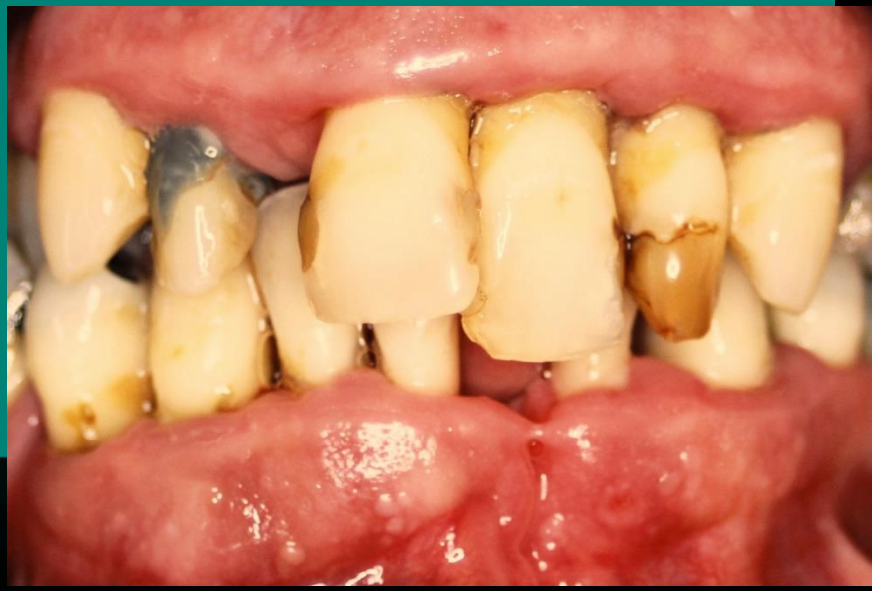
- Oral hygiene
  Local plaque retentive factors
  Bacterial specificity
  Systemic immune status
  Diabetes mellitus
  Tobacco smoking
  Osteoporosis
  Etnic background
  Age
  Diet
  Genetics
- Stress
  Social-economics



Tobacco smoking

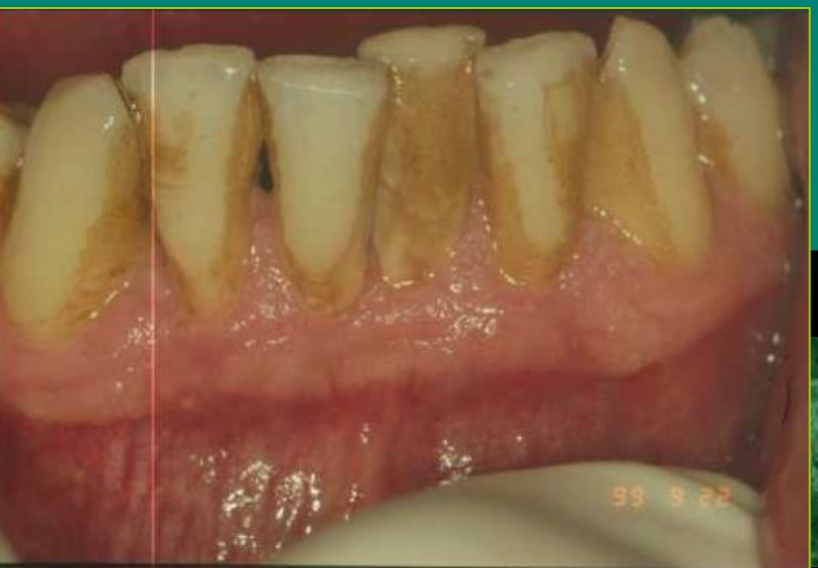
In the past - we believed that only the inferior oral hygiene of smokers was responsible for the worse periodontal status

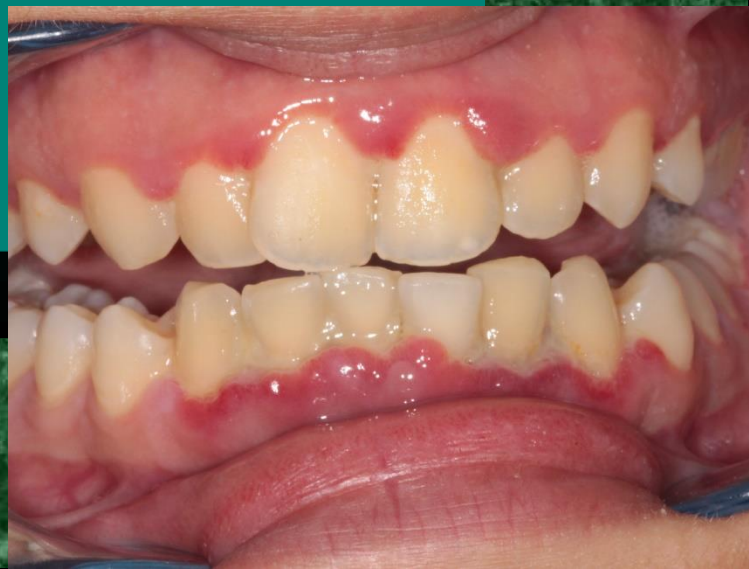
Now - several evidences are indicating a direct metabolic effect of smoking on periodontal health state



Smoking → Gingivitis

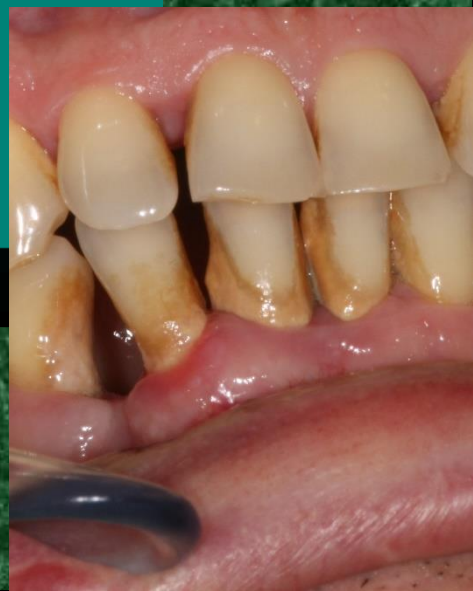
- Close correlation between smoking and ANUG
- Humane experimental gingivitis studies showed a marked reduction in inflammatory response in the gingiva to plaque accumulation in smokers
  Much less severe marginal gingivitis develops in smokers than in non-smokers
- Cross sectional studies also indicated that smokers have less severe gingivitis than non smokers



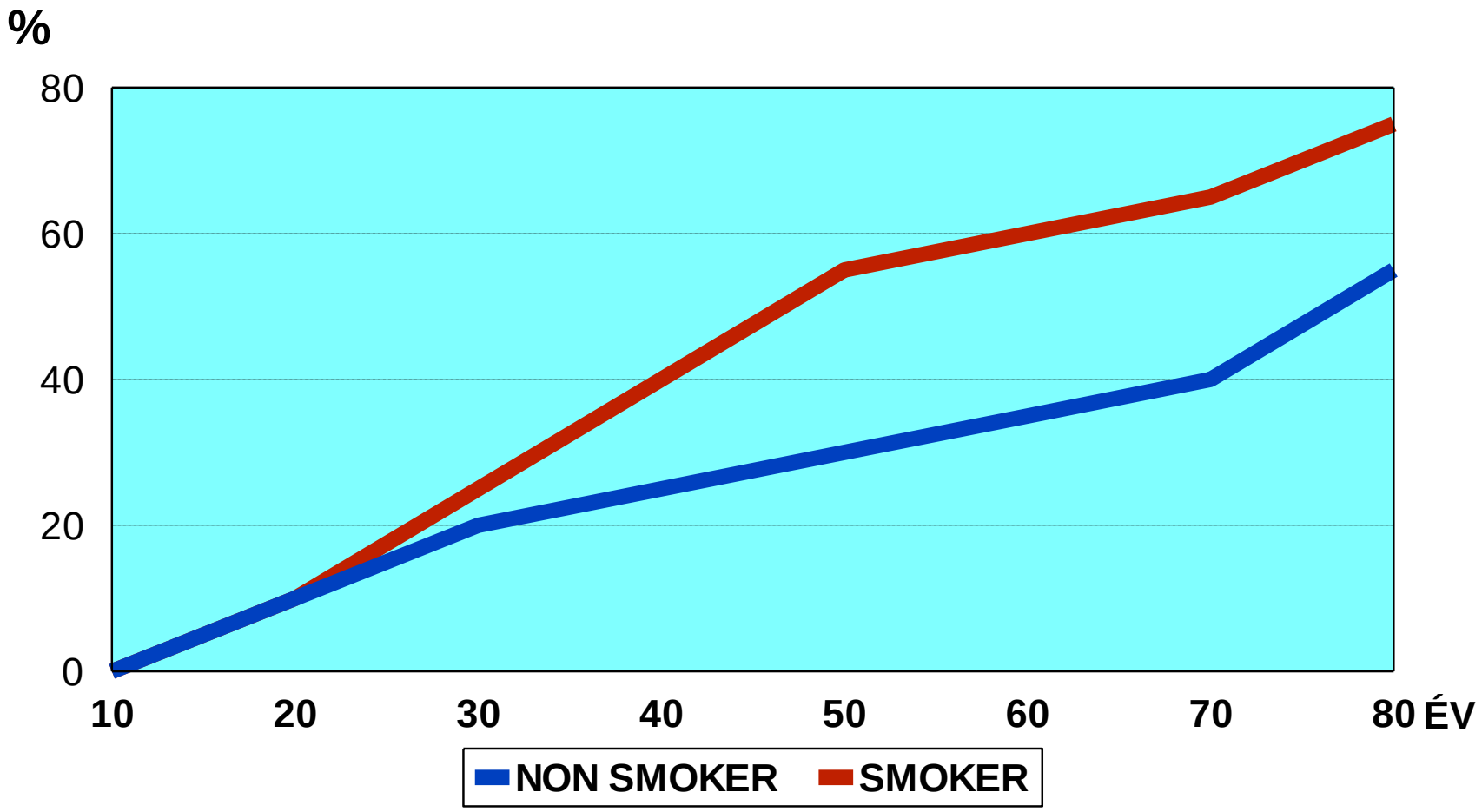


Smoking → Periodontitis

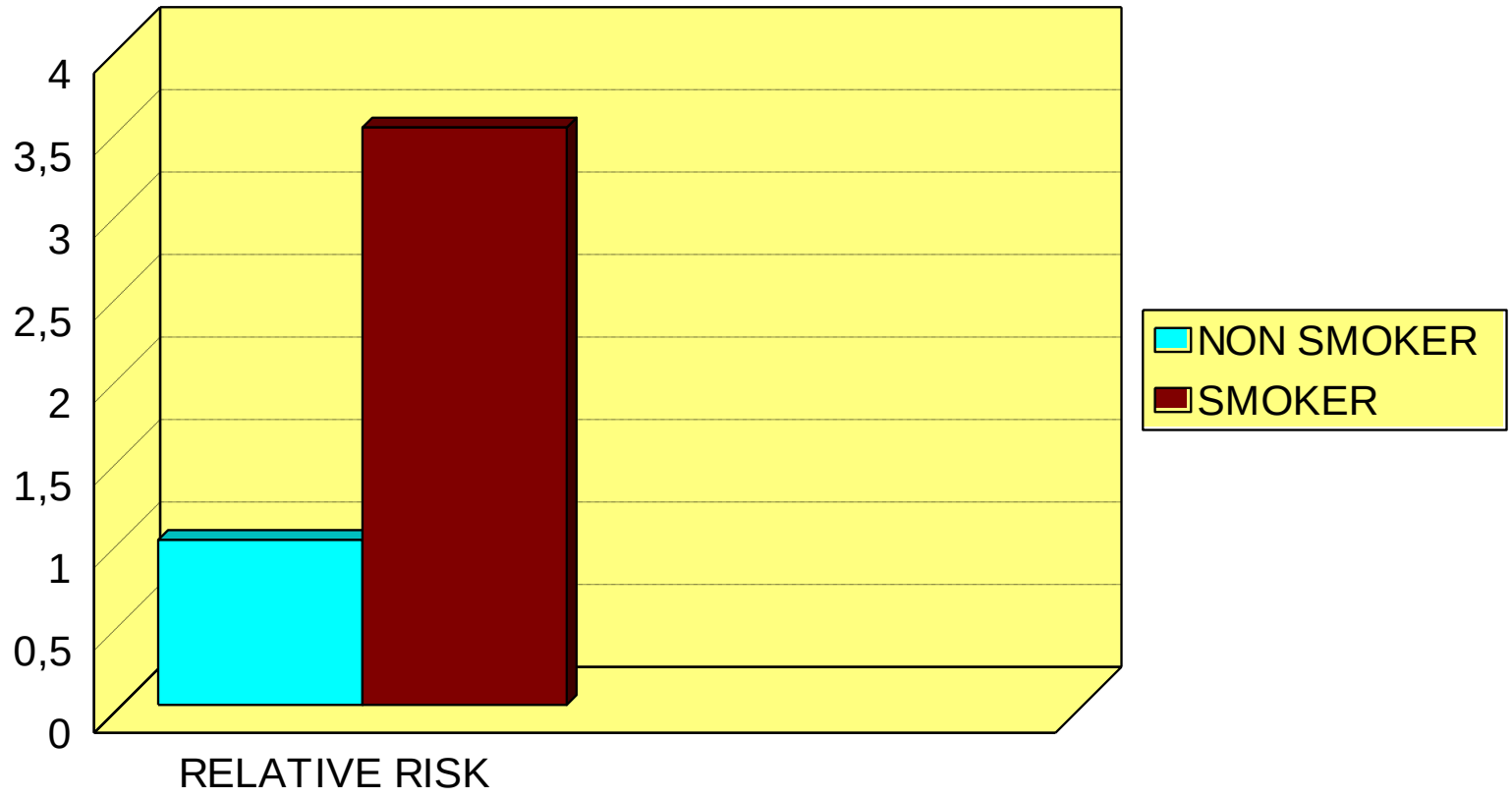
- Severe destructive periodontitis is more common in smokers than in matched non smokers
- The age matched smoker population shows a significantly higher average periodontal index score than non smokers
- The extent and severity of periodontal bone loss is more severe in smokers than in age matched non smokers



THE RATE OF ALVEOLAR BONE LOSS RELATED TO THE AGE



THE ATTRIBUTABLE RELATIVE RISK FOR SMOKING IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTITIS



Oral health in young adults in the German army.

- Oral hygiene (QHI) was poor in the entire study population
- In both females and males half of the participants were Smokers (56.7%)
- Smoking had a negative effect on periodontal health
- Men showed poorer oral health than women.
- The great proportion of young adults requiring periodontal treatment is high in Germany.

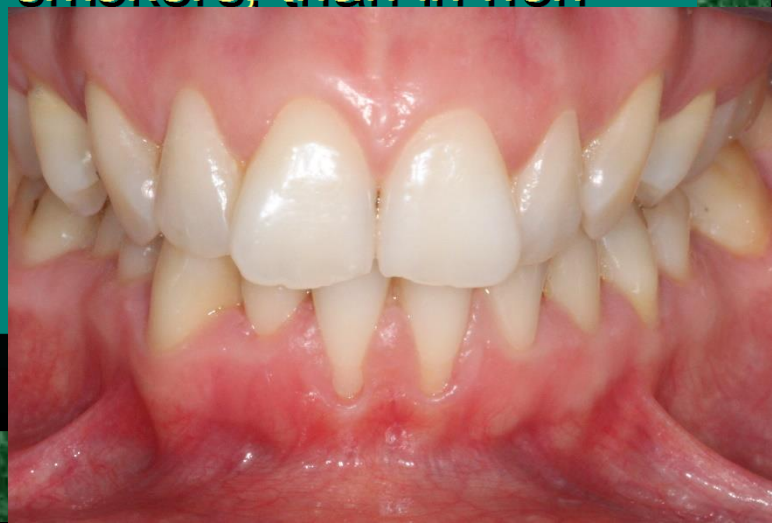
Ziebolz D, et al.: **Oral health in young adults in Germany--a comparison between women and men of the German army.** Schweiz Monatsschr Zahnmed. 2008;118(10):944-50

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 → Periodontitis

- 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 n



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.
- Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis and Prevotella intermedia no significant differences in the prevalence of these microorganisms between smokers and nonsmokers in deep pockets.
- Enhances the accumulation of periodonto-pathogenic microorganisms in relatively shallow pockets (>4mm)
- The relative and absolute number of anaerobic microorg anisms are increased in deep pockets by smoking

Smoking → Physiology

- Decreased vascular responses
- Decreased subgingival temperature
- Slower tissue responses

Smoking → Immunology

- PMN lekocyte count increased
- CRP increased in smokers
- PMN leukocytes chemotactic activity is decreased
- Increased local TNFa, PGE production
- Increased MMP-8 production by PMN cells
- Responding to LPS stimuli the monocytes produces more PGE

The effect of smoking on the outcome of periodont 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

The effect of smoking on the outcome of periodont therapy and periodontal regeneration

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

RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

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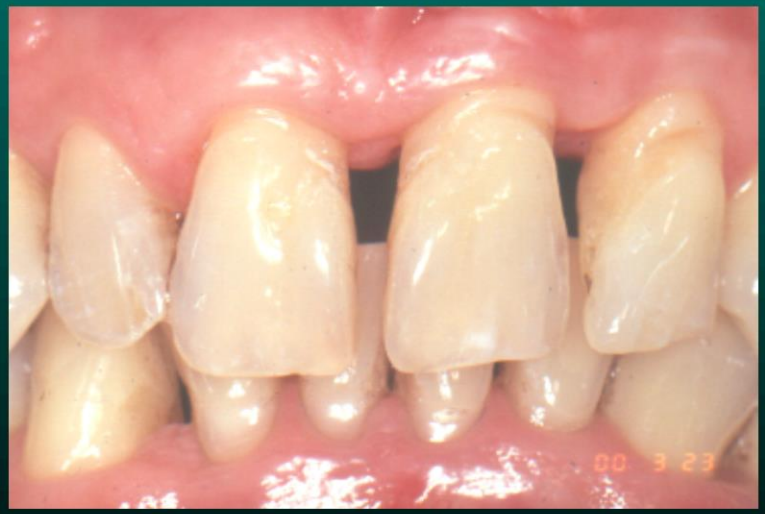


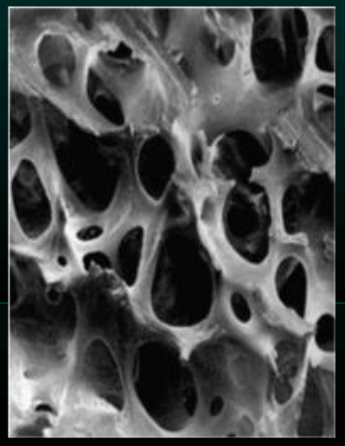
Osteoporosis

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

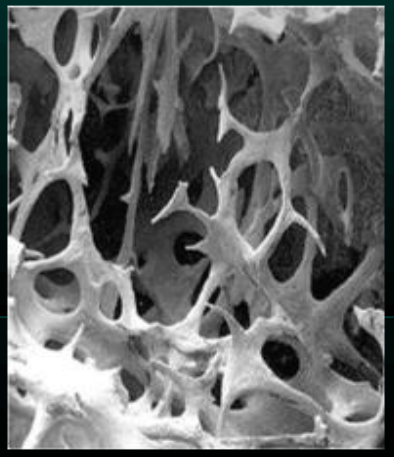
45% of women with severe generalized osteoporosis were edentulous
while only 15% were edentulous among non osteoporotic matched controls

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



Intact Trabecular Structure



Deteriorated Trabecular Structur

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
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 osteoclats 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.

Smoking → Osteoporosis

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

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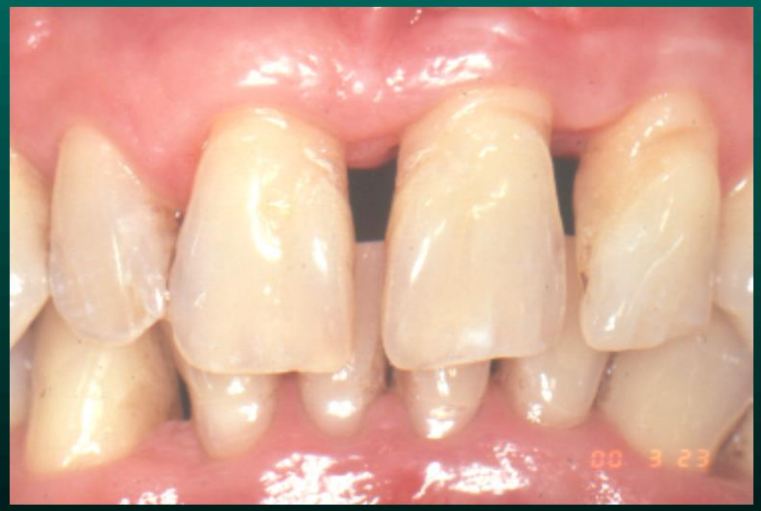


Ethnic group

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.

Ethnic group

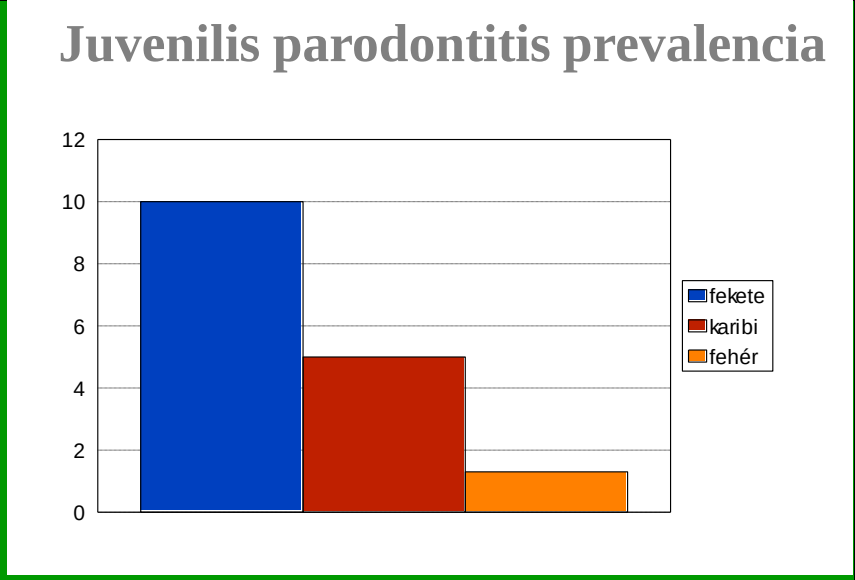
According to another US study the occurrence of 5mm deep pocket was 330% more frequent among blacks than in whites

The prevalence of the juvenile aggressive periodontitis is 10 % among black teenagers while only 5% among Caribbeans and only 1,3 among Caucasians.

juvenie parodontitis

BLACK TEENAGERS 10%-os

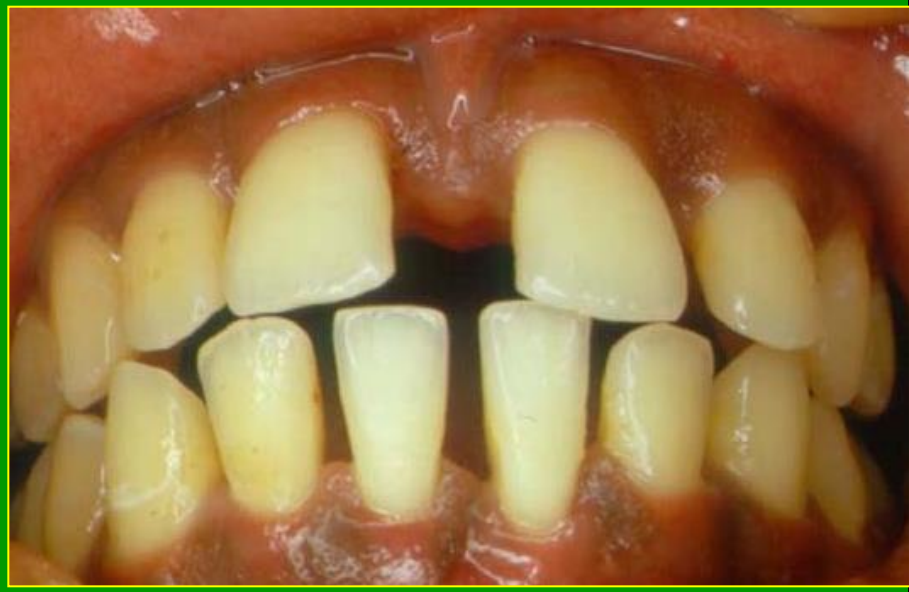
WHITE TEENAGERS 1,3%



ALBANDER JM. Periodontal disease in North America. *Periodontol 2000* 2002;29:31-69.

.ALBANDER JM, BROWN LJ, LÖE H. Clinical features of early-onset periodontitis. J *Am Dent Assoc* 1997;128: 1393-1399.

.LÖE H, BROWN LJ Early onset periodontitis in the United States of America J Periodontol 1991;62: 608-616



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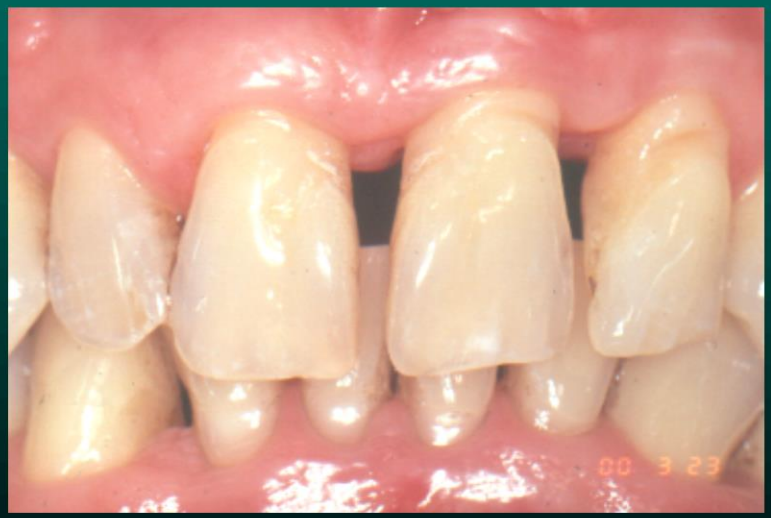


Age

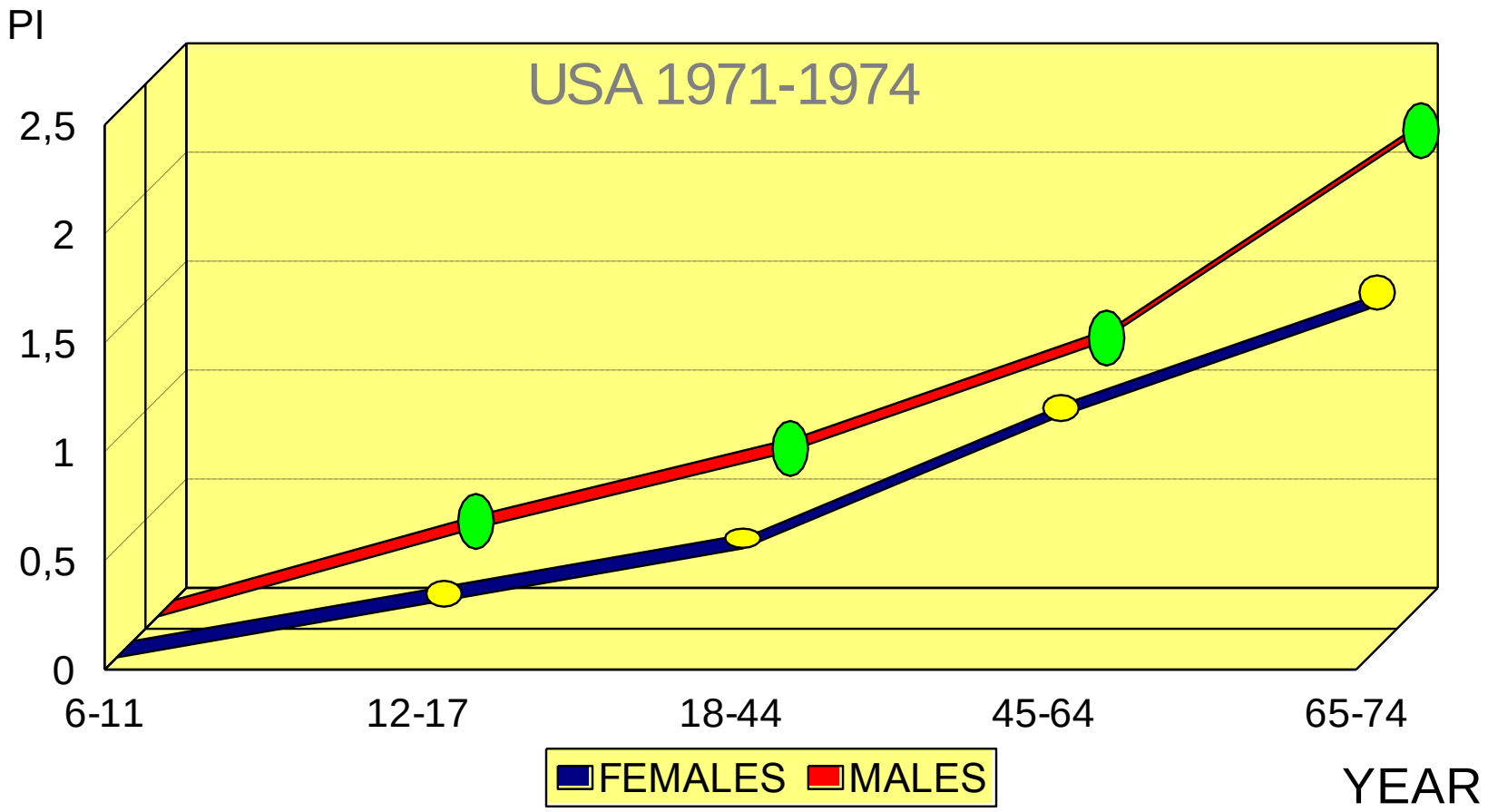
The average attachment loss is increasing parallel with the ages

The average pocket depth does not follow this trend

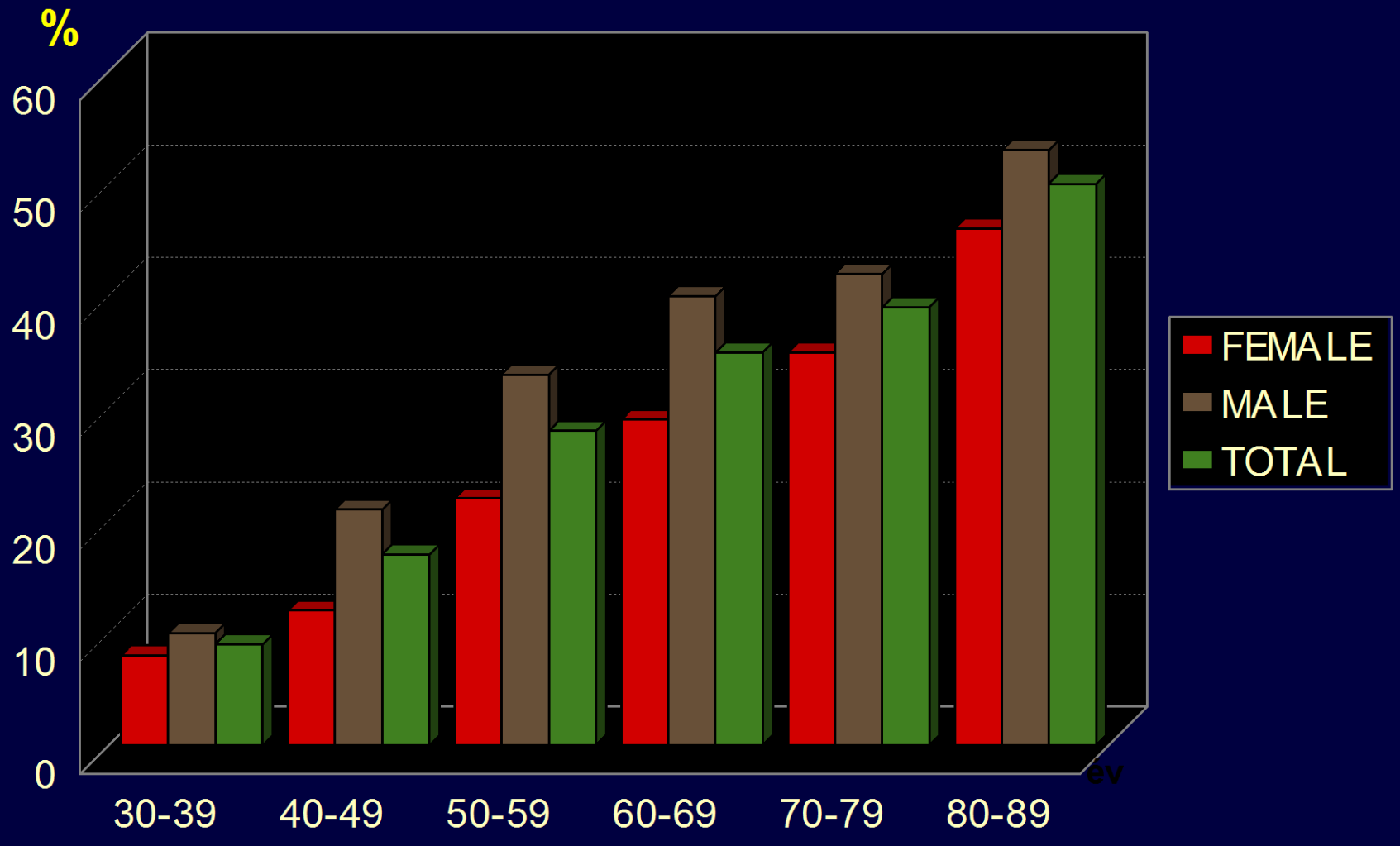
This is due to the gingival recession

The prevalence of the individuals with >4mm pocket depth does not increase with the 50-80 age period

RUSSELL PERIODONTAL INDEX ACCORDING TO AGE GROUPS

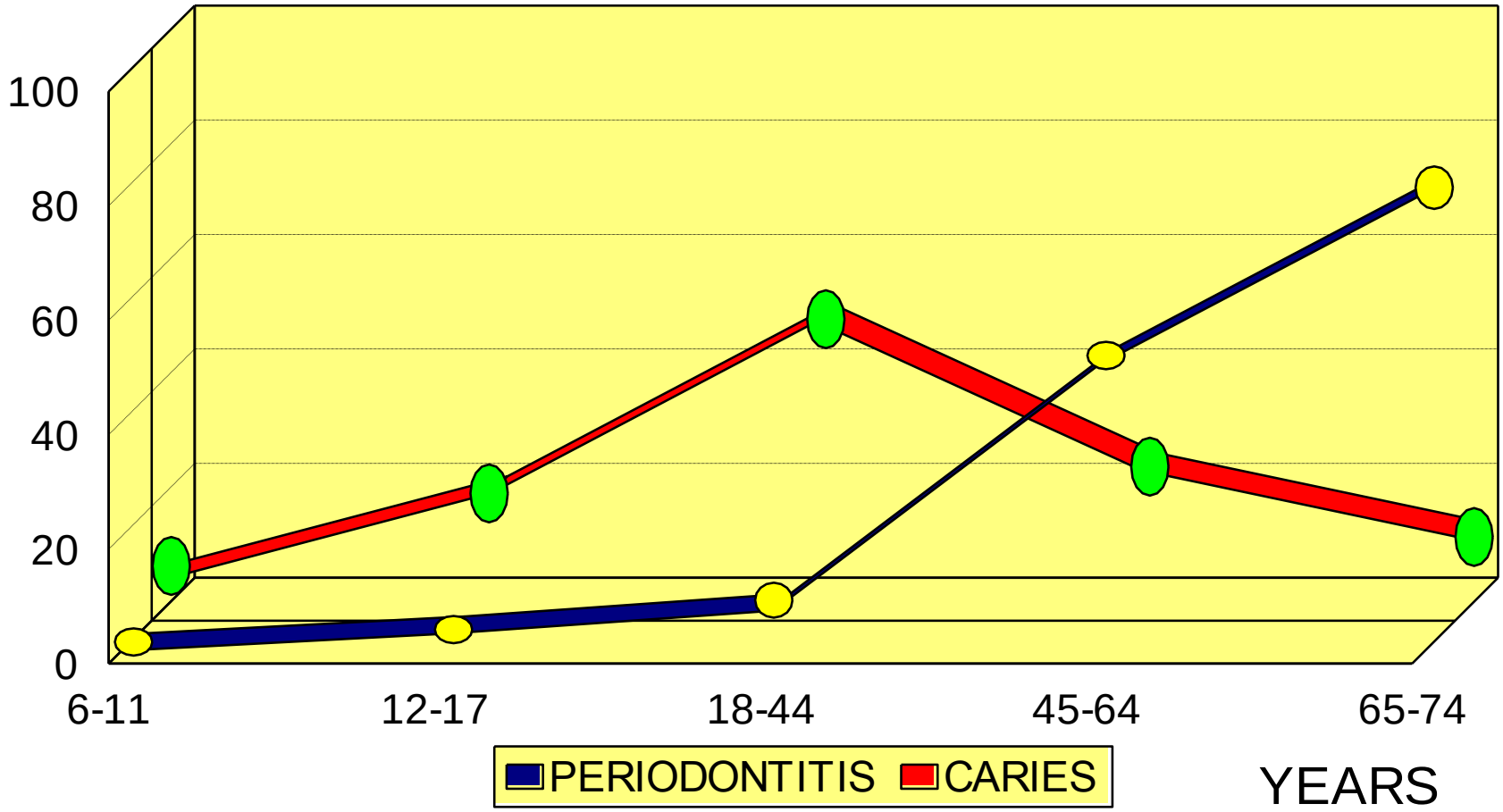


PERCENTAGE OF TEETH WITH ≥ 3 mm ATTACHMENT LOSS BY AGE



Albander et al. Destructive periodontal disease in adults 30 years of age and older in the US 1988-1994 J. Periodontol 1999;70-13.

TOOTH LOSS DUE TO CARIES OR PERIODONTAL DISEASE IN AGE GROUPS



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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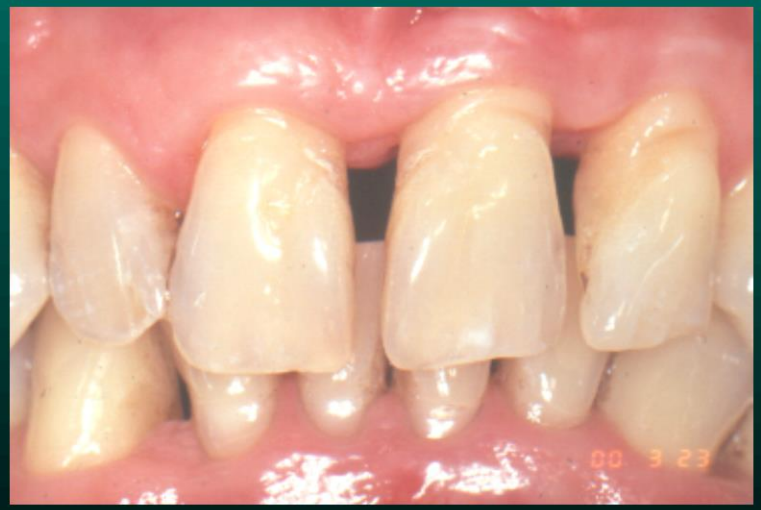


Diet

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 .

the physiological role of Vitamin C

Improved PMN cell functions

Vascular endothelial functions

Collagen matrix production- wound healing

VITAMIN –C deficiencies

Compromised periodontal collagen metabolism -regeneration

Decreased bone formation

Increased epithelial permeability –LPS

Sever vitamin C deficiencies - scorbutic

Compromised collagen remodelling

Decreased osteoid synthesis

Decreased osteoblast functions

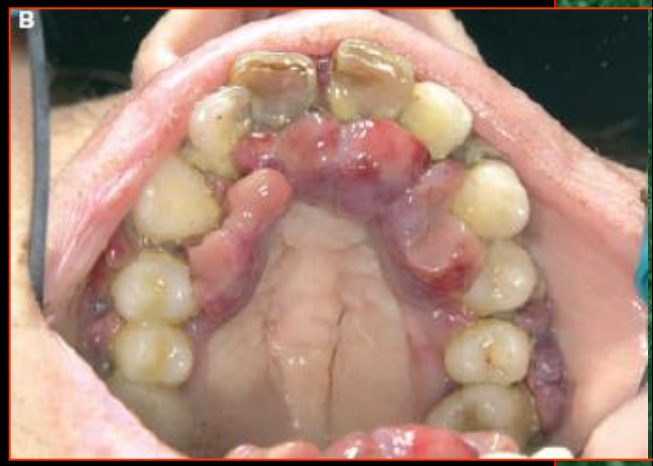
Increased capillary permeability

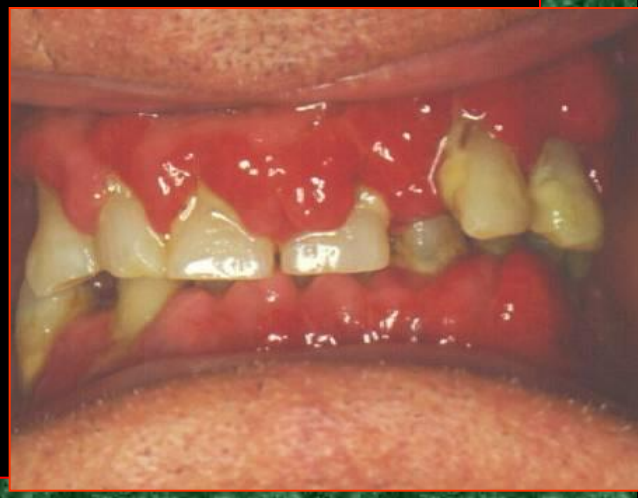
and capillary growth

Very severe gingival hyperplasia

Severe periodontal disease

Tooth loss





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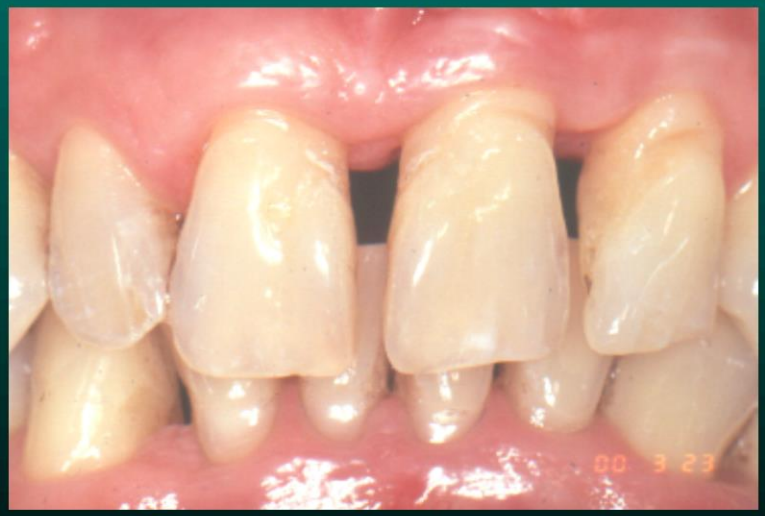
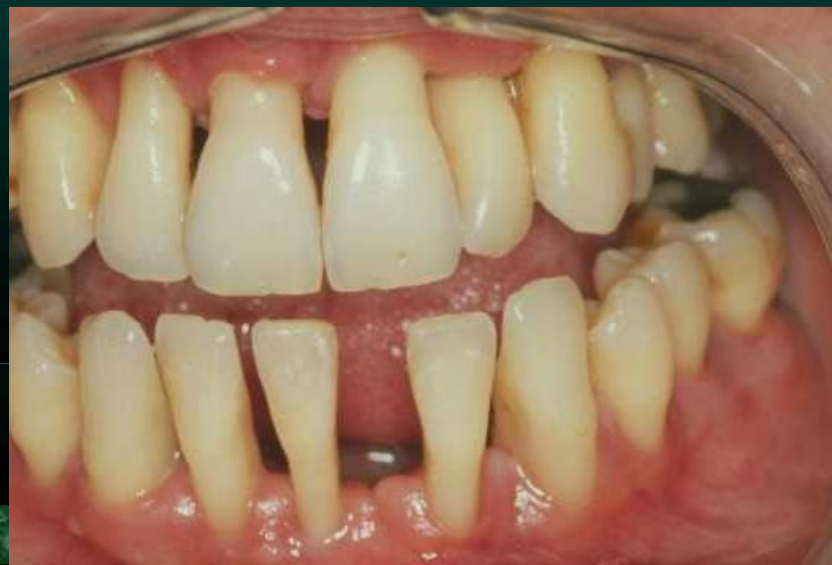


Genetic factors

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

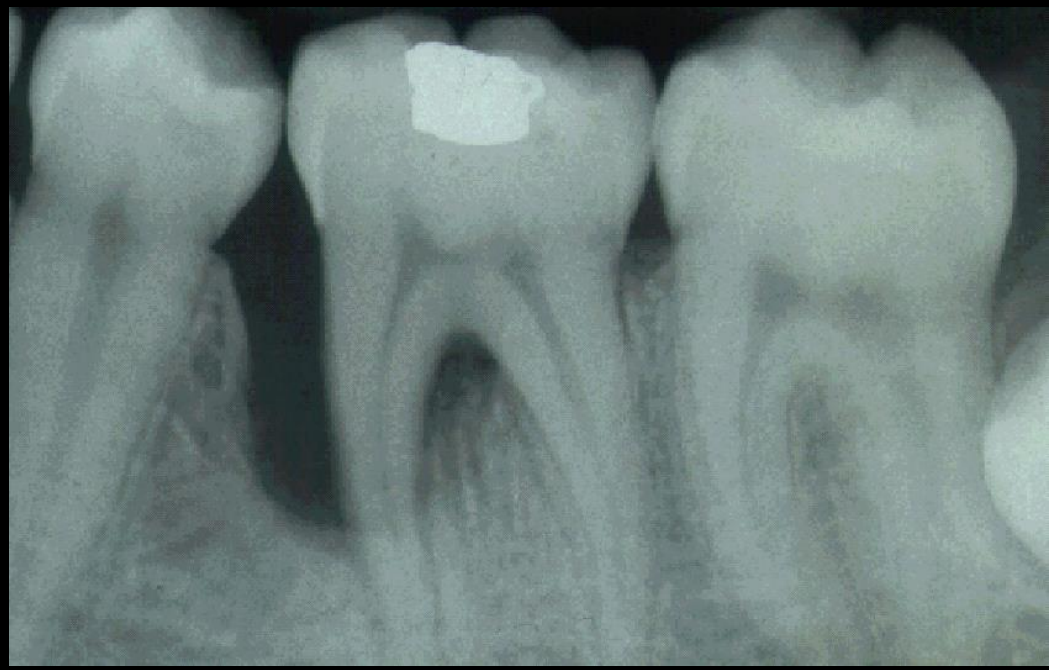
The role of genetic factors can easily be studied among identical twins

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

According to a survey in the (1994) the incidence of juvenile aggressive periodontitis ranged between 0,16 és 2,4% , while in certain families it was 40-50%.

LÖE H, BROWN LJ Early onset periodontitis in the United States of America

J Periodontol 1991; 62: 608-616



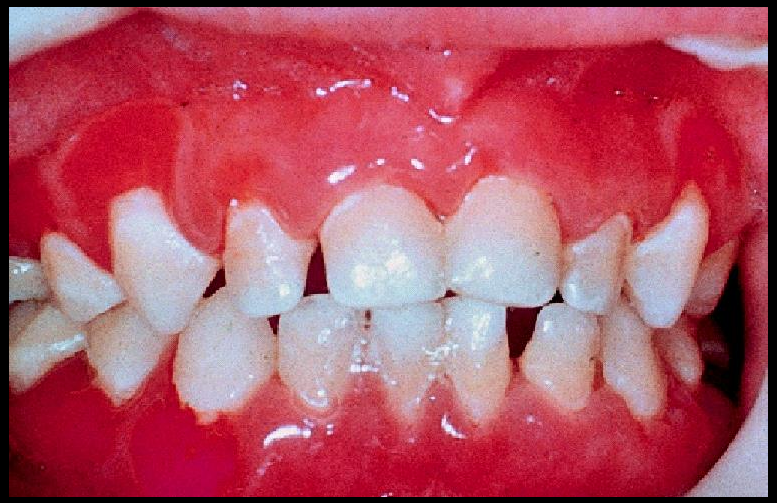
family tree study

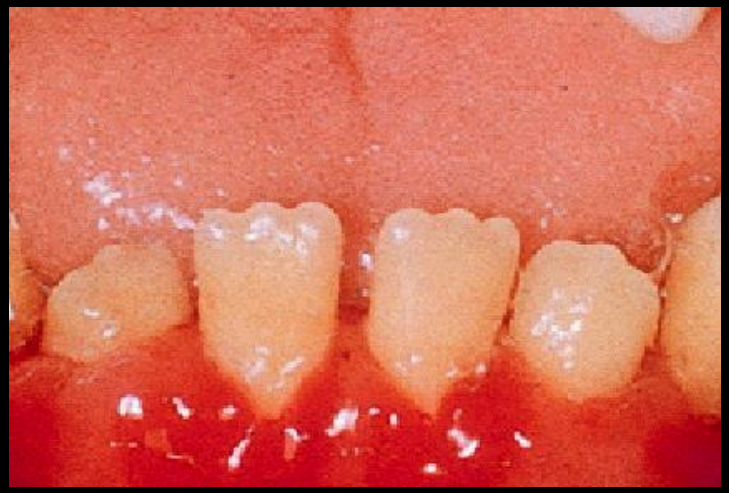
aggressive periodontitis

227 families - mainly Afro-Americans

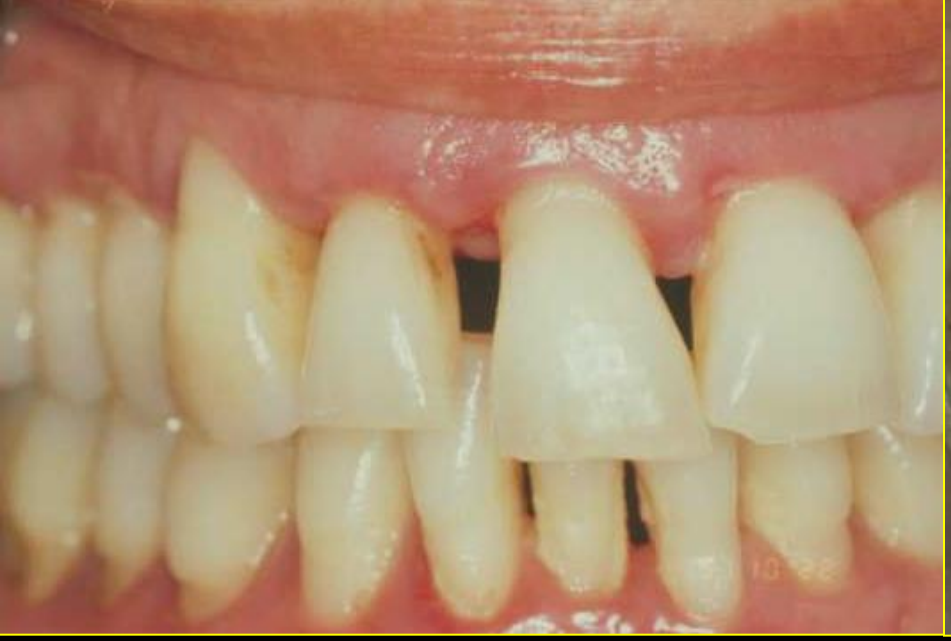
autosomal dominant trait (90%)

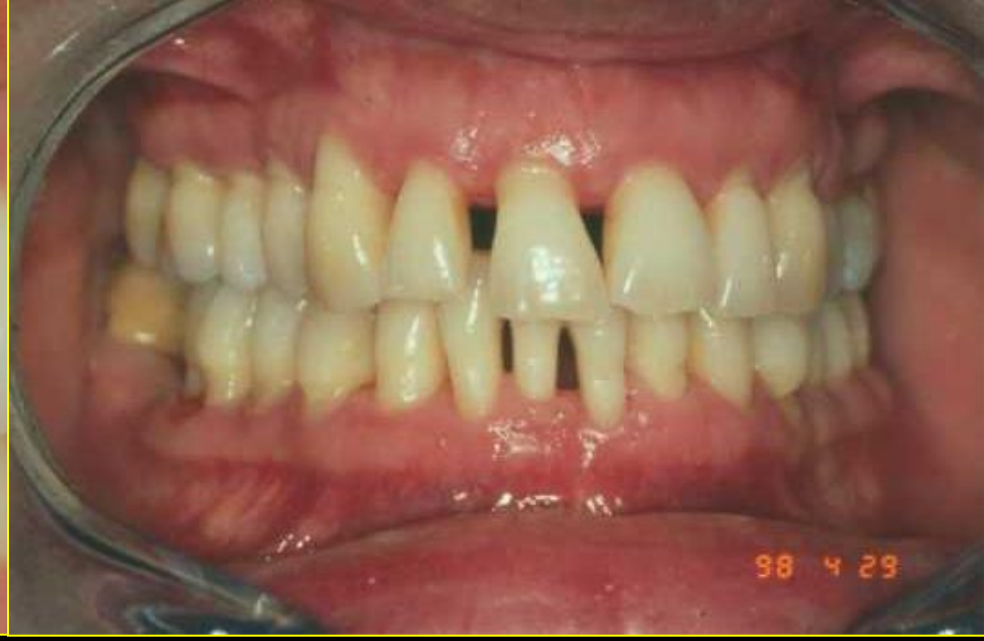
Marazita ML, Burmeister JA, Gunsolley JC, Koertge TE, Lake K, Schenkein HA. Evidence for autosomal dominant inheritance and race-specific heterogeneity in early-onset periodontitis. *J Periodontol* 1994; **65**: 623–630. .





Theoretically any gene combination that has effect on the innate or adaptive immune response might be either a susceptibility or severity risk factor

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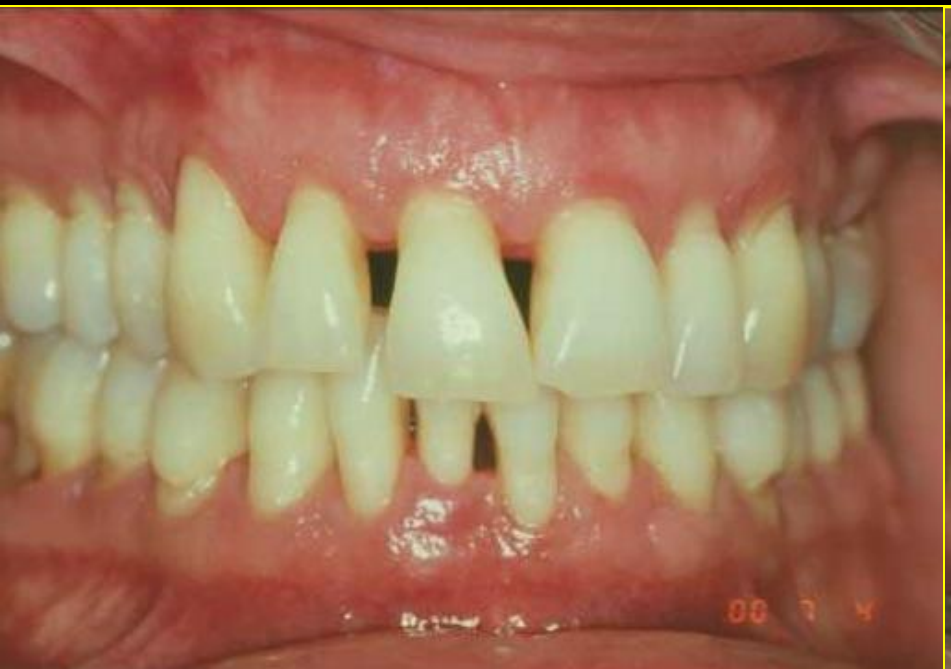

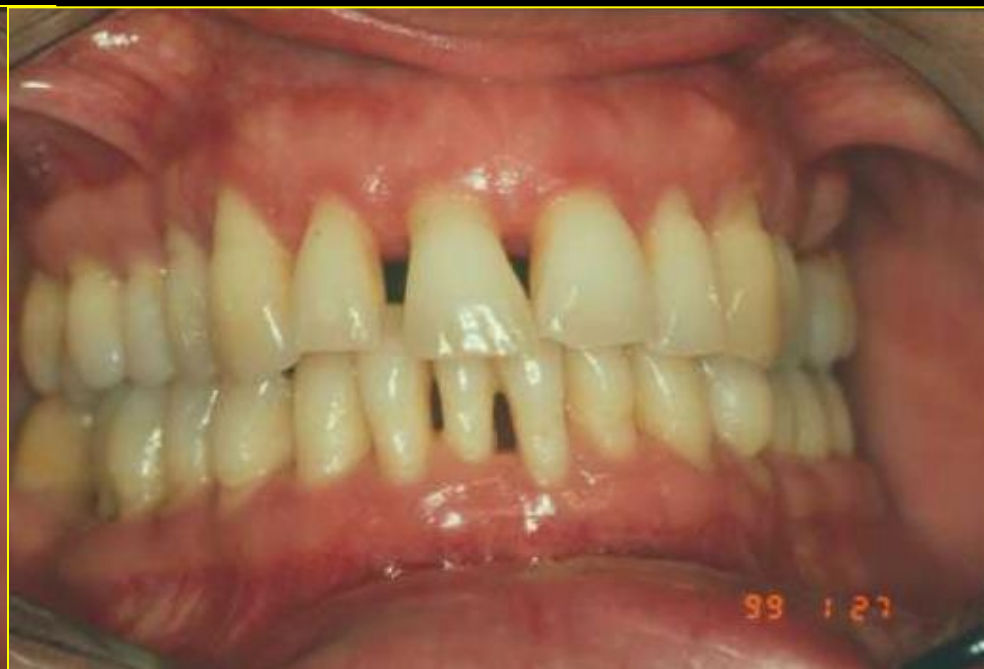

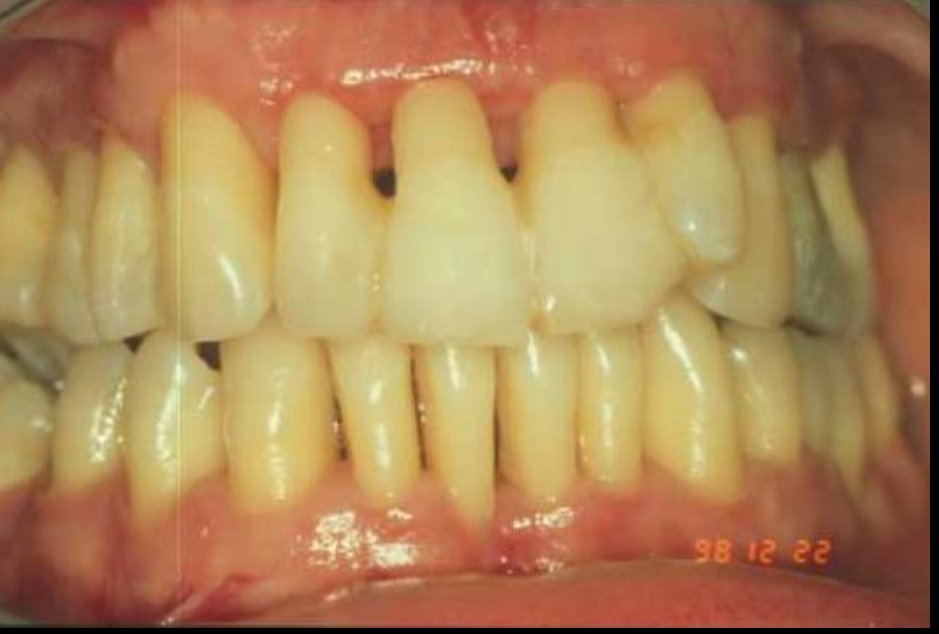
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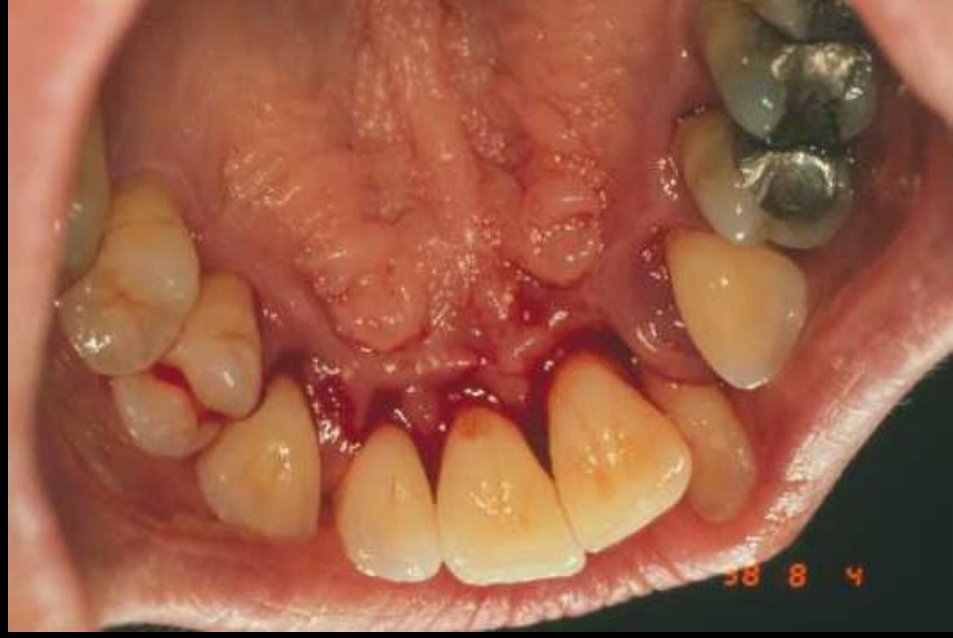
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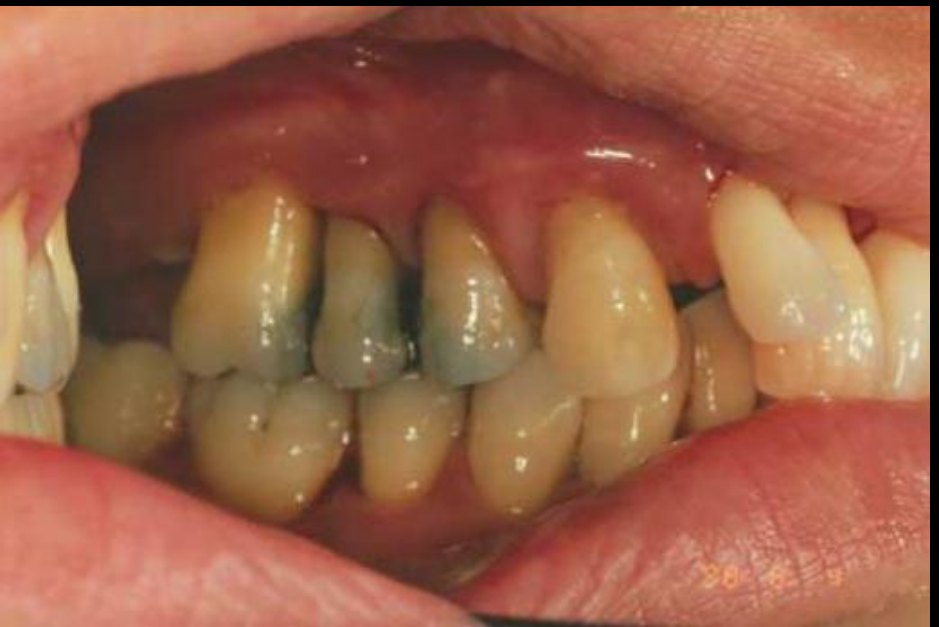

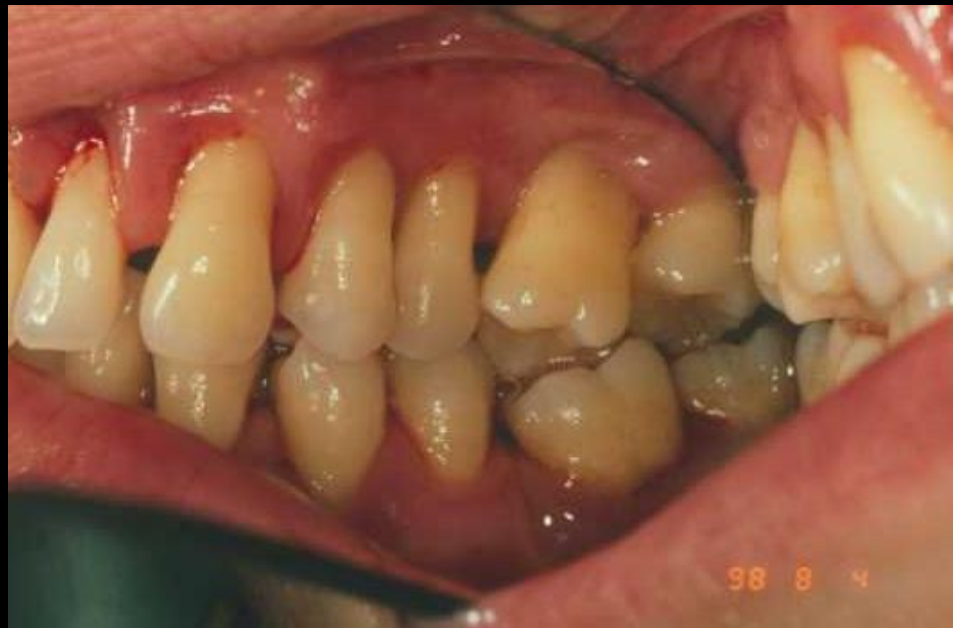
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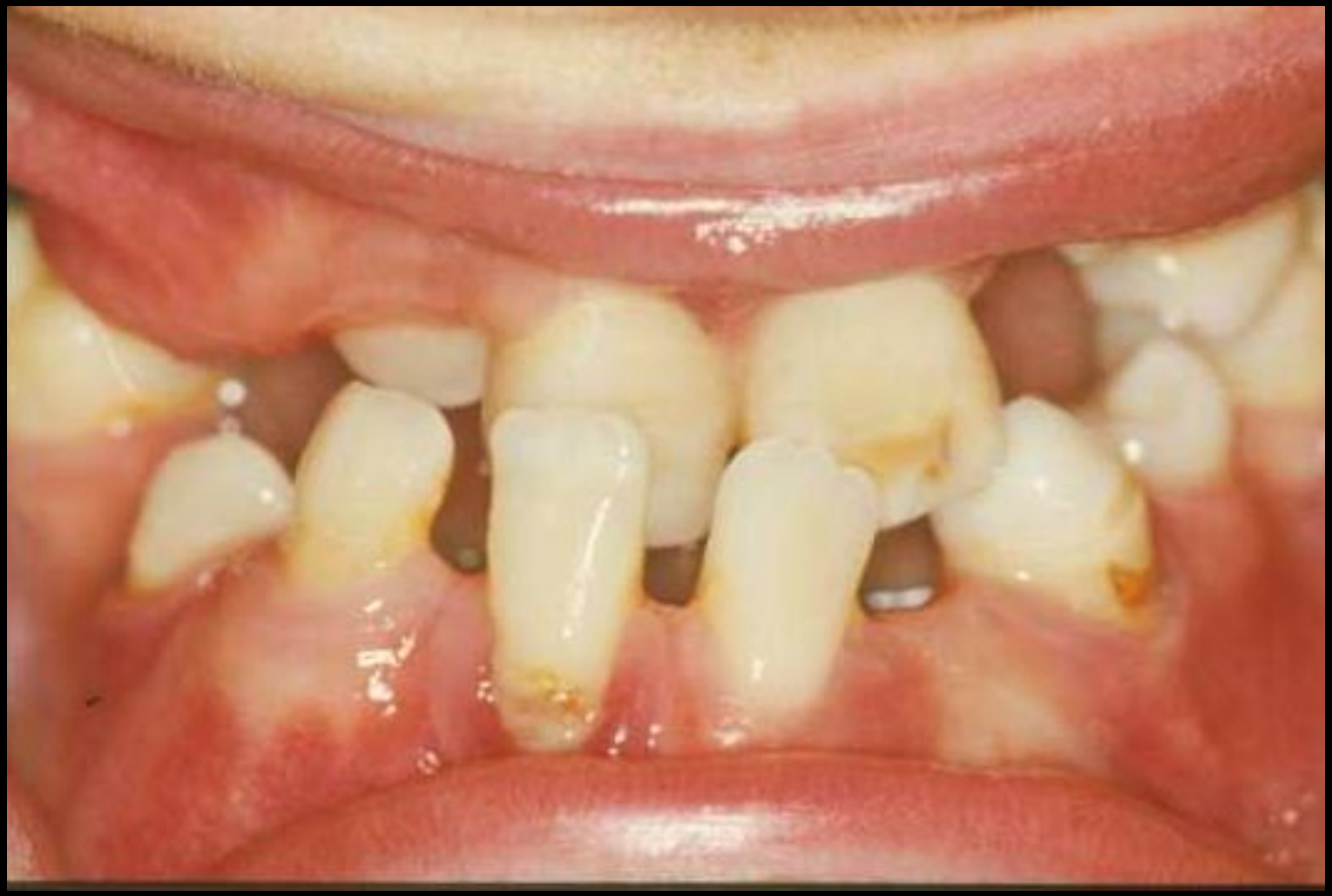
Most of the periodontitis with certain genetic backgrounds are polygenic

These will not follow the rule of the Mendelian inheritance

susceptibility allele

severity allele

DOWN-SYNDROME , (21-es chromosoma trisomy)



Down-syndrome, (21-es chromosoma trisomia)

•At first *Talbot* described in1899 that Down-disease (mongoloidism) associates with severe periodontal disease .

• *Ehmer* in 1976 studies the incidence of severe periodontal disease among Down-syndrome patients -

• between 3-6 years incidence 25%-ban,

• up to 15 years of age 81%

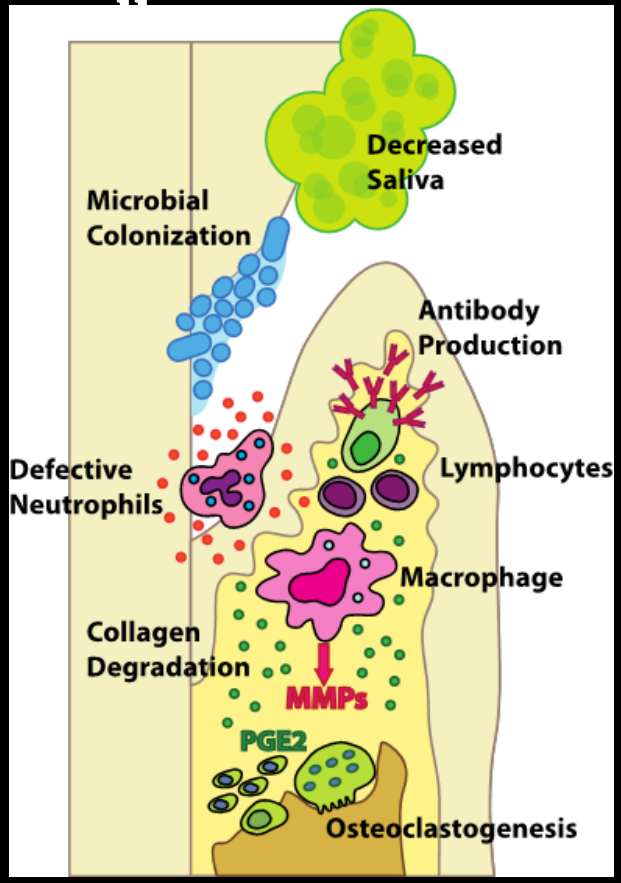
•More than 15 years 93,5%-

1. EHMER U: Condition of periodontal and oral higiene in trisomy 21. Zahn und Mund-Kiefer Helkd 1976; 63: 547-559.

Down-syndrome ,

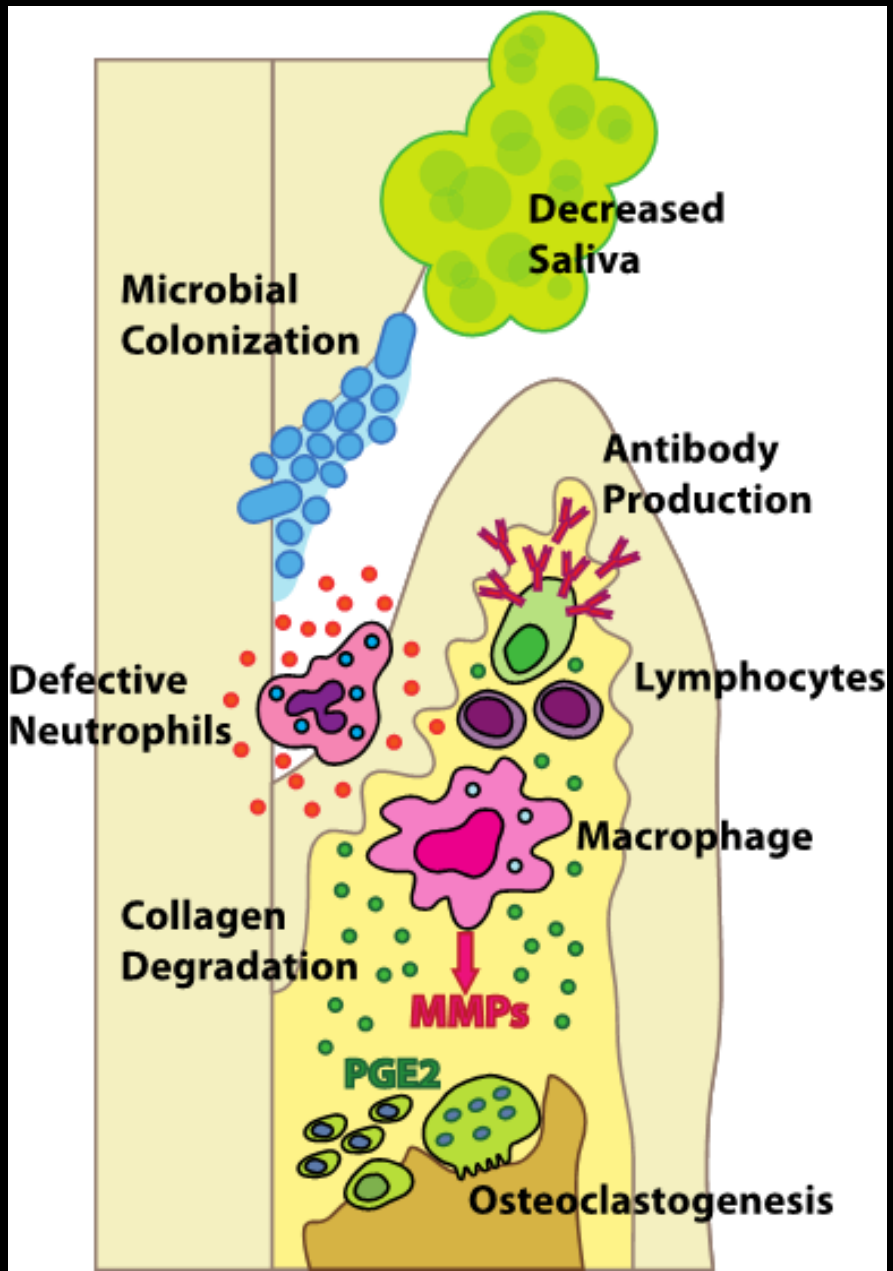
Compromised PMN leukocyte chemotactic activity and intracellular bacterium killing mechanisms

Decreased cellular immune response , decreased number of circulating T cells and decreased number of crevicular PMN



Down syndrome increased MMP-8 (neutrofil collagenaze) production that is also responsible for the periodontal destruction

Down-syndrome ,



- PMN LEUKOCYTES
  - **non matured**
  - **Shorter life cycle**
  - **Hampered chemotaxis**
  - **Hampered phagocytosis**

- **Enhanced PMN cytokin and MMP production**

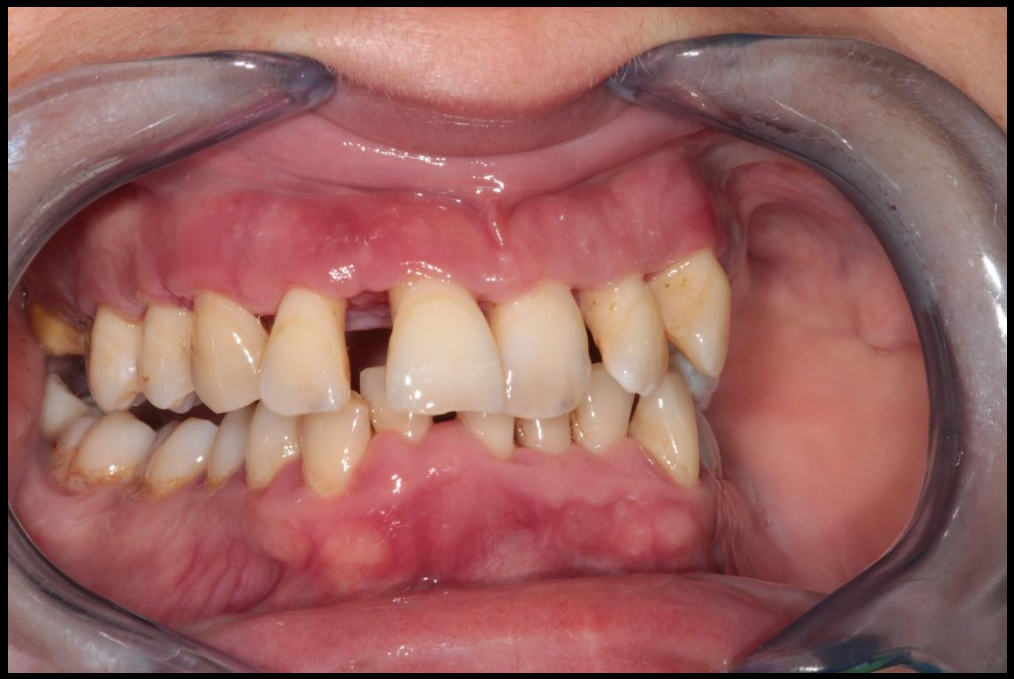
MENDELIAN INHERITANCE

Several monogenic syndromes associated with severe early onset aggressive periodontitis

Syndrome	Mutated gene	Chromosome region
Papillon–Lefèvre	Cathepsin C (*CTSC*)	11q14.1–q14.3
Chediak–Higashi	Lysosomal trafficking regulator *CHS1/LYST*	1q42.1–q42.2
Hypophosphatasia	*ALPL*	1p36.12
Congenital and cyclic neutropenia	*ELANE*	19p13.3
Leukocyte adhesion deficiency type I	Beta-2 integrin chain	21q22.3
Leukocyte adhesion deficiency type II	GDP-fucose transporter-1	11p11.2
Glycogen storage disease	*SLC37A4*	11q23.3
Ehlers–Danlos	Collagen alpha-1(V) gene (*COL5A1*) or the collagen alpha-2(V) gene (*COL5A2*)	9q34, 2q31

AUTOSOMAL DOMINANT TRAIT

- IT IS MANIFESTED EVEN IN HETEROZYGOTES
- IN HOMOZYGOTES THE MANIFESTATION IS MUCH MORE SEVERE
-



AUTOSOMAL DOMINANT TRAIT

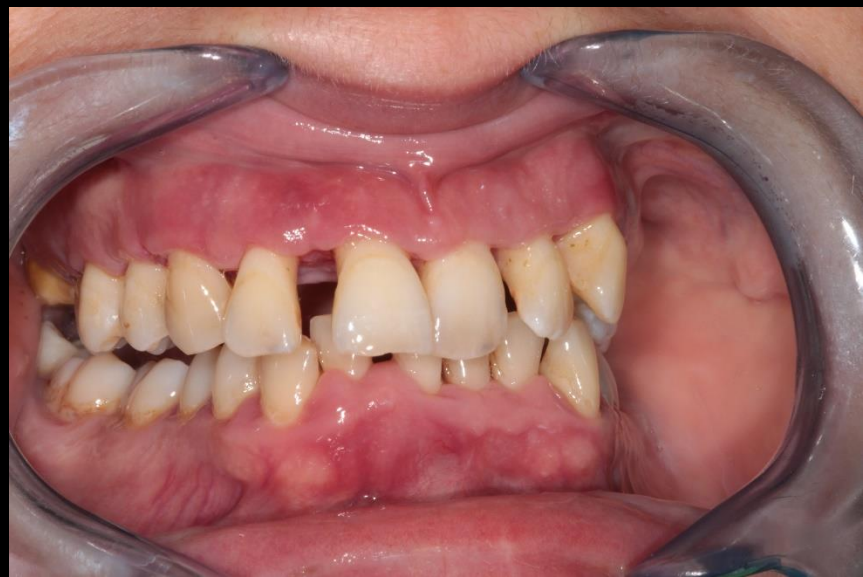
- ***Familiar chronic benign neutropenia-***

- ***. Severe congenital neutropenia***
  Severe congenital neutropenia 1:
  Severe congenital neutropenia 2:
  Severe congenital neutropenia 3 Morbus Kostmann

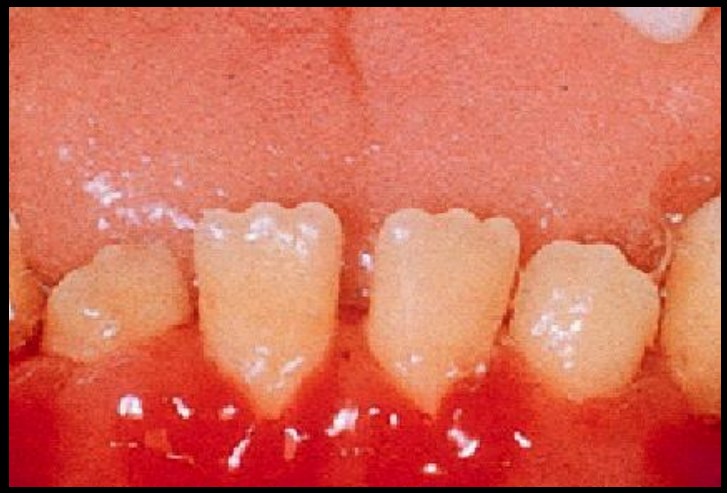
- ***Cyclic neutropenia***

- ***Ehlers-Danlos Syndrome (EDS)***
- ***Hypohosphatasia***

- ***Lazy leukocyta syndrome***



Familiar chronic benign neutropenia-

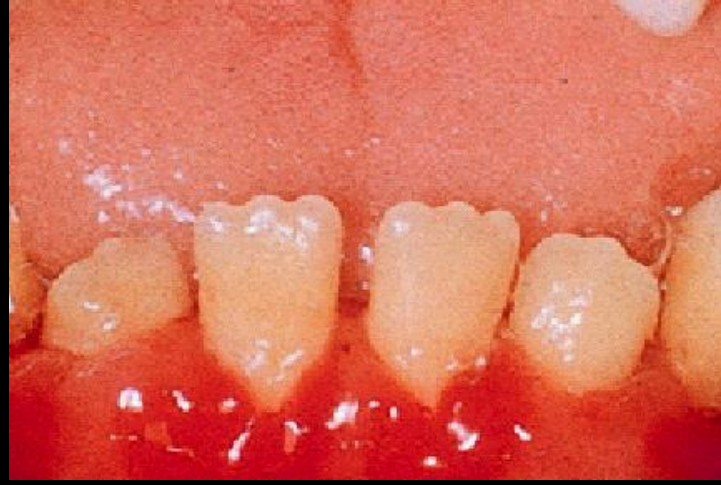


PMN count is less than 500/mm^3. ,

The other white blood cells are normal.

It might be an auto-immune disease in which anti-neutrophil IgG produced

Familiar chronic benign neutropenia-



Characteristics : very severe aggressive periodontitis in the early childhood

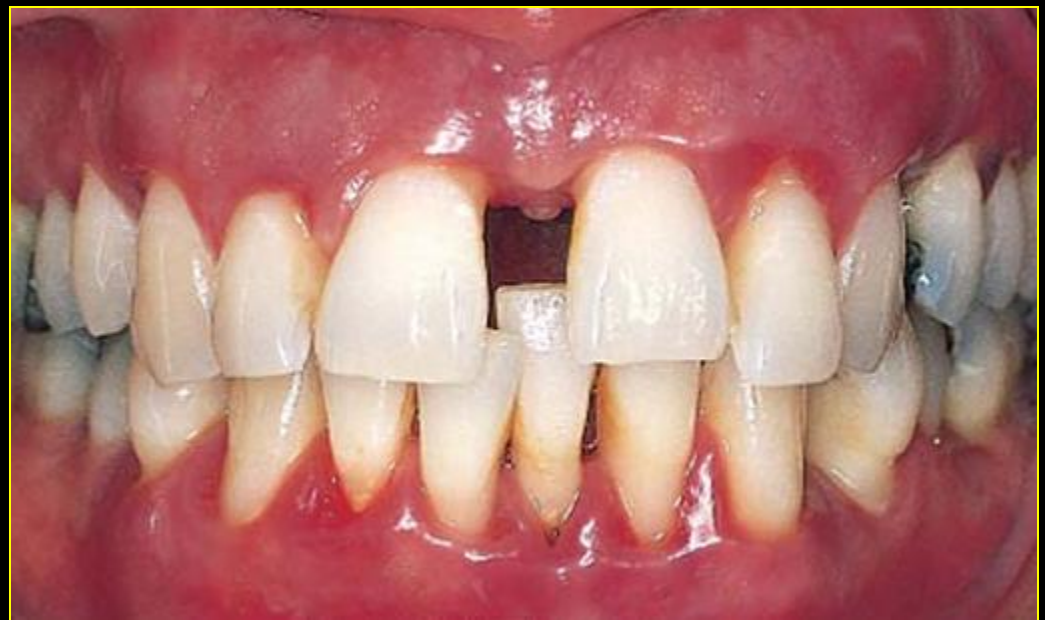
Severe attachment loss and gingival enlargement

REICHERT PA, DORNOW H. Gingivo-periodontal manifestation in chronic benigne neutropenia.

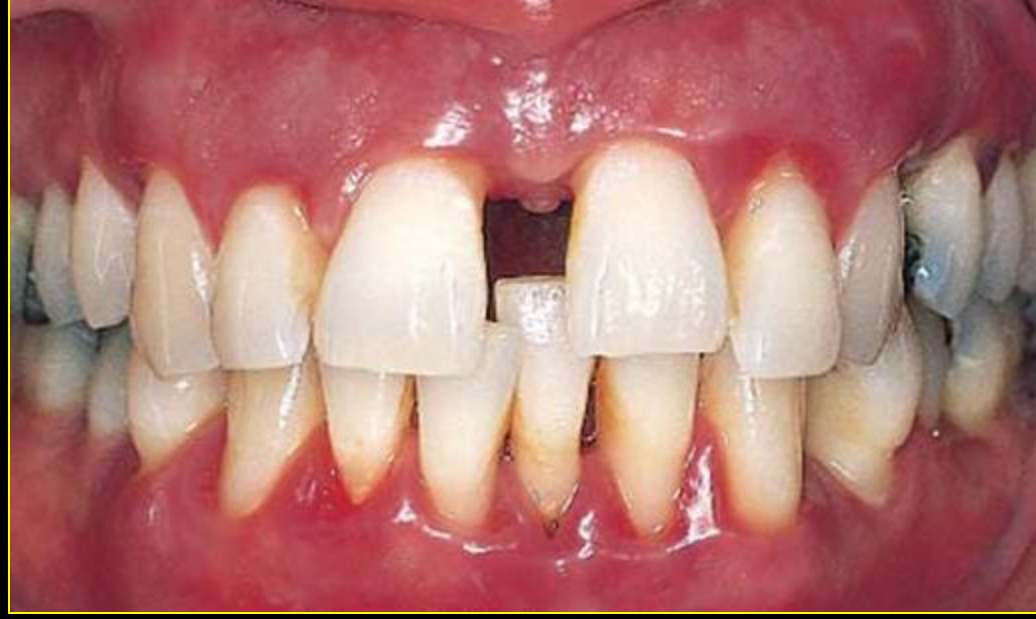
J Clin Periodontol 1978;5:74-80.

Severe congenital neutropenia

- Severe congenital neutropenia 1:
- Severe congenital neutropenia 2:
- Severe congenital neutropenia 3 Morbus Kostmann

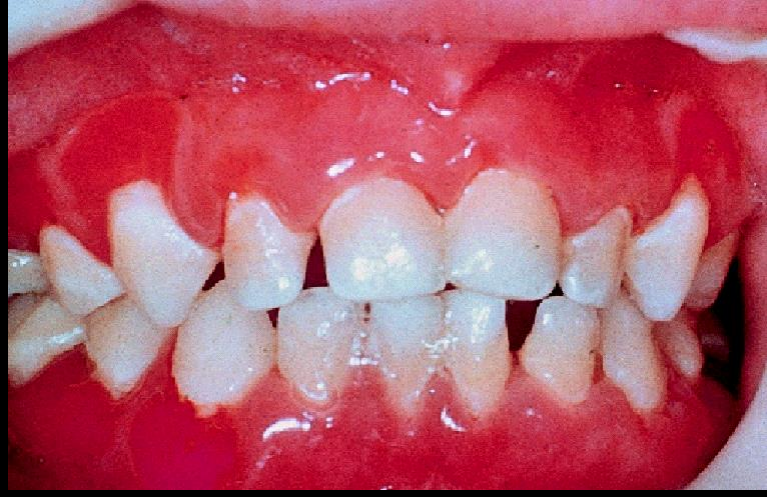


severe congenital neutropenia 1



- is caused by mutation of the neutrophil elastase gene (*ELA2*). in granules of neutrophils and monocytes
- severe congenital neutropenia
- affected its proteolytic activity.

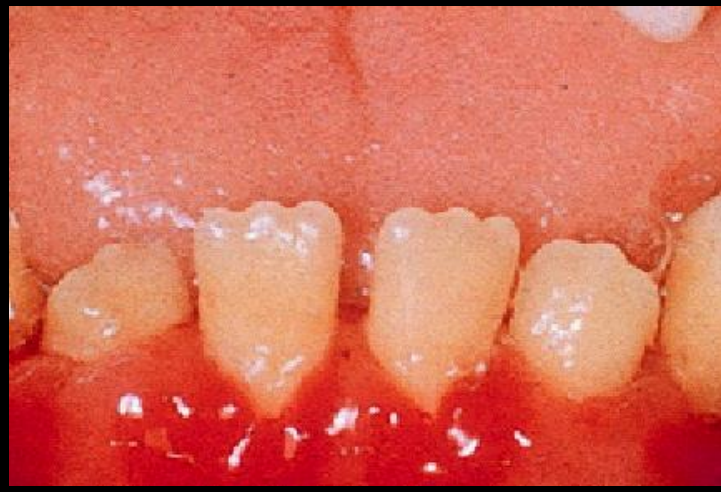
Severe congenital neutropenia 2



- Growth factor-independent 1 protein gene mutation
- myeloid progenitor cells fail to differentiate to mature neutrophils,

  causing the accumulation of monocytes and abnormal granulocytes

Severe congenital neutropenia 3 Kostmann syndrome



was first described in a large, intermarried Swedish family

Congenital neutropenia (Kostmann syndroma) is an autosomal-dominant/recessive condition caused by mutation of a hematopoietic cell-specific protein X1 (HAX1)

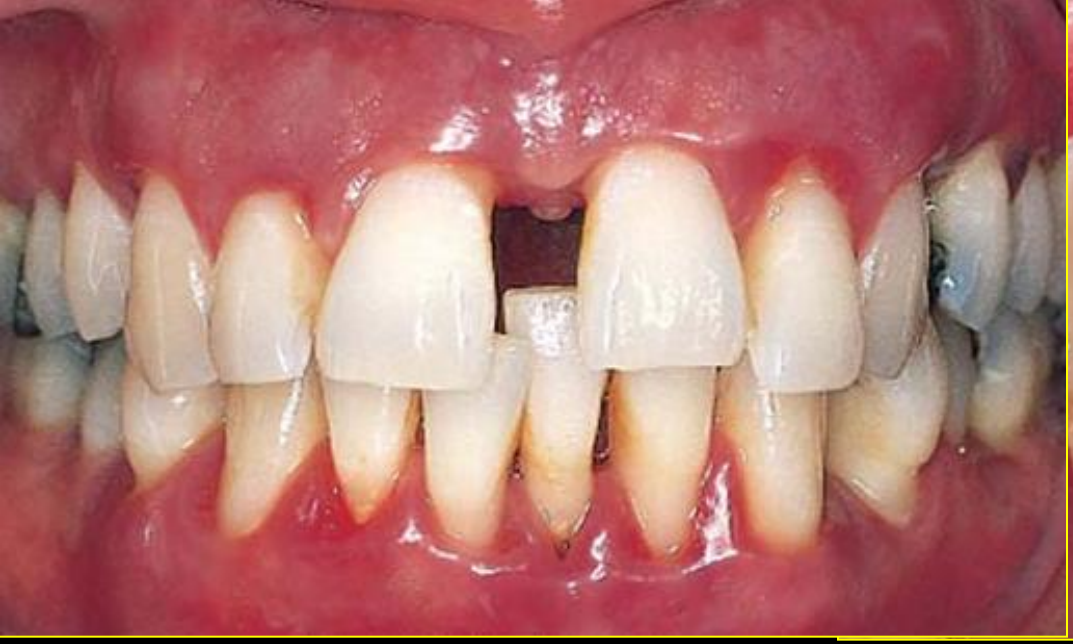
PMN count is less than 200/mm^3.

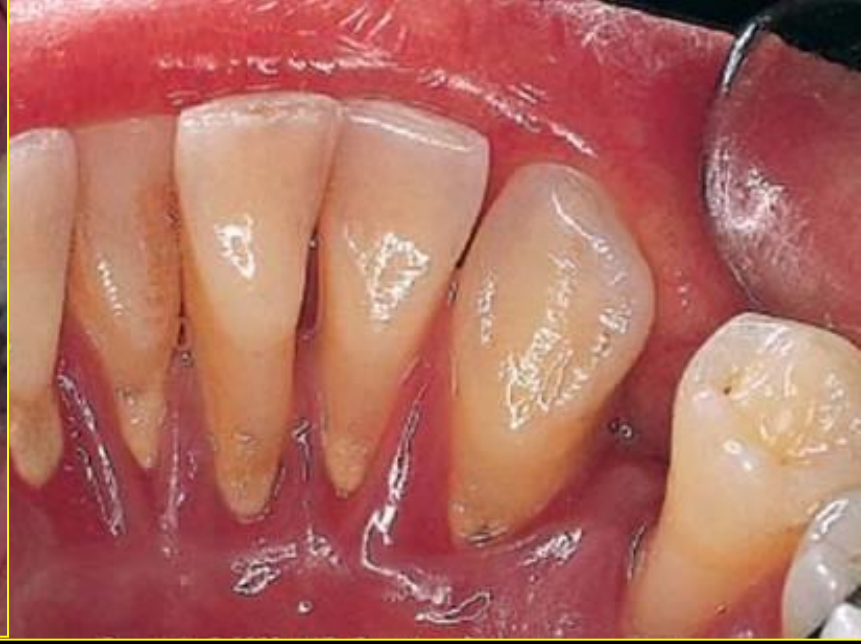
- Mutations in the granulocyte–colony-stimulating factor gene
- Formally this was a lethal condition

- most patients respond to recombinant human granulocyte–colony stimulating factor.

- Hematopoietic stem cell transplantation is the only treatment for patients refractory to granulocyte–colony stimulating factor treatment

ZEIDLER C, BOXER L, DALE DC, FREEDMAN MH, KINSEY S, WELTE K. Management of Kostmann syndrome in the G-CSF era. *Br J Haematol* 2000;109:490-495.





Formally this was a lethat condition

ZEIDLER C, BOXER L, DALE DC, FREEDMAN MH, KINSEY S, WELTE K. Management of Kostmann syndrome in the G-CSF era. *Br J Haematol* 2000;109:490-495.

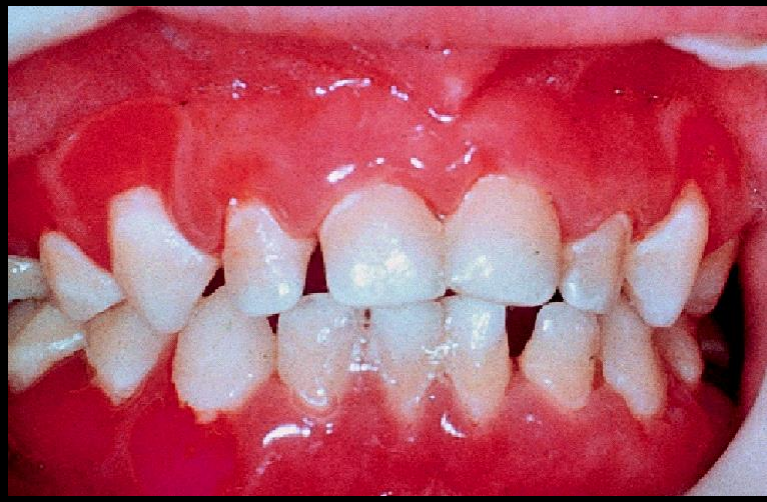
Congenitalis neutropenia (Kostmann syndroma)

Despite the successful CSF therapy the periodontal condition will not improve

The gingival inflammation and periodontal attachment loss is dependent on the number of functional circulating neutrophils

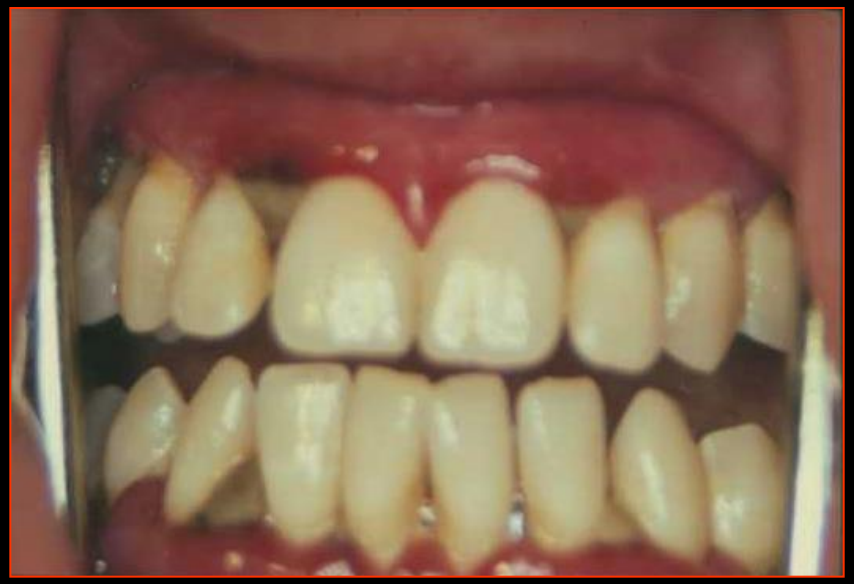
.CARLSSON G, FASTH A. Infantile genetic agranulocytosis, morbus Kostmann: Presentation of six cases from the original “Kostmann family” and a review. Acta Paediatr 2001;90:757-764.

Cyclic neutropenia autosomal dominant inheritance



- Cause: neutrofil elastaz gene (19p13.3) mutation
- manifested by transient severe neutropenia
- periodically non responding to *granulocyta colony-stimulating factor*
- that recurs approximately every 21 days
- Circulating neutrophils vary between almost normal numbers and zero.
- During intervals of neutropenia, affected individuals are at risk for opportunistic infection

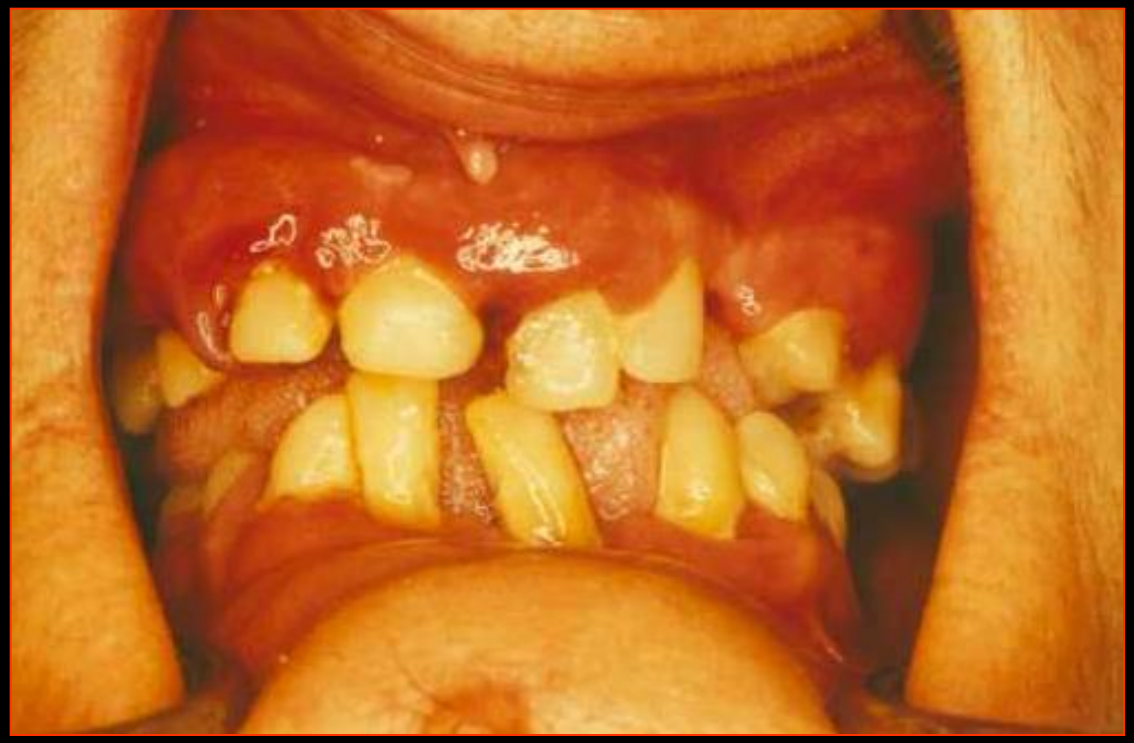
Cyclic neutropenia



The bone marrow is full with PMN progenitor cells but it periodically non responding to *granulocyta colony-stimulating factor*

DALE DC, HAMMOND WP. Cyclic neutropenia: a clinical review. *Blood Rev* 1988;2:178-185

HYPOPHOSPHATASIA



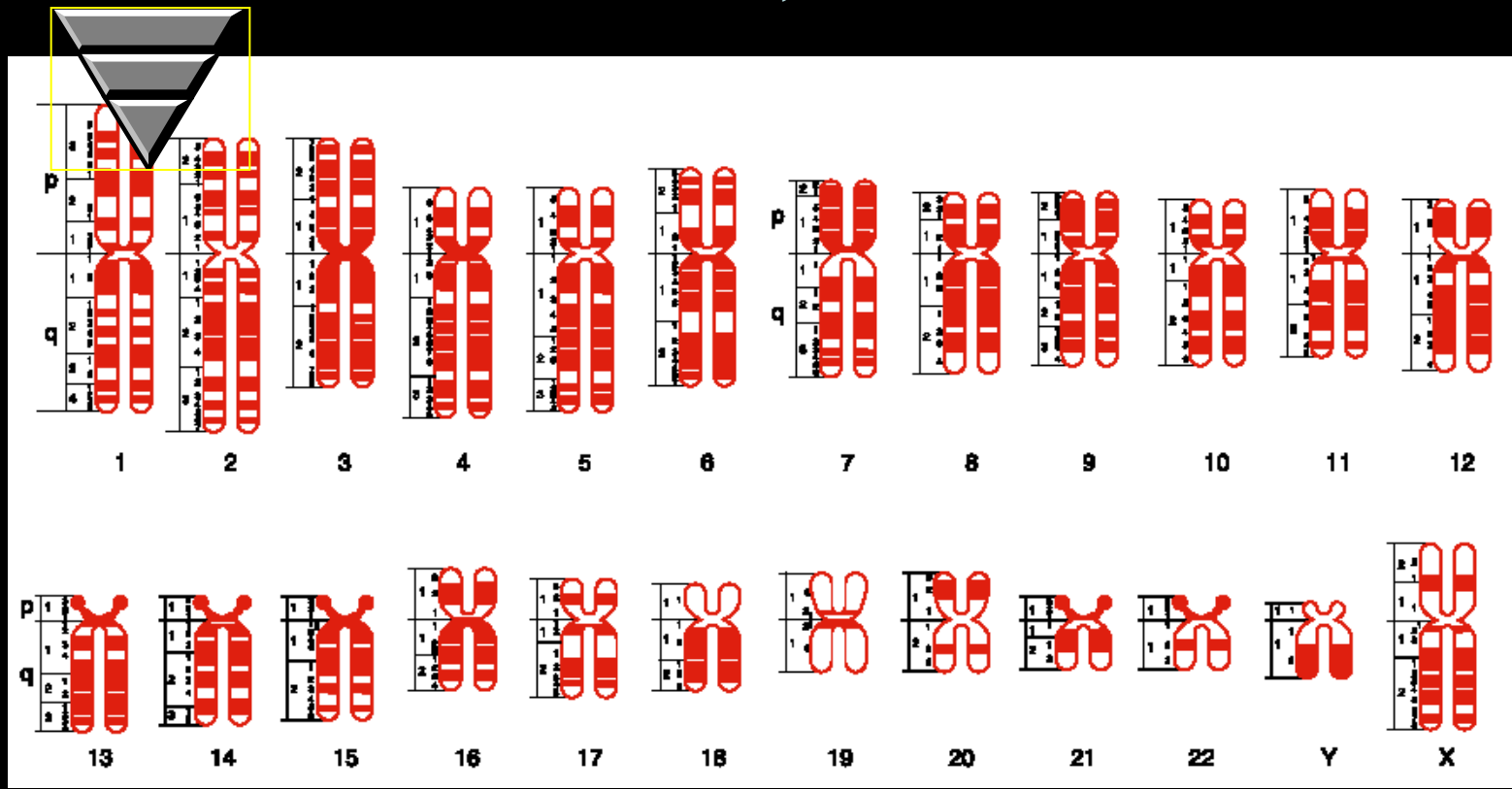
1. CHAPPLE IL, THORPE GH, SMITH JM, SAXBY MS, és mtsai. Hypophosphatasia: a family study involving a case diagnosed from gingival crevicular fluid. J Oral Pathol Med. 1992;21:426-31. Review.

Hypophosphatasia

tissue alkalic phosphatasa enzyme
(1p36.1-p34) gene mutation

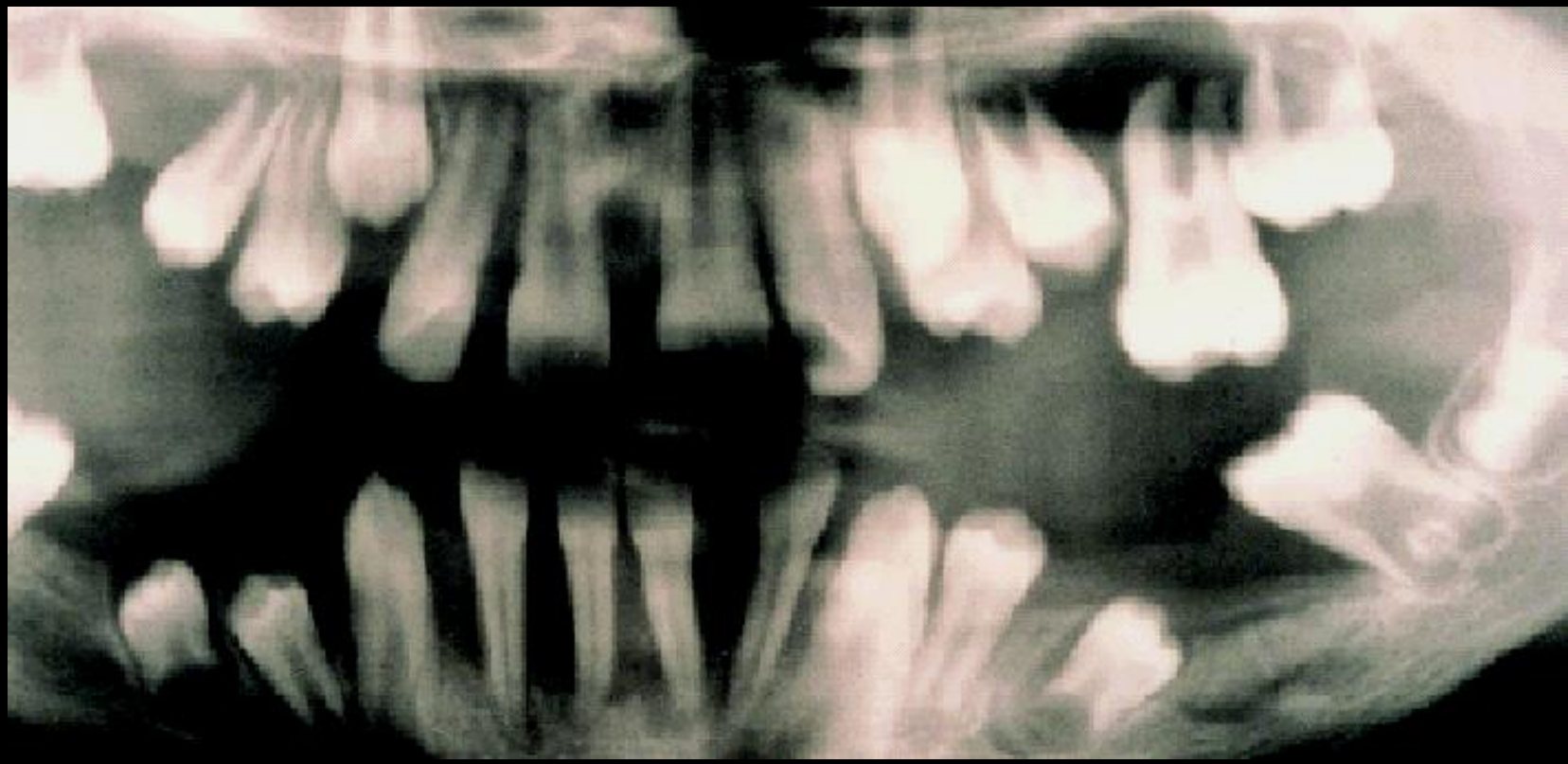
Alkalic phosphatase enzyme function is diminished

ROOT AW Recent advances in the genetics of disorders of calcium homeostatis Adv Pediatr. 1996; 43: 77-84

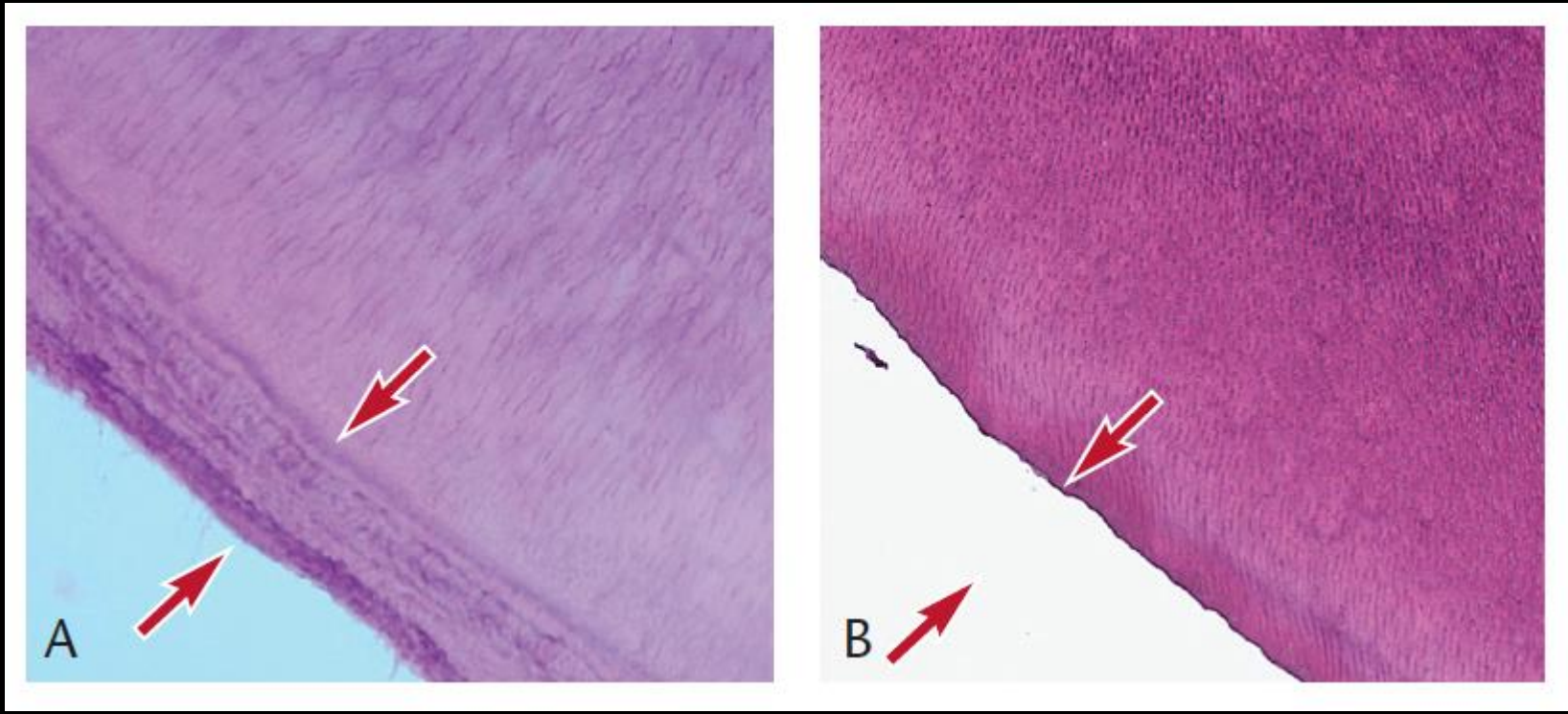


Hypophosphatasia

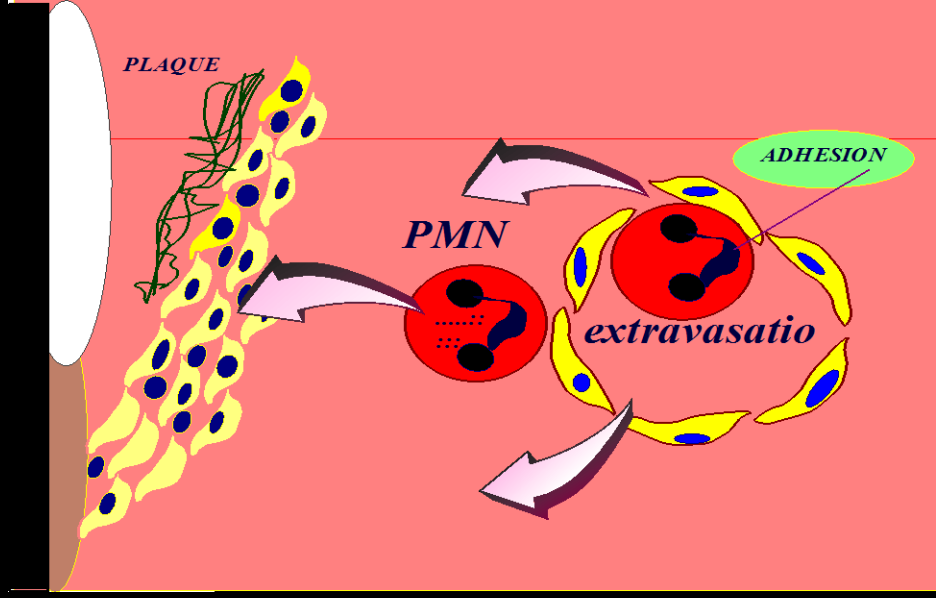
- **Severe bone formation discrepancies**
- **severe cementum formation discrepancies**
- **The most severe cases are lethal in the early ages**
- **In mild cases very severe early onset aggressive periodontitis and early tooth loss occur**



severe cementum formation discrepancies



Lazy leukocyte syndrome



Defective function of the PMN cell's cytoskeleton built up by actin/myosin microfilaments .

The PMN cell's migration ins hampered

PMN cells are not able to leave the bone marrow .

In the tissue PMN cells' migration and chemotactic response is also weak

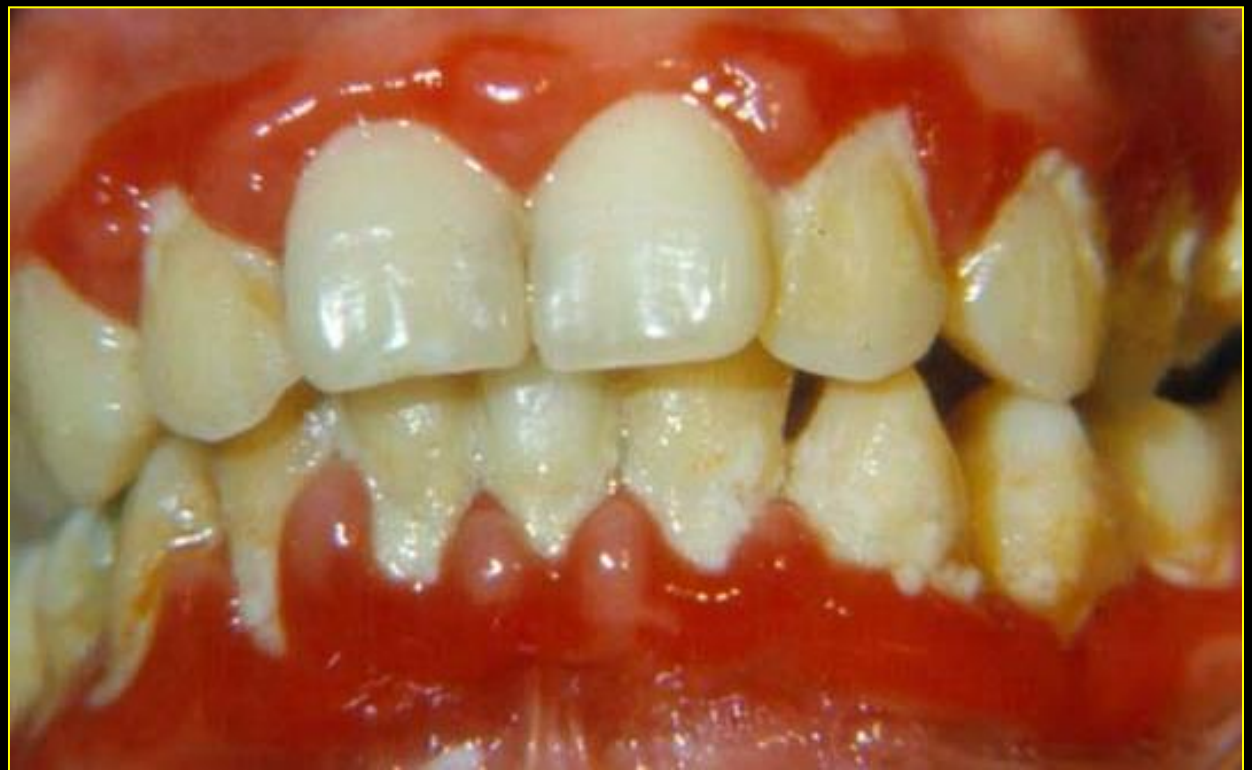
other very rare autosomal dominant syndromes associated with severe periodontitis

.

- **Ehlers-Danlos syndrome**
- **Marfan-syndrome**
- **Cohen syndrome**
- **Hajdu-Cheney syndrome**

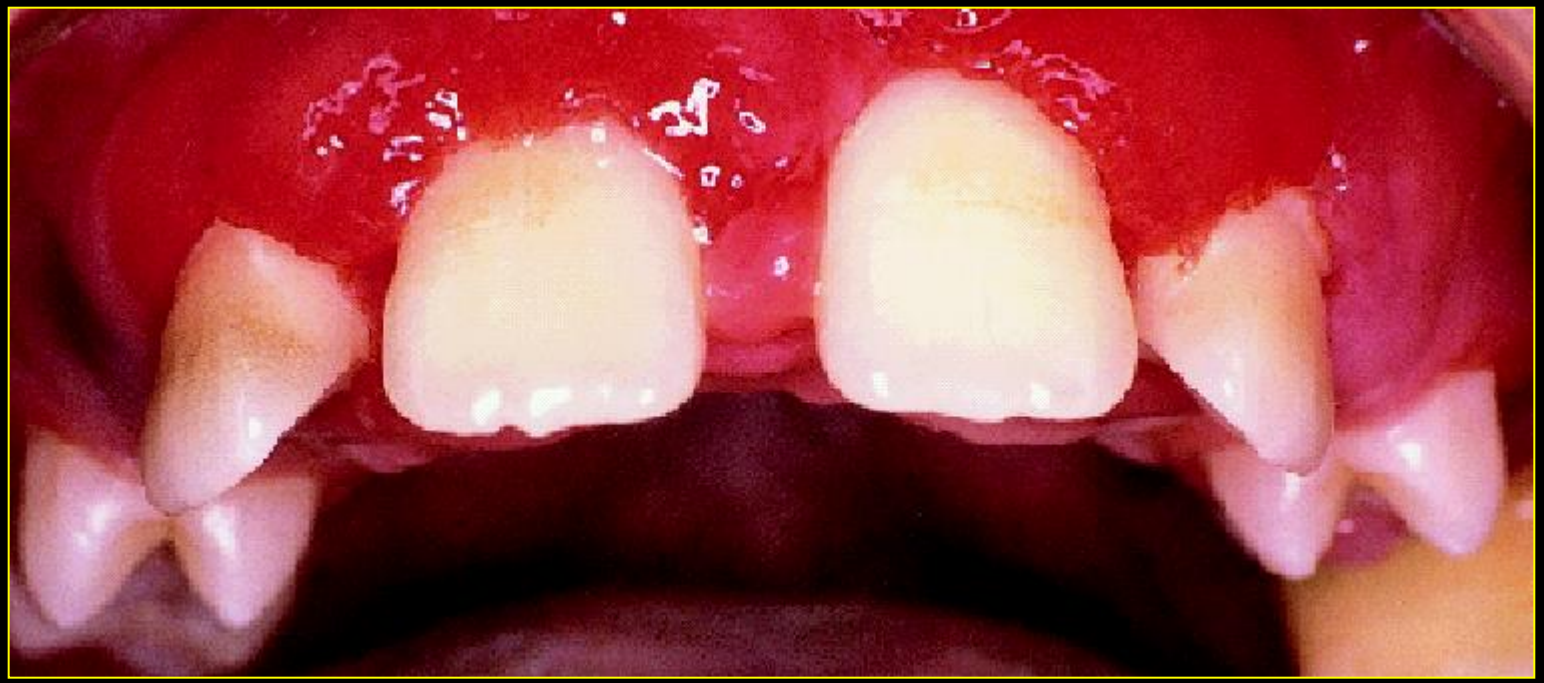
AUTOSOMAL RECESSIVE SYNDROMES

- **It can be manifested only in homozygtes**
- The majority is enzymopathy
- The incidence shows great variations in different ethic groups

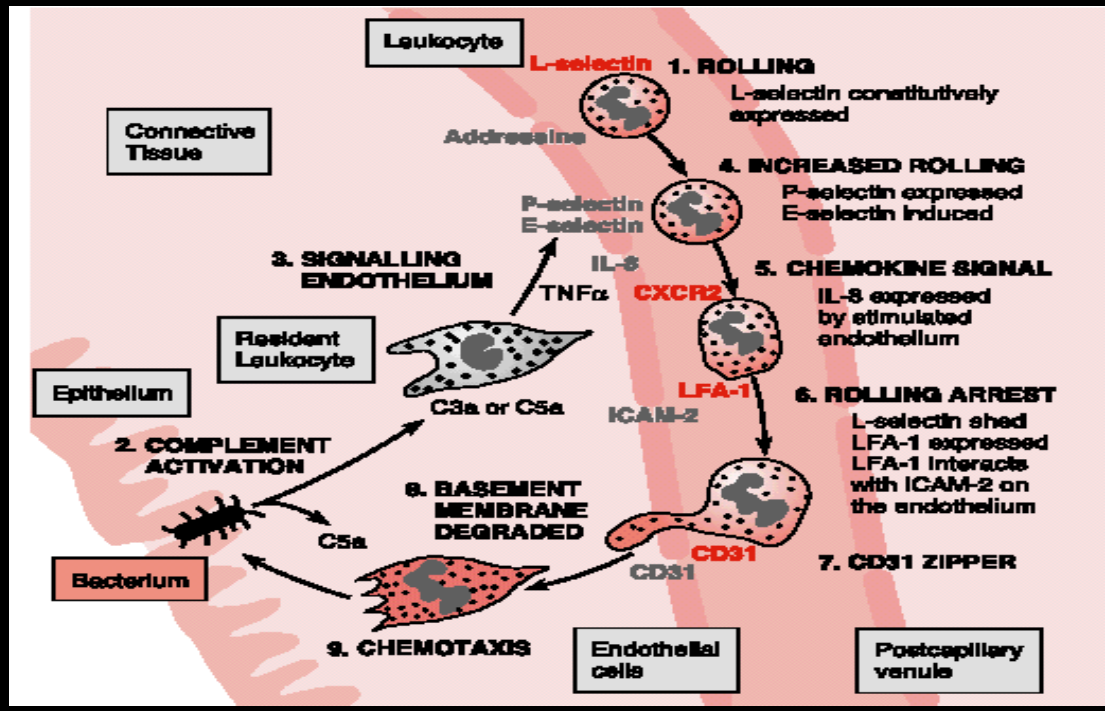


AUTOSOMAL RECESSIVE SYNDROMES

- LAD I
- LAD II
- Papillion-LeFevre syndrome

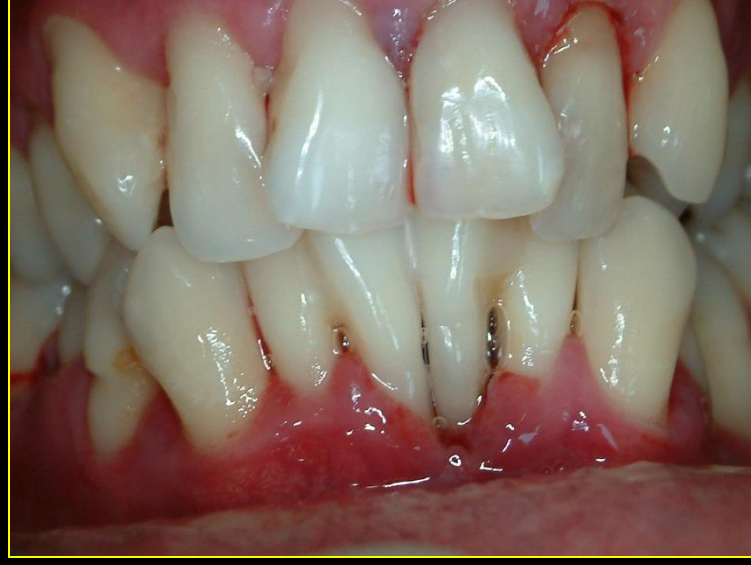


THE ROLE OF THE PMN LEUKOCYTES .



- ROLLING
- ATTACHMENT TO THE ENDOTHELIAL CELLS
- MIGRATION TOWARDS THE CHEMOTACTIC SIGNAL
- BINDING ANTIGENS AND MICROORGANISMS
- PHAGOCYTOSIS
- INTRACELLULAR BACTERIAL KILLING

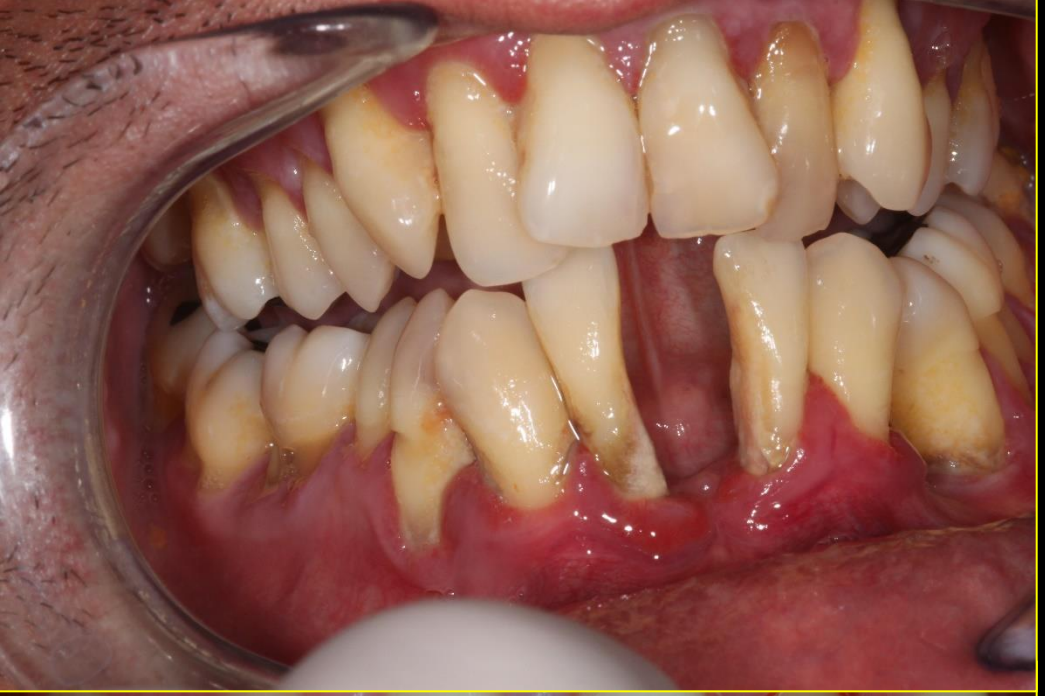
Leukocyte adhesion deficiency type 1



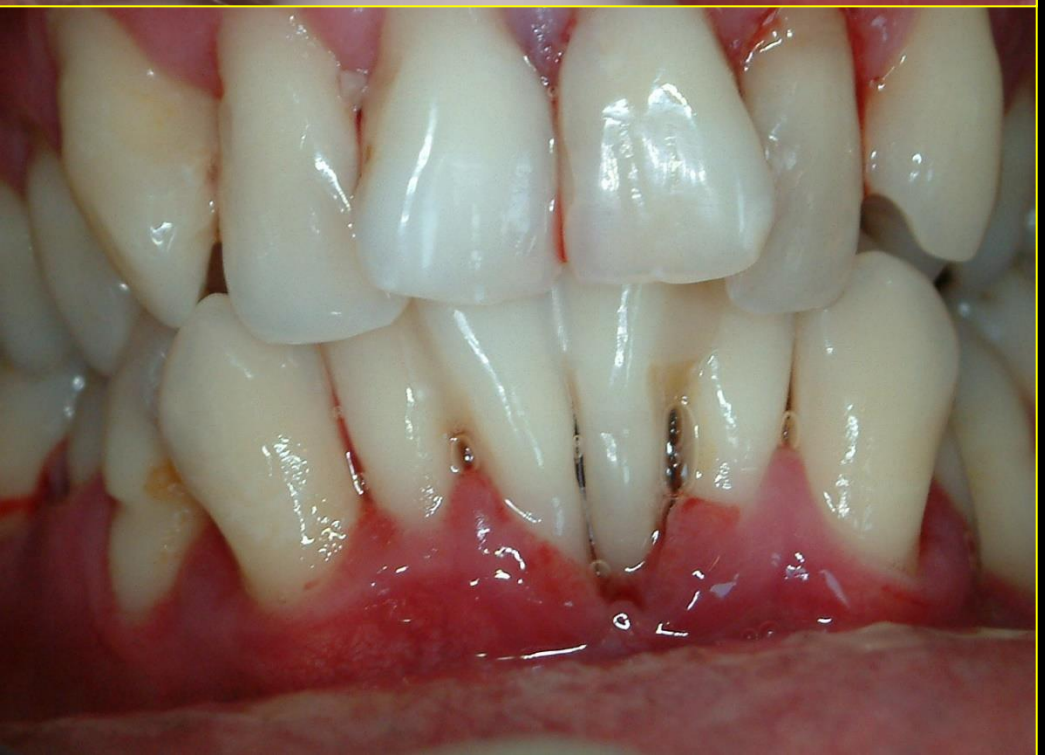
An autosomal-recessive condition, the most common form of leukocyte adhesion deficiency.

It is also the most severe form

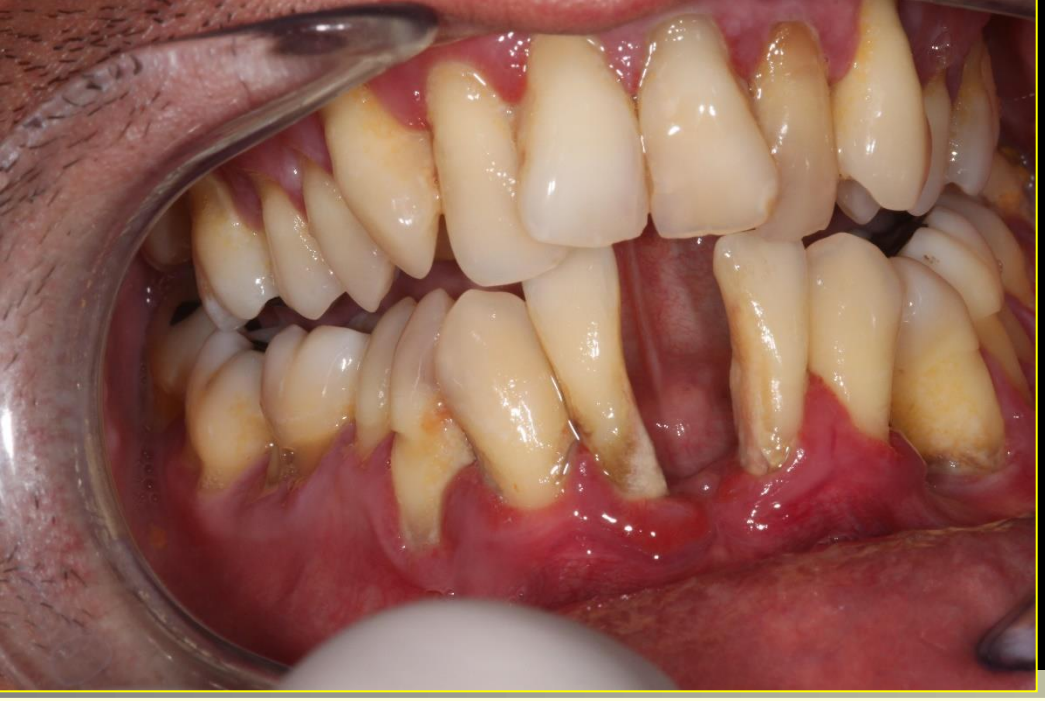
may be fatal without treatment such as bone marrow transplant.



Brother 21 y



Sister 16 y



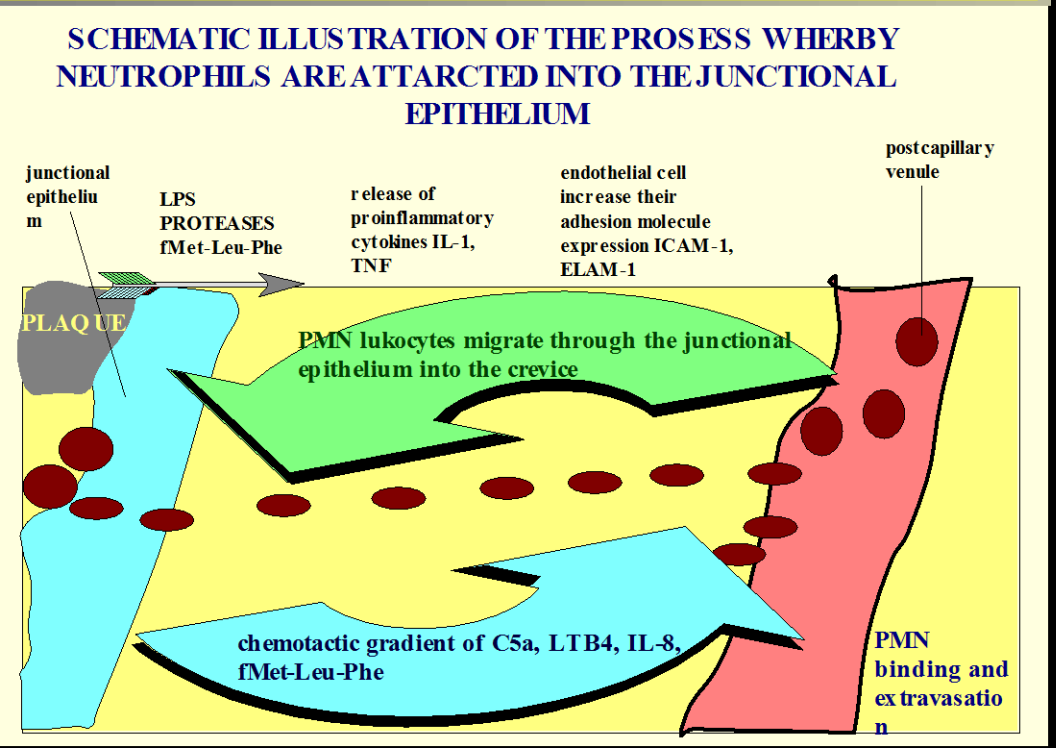
Leukocyte adhesion deficiency type 1

Selectin CD-15

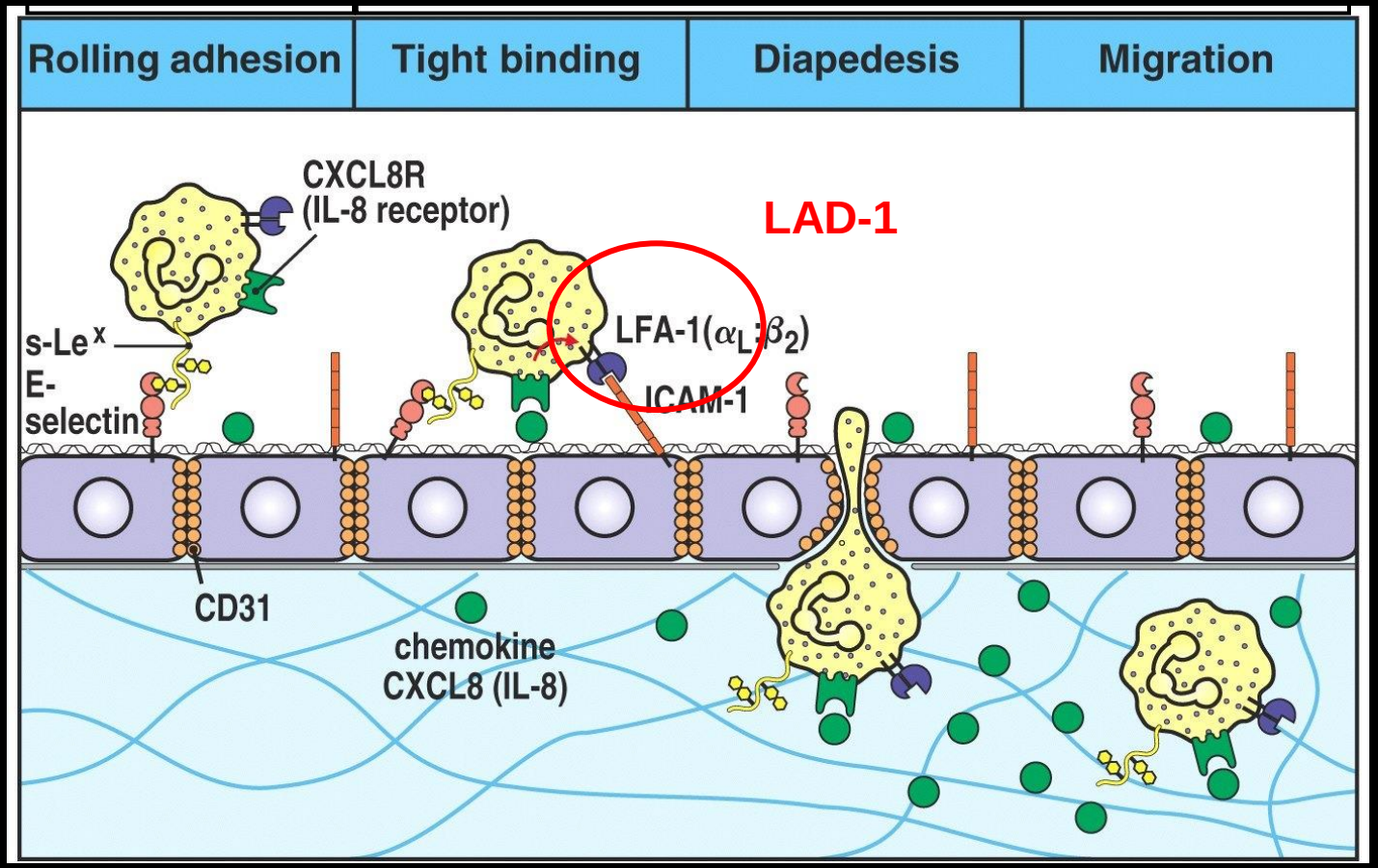
Integrin

CD-11 a,b,c

CD-18

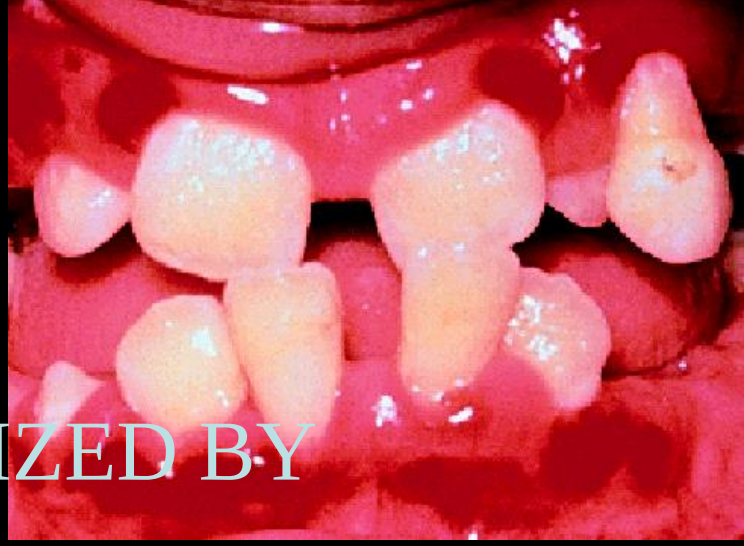


Selectins **Integrins**



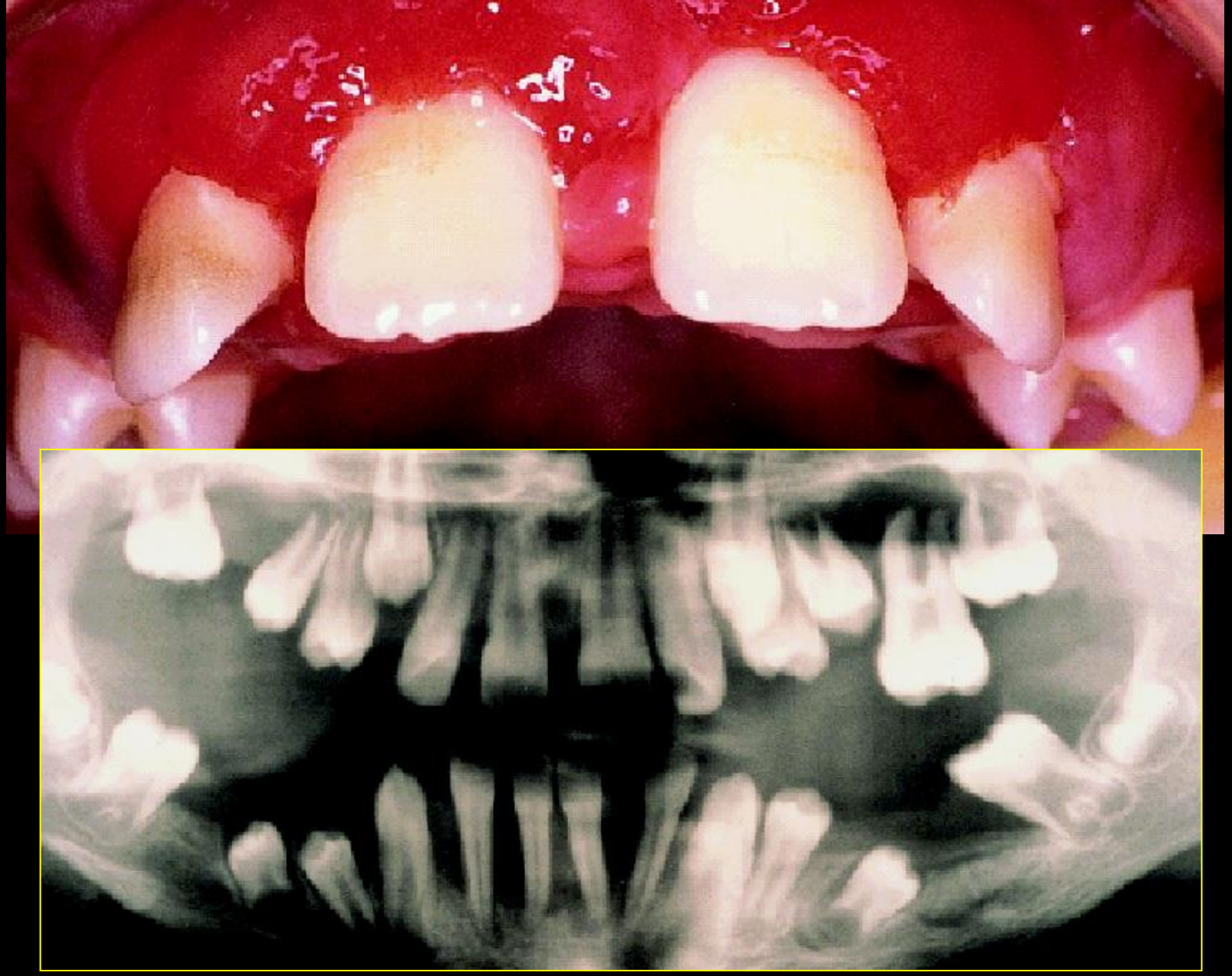
- **Integrin mutations:**
  - prevents neutrophils from **adhering** to the vessel wall at the site of an infection

Leukocyte adhesion deficiency type 1



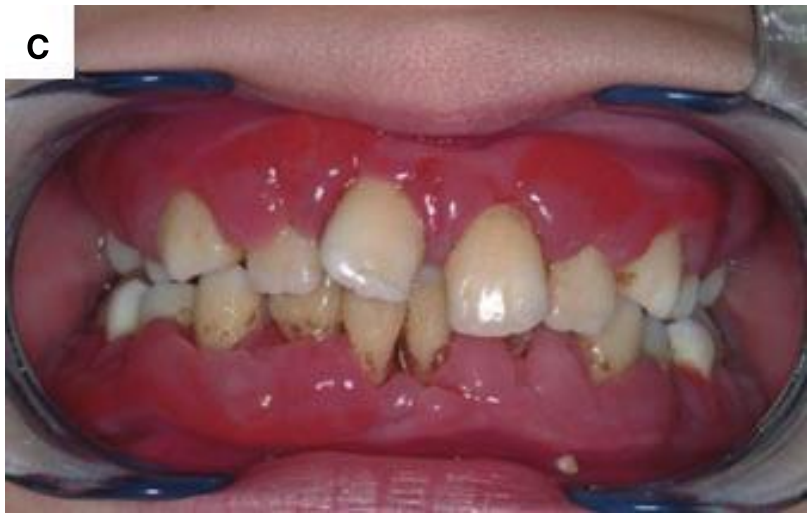
CLINICALLY CHARACTERIZED BY

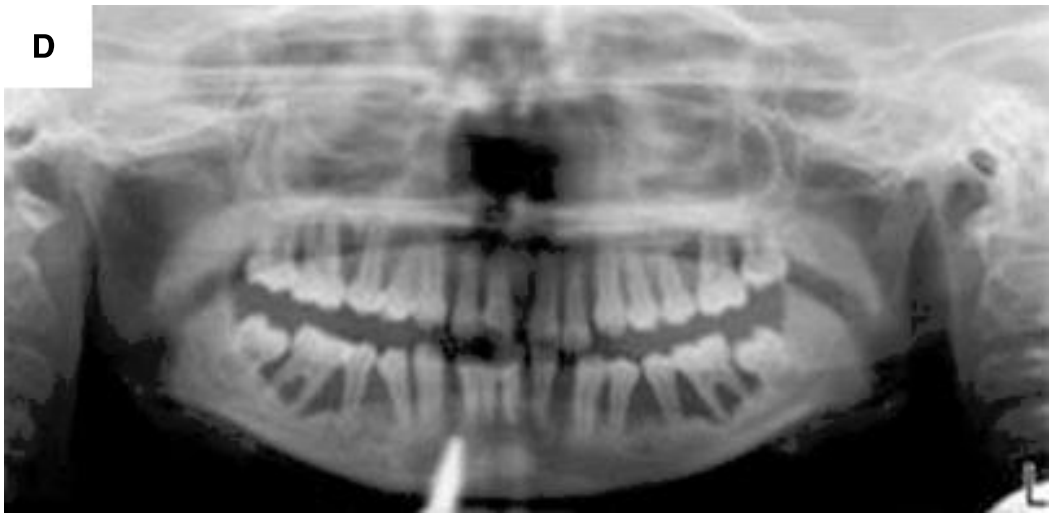
- recurrent bacterial infections,
- impaired pus formation,
- impaired wound healing,
- a wide range of abnormalities of adhesion-dependent functions of granulocytes, monocytes, and lymphocytes.
- Aggressive periodontitis



Majorana A, Notarangelo LD, Savoldi E, et al: Leukocyte adhesion deficiency in a child with severe oral involvement. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 87:691.)

LAD-1 clinical and radiological signs

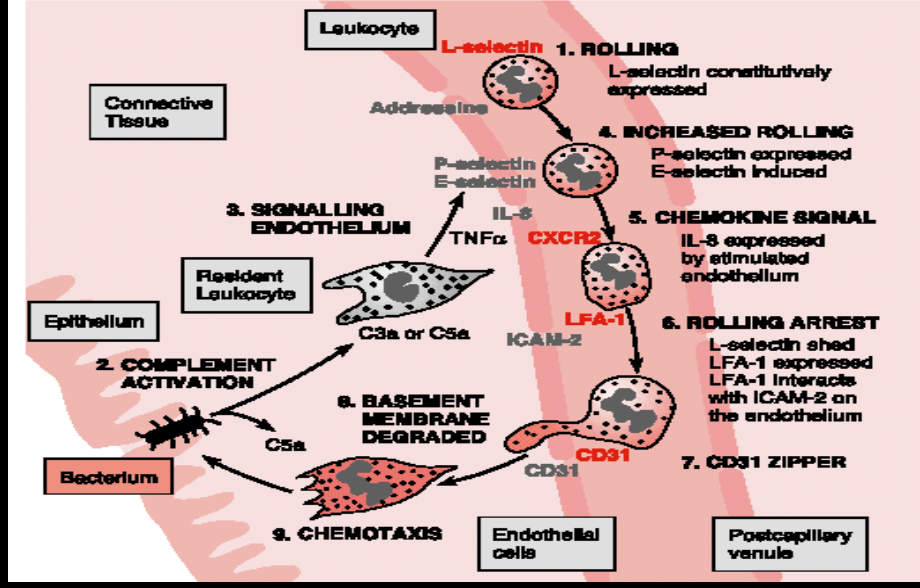




Oral findings include acute gingival **inflammation** of both primary and permanent dentition, gingival **proliferation**, **recession**, tooth **mobility**, pathologic **migration**, **advanced bone loss**, and early tooth **loss**

(Hart TC, Atkinson JC. Mendelian forms of periodontitis. Periodontology 2000 2007: 45)

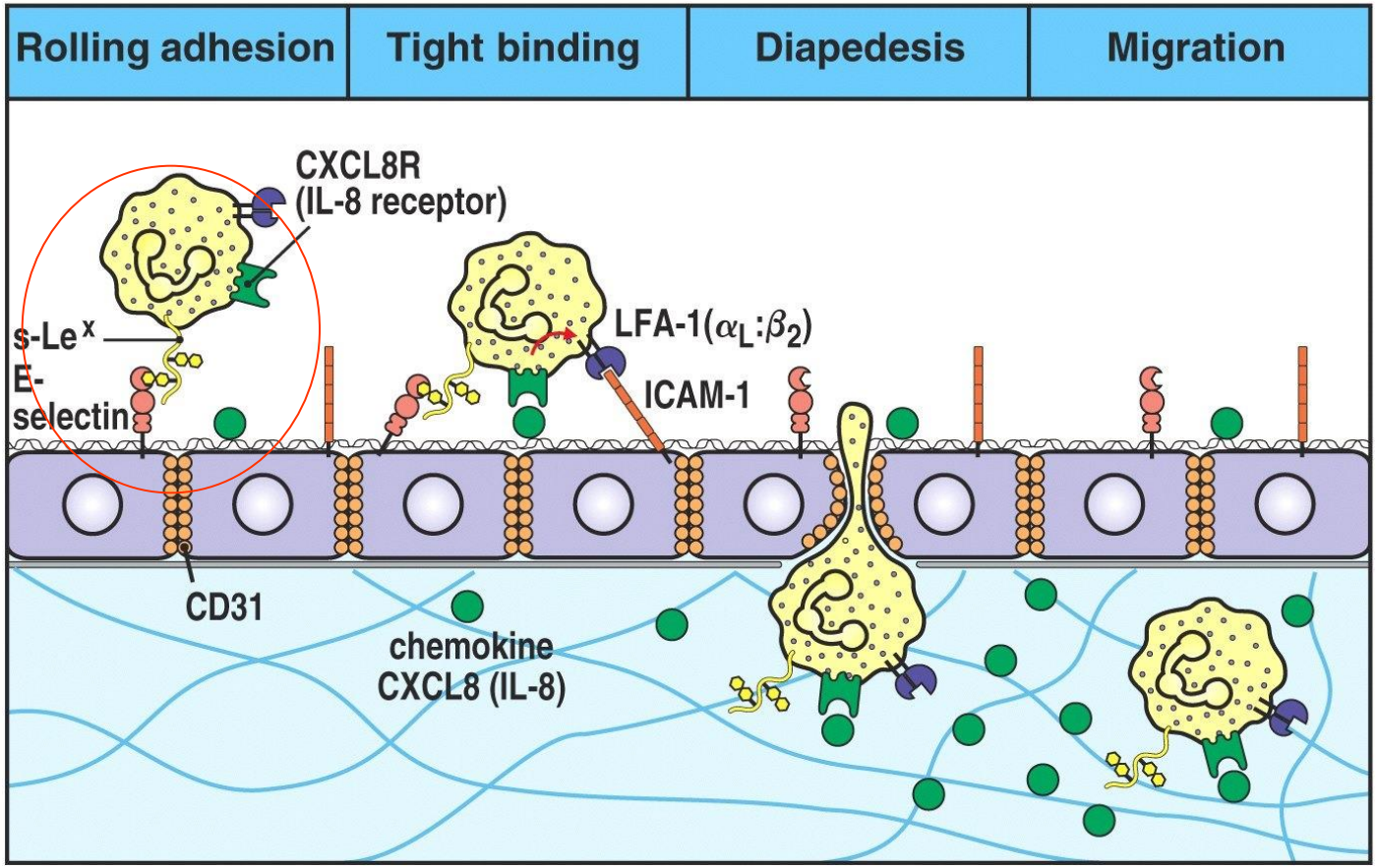
Leukocyte adhesion deficiency type 2



A ***LAD-II*** is rare and less severe

Several reports of leukocyte adhesion deficiency 2 describe mental retardation, short stature, distinctive facial appearance, and recurrent episodes of bacterial infections, mainly pneumonia, periodontitis, and otitis media

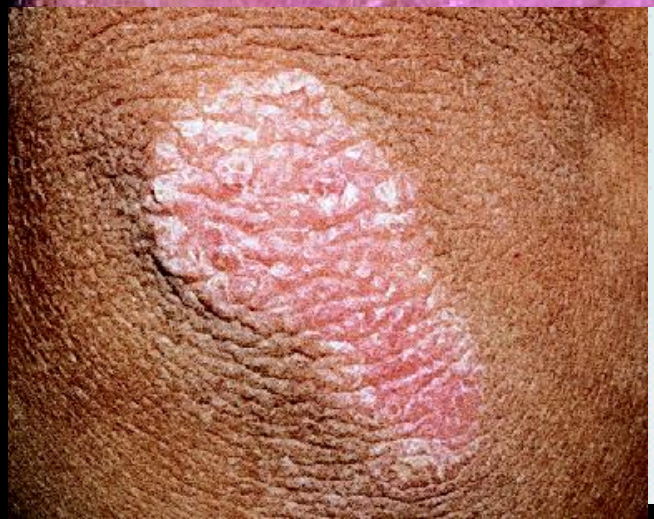
leukocyta adhéziós deficienca, (LAD II)

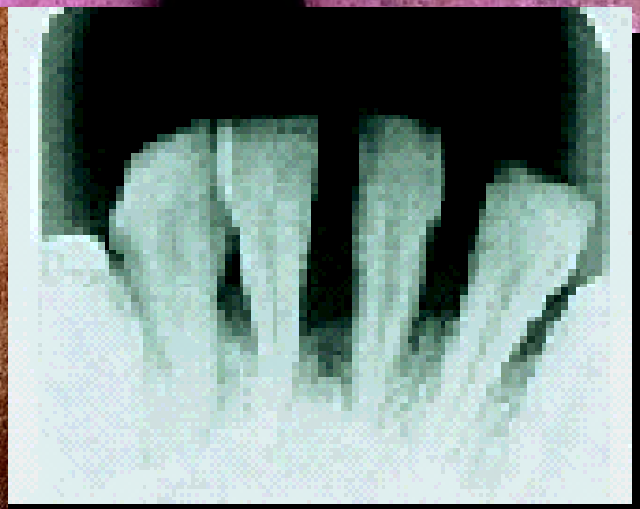


A *LAD-II* forma még ritkább,

oka a fehérvérsejtek selectin molekuláinak (CD15s receptor) hibás működése és ennek következtében a fehérvérsejt-marginalizáció első szakaszának, az un. rollingnak az elmaradása
Ennek eddig parodontális tüneteiről nem számolt be az irodalom

Papillion-LeFevre szindroma (PLS)



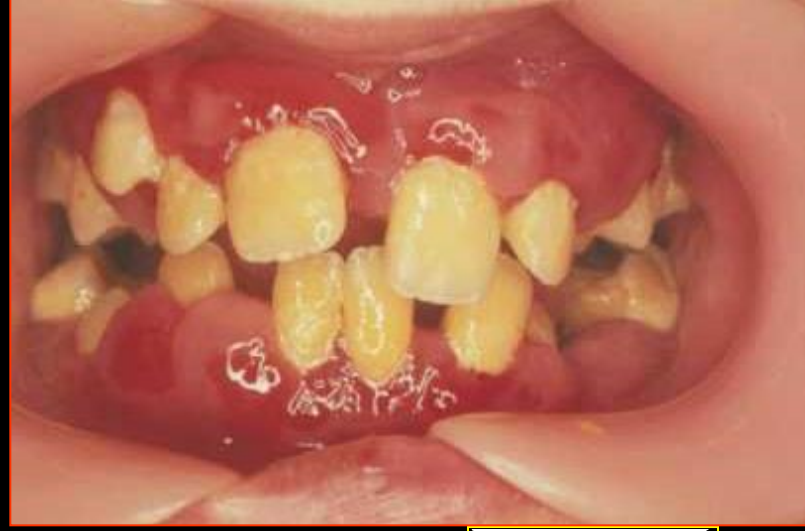


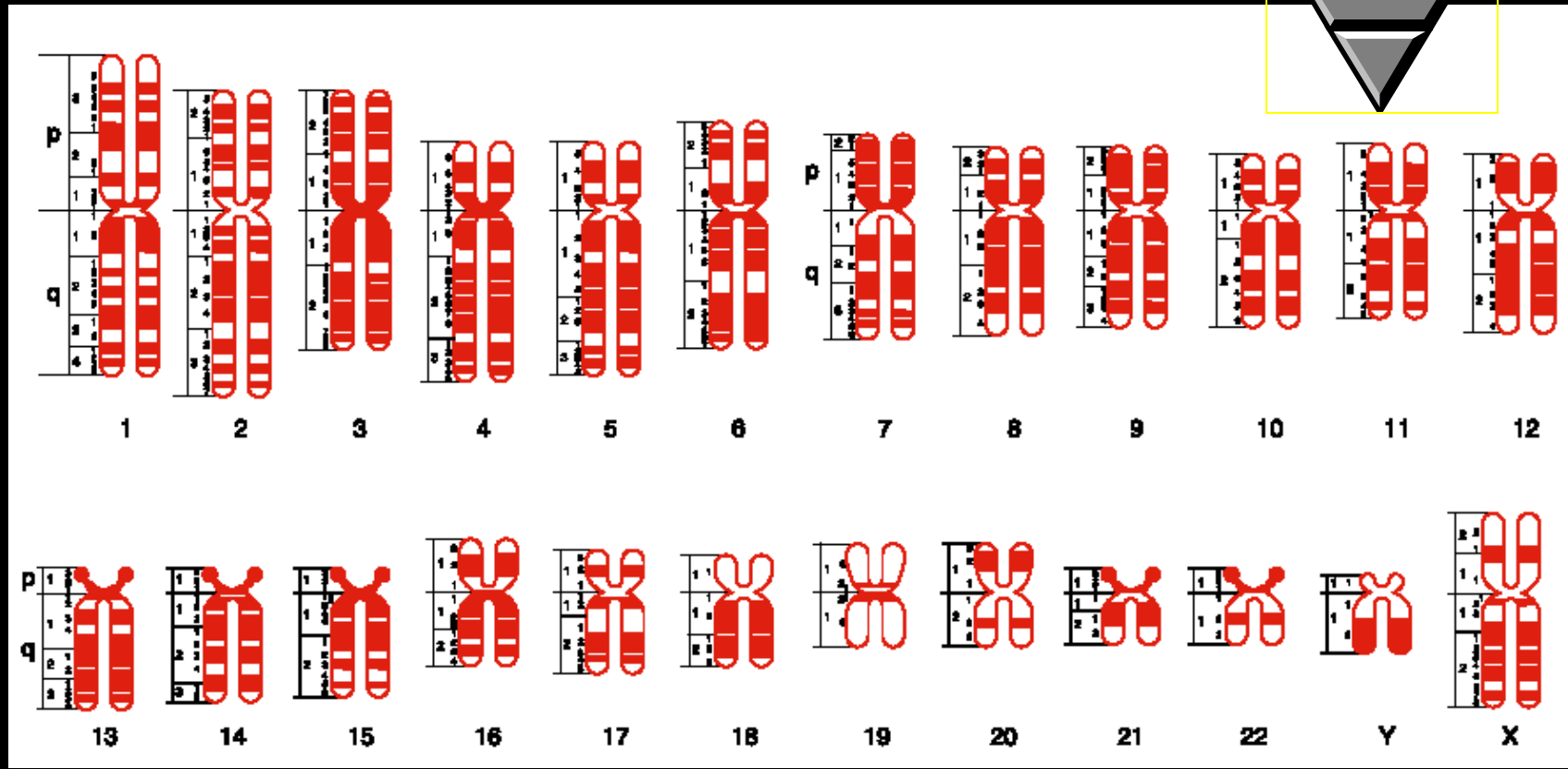
Papillion-LeFevre szindroma (PLS)

Papillon–Lefèvre syndrome is an autosomal-recessive palmoplantar keratodermal disorder

PAPILLIONMM, LEFEVRE P. Deux cas de keratodermie palmaire et plataire symmetrique familiale (maladie de Medea) chez le frere et la soeur. Coexistence dans les deus cas d'alteration dentaires graves. *Bull Soc Fr Dermatol Syphiligr* 1924;31:82-87.

11 chromosom (11q14-q21) cathepsin C gene mutation

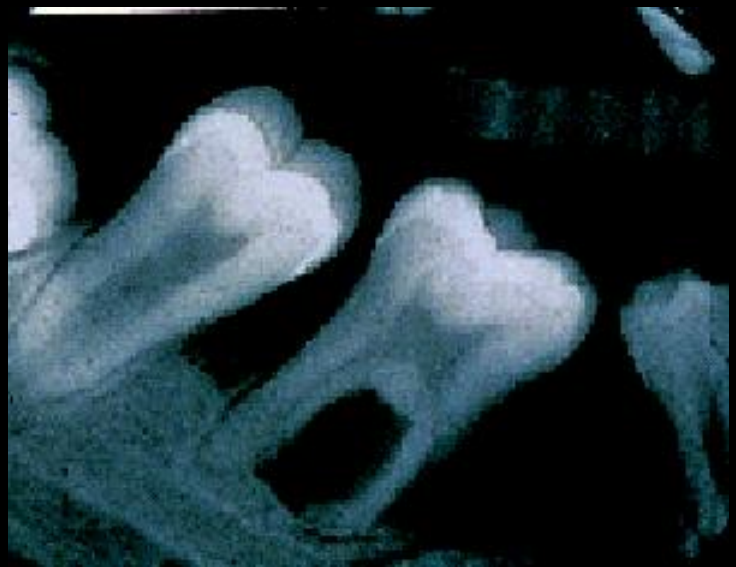


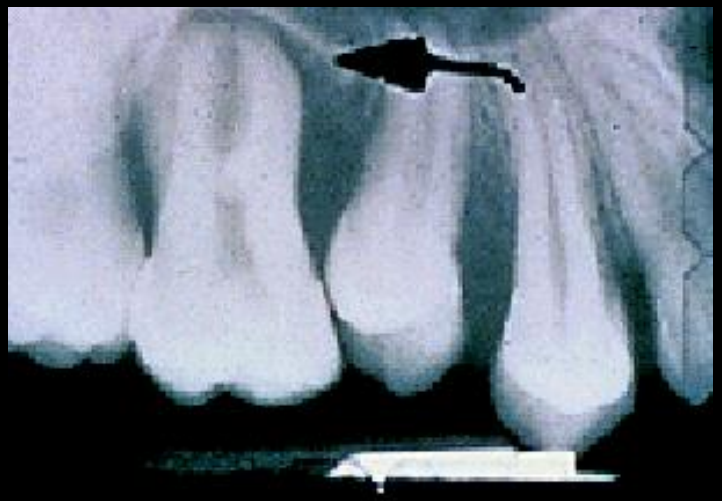


11 chromosom (11q14-q21) cathepsin C gene mutation

.HART TC, HART PS, BOWDEN DW és mts. Mutation of the cathepsin C gene are responsible for Papillion-Lefevre syndrome *J Med Genet* 1999; 36:881-888

.HART TC, ZHANG Y, FIRATI E és mts. Identificaton of cathepsin C mutations in ethnically diverse Papillion-Lefevre syndrome patients *J Med Genet* 2000; 37: 927- 931





- Cathepsin C, is a lysosomal protease .
- Functional cathepsin C is needed to activate a number of important neutrophil serine proteases.

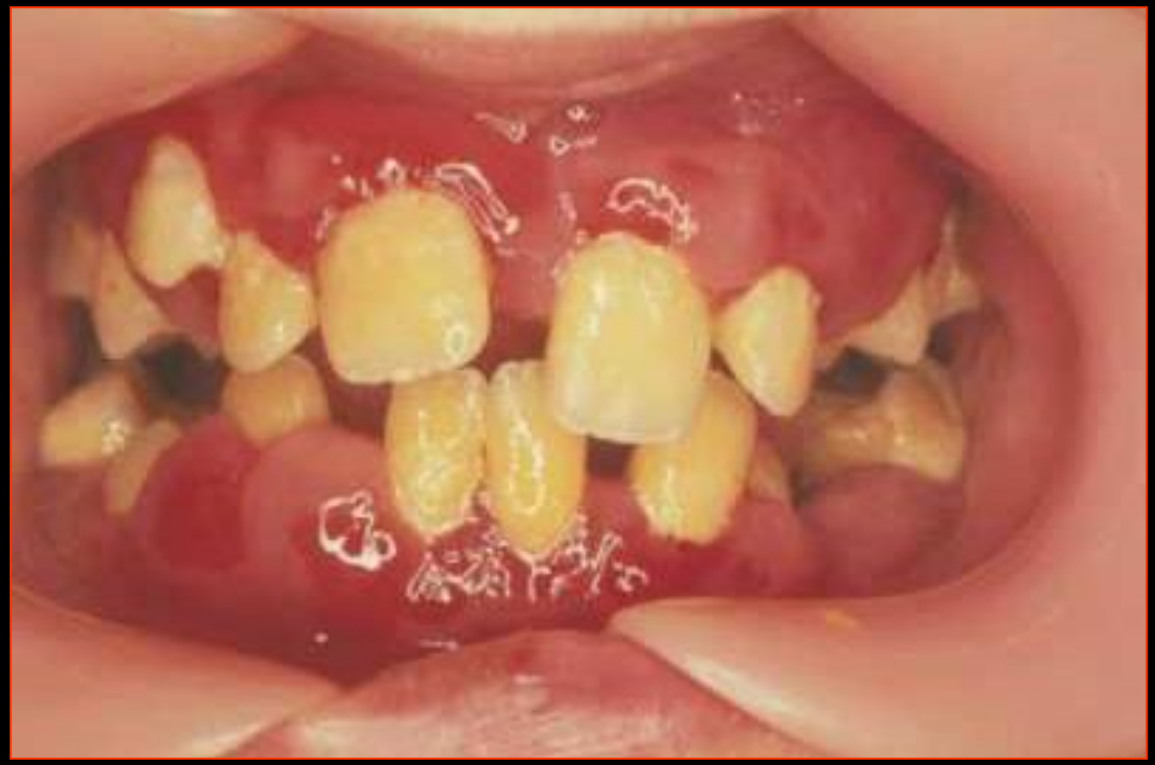
not able to activate the neutrophil serine proteases cathepsin C, neutrophil elastase, and proteinase 3

DJAWARI D. Deficient phagocyte function in Papillion-Léfevre syndrome. *Dermatologica* 1978;156:189-192.

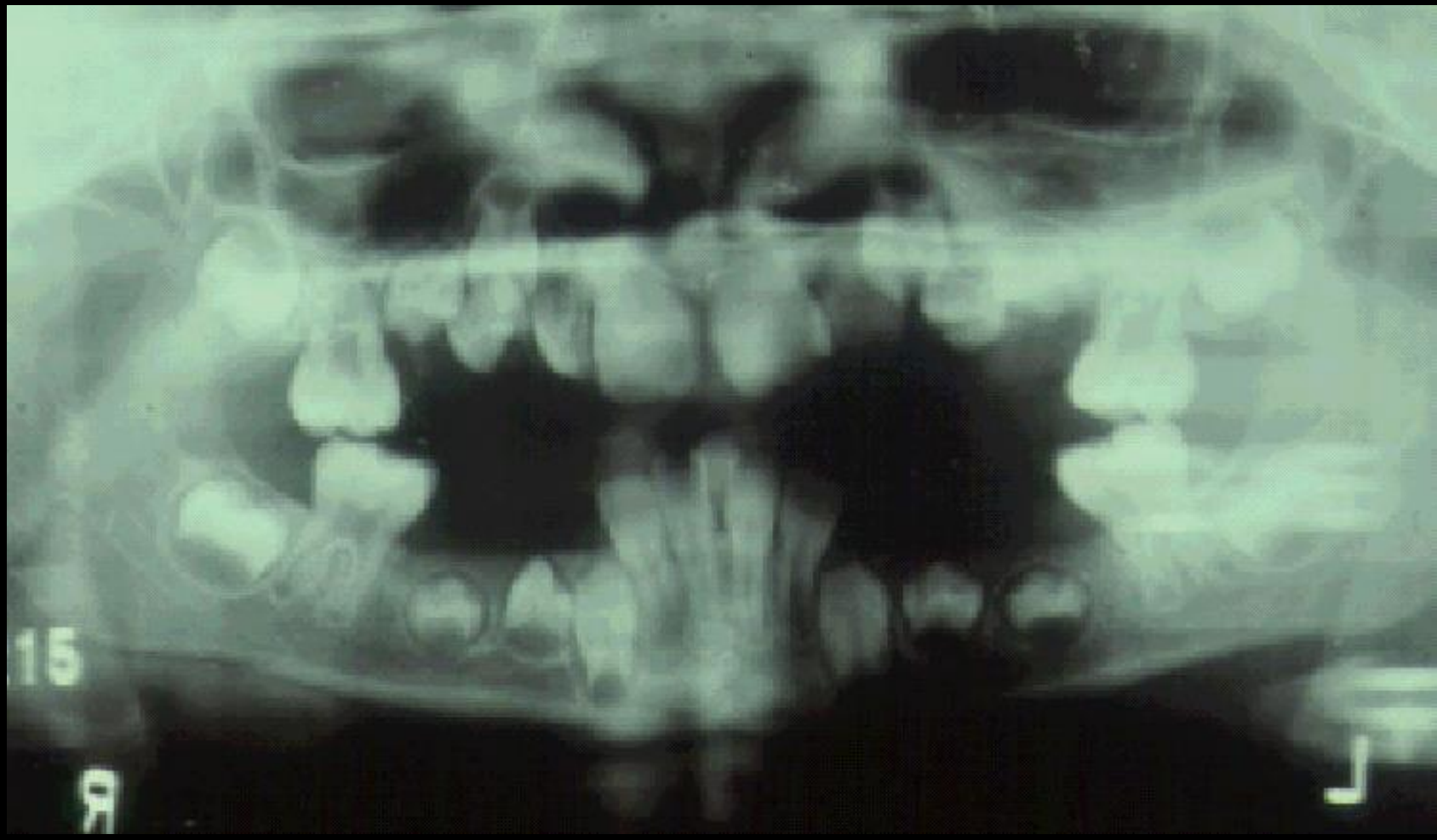
VAN DYKE TE, TAUBMAN MA, EBERSOLE JL, HAFFAJEE AD, SOCKRANSKY SS, SMITH DJ, GENCO R. The Papillion-Lefevre syndrome: neutrophil dysfunction with severe periodontal disease *Clin Immunol Immunopathol* 1984; 31: 419-429

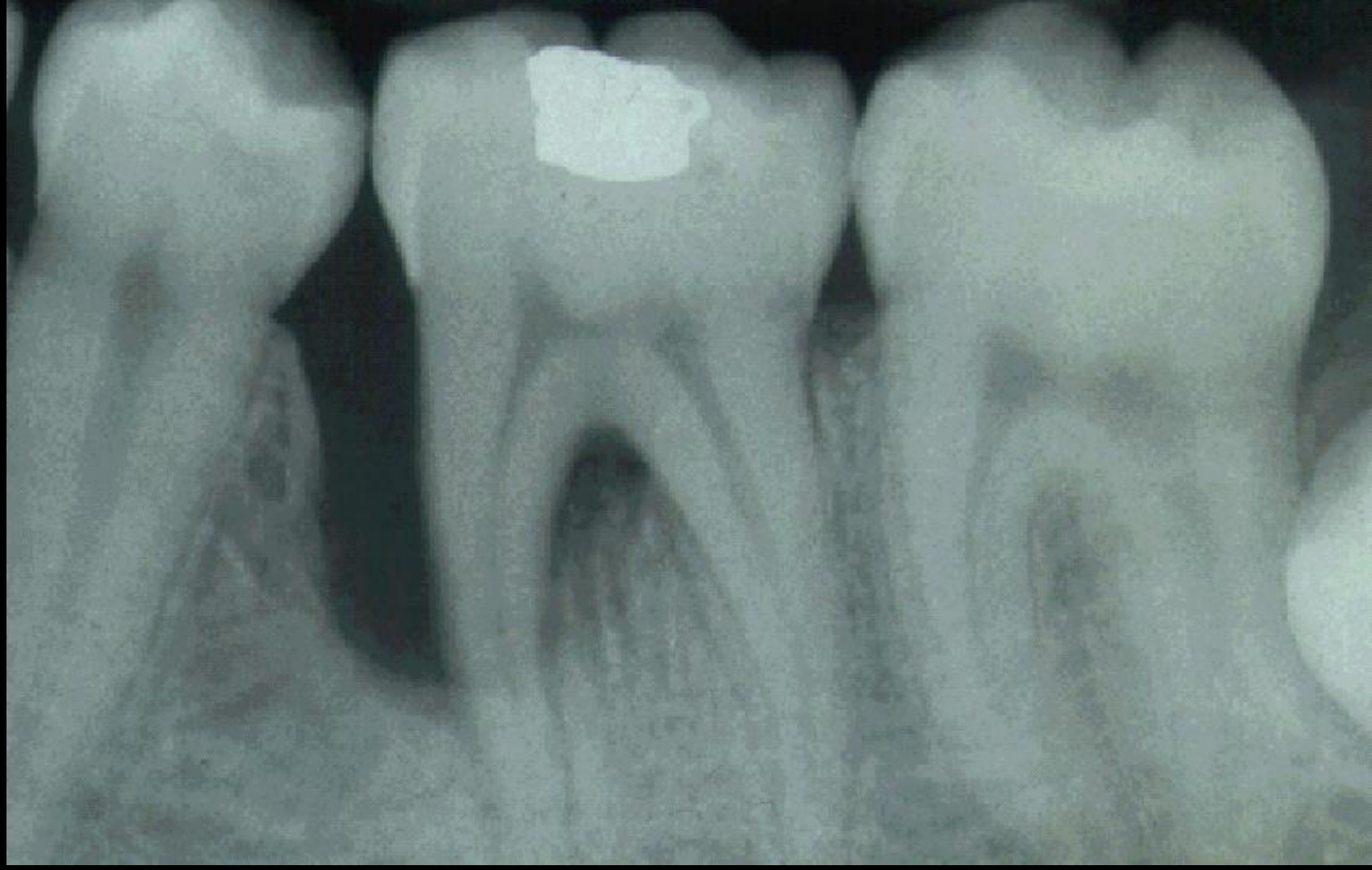
Papillion-LeFevre szindroma (PLS)

The cardinal clinical features of the condition are palmoplantar hyperkeratosis and severe early-onset periodontitis that affects both primary and permanent dentitions

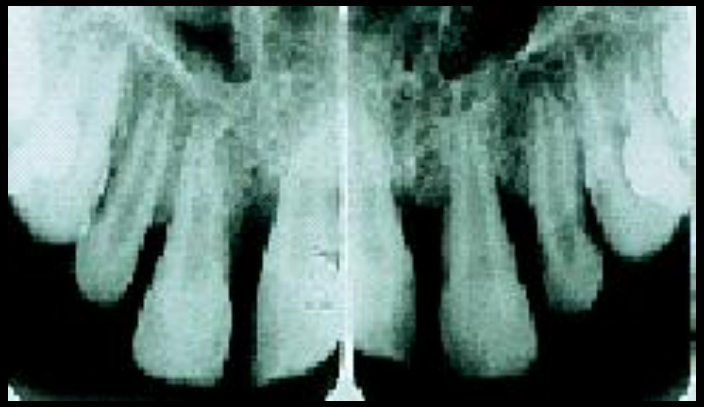


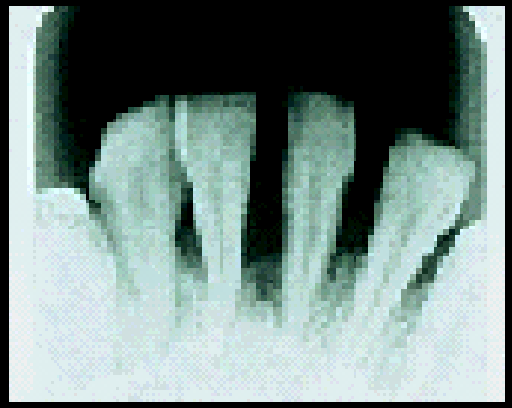
Papillon-Lefevre syndroma.

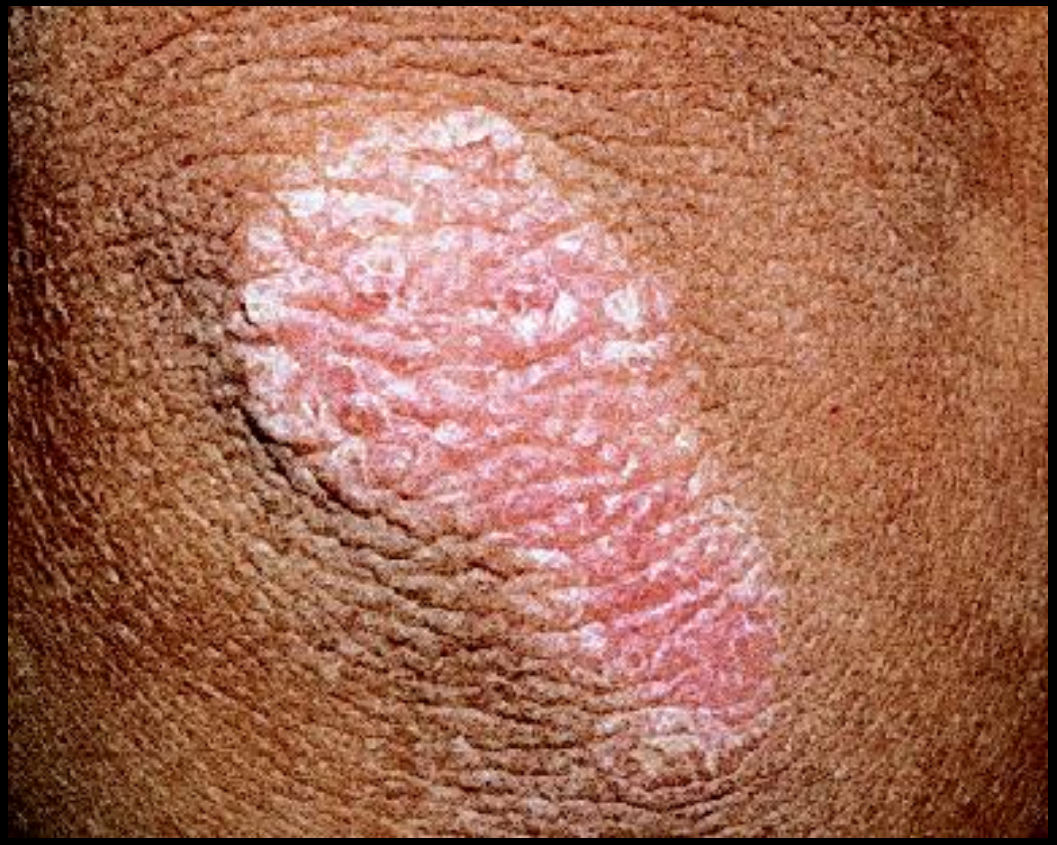




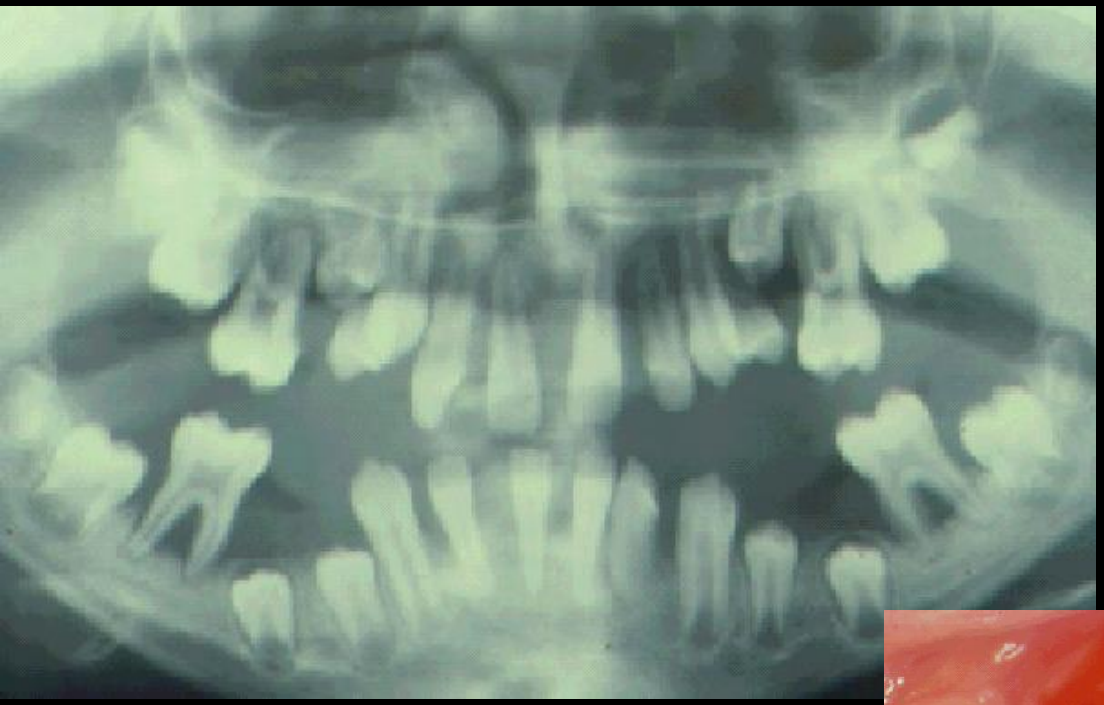
Radiograph demonstrating deep localized circumferential bone loss on first molar in 18-year-old.



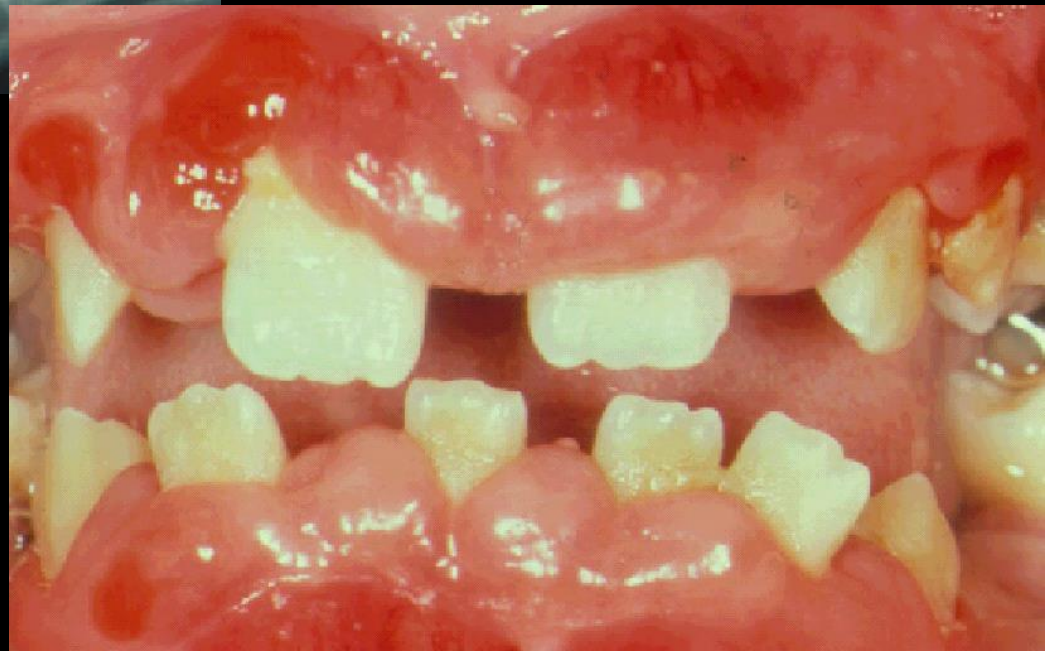




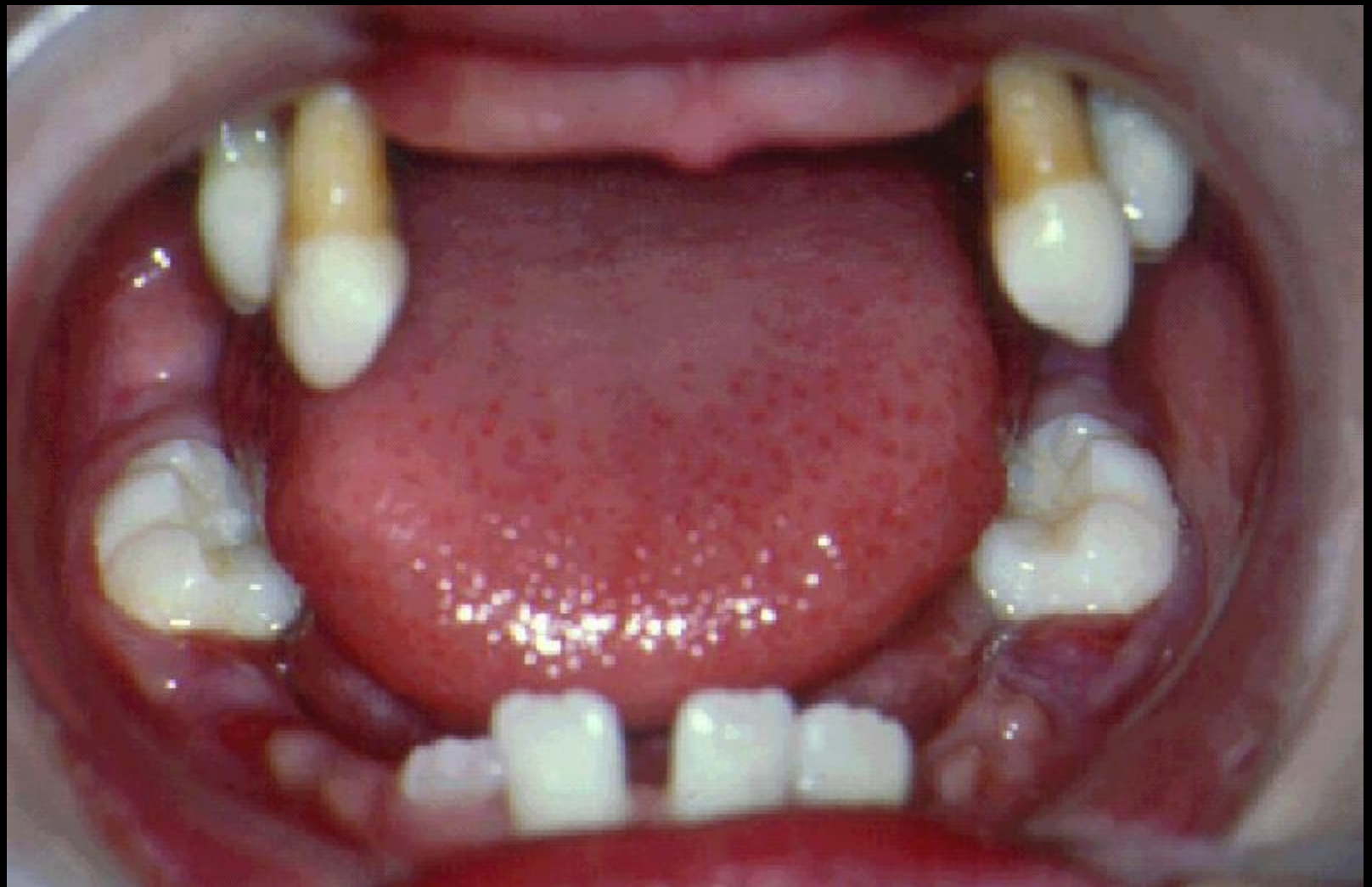
Rapidly progressive adult periodontitis



ALBANDER JM. Periodontal disease in North America. *Periodontol 2000* 2002;29:31-69.



10 years old patient with Papillon-Lefevre syndrome.

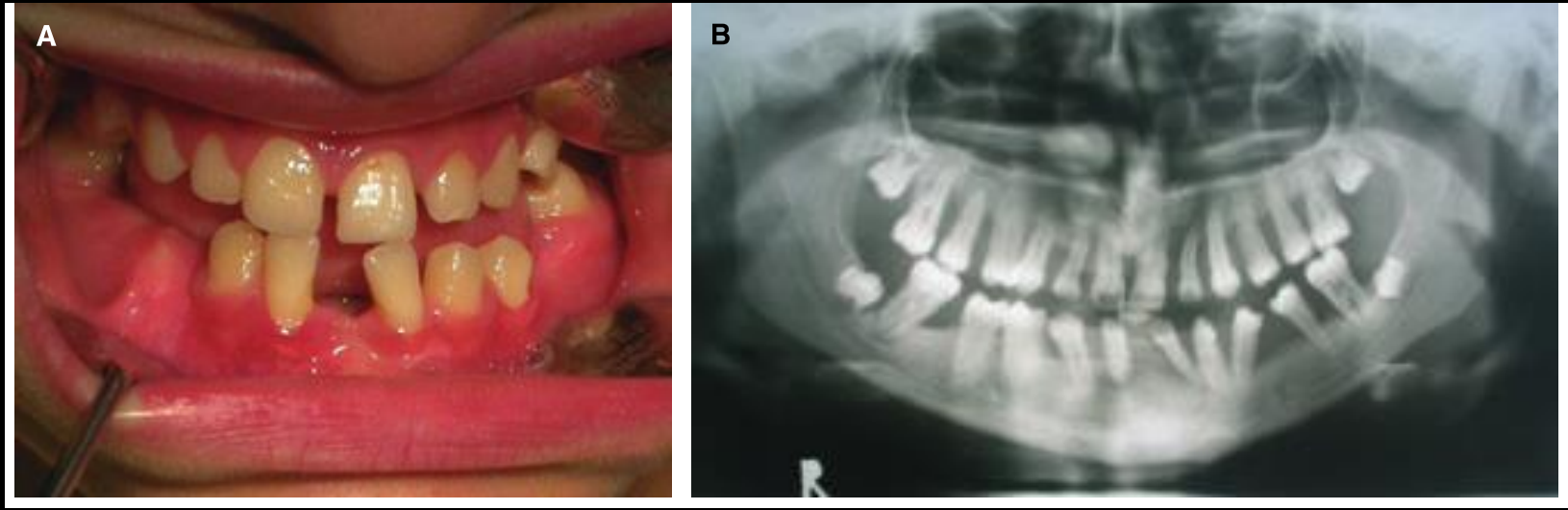


PLS patients' *Actinobacillus actinomycetemcomitans* content is significantly higher in the biofilm

They are more susceptible for *Actinobacillus actinomycetemcomitans* infection

SCHROEDER HE, SEGER RA, KELLER HV, RATEISHAK-PLÜSS Behaviour of neutrophilic granulocytes in a case of Papillion-Lefevre syndrome *J. Clin Periodontol* 1983; 10: 618-635.

Clinical Presentation of PLS



Oral findings include generalized rapid destruction of the periodontal attachment apparatus resulting in **severe early-onset periodontitis**, **premature loss** of both primary and permanent teeth, and alveolar bone loss.

Hart TC, Atkinson JC. Mendelian

Papillon-Lefévre syndrome
different stages

- based on the severiry of cathepsin C gene mutation

  - PLS
- Haim-Munk syndrome (OMIM 245010)
  - Aggressive periododntitis type I (OMIM 170650),

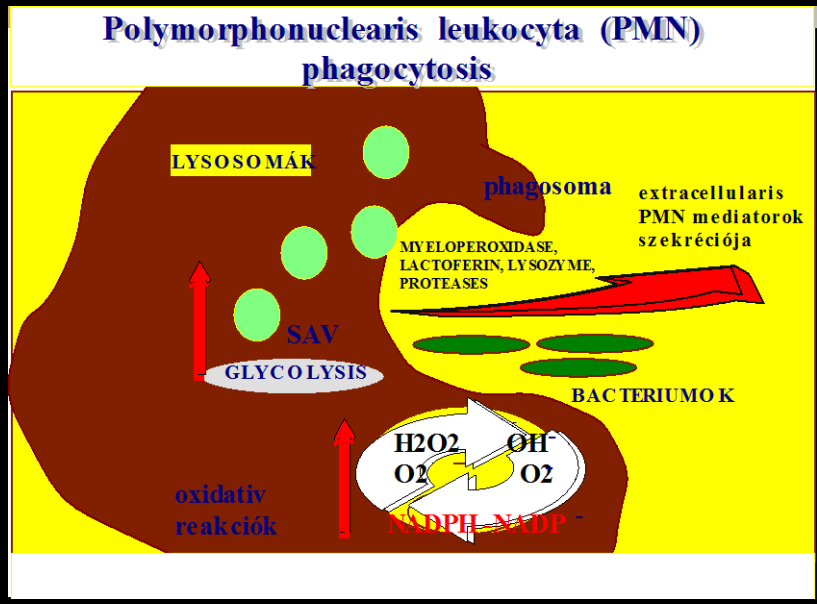
- Hart TC, Hart PS, Michalec MD, et al. : Haim-Munk syndrome and Papillon-Lefevre syndrome are allelic mutations in cathepsin C. *J Med Genet* 2000.; 37: 88-94.

Hewitt C, McCormick D, Linden G, et al.: The role of cathepsin C in Papillon-Lefevre syndrome, prepubertal periodontitis, and aggressive periodontitis. *Hum Mutat*. 2004; 23: 222-228.

The intracellular killing mechanisms of PMN leukocytes are mainly depend on the function of the lysosomal enzymes stored in specific granules and also the intracellular oxydative mechanism

Chediak–Higashi syndrome

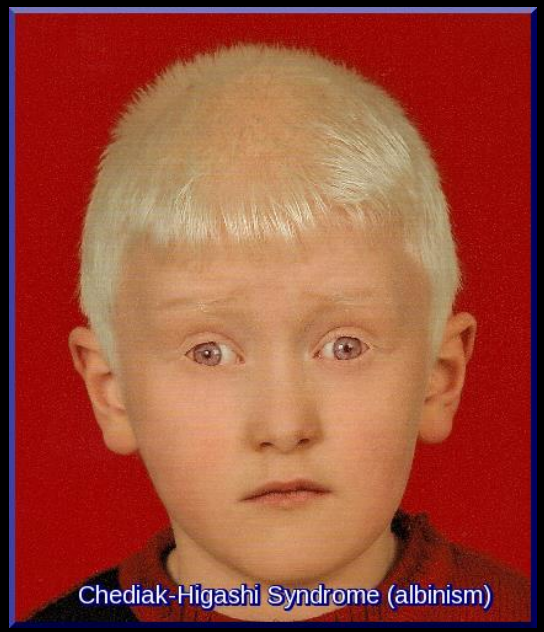
Chronic granulomatous disease .

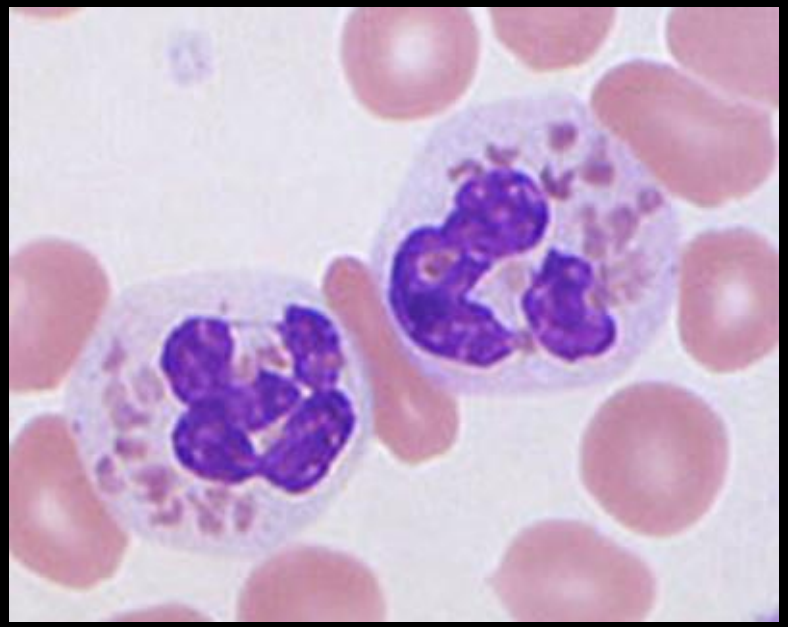


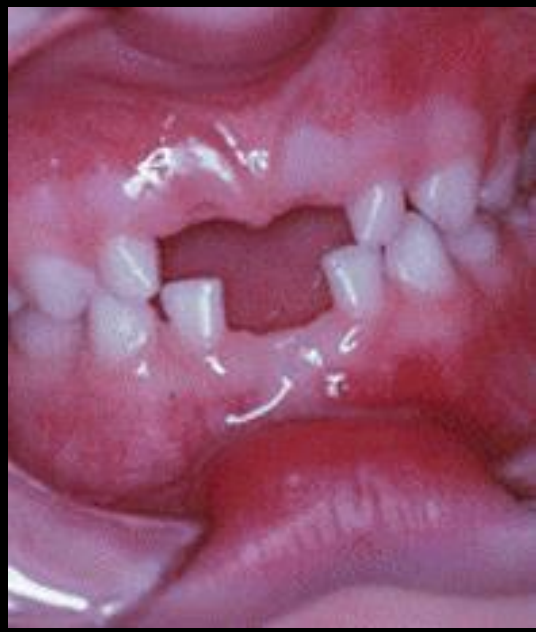
Chediak–Higashi syndrome

Chediak–Higashi syndrome is a mutation in the lysosomal trafficking regulator gene

The decrease in phagocytosis results in severe pyogenic infections, periodontitis, albinism and neuropathy







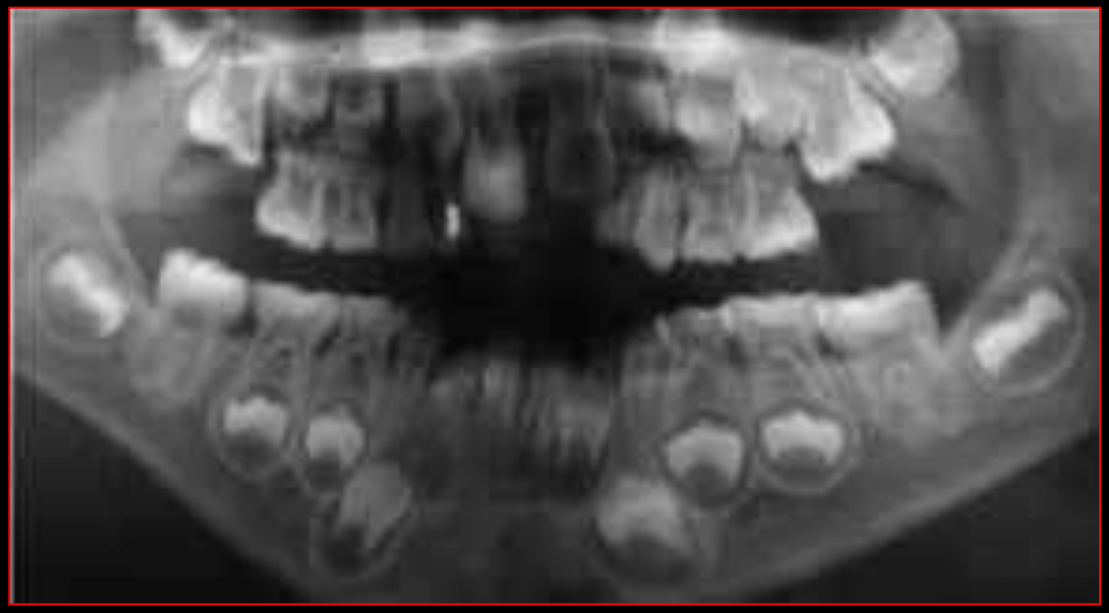
HIGHASI O. Congenital gigantism of peroxidase granules: the first case ever reported of qualitative abnormality peroxydase *Tohoku J Exp Med 1954;59:315-332*

CHEDIAK M. Nouvelle anomalie lukocytarie de caractere constitutionnel et familiel. *Rev Hematol* 1952;7:362-367.

Chediak–Higashi syndrome

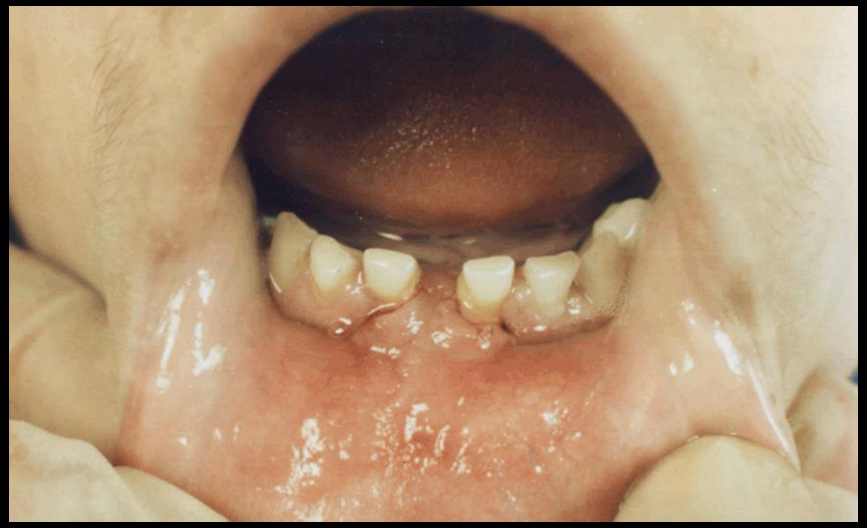
Periodontitis is typically aggressive and unresponsive to conventional periodontal therapy

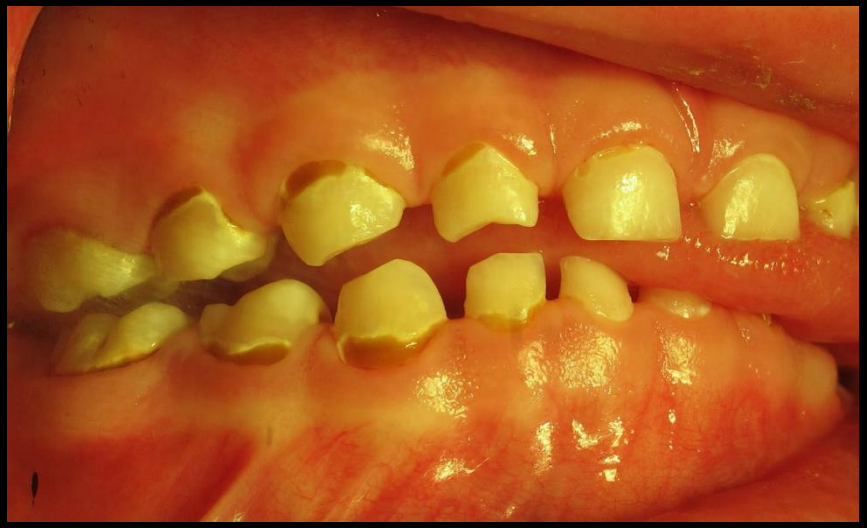
Oral ulcerations can also occur



DELCORT –DEBRUYNE EMC, BOUTIIGNY HR, HILDEBRAND HF. Features of severe periodontal disease in teenager with Chediak-Higashi Syndrome. *J Periodontol* 2000;71:816-824

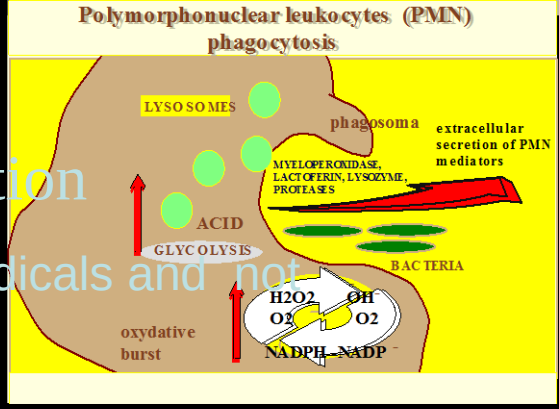
Clinical Presentation of CHS





Oral findings include severe gingivitis, ulcerations of the tongue and buccal mucosa, and **early onset periodontitis** leading to premature loss of both deciduous and permanent dentitions

Chronic granulomatozus disease



PMN leukocytes NADPH oxydaze enzyme disfunction

The PMN cells are not able to produce reactive free oxygen radicals and not capable of killing phagocytosed bacteria

Myeloperoxidase deficiency –

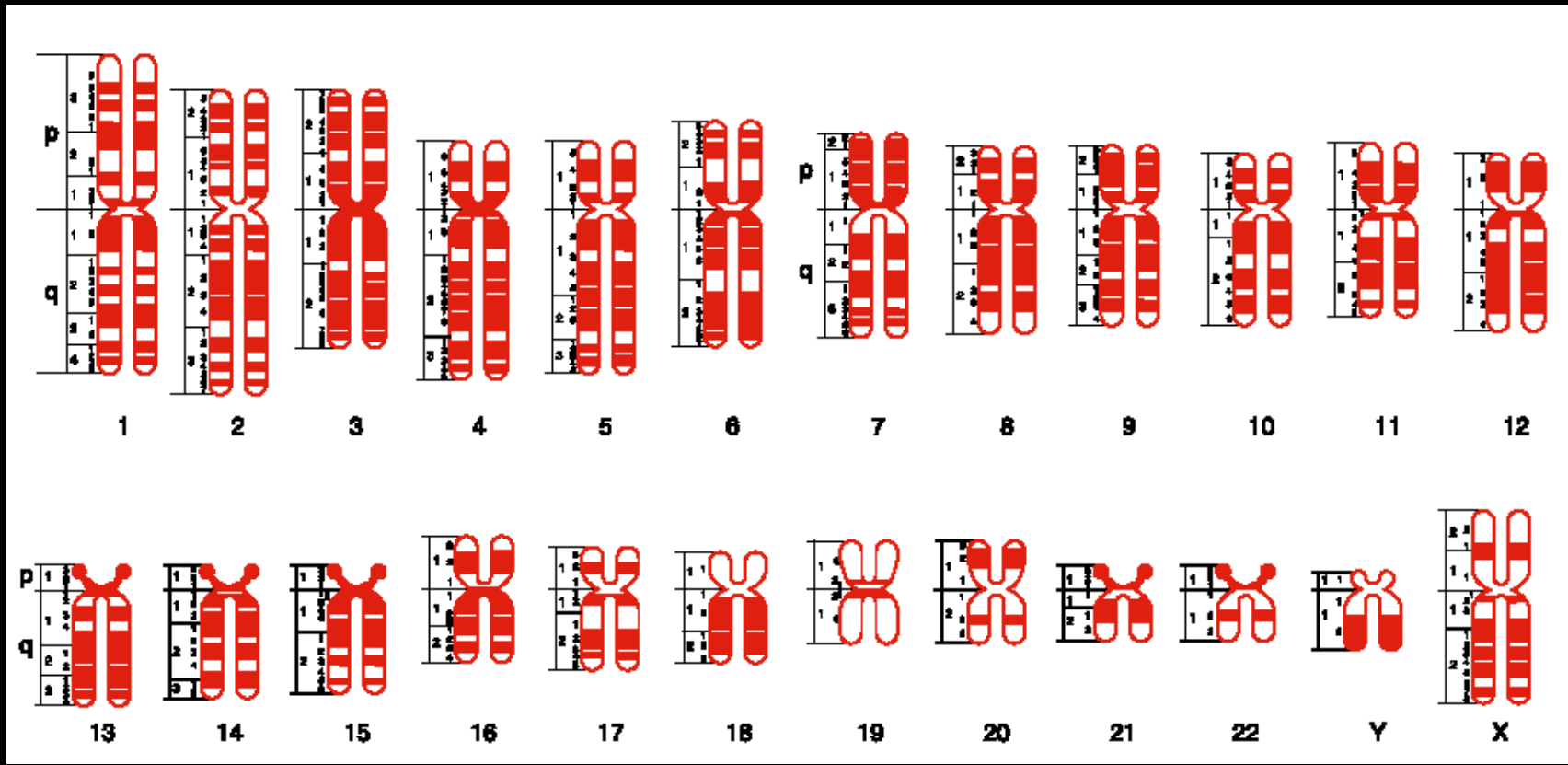
Myeloperoxidase enzyme produces hypochloric acid , HOCl- it is one of the most aggressive oxygen dependent killing mechanisms

Acatalasia decreased catalase enzyme production in the white and red blood cells. The catalase enzyme splits the H_2O_2 to O_2 and H_2O

If defected it cannot neutralize H2O2 - molecules and this will damage the tissues

NORMAL GENETIC VARIATIONS – GENE POLYMORPHISMS .

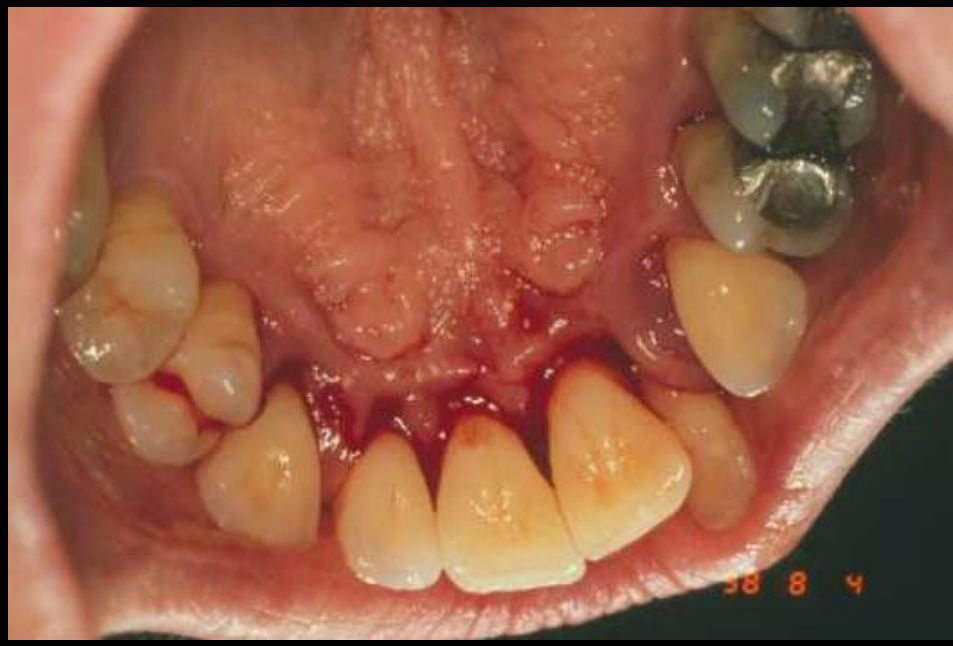
A given function and protein structure can be determined by different but approximately similar nucleotide sequences
Nevertheless the minor differences in the gene sequence might result in slightly different reactions and susceptibility



Genetic polymorphismus (single nucleotide polymorphisms) in both chronic and aggressive periododntitis

- interleukin-1, interleukin-6,
- tumor necrosis factor,
- interleukin-10,
- E-selectins,
- Fc-gamma receptor,
- CD14,
- toll-like receptors,

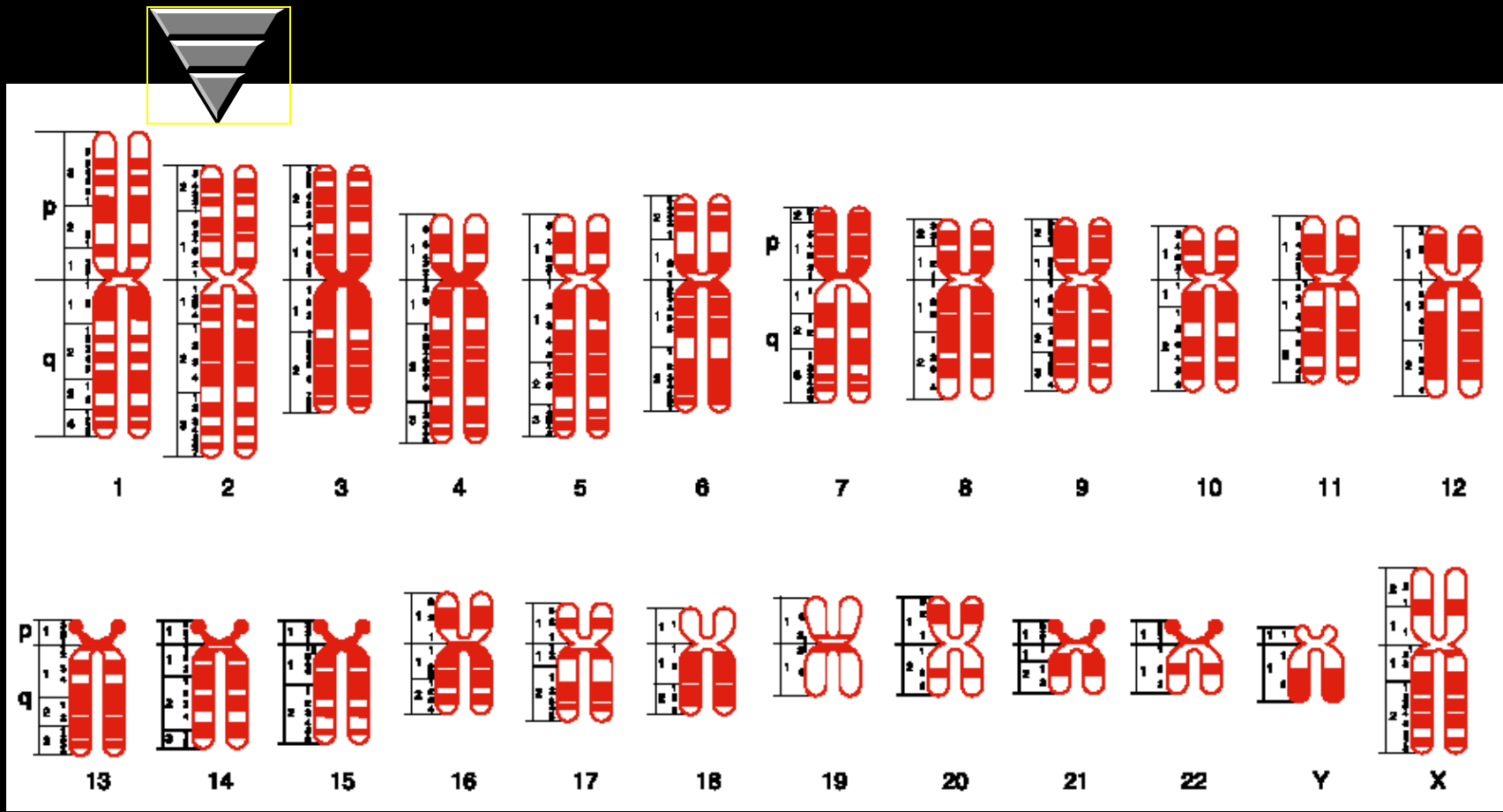
vitamin D receptor.



Interleukin –1 polymorphism

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

(IL-1A, IL-1B és IL-1RN gének).



Significant positive correlations were detected between "*periodontitis associated genotype*" (PAG) and the crevicular fluid IL concentration

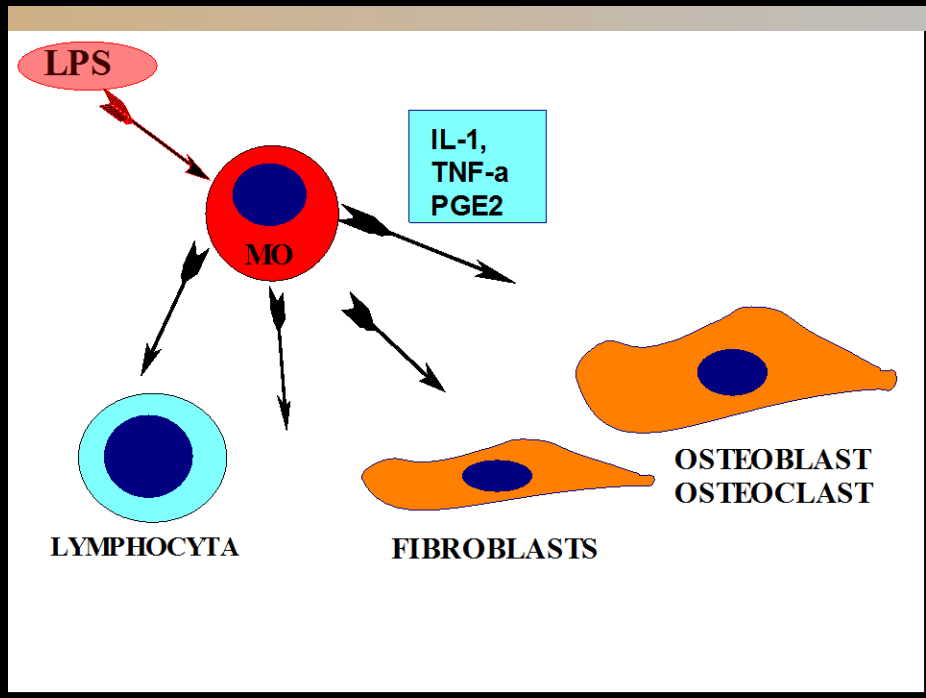
ENGEBRETSON SP, GRBIC JT, SINGER R, LAMSTER IB. IL-1 profiles in periodontal disease. *J Clin Periodontol* 2002; 29: 48-53.

SHIRODARA S, SMITH J, MCKAY IJ és mts. Polymorphisms in the IL-1A gene are correlated with level of interleukin-1alpha protein in gingival crevicular fluid of teeth with severe periodontal disease *J. Dent Res.* 2002; 79: 1864-1869.

Monocytes from collected from different individuals can respond with markedly different interleukin production after being stimulated by lipopolysaccharide (LPS)

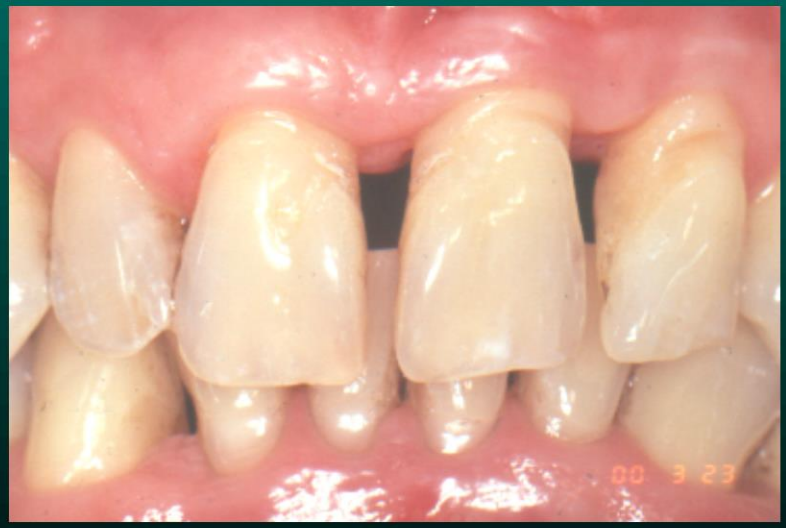
These differences can be determined by different gene polymorphisms.

MOLVIG J, BAEK L, CHRISTEN P. et al.: Endotoxin stimulates human monocyte secretion of interleukin-1, tumor necrosis factor –α and prostaglandin E_2 shows stable inter-individual differences. *Scand J Immunol* 1988; 27: 705-716



RISK FACTORS IN THE ETIOLOGY OF DESTRUCTIVE PERIODONTAL DISEASE

- Oral hygiene
  Local plaque retentive factors
  Bacterial specificity
  Systemic immune status
  Diabetes mellitus
  Tobacco smoking
  Osteoporosis
  Etnic background
  Age
  Diet
  Genetics
- Stress
  Social-economics



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 the adrenal cortex, that 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 andidepressants will cause xerostomia, that promote plaque accumulation and consequently periodontal disease

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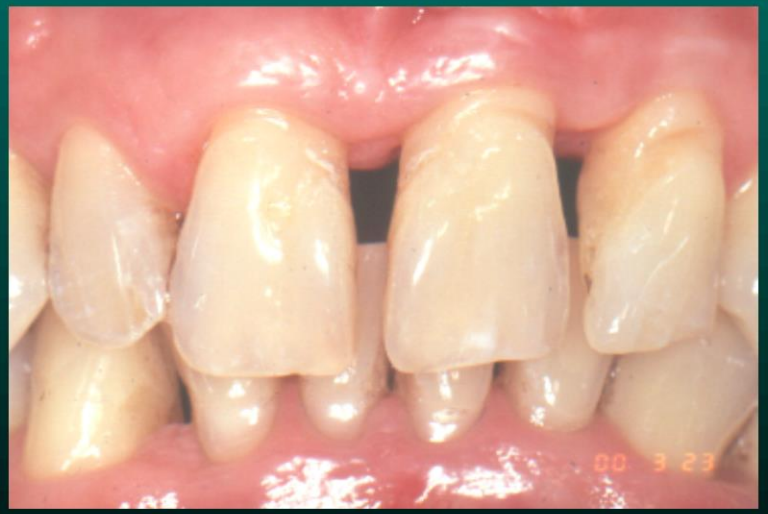
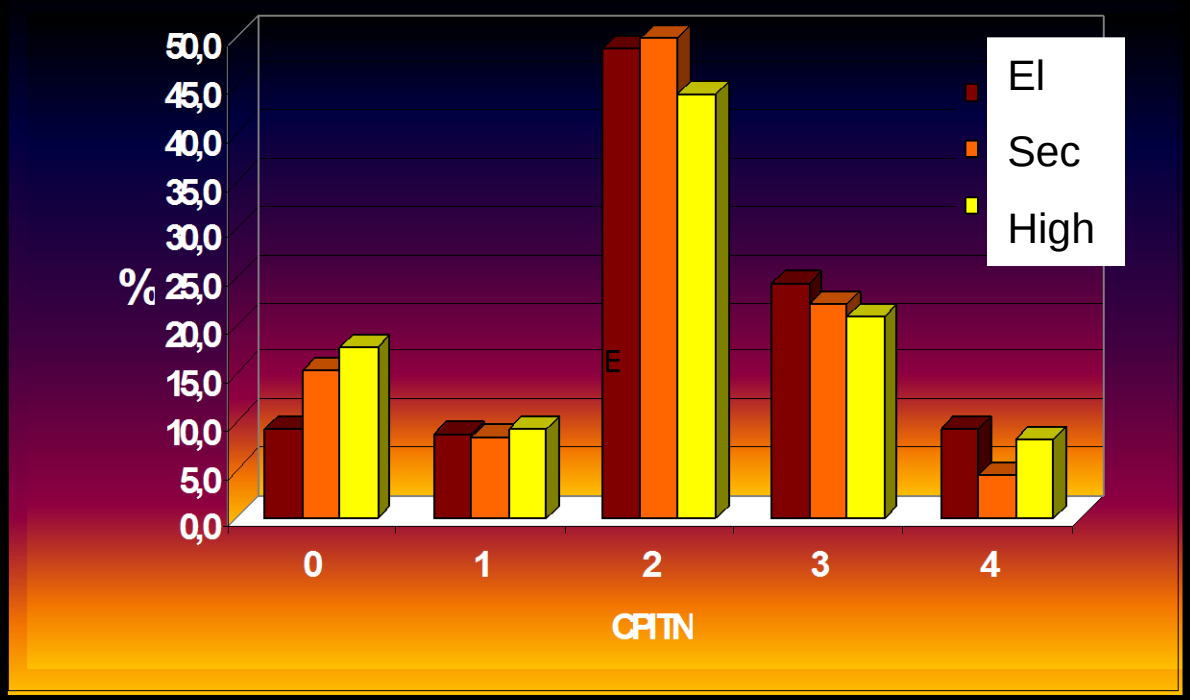


Socio-economic factors

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)

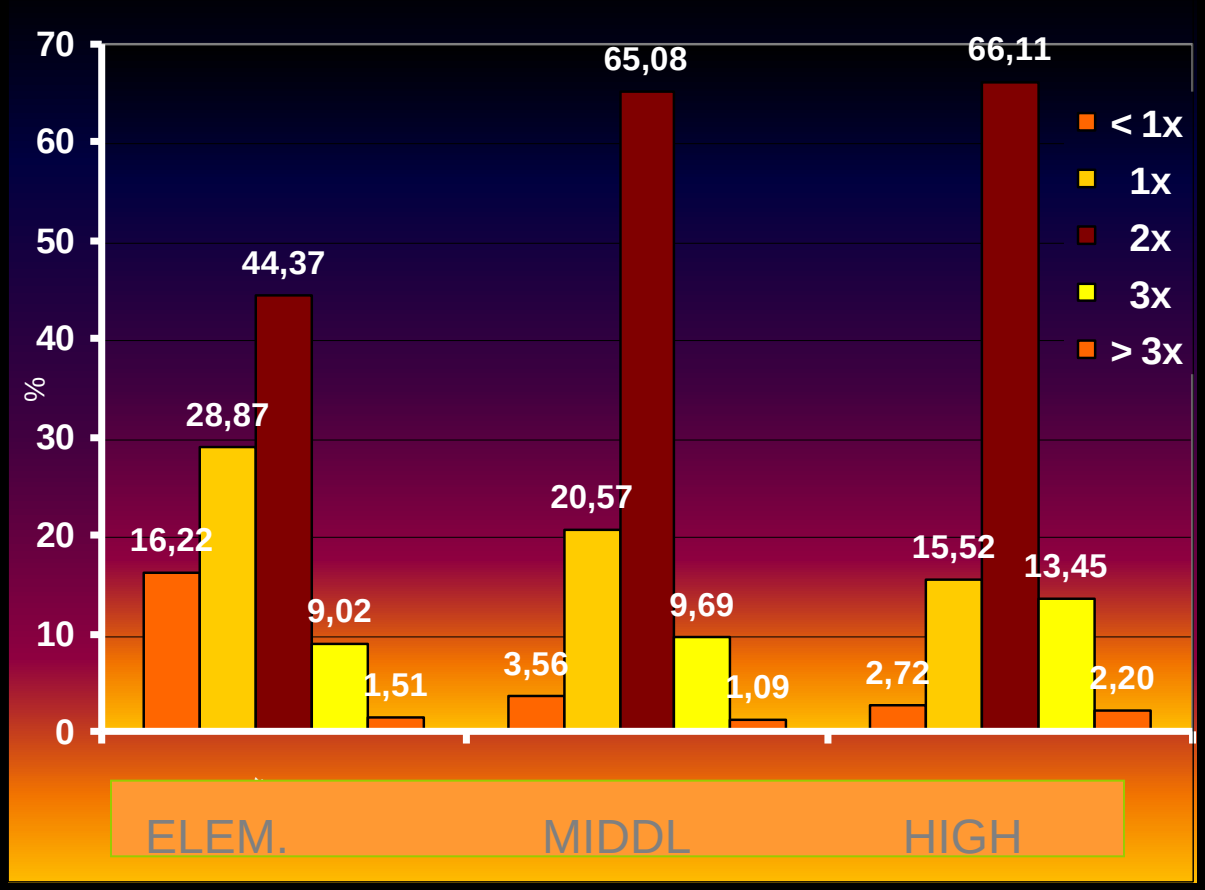
CPITN BY EDUCATION



szignificant differences between the prevalence of CPI degrees in different education levels (p=0,000) .

Hermann P, Gera I, Borbely J, Fejerdy P, Madlena M. Periodontal health of an adult population in Hungary: findings of a national survey. J Clin Periodontol 2009; 36: 449–457.

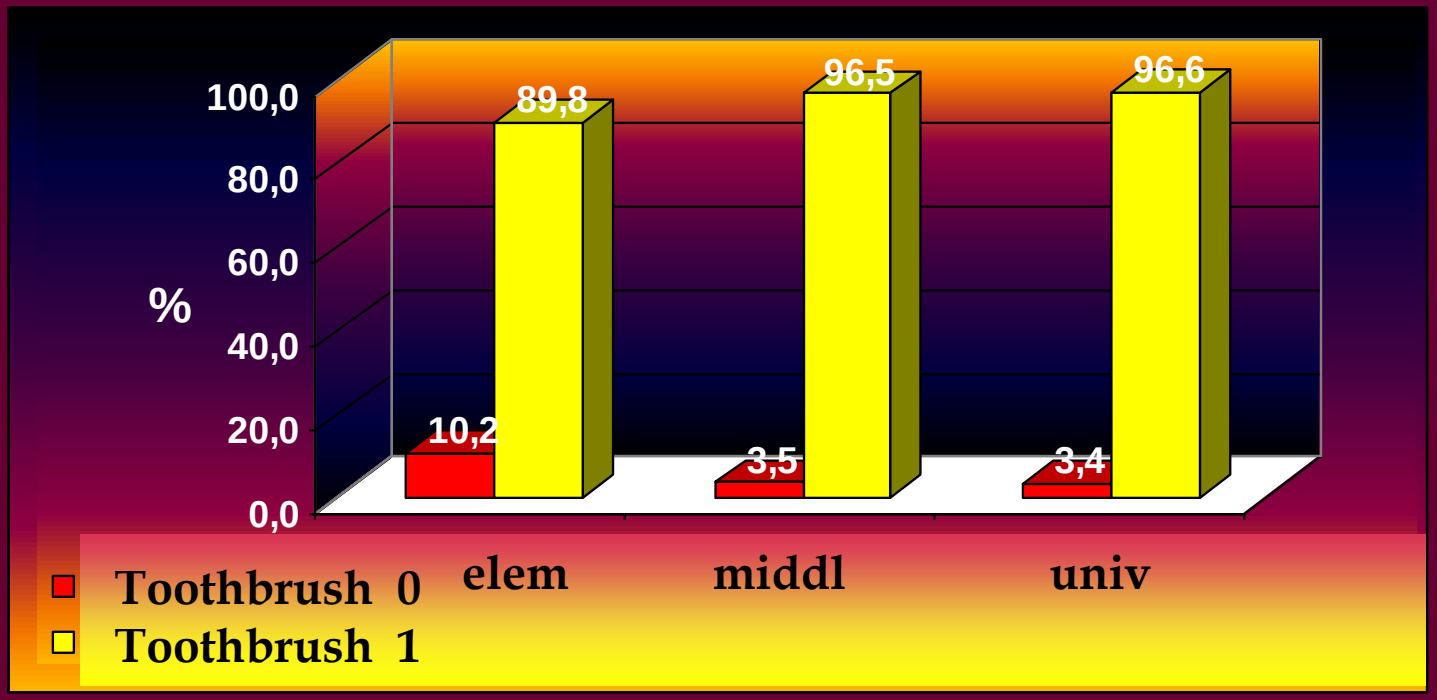
TOOTHBRUSHING BY EDUCATION

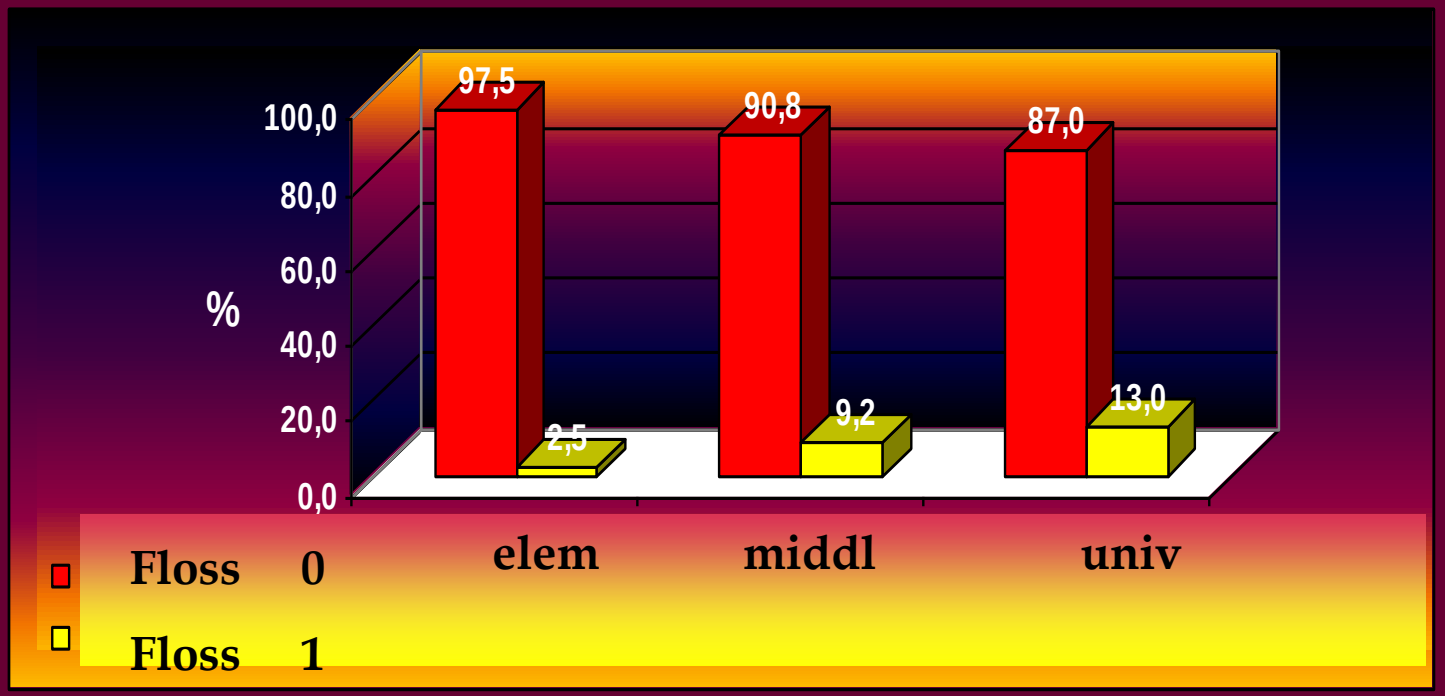


The were significant differences in daily tooth brushing frequency among different educational levels (p=0,000) .

Hermann P, Gera I, Borbely J, Fejerdy P, Madlena M. Periodontal health of an adult population in Hungary: findings of a national survey. J Clin Periodontol 2009; 36:449–457.

THE USE OF TOOTHBRUSH AND DENTAL FLOSS BY EDUCATION





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